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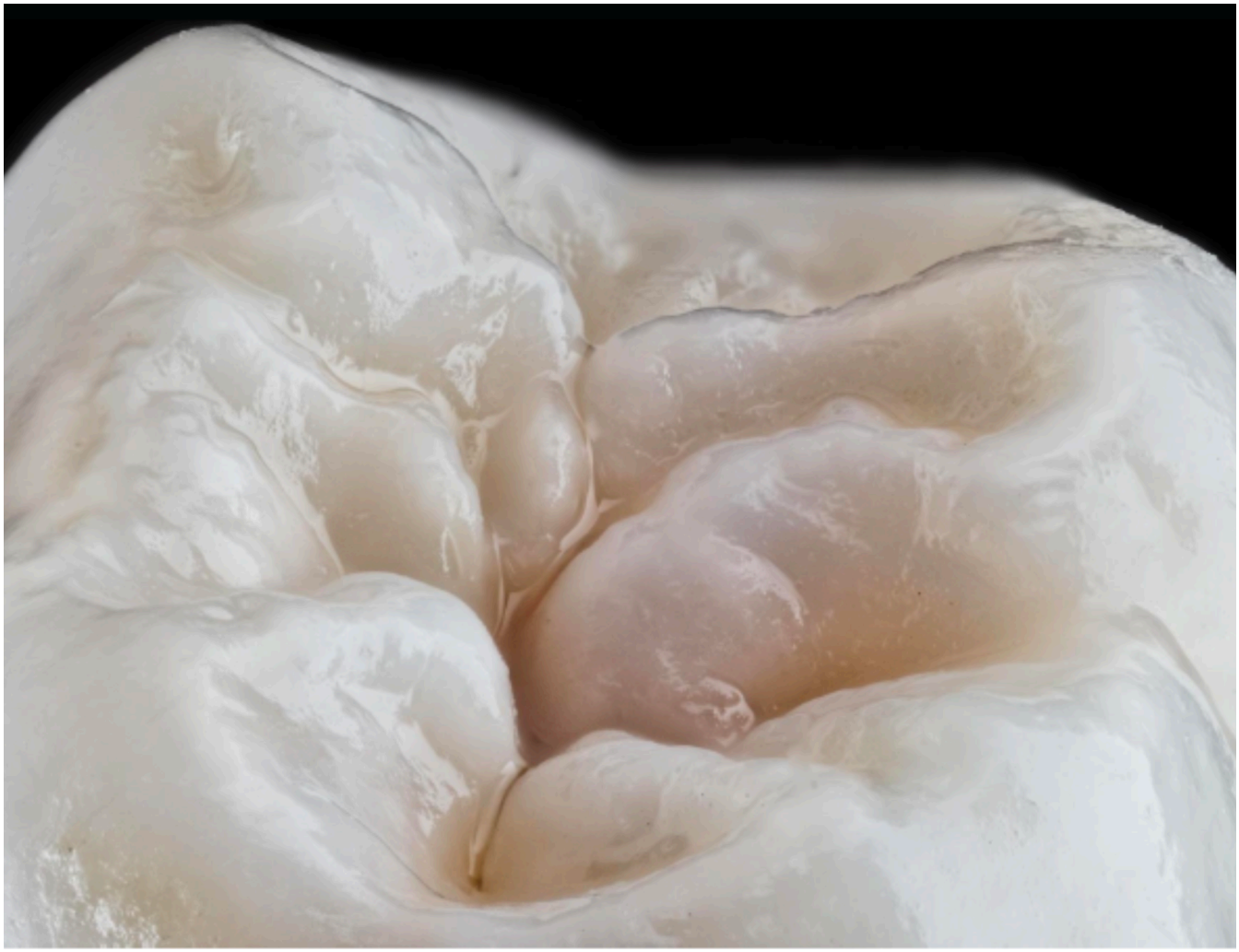
Bachelor's Degree in Dentistry

# Dental Pathology

## Booklet



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Dr. José Manuel Granero Marín
Dr. Raúl Jiménez Soto
Dr. Diego Riera Álvarez
Dr. Raúl Melendreras Ruíz
Dr. Manuel Maíquez Gosálvez
Dra. Carmen Marin Casero
Dra. Mª Rosario Sánchez-Carreño López

# A

# AUTHORS


## Dr. José Manuel Granero Marín

Vice dean Bachelor´s Degree in Dentistry.
Dental Pathology and Therapy I. Coordinating Professor.
Dental Pathology and Therapy II. Coordinating Professor.
Adhesive Aesthetic Dentistry. Coordinating Professor.
Digital Dentistry and New Technologies. Coordinating Professor.
UCAM Universidad Católica San Antonio de Murcia.

## Dr. Raúl Jiménez Soto

Dental Pathology and Therapy I Professor (Spanish Degree)
Prosthesis II Professor (Spanish Degree)
Practicum I Professor (Spanish Degree)
UCAM Universidad Católica San Antonio de Murcia.

## Dr. Diego Riera Álvarez

Dental Pathology and Therapy I Professor (Spanish Degree)
Practicum I Professor (Spanish Degree)
UCAM Universidad Católica San Antonio de Murcia.

## Dr. Raúl Melendreras Ruiz

Dental Pathology and Therapy I Professor (Spanish Degree)
Preventive and Community Dentistry (Spanish Degree)
UCAM Universidad Católica San Antonio de Murcia.

## Dr. Manuel Máiquez Gosálvez

Dental Pathology and Therapy I Professor (Spanish Degree)
Sport Dentistry (Spanish Degree)
UCAM Universidad Católica San Antonio de Murcia.

Translated and interpreted by:

## Dra. Carmen Marin Casero

Dental Pathology and Therapy I Professor (Bachelor´s Degree)
Dental Radiology and Radioprotection Professor (Bachelor´s Degree)
Ergonomics, Dental Materials and Instrumentation Professor (Bachelor´s Degree)
Preventive and Community Dentistry (Bachelor´s Degree)
Preventive and Community Dentistry (Spanish Degree)
UCAM Universidad Católica San Antonio de Murcia.

## Dra. Mª Rosario Sánchez-Carreño López

Dental Pathology and Therapy I Professor (Bachelor´s Degree)
UCAM Universidad Católica San Antonio de Murcia.

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To our alumni

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# Part I

## DENTAL PATHOLOGY OF EMBRYONIC ORIGIN

# 1

# ANOMALIES OF DENTAL HISTODIFFERENTIATION

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## Introduction

Dental formation begins with the development of deciduous teeth in the sixth week of fetal development and lasts until adolescence. It is the result of several complex processes through the histological and functional modification of stem cells that allow teeth to erupt. This process is carried out basically in two phases, the histogenesis or histodifferentiation, and the morphogenesis or morphodifferentiation. Both phases require a series of stepped and overlapping stages, with the process of forming the pattern of the dental crown one of the most important and complex.

Throughout these processes, the influence of etiological, genetic, or environmental factors can lead to abnormalities in the shape, number, size, colour or structure of the teeth causing abnormalities in dental morphodifferentiation.

Histogenesis or cytodifferentiation is the phase in which the process of formation of dental tissues occurs: enamel, dentine, and pulp from the patterns of the crown and tooth´s root. Any anomaly in this process will give rise to alterations in the structure of the dental tissues, the disorder varies depending on the structure that is affected.

## 1.1 Anomalies in the development of the enamel.

The ameloblasts are cells responsible for the formation of the enamel. They are highly differentiated cells and extremely sensitive to the effects of a variety of etiological agents, resulting in alterations or dysplasias in the formation of enamel.

The formation of the enamel has two phases. The first one the secretion phase, in which the ameloblast secretes the protein matrix through the Tomes fibres. The second one is the mineralization phase in which the protein matrix begins to calcify and the nuclei of the hydroxyapatite crystals that later mature and form the prisms. This process will give rise to the enamel as we know it.

The tooth mineralization phase always begins after the secretion phase, and both phases coexist in the tooth in different areas, (i.e. at any given time the occlusal surface of a molar may be mineralising and at the same time the secretion of the matrix in the cervical areas may occur). The mineralisation has a centrifugal pattern that begins in the innermost part of the enamel at the incisal level and continues in two directions towards the outside of the enamel and towards the tooth cervix.

### 1.1.1 Classification of enamel dysplasias.

Enamel dysplasias can be classified according to their histopathology, location, or aetiology.

1.1.1.1 According to its histopathology

A) Quantitative defects: hypoplasia and aplasia.

These are alterations that occur during the secretion phase of the enamel matrix and result in a deficit in its volume that can be partial (hypoplasia) or total (aplasia). Within the quantitative defects we will find different clinical forms of appearance:

- Points or pits: the appearance of points, pits, or holes with a usual linear arrangement and variable depth and length that may appear pigmented.

- Bands: they appear as linear spots or depressions with a horizontal pattern, parallel to the Retzius striae and perpendicular to the dental axis.

- Areas: they take irregular forms with more or less extensive affectation where the enamel is of smaller thickness.

Hypoplastic signs are often followed by alterations in colour (yellow or brown) due to factors such as staining, deposits, cavities, or remineralisation processes.

B) Qualitative defects: hypocalcification.

These are alterations that occur during the mineralisation or maturation phases. Within the qualitative defects we will find different clinical forms of appearance:

- Circumscribed: with well-defined and delimited edges, the adjacent enamel is normal and well-differentiated from the anomalous one.

- Diffuse: The damage is not clearly delimited and overlaps with the healthy enamel that surrounds it.

1.1.1.2 According to its location.

- Located, which affects one or more teeth.

- Widespread, they affect the entire dentition.

1.1.1.3 According to its aetiology

- Genetic or primary. Those with a hereditary transmission pattern, where mutations in the genes involved in amelogenesis are likely to exist. They affect both dentitions, a single tissue (enamel or dentin), and all teeth with vertical or diffuse distribution. There is no family history and the causative agent is not detectable.

- Environmental or secondary. They are linked to external or environmental factors that cause injuries to teeth and surfaces, temporarily limited to the time at which the causative agent acts. They affect one of the two dentitions; it can affect several tissues (enamel and dentin) and one or more teeth with a horizontal distribution of the injuries. There is no family history and the causative agent is detectable.

### 1.1.2 Amelogenesis imperfecta.

Amelogenesis imperfecta is a process included in a group of disorders with a genetic and hereditary origin. It seems to have no apparent systemic involvement that includes all phases of enamel development both in the secretion of the organic matrix and in the mineralization and maturation of enamel. It produces disorders in the structure that are manifested in the clinical appearance and may affect one or both dentitions. Its prevalence is estimated at 1/4000 and 1/14000.

#### 1.1.2.1 Etiopathogenesis

It is a disease with a complicated genetic origin that can manifest itself with different phenotypic expressions and with variable inheritance patterns between dominant and recessive sex-linked. Currently, 5 genes involved in amelogenesis imperfecta are known, each of which can lead to different clinical manifestations.

#### 1.1.2.2 Classification

Weinman in 1945 established the first classification with two unique types, the hypoplastic and the hypocalcified one. Years later in 1988, Witkop established three groups based on the histopathology of the injuries giving rise to hypoplastic, hypocalcified, and hypomature injuries. Later on, a fourth group was added and was associated with Taurodontism, leaving a classification of 4 groups and 15 different subtypes. With current and future studies and the general advancement of the medical sciences, new classifications are more likely to appear more accurate and complete than the current ones. This classification includes: hypoplastic form (type 1), hypomature form (type 2), hypocalcified form (type 3) and hypomature-hypoplastic form associated with Taurodontism (type 4).

A) Type 1: Hypoplastic form

There is an inadequate deposition of the enamel matrix, but correct mineralization of the same. Radiographically the enamel contrasts with the dentine.

a) Punctiform variant: hypoplastic enamel with small pits, especially on facial areas and often arranged in rows and columns. With dominant inheritance.

b) Dominant localised form: hypoplastic enamel, located in pits and grooves, with a horizontal pattern in the middle third area. Not all teeth are affected. With dominant inheritance.

c) Localised recessive form: hypoplastic enamel, localised but more severe. Hypoplastic enamel may be hypocalcified. All teeth are affected and their inheritance is autosomal recessive.

d) Dominant smooth variant: smooth thin hypoplastic enamel with changes in colour. There is also a loss of contact points. It is associated with dominant inheritance.

e) Recessive smooth variant: affects in a different way according to gender. In men, the hypoplastic enamel is smooth, thin, and with changes in colour and in women, it has alternating vertical bands of normal and hypoplastic enamel. It is associated with an open bite. Associated with recessive inheritance.

f) Rough variant: hypoplastic, rough, thin, hard, and granular-appearance enamel. There are no contact points. It is associated with an open bite and its inheritance is dominant.

g) Enamel agenesis: rough and yellowish-brown tooth surface. There are no contact points. Teeth are frequently retained and reabsorbed. It is associated with an open bite.

B) Type 2: Hypomature form.

In these cases, there is a defect in the maturation of the hydroxyapatite crystals, teeth have a normal shape, and white or brown discolourations may occur. The enamel is softer, splinters, and peels off as the explorer passes through. Radiographically the density is similar to dentine, therefore, it has a lower radiographic density than normal enamel.

a) Brown pigmented form: the enamel fractures and separates from the dentine. With an autosomal recessive inheritance.

b) White - yellowish: alternation between normal bands and hypomature ones. With X-linked recessive transmission. In males, the enamel is whitish or yellowish and mottled. In women, it has alternating bands of normal enamel and opaque and hypomature ones. Its colour is white or yellowish.

c) Mottled teeth: the enamel is opaque white, covers incisal and coronal thirds. With X-linked inheritance.

d) Mottled tooth, with partial discolourations with anterior or posterior involvement (not global). Probably autosomal dominant inheritance.

C) Type 3: Hypocalcified form

There is a failure in the process of calcification. Teeth have a normal morphology with a yellow-brown discolouration, which in aggravated processes changes to dark brown-black. Tartar is deposited on it rapidly and the enamel is quickly lost. There is less

involvement of the cervical enamel, which has better calcification and delays more in the fall. This type of amelogenesis is related to dental inclusions and open bite. Radiographically the radiodensities of dentine and enamel are the same.

a) Autosomal dominant variant: it has a hypocalcified enamel with an initial colour yellow-brownish or orange. It may have a soft consistency and a loss of the same to slight mechanical stimuli. Also associated with an open bite.

b) Autosomal recessive variant: Similar to the dominant variant but presenting exacerbation of the clinical picture.

D) Type 4: Hypomature-hypoplastic form associated with Taurodontism

In this form, there is a combination of hypoplastic and hypomaturative aspects predominating one or the other according to the variant that appears. It occurs diffusely in both dentitions and is associated with Taurodontism at the dental level and trichodental syndrome (also trichodental dysplasia) at the systemic level.

a) Hypomaturative variant: there are hypoplastic components in the form of pits in the facial surfaces. With autosomal dominant inheritance.

b) Hypoplastic variant: where the hypoplastic components with hypomature areas predominate. With autosomal recessive inheritance.

1.1.3 Environmental enamel dysplasias

1.1.3.1 Prevalence

Scientific research was performed on 1500 children aged 12 to 15 years in permanent dentitions, finding that 67.2% of patients had opacities and 14.6% had enamel hypoplasia, with an average involvement of 3.6% of all the pieces under study.

1.1.3.2 Treatment

Within the anomalies, in the development of enamel due to environmental causes we can find different degrees of affectation, limited to the intensity and time at which they occur. Treatment will depend on the severity of the condition, in mild cases may be sufficient with preventive measures, such as instructions for proper oral hygiene and fluoridation. In many cases, the problems are predominantly aesthetic and can be treated with full coating crowns or even with porcelain or composite facets (also veneers). These treatments should be performed as soon as possible to avoid the negative psychological impact that this disease has on patients.

1.1.3.3 Aetiology

Its causes are many and among them we highlight:

A) By traumatic processes in childbirth:

Observing what is called the neonatal line, we can see the accentuation of stria of Retzius or the presence of an abnormal band of defective enamel. The alterations are located on the enamel of the incisors in the middle third, canine and molar cusps, always observing these defects in the temporary dentition and being able to present manifestations of hypoplasia, hypocalcification or hypomature processes.

B) For nutritional alterations:

a) Rickets. There are many vitamins whose deficiency is associated with problems in the development of enamel, but it is only evidenced by the deficiency of vitamin D. This deficiency can lead to dysplastic enamel alterations always located in permanent dentition as during pregnancy the foetus acquires all the elements necessary for the formation of the enamel through the mother.

b) Fluorosis. Ingestion of more than 1.8 ppm of fluoride in water results in a dysplastic condition in the enamel known as fluorosis. Fluoride on a developing enamel causes degenerative processes in ameloblasts and makes it difficult to remove amelogenins. It could present as a mineralization disorder or as a hypoplastic process. Fluorotic enamel is less susceptible to caries than normal enamel except in the most severe cases, being important to make a correct differential diagnosis with amelogenesis imperfecta. The distribution of fluorosis shows more horizontal striations and changes in colouration, and although several members of the same family may suffer from it, it has no genetic pattern.

c) Celiac disease: celiac disease consists of an intolerance to gluten proteins that runs with the atrophy of the mucosa of the small intestine. Its association with enamel hypoplasia and also with delays in a rash and recurrent aphthosis has been described.

C) For debilitating diseases:

The most common is febrile hypoplasia, where the increase in temperature has an impact on ameloblasts causing narrow horizontal bands of dysplastic enamel. The number and type of teeth affected indicate the time of onset of the disease and the involvement is usually bilateral and symmetrical.

D) By infectious processes:

a) Apical infection in temporary teeth. They give rise to the so-called Turner´s teeth, whose aetiology lies in processes of periapical infection, usually in

molars. It can also occur from trauma to temporary teeth, especially anterior ones. Permanent teeth have white, yellow, or brown discolouration processes, with hypoplastic horizontal bands and may be associated with dilaceration.

b) Prenatal infections. If the mother contracts rubella in the first trimester of pregnancy, it causes several alterations in the child of anatomical and neurological nature. At the dental level, there is no specific pattern, but it usually presents as a form of generalised hypoplasia in the enamel. In case the mother has syphilis, the child will have a series of three clinical manifestations called Hutchinson's triad which is: neurological deafness, interstitial keratitis, and dental disorders. At the dental level, the anterior teeth are in the form of a screwdriver and are observed hypoplasia in incisal areas and the presence of diastemas. Posterior teeth are blackish, also called Fournier.

E) For iatrogenic causes

a) Tetracyclines. Lesions in the form of pigmentation are present, which may be accompanied by dysplastic lesions with an undefined pattern. Tetracycline is incorporated into the enamel matrix in the calcification phase forming a calcium phosphate-tetracycline complex and the exposure of these complexes to sunlight results in a discolouration that ranges from light yellow to brown, accompanied by varying degrees of decalcification.

b) Radiotherapy: It has an effect when applied in children under 12 years being more evident in children under 5 years, radiotherapy has hypoplastic defects and can be generalised or localised without a specific pattern. It is associated with processes of hypodontia, microdontia, root hypoplasia, dentin disorders and mandibular hypoplasia.

c) Chemotherapy: They usually manifest as hypoplastic alterations, with discolourations and associated with microdontia and root hypoplasia.

1.1.3.4 Degrees of affectation

Finally, it is important to know the classification of the severity of involvement in hypomineralization proposed by Mathu-Muju and Wright in 2006:

-Mild: Opacities in non-functional areas, negative sensitivity, absent caries, unaffected or mild involvement incisors.
- Moderate: Opacities in functional areas, negative sensitivity, caries or PEB (post eruptive breakdown due to MIH: molar incisor hypomineralization) up to two surfaces, no cusps are affected, some aesthetic compromise, and intact fillings.
- Severe: PEB or extensive caries, positive sensitivity, pulp involvement and defective fillings.

## 1.2 Abnormalities in the development of dentine.

Dentine is formed after the differentiation of dental bud cells into odontoblasts induced by preameloblasts of the enamel organ. Dentinogenesis consists of two phases.

During the secretion phase, odontoblasts synthesize proteins from the dentine matrix and secrete it to the outside by cytoplasmic prolongation or odontoblastic process. The odontoblastic secretory activity gives rise to predentine.

During the mineralisation phase, the deposition of hydroxyapatite crystals occurs which are secreted in the form of vesicles. The mineralized zone is preceded by an unmineralized predentine front. The mineralisation pattern of dentine is twofold, a globular pattern in which crystals bind into mineral aggregates called calcospherites, which then bind to each other and another pattern according to a linear front. At the same time, all the components of the peritubular and intertubular dentine are mineralised.

Histopathologically, the alterations that we can have in the dentine when there are alterations in the function of its odontoblasts are of two types:

- Alterations in the tubular pattern, assume the presence of irregular tubules in shape and size. If the damage is more severe, the odontoblast dies with the consequent reduction in the number of tubules. Dentine can produce subsequent differentiation of mesenchymal cells in odontoblasts with regular dentine production after the process.

- Alterations in mineralization, are in relation to the severity of the process, so they appear from minimal mineralisation defects to severe conditions. In the process of normal dentine calcification, the spheroidal calcification nuclei (calcosphere) fuse with each other. In the case of calcification disorders, they do not fuse, generating non-calcified areas of matrix between the calcospherites that delimit them, giving them a typical "holly leaf" shape, this is what is known as interglobular dentine.

### 1.2.1 Classification

| Genetics | Environmental |
|---|---|
| Dentinogenesis imperfecta type I, II and III | Rickets |
| Dental dysplasia | Calcinosis |
| Associated with Syndromes | Use of tetracyclines |
| Hypophosphatasia | |
| Ehlers-Danlos syndrome | |

Table 1. Aetiological classification of abnormalities in the development of dentine.

### 1.2.2 Dentinogenesis imperfecta

It does not usually have systemic involvement. Its incidence is around one case per 8000 inhabitants and usually responds to a dominant genetic pattern of high penetrance, related to the alteration of the long arm on chromosome 4, a subtype that can be associated with osteogenesis imperfecta. The dentine adopts a characteristic opalescent appearance which is why this process has been called hereditary opalescent dentine.

#### 1.2.2.1 Histopathology

The scalloping of the amelodentinal junction is not observed, so it appears smooth and uniform although the dentine may be normal in the vicinity of the enamel. In the rest, the histological lesions present typical alterations of the development of the dentine: alterations in the pattern of the tubules and in the calcification, there is a significant reduction of dentinal tubules, being in addition small and irregular, anarchic, and frequently obliterated. There are areas of non-tubular dentine and an increase in interglobular dentine. There is a greater amount of organic material and water, implying a reduction in the inorganic material.

The pulp chamber is very small or completely obliterated, with the presence of calcified bodies (pulp stone). Odontoblasts are few and atypical and may even be absent.

#### 1.2.2.2 Clinical manifestations

It affects all the teeth of both dentitions, with greater involvement in the temporary dentition. The dentine takes on a characteristic opalescent and translucent appearance and there are discolourations ranging from bluish, grey and brown and the translucency of the tooth is characteristic.

In spite of not existing alterations in the enamel, this one comes off easily since it lacks the support that confers the healthy dentine to him, because it has lost the scalloped one of the dentinoenamel joint and the dentine is softer, producing attrition of very fast progression which has led to call this alteration, "teeth without a crown." Morphologically we see the premolars and molars with a bulbous shape, with constriction at the cervical level giving a bell-shaped shape to the pieces and their roots are usually short, thin and translucent.

Radiographically, there is a greater radiolucent of the dentin due to its higher organic and water content. There is also a reduction of the pulp chambers to points where they are not visible, making it difficult to treat the ducts of these pieces, being also the roots of these, short and thin or bulb-shaped.

Wiktop classifies the clinical forms into two types: dentinogenesis imperfecta, which refers to processes associated with osteogenesis imperfecta, and another that qualifies as hereditary opalescent teeth without genetic association to osteogenesis imperfecta. The classification we will use is that of Shield, which classifies

dentinogenesis imperfecta into 3 types: type I associated with osteogenesis imperfecta, type II, and type III in Brandywine, Maryland.

A) Type I dentinogenesis imperfecta.

It is considered another manifestation of osteogenesis imperfecta. Altered genes have been identified and are related to the synthesis of type I collagen (COL1A1 and COL1A2) and may follow a dominant or recessive hereditary pattern.

There is considerable variability in affectation, due to phenotypic variants of the same gene. All teeth can be affected or only some of them, and there may be different degrees of involvement in different members of the same family. Currently, some authors tend to call this entity osteogenesis imperfecta with opalescent teeth, referring to dentinogenesis imperfecta for only dental involvement (type II).

B) Type II dentinogenesis imperfecta.

There is only dental involvement and it is due to a mutation in the DSPP gene (or dentinal sialophosphoprotein. DSPP gene provides instructions for synthesising this protein). It is transmitted by autosomal dominant inheritance, has an incidence of approximately 1 / 8,000 and is one of the most typical and clear cases of autosomal dominant inheritance, with a penetrance of almost 100%, although with a variable expressiveness.

The clinical, histological and radiographic aspects are the same as in type I; the differences lie in:

- There are no bone or other alterations such as those present in osteogenesis imperfecta.
- There is less variability in the affectation within the same family so that there are similar degrees of colouration, severity and wear.
- Both dentitions are affected with equal intensity.

C) Type III dentinogenesis imperfecta

It is a severe and uncommon form characterized by both opalescent dentitions with marked dentine attrition, large pulp chambers, multiple pulpal exposures, and a characteristic radiographic appearance in the shell of the teeth (shell teeth).

### 1.2.3 Dental dysplasia

It is an alteration in the development of hereditary transmission dentine with an autosomal dominant pattern, which affects the dentine in isolation. It has a very rare presentation (1 / 100,000) and affects both dentitions, but more severely to the temporary.

A) Type I or Radical Dental Dysplasia

The crowns are of normal colour, size and shape in both dentitions. They are called rootless teeth because the roots are very short, which results in excessive tooth mobility, malpositioned teeth, and premature tooth loss. Pulp chambers in the permanents are often obliterated by masses of tubular dentin in a horizontally stratified manner. So, the remains of pulp tissue are radiographically observed as horizontal or crescent-shaped radiolucent areas, giving them an appearance called "cascade". This obliteration occurs very prematurely, even before the eruption occurs. Periapical radiolucent lesions or areas are common in caries-free teeth.

B) Type II or Coronal dentinal dysplasia

There are clinical differences between temporary and permanent dentition. The temporary teeth are the same as those of dentinogenesis imperfecta: very susceptible to attrition, opalescent, with bulbous crowns and with obliterated pulp chambers with abnormal dentine.
Permanent teeth, on the other hand, have a normal colour and also normal pulp chambers, even enlarged, due to the root extension of the pulp chamber. Unlike type I, the length of the roots is normal.
Histopathology is typical of dentine abnormalities: alterations in the tubular pattern, non-tubular areas and interglobular dentine alternating with areas of healthy dentine.

1.2.4 Alterations in the development of dentine associated with syndromes

A) Ehlers-Danlos syndrome: it presents with dentinal alterations and obliterated pulps.

B) Goldblatt syndrome or spondylometaphyseal dysplasia: The temporary teeth look like a dentinogenesis imperfecta type I, while the permanent dentition is completely normal.

C) Schimke's syndrome or immuno-osseous dysplasia: It presents a yellowish-grey alteration, bulbous crowns and obliteration of the dental pulp.

**1.3 Anomalies in the development of cement.**

Cement is an avascular connective tissue with a much lower level of cell differentiation than enamel and dentine, making it less susceptible to developmental failures. Anomalies that affect only cement without other dental structures being involved are rare.

1.3.1 Classification

1.3.1.1 According to its histopathology

A) Cement hypoplasia. Deposition of cellular cement on the acellular occurs and is associated with cleidocranial dysostosis.

B) Aplasia of the cement. There is an absence of cement with premature loss of temporary incisors and alveolar bone in the anterior region. The teeth have large pulp chambers and are associated with hypophosphatasia

1.3.1.2 According to its aetiology

A) Genetics

a) Hereditary multiple cementosis. The presence of radiopaque diffuse masses in the periapical area is observed radiographically. It runs asymptomatically. Histopathologically, it is a dense, acellular and highly calcified cement, with little vascularization. Transmission is autosomal dominant and its incidence is very rare.

b) Cleidocranial dysostosis. It is an autosomal dominant inherited disease that occurs with clavicle aplasia and a large development of the transverse diameter of the skull with delayed ossification of the fontanelles. It is often associated with the presence of supernumerary teeth and disorders of tooth eruption or retention. There seems to be an absence of cell cement in both dentitions, the lack of eruption could be due to the absence of junction between the dental follicle and the oral mucosa. In addition, the fibrous connective tissue is formed between the follicle and the mucosa that act as a barrier to the rash.

c) Hypophosphatasia. It is a congenital metabolic disorder that is inherited in an autosomal recessive manner. There are low concentrations of alkaline phosphatase in the blood, accompanied by skeletal abnormalities because the bone is not mineralized properly, there are osteoporosis and bone fragility. At the dental level, there is a premature loss of the primary teeth because cement is not formed properly. The lower incisors are especially affected. Teeth acquire mobility with minimal trauma. Sometimes tooth loss is the first sign of the disease. There are other associated dentinal abnormalities, along with a reduction in the thickness of the cement there is a reduction in the thickness of the dentine. The teeth have large pulp chambers and wide root canals.

B) Environmental

a) Concretion. It is the union of two teeth by the cement at the level of the roots, without a union of the dentine. The tooth retains its normal morphology.

b) Hypercementosis. It consists of the excessive formation of root cement as a physiological reaction to various aggressions: infectious, inflammatory or traumatic. The consequences of hypercementosis are of two types, on the one hand, there may be a delay in root resorption eruption or ankylosis with the underlying bone, and on the other hand, a deformation of the root may occur.

c) Paget's disease. It consists of an increase in the volume of the bones, which show a greater radiolucency with a cottony appearance. In Paget's disease, a special type of hypercementosis occurs which is limited to the apical third of the root.

**1.4 Anomalies in the development of enamel and dentine.**

During the formation of the tooth, there is an interaction and a union between the different components of the enamel and the dentine that can lead to alterations that affect both tissues. The most representative entity is odontodysplasia.

1.4.1 Odontodysplasia

Odontodysplasia is a developmental disorder that affects a certain group of teeth and without a genetic cause, affects one or more teeth within the same dental quadrant, hence the name regional odontodysplasia, although some authors also call it odontogenesis imperfecta. The cause of this alteration is not known, although it is believed that it is due to problems of vascular origin, which would explain the limited nature of these alterations. It can affect only the permanent or both dentitions and is a disease with a very low incidence.

1.4.1.1 Histopathology

The alterations are the typical ones observed in alterations of the development of the dentine, alterations in the calcification, interglobular dentine, anomalous tubular patterns with irregular passages, non-tubular zones, etc. The enamel is also affected and areas of hypoplasia and hypocalcification may appear.

1.4.1.2 Clinical manifestations

It is usually accompanied by eruptive disorders, which should alert us, as it can be the first sign of this disease. The crowns are small with altered shapes and have discolourations. The enamel and dentine are very fine and poorly mineralized. The pulp chambers are enlarged and the roots are short and with little definition, which radiographically gives the tooth a very radiolucent appearance, being called by this image “ghost tooth”. Of course, enamel fractures are very common and are very prone to caries, sometimes with periapical complications even in the absence of deep caries.

**1.5 Anomalies in the development of dentine and cement.**

1.5.1 Rhizomelia
Trauma or infection in a temporary tooth that affects the permanent developing.

**1.6 Anomalies in the development of enamel, dentine and cement.**

In addition to the environmental causes of tetracyclines in all mineral dental tissues, there are other genetic entities that may also affect this triad of dental tissues.

### 1.6.1 Inherited epidermolysis bullosa

It runs with fine enamel and a superficial spotting, simulating an amelogenesis imperfecta and sometimes existing root folds.

### 1.6.2 Congenital ichthyosis

The entity that runs at the oral level with dilaceration and resorptions. Hyperkeratosis is observed in several areas of the oral cavity, deformations of the maxilla and mandible and alterations in the position of the teeth.

# 2

# ANOMALIES OF DENTAL MORPHODIFFERENTIATION

2.1 Number anomalies.
2.2 Size anomalies.
2.3 Shape anomalies.
2.4 Anomalies due to union.

**Introduction**

Morphogenesis, or morphodifferentiation, is the phase in which the formation processes occur that will give rise to the crown of the tooth and later to the root. Any anomaly in this process will give rise to alterations in the size, number or shape of the teeth.

To classify the anomalies in dental morphodifferentiation we can rely on the embryological origin of each entity or the clinic depending on the final shape of the tooth. This is more useful and simpler although in many cases the embryological origin will help us understand the anatomical consequences.

**2.1 Number anomalies.**

They occur in the dental lamina stage before dental germs form. By default, they are called agenesis and by excess hypergenesis.

2.1.1 Isolated agenesis or hypodontia

In isolated agenesis or hypodontia, there is an absence of one or more teeth (up to 5). The prevalence of isolated agenesis is common and is around 20% of the population. Within each dental group, agenesis is more likely to occur in the most distal tooth. The agenesis of the upper lateral incisors is the most frequent one, followed by second premolars and chordals. According to Dahlberg's theory, there is an evolutionary tendency to reduce the dental formula at the expense of the loss of the most distal tooth of each group.

The genetic pattern of appearance is very marked and its expression is variable, so it is common to find hypodontia, microdontia or conoid teeth in different individuals in the same family.

2.1.2 Oligodontia

This term refers to a dental reduction of 6 teeth or more. It can be sectorial (grouped absences in the same quadrant) or scattered (absences in several quadrants). They can also appear in any dental group, although it does not usually affect the first teeth of each group because they are phylogenetically more stable teeth.

2.1.2.1 Aetiology

- Localised: due to an affectation of a certain area of the dental lamina preventing its development, it can be due to trauma, vascular alteration, lack of space ...

- Systemic: there is a general affection during pregnancy coinciding with the developmental stage of dental germs. Among the causes we can find infections, poisoning, radiation ...

- Genetics: linked to general syndromes. They are usually associated with alterations in the shape and size of the teeth. They are the most common.

### 2.1.3 Anodontia

The total absence of teeth is very rare, as the agenesis of the first teeth of each series is very rare given its high phylogenetic stability. Anodontia is associated with anhidrotic ectodermal dysplasia (AED), a disease that affects all tissues derived from the ectoderm.

### 2.1.4 Hypergenesis

Hypergenesis is relatively common, as its frequency is between 0.5% and 3% and is due to the presence of teeth that exceed the usual number that corresponds to each dentition, 20 in the temporary and 32 in the permanent one. These extra teeth are also called supernumeraries. Given the hereditary nature of anomalies in the number and variability in gene expression, it is possible to find in the same individual agenesis in one dental group and hypergenesis in another. This condition would not produce variation in the total number of teeth, for this reason the alterations in the number of teeth must be evaluated by dental groups. They are more common in permanent maxillary teeth, especially in the midline and distal to the third molar.

#### 2.1.4.1 Aetiology

Its origin is generally hereditary, although it has also been associated with atavistic or ancestral causes, that is, it comes from remote ancestors and was latent in immediate generations.

When a supernumerary tooth appears, it is usually a single one. But, when several ones appear, it is because its aetiology is associated with syndromes such as cleidocranial dysplasia or Gardner syndrome. It can even appear associated with disorders in the development of the dental lamina, in which case the patient has other oral pathologies, such as cleft lip or cleft palate.

#### 2.1.4.2 Clinical manifestations

The clinical importance of supernumerary teeth is due to the fact that the increase in the number of teeth generates a lack of space in the arch. This clinically produces eruptive alterations, malpositions, diastemas, crowding or reabsorption of contiguous teeth.

By its form we can distinguish clinically two types. The supplementary ones, that have the shape of a normal tooth, generally of the contiguous one. And the accessories have abnormal shapes like cone shape, pin shape...

Depending on their location, the supernumerary teeth give in some cases a specific clinic that receives its own name, among which we highlight:

A) Mesiodens. It is located between the central maxillary incisors, at the level of the midline. It is usually an accessory tooth (although there may be several) with a conical shape that causes alterations in the rash, diastemas or malpositions, and may even be retained or move inside the nose (nasal tooth).

B) Peridens. Teeth with normal or abnormal morphology that appear generally by the facial surface of the mandibular premolars.

C) Paramolar. It appears more frequently among the definitive molars positioned by both the facial and lingual surfaces. In some cases, it may appear fused to one of the molars generating the so-called paramolar tuber.

D) Distomolar. It appears distal to the third molar; it is usually a random radiological finding.

2.1.4.3 Treatment

We can find two different situations, in case they hinder the eruption of other teeth, their correct position or are misaligned in the arch, which can cause occlusal alterations or hinder hygiene. In these situations, the treatment would be an exodontia. If, on the other hand, the supernumerary teeth are well aligned in the arch, we can leave them or carry out an aesthetic treatment to adapt their shape to their position in the arch.

**2.2 Size anomalies.**

After the development of the dental lamina, the dental germs begin to develop individually, the alterations that have occurred in this phase can lead to alterations in the size of the resulting teeth, either by excess (macrodontia) or by default (microdontia). Both macrodontia and microdontia are difficult alterations to establish by comparing the size of the teeth between individuals, as there is great variation from each other, so the relationship between the size of the teeth and the maxilla that contains them must be established isolated for each individual.

The widespread discrepancy in size between the teeth and the maxilla or mandible that house them can be due to three reasons:

- Physiological cause, since the size of the teeth is determined by genes different from those that determine the size of the maxilla and mandible and are therefore transmitted differently. So, the phenotypic expression of these genes can give an incongruous result without that this is due to an alteration during development.

- Skeletal cause. Teeth are normal, but they are in a maxilla or mandible with an alteration in their development, either of hyperplastic type or of hypoplastic type.

- Dental cause. In this case the size of the maxilla and mandible is normal, and it is the teeth that show an alteration in size. In this case, the alteration of the size of the teeth is associated with other developmental syndromes with systemic manifestations.

### 2.2.1 Microdontia

We call microdontia to the clinical situation in which the patient has a reduction in the size of the teeth resulting in the presence of diastemas. This can be of several types:

A) Relative microdontia. Teeth of normal size in the maxilla or hyperplastic jaw.

B) True or absolute microdontia. Small teeth in normal maxilla and mandible. Depending on the number of teeth affected, true microdontia can be of two types:

- Total or generalised. Affects all teeth and is very rare. It is associated with developmental disorders such as hemifacial microsomia or syndromes such as pituitary dwarfism and in some cases Down Syndrome.

- Partial or local. Affects a tooth or group of teeth. It is more common than the true one and is associated with an autosomal dominant hereditary origin. Due to the variability in the phenotypic expression of these genes it may be accompanied by alterations in shape, especially in maxillary lateral incisors that usually take on a conoid shape. We can even find in the same individual microdontia on one side and agenesis on the other.

The most frequently affected teeth by microdontia are also the last of each series. In order of frequency they would be lateral incisors, third molars and premolars.

The treatment of microdontia has the aesthetic and functional purpose of giving the tooth the right size, this increase in size can be done using composite resins or ceramics.

### 2.2.2 Macrodontia

We call macrodontia the clinical situation in which the patient has an increase in the size of the teeth resulting in the presence of crowding, malpositions or eruptive alterations. Macrodontia can be of several types:

A) Relative macrodontia. Teeth of normal size in the maxilla or hypoplastic jaw.

B) True or absolute macrodontia. Teeth of greater reduction in normal maxilla and mandible. Depending on the number of teeth affected, true macrodontia can be of two types:

- Total, generalised or macrodontism. Affects all teeth and is associated with developmental disorders such as hemihypertrophy or syndromes such as pituitary gigantism.

- Partial or local. Affects a tooth or group of teeth, are uncommon and can be confused with dental fusion or twinning.

Treatment of macrodontia may require exodontia and / or orthodontics to correct crowding and malpositions.

## 2.3 Shape anomalies

The anatomical characteristics of the teeth are not the same in all humans. There are variations that are associated with certain races or ethnic groups being normal among the individuals that make it up. However, outside that group can be considered an anomaly of shape. Shape abnormalities can affect the tooth as a whole or in isolation from the root or the crown.

2.3.1 Abnormalities of the complete dental shape (affect the crown and the root)

2.3.1.1 Conoid teeth (also “peg laterals”)

Both the root and the crown have a conical shape, the tooth as a whole has the appearance of two cones joined at the base at the level of the amelo-cement line. It is a very common alteration that usually affects the upper lateral incisors and is usually bilateral, although it can also occur unilaterally or in association with other alterations such as agenesis, microdontia…

The conical anatomy of the crown generates diastemas and aesthetic problems, which are easily solved by giving the tooth the anatomy that corresponds to it according to its position in the arch by means of composite resins or total or partial ceramic coating.

2.3.1.2 Taurodontism

In this case the teeth resemble bovine molars. It affects premolars and molars and is characterised by having large pulp chambers, elongated in the corono-radicular direction. Clinically, the crown is normal, so the finding is usually accidental, so radiographically we find a large pulp chamber with short roots and a spaced furcation from the cementoenamel junction in the apical direction.

Taurodontic teeth are common in the paleontological findings of primitive men and in some breeds such as the Eskimos. It is less common in modern men, where the frequency does not reach 1% of the population.

It can be associated with ectodermal hypoplasia syndromes, X chromosome aneuploidies, size changes, shape changes…

2.3.1.3 Invaginated tooth or “dens in dente”

It occurs because during the process of tooth formation the layers of ectodermal cells of the enamel organ instead of expanding outwards, undergo a folding inward of the dental papilla, resulting in a tooth whose tissues give the feeling of being invaginated or folded inwards. Radiographically the image is of one tooth inside another. It can be unilateral or bilateral and is more common in definitive maxillary lateral incisors (60% of cases, with an incidence of between 3% and 10%) and sometimes appears in supernumeraries, being rare to find in the jaw and in temporary teeth.

Depending on the depth of the invagination, the involvement can be only of the crown or also affect the root. It is divided into three types:

- Type 1. Affects only the crown of the tooth, with or without pulpal involvement.

- Type 2. Invagination exceeds the cementoenamel junction without reaching the periodontal ligament.

- Type 3. The invagination exceeds the cementoenamel junction reaching the periodontal ligament, generating a communication between the surface of the tooth and the periodontal ligament that can compromise its stability.

The treatment consists of sealing the entrance to the invagination as soon as possible, since if a caries with pulpal involvement occurs, the presence of enamel inside the crown would make it difficult to treat the ducts.

2.3.1.4 Crown-root syndesmo or palato-radicular groove (PRG).

The tooth has a folding of its outer wall, usually in the lingual surface of the upper lateral incisors, resulting in a cleft in the surface that extends apically and generates a periodontal defect that usually requires the exodontia of the tooth. If the cleft is very deep in the direction of the tooth axis, it can communicate with dentin or pulp, generating an untreatable pulpal pathology.

2.3.2 Anomalies of the crown shape

2.3.2.1 Accessory cusps

They are supernumerary cusps in the crown and can affect any tooth on any surface. It can lead to aesthetic problems difficult to treat as they cannot be removed because being cusps, there can be a pulpal horn inside. Its existence also generates an increase in the susceptibility to tooth decay that presents it, since between the accessory cusp and the surface of the tooth a deep groove is generated.

The accessory cusps may be alterations in the shape or anatomical characteristics of the breed, depending on how we establish the comparison. In Caucasians it is common to find an accessory cusp in the mesiopalatal area of the first upper molar (cusp of Carabelli), while in Mongols we can find a similar buccal or facial cusp (Bolk's cusp).

2.3.2.2 Evaginated tooth (dens evaginatus)

It is a very rare type of accessory cusp that is produced by the elevation of the enamel in the central groove or by lingual of the facial cusp of permanent molars and premolars.

It is caused by a hyperplasia of the pulpal mesenchyme that generates an elevation of the enamel epithelium, so its interior contains pulpal tissue as well as an accessory cusp, and like these can increase the susceptibility of caries and cause aesthetic problems. The fact that they appear in the central groove can cause an alteration of the occlusion that is not easy to solve, since if we cut the evaginated tooth we can make a pulpal exposure. So, in case of an occlusal problem it is better to make an occlusal adjustment via selective cutting.

2.3.2.3 Hutchinson´s tooth and mulberry molar.

Clinically, they are alterations in the enamel that are a result of the histodifferentiation phase, as described in the previous chapter.

2.3.2.4 Crown anomalies in incisors.

They are anomalies in the shape of the crown of the incisors due to a change in the ratio of their mesio-distal width, given that it is normal for the incisors to have the mesial and distal walls diverging towards the incisal surface. The anomalies can be:

- Piano key teeth. The mesial and distal walls are parallel, resulting in a rectangular tooth.

- Screwdriver-shape tooth. The mesial and distal walls are convergent towards the incisal surface

- Oval tooth. The mesial and distal walls are curved, the crown has its maximum contour in the middle third area.

- Shovel-shape tooth. From the buccal surface a normal crown is observed, but the lingual surface has an increase of concavity due to an increase in the size of the marginal ridges.

2.3.3 Anomalies in root shape.

It is difficult to establish anomalies in the root´s anatomy due to the great variability that we can find both in shape and number within normality.

2.3.3.1 Enamel pearls or enamel nodules.

They are deposits of rounded enamel attached to the root cement. They are usually formed only by enamel, although they may contain inside dentin and pulp tissue that extends from the dentin and root pulp.

2.3.3.2 Enamel spurs

They are extensions of the coronal enamel with a triangular shape that exceed the cementoenamel junction at the level of the furcation, especially in mandibular molars by the buccal surface. They can cause periodontal disease in the area of the furcation because they can prevent the correct sealing of the soft tissues at the level where the spur is located.

2.3.3.3 Supernumerary roots

A tooth has supernumerary roots when due to a hyperactivity of the sheath of Hertwig has more roots than those corresponding to its dental group. These can be normal or hook-shaped. Depending on the affected tooth we can find:

- In mandibular incisors, one facial root and another lingual one.

- In canines, a mesial root and a distal root in temporary teeth and a buccal and a lingual root in permanent teeth.

- In upper premolars, two buccal roots and another lingual one.

- In lower premolars, one mesial root and one distal (20% of cases of Turner Syndrome).

- In lower molars, hook-shaped roots by lingual or buccal of the mesial or distal root and great variability of the number of roots in the chordals.

2.3.3.4 Reduction of the number of roots

It is due to a fusion of the roots giving the appearance of a single pyramidal root. It usually affects the second and third upper molars and may have a single pulpal duct.

2.3.3.5 Excessive angles

They have their origin during the process of root formation due to interferences in the correct development of the Hertwig sheath, which due to a mechanical obstacle, lack of space or a trauma changes its direction of development.

- Dilaceration itself is an excessive angulation of the root with respect to the crown, so it can be considered a total shape anomaly.

- Root bending is a pronounced angulation of the root in any area. Generally, and physiologically all teeth have the roots slightly curved distally, we only consider that there is root bending when this angle is excessive.

- Bayonet root form is a double root bend, i.e. the root has two bends with different directions.

## 2.4 Anomalies due to union

2.4.1 Fused teeth

It occurs when two tooth germs are joined. It is usually by the dentine, totally (crown and root) or partially (crown or root) depending on when the joint occurs. The less complete the joint the more advanced the process of development is at the time it occurs. It is more common in anterior temporary teeth, with unilateral or bilateral involvement, and can occur with supernumerary teeth.

The aetiology is not clear. It can be both due to hereditary factors and be the result of a lack of space for the development of dental germs that ends up forcing their union.

2.4.2 Gemination

It occurs when a tooth germ splits in two, partially or completely. If the division is complete it is called twinning because it gives rise to another identical supernumerary

tooth, it would therefore be an alteration in the number. If the division is incomplete, we would have two crowns and a single root.

### 2.4.3 Concrescence

The roots of two teeth are joined by the cement. It is common between the second and third molar or even between a normal tooth and a supernumerary one because these are cases where the roots have less room for development. Clinical crowns are normal, so the diagnosis is radiological, as there is no periodontal ligament or bone tissue between the roots. This way, to perform the exodontia of one of the teeth we must first separate it by odontosection.

#### 2.4.3.1 True concrescence

It takes place during the root formation phase. The lack of space between the roots of different teeth causes them to fuse during their development.

#### 2.4.3.2 Acquired concrescence

It takes place in roots formed between which the interradicular bone disappears and cement is formed in the repair process that joins the roots. There are frequent cases of acquired concretion in diseases that present with hypercementosis, infections or Paget's disease.

### 2.4.4 Coalescence

The roots of two different teeth are joined by dense bone or fibrous tissue. Possibly due to trauma, an infectious process, or lack of space. Radiographically no periodontal ligament or interradicular bone is observed.

# Part II

## DENTAL PATHOLOGY OF BIOLOGICAL ORIGIN

# 3

# DENTAL DISCOLOURATIONS

3.1. Deposits
3.2. Stain

**Introduction**

The natural colour of the teeth can be altered by deposits or stains. The deposits are originated extrinsically by substances that are deposited on the teeth, while the stains have an intrinsic origin and they are due to alterations that can occur either during the formation of the dental tissues or later and they produce changes in colour. The deposits, being of external origin, can be cleaned with cleaning systems, such as brushing or ultrasounds, while staining requires more complicated treatments, such as bleaching. Most of the time, stains only cause aesthetic problems and dental health is not compromised.

## **3.1 Deposits** (extrinsic tooth discolouration or extrinsic stains)

The surface of the teeth can be affected by deposits of different kinds, which can come from food debris, bacteria, saliva, exogenous habits, products of serum origin or even the bacteria that settle on the teeth (cause of the most frequent human diseases: tooth decay and gingivitis).

Deposits on the teeth can vary between individuals as they depend on the oral environment and this is conditioned by the following elements:

- Saliva. Composed mainly of water. It also has organic and inorganic components. It is slightly alkaline (normal pH between 5.6 and 7.9 according to the International Journal of Drug Testing) but on an empty stomach it tends to be more acid.

- Mucin. Mixture of proteins and polysaccharides that play an important role in the formation of plaque.

- Germs. The oral flora is extremely varied and it is in biological balance thanks to the individual's own defenses.

- Epithelial cells. From the buccal and gingival epithelium.

- Degenerate elements. Leukocytes, red blood cells and food debris.

- Self-cleaning. Produced by the action of chewing, the tongue, cheeks, lips and saliva, which remove a large part of the deposits due to friction and the sweeping effect they have on dental surfaces. The oral flora also plays an important role in self-cleaning with the production of enzymes that are able to dissolve deposits in a short period of time.

### 3.1.1 Dental plaque

Dental plaque is the biofilm on the surface of the tooth. It is a microbial ecosystem formed by bacteria adhered to the substrate or interface that can be alive or lifeless. The bacteria are enclosed in the adhesive and protective extracellular matrix of polymers that they themselves have produced. Bacteria show a different phenotype from planktonic cells also having different requirements and organizations and making necessary for them to synchronize.

Dental plaque can be removed from the tooth surface by mechanical and/or chemical control procedures.

The mechanical control would be the sweeping of the dental surface with manual brushing, interproximal hygiene devices, electric brushes, etc.

Chemical control can be carried out using products that have some action on the bacteria, but since bacteria in a biofilm are more resistant to antimicrobial products than planktonic bacteria, mechanical removal is more effective than the use of antimicrobial agents.

### 3.1.2 Materia Alba

It is a whitish, sometimes yellowish deposit composed of bacteria, cellular debris, leukocytes and mucin on the tooth surface with a soft, sticky consistency and less adhesive than dental plaque, from which it differs due to the lack of internal architecture. It is normally arranged in the contact areas of the gum and the tooth (gingival thirds and interproximal areas) where self-cleaning is more difficult. It is also visible on fillings, calculus, gums and dental prostheses.

It occurs by sedimentation of saliva in periods of chewing rest and by salivary chemical alterations and it is sometimes responsible for halitosis. It is easily removed with the water spray from the dental chair due to its lack of adhesion, but its complete removal must be carried out by mechanical means.

### 3.1.3 Calculus

Adherent deposit that forms on the teeth surface, dental restorations and dental prostheses and that comes from the calcification of dental plaque. Dental plaque does not always become mineralized but when it does, mineralization begins in the first hours after its formation and it can become fully mineralized in about 2 weeks. Depending on its location the calculus is classified into supragingival and subgingival.

3.1.3.1 Supragingival calculus

It is visible and whitish in colour although sometimes it appears stained by deposits of another type. It has a hard consistency and it is firmly adhered to the dental surfaces. When there are bulky deposits, they can be easily detached with the use of cleaning instruments and some fragments can even come off spontaneously.

The mineral salts that form the calculus come from the saliva. This is the reason why calculus generally appears on the lingual aspect of the lower anterior teeth due to the proximity of the Wharton canal and on the buccal aspects of the upper molars due to the proximity of the Stenon canal. It also accumulates in areas free of self-cleaning such as dental malposition, antagonist teeth to a painful process that prevents chewing for a long period of time and even in the grooves of the occlusal surfaces in molars.

It is closely related to saliva and appears from an early age in children with poor oral hygiene, although it tends to appear from adolescence, affecting 86% of the population at the age of 40.

3.1.3.2 Subgingival calculus

It is located below the gingival margin, so it is not well observed with a simple visual inspection. A good periodontal examination is required to locate it. It is very hard, stony in consistency, dark, brown or blackish in colour and it is very adherent.

The mineral salts that form the subgingival calculus come from the serum fluid of the gingival crevicular fluid, so it can be found in all teeth and it is not related to the position of the draining canals of the salivary glands.

3.1.4 Dental pigmentations

They are coloured deposits on the acquired pellicle with no difference in their appearance with respect to sex. Its frequency increases in irregularities of the dental surface, such as grooves and depressions and it is influenced by the thickness of the acquired pellicle. Its origin is extrinsic, and its aetiology depends on the agent that gives the colouring.

3.1.4.1 Pigmentations caused by bacteria

- Black pigmentation. It appears as a black line, about 1 mm wide, located in the gingival third of the buccal and lingual surfaces of the teeth. Next to the

interproximal spaces this black line is broader and it can look like a stain. It is not related to the patient's hygiene so it also appears in individuals with good oral hygiene. This pigmentation is due to the activity of certain chromogenic bacteria (pigment producing Actinomycetes and Bacteroides) that decompose ferrous salts from food producing ferrous oxide on contact with the water in saliva. It is adherent and normal brushing does not remove it, requiring the use of cleaning cups and abrasive paste for its removal.

- Green pigmentation. It appears from the gingival margin to the middle of the tooth surface, especially in children and young people due to the action of fluorescent bacteria and fungi. The cause is a pigment called phenazine which is produced by bacteria and fungi, although there are authors who defend that this is a pigmentation from haemoglobin that comes from bleeding gums. It is frequently related to children with poor oral hygiene and due to lack of brushing it can increase in size and thickness. It usually disappears at puberty due to changes in the flora of the bacterial plaque. Its removal is easy with cleaning cups or brushes and abrasive paste.

- Orange pigmentation. It appears as small irregular orange spots or stains on the buccal surfaces of the anterior teeth on the gingival third. It can be found affecting only one tooth. They are bacterial colonies formed on the dental plaque so they simply disappear with brushing.

3.1.4.2 Pigmentation due to consumption of coloured substances

-Pigmentation caused by habits. The repeated intake of some foods or certain habits deposit substances on the teeth surfaces, as it occurs with coffee, tobacco, tea or marijuana for example. The substances are retained in places where there is no self-cleaning. That is why they are more evident in interproximal areas and on the buccal surfaces of retruded or rotated teeth. To eliminate these pigments, it is usually necessary to use ultrasounds since the substances are quite adherent.

-Metal pigmentation. It is closely related to certain professions and it is generally due to the inhalation of metal salts in industry. This metallic dust binds to the acquired pellicle and it generates greenish (lead, nickel), brown (iron) or black (silver, manganese) pigmentation. To remove it, you must have a strict brushing regime because these deposits can penetrate through the enamel and they can produce permanent discolorations.

- Iatrogenic pigmentation. They are those that health professionals produce when prescribing some type of medication to treat a pathological process and that are caused as an unwanted collateral effect. Chlorhexidine generates greyish-brown spots. Medications for anaemias and vitamin complexes cause black deposits. Fluoride mouthwashes can also cause

coloured deposits. They are removed with hygiene cups or brushes and abrasive paste.

**3.2 Stains (Intrinsic tooth discolouration or intrinsic stains)**

They are of intrinsic origin since they occur inside the dental tissue affecting the dentine and/or the enamel. Being intrinsic, they cannot be removed by brushing, ultrasound or abrasive pastes since they are not adherent to the tooth surface.

Staining of these structures can occur during the formation and development of teeth or when they are already formed.

3.2.1 Fluoride stains

Fluorosis occurs as a consequence of an excessive intake of fluoride during the period of development and calcification of the dental enamel, that is, from the sixth month of gestation to 8 years of age.

The fluoride can come from the water daily intake or from the fluorides used in caries prophylaxis. The degree of affectation is related to the concentration of absorbed fluoride. From 1 ppm, it can cause fluorosis.

It affects the permanent dentition more than the temporary one because the greater mineralization of the temporary teeth occurs during the gestation and the placental barrier plays an important role.

To assess the severity of fluorosis, Dean's criteria are applied, which quantify it as follows:

- Normal enamel: enamel shows normal translucency, the surface is smooth and shiny and, generally, it is pale cream coloured.

- Questionable: small aberrations in the translucency of normal enamel that can range from a few whitish shades to white spots of 1 or 2 mm in diameter.

- Very mild: irregular opaque white areas on the buccal surface of the tooth (<25% of the tooth surface).

- Mild: opaque areas affecting half of the tooth surface, with a marked occlusal attrition.

- Moderate: there are no changes in the shape of the teeth and in general all the tooth surfaces are affected, showing marked wear on the surfaces subject to attrition.

- Severe: hypoplasia is so severe that the shape of the teeth may be affected, forming large irregular hypoplastic areas.

The severity of fluorosis is influenced by the age at which it begins and the duration and the termination of enamel mineralization. The hypoplastic manifestation of the enamel will be more severe the later the tooth erupts.

### 3.2.2 Tetracycline staining

The mechanism of action corresponds to the chelating power of tetracycline on calcium, forming tetracycline orthophosphate, initially colourless, but which under the action of ultraviolet radiation darkens the teeth. This justifies that the anterior teeth present more discoloration than the posterior ones. The intensity of the lesions is consistent with the amount ingested, the repetition of the treatments and the age at which the antibiotic treatment occurred.

According to Jordan, 4 degrees are distinguished:

- Grade I: minimal involvement, simply a yellowish-brown or greyish colouration is seen on the entire tooth surface.

- Grade II: no bands but with a coloration similar to the previous one but more intense.

- Grade III: horizontal bands appear, especially in the gingival third, dark grey or bluish in colour.

- Grade IV: the entire crown is affected with an intense blue or characteristic black colour. It is located mainly in the dentine and responds with greater intensity of fluorescence than the previous degrees to illumination by ultraviolet light. This is a diagnostic test in this type of staining.

### 3.2.3 Staining of pulp origin

Pulpal necrosis and dental trauma with or without intrapulpal haemorrhage are processes that involve dental staining. The frequency of those of traumatic origin is higher in the anterior and superior group and especially in childhood. In most cases only one tooth is affected.

Staining occurs as a consequence of the degradation of haemoglobin to hemosiderin, when there is haemorrhage and the entry of these products into the interior of the dentine tubules, or by products of protein degradation in pulp degenerations.

The coloration is more intense when there is bleeding and varies from brown, grey or black. In both cases these products that are generated in the pulp chamber and penetrate the dentine tubules causing this intrinsic staining that is difficult to remove.

### 3.2.4 Iatrogenic staining

They are produced by dental professionals while treating the teeth and they can occur for different reasons:

- Amalgam: it undergoes corrosion over time causing staining in the dentine tubules, giving the tooth a greyish or blackish colouration that will increase over time.

- Pulp remains: this happens when the dental pulp is not fully removed when opening the pulp chamber to perform endodontic treatment. This is very frequent in the upper incisors as the pulp horns are not included in the opening.

- Root filling cements and gutta-percha remains: if they are not properly removed from the pulp chamber when the tooth is to be filled. They also cause gingival staining.

- Some drugs used in the past such as argentophenol and argentochrome also produced stains.

# 4

# REACTIVE GROWTH OF HARD DENTAL TISSUES

4.1. Reactive processes of the dentine-pulp complex.
4.2. Reactive cement processes.

**Introduction**

The reactive processes of the dental hard tissues are defensive response mechanisms of the tooth against chronic irritative stimuli, both physiological and pathological, which result in the creation of new dental tissues by the cells they contain. Enamel, being an acellular structure, lacks reactive capacity, so these defensive processes will only appear in dentine and cementum.

Dentine and pulp form the dentine-pulp complex, which is why we consider them as a unit.

## 4.1 Reactive processes of the dentine-pulp complex

They are mechanisms aiming to protect the pulp from chronic irritative stimuli. These irritative stimuli activate the odontoblasts, producing a greater amount of dentine and they can also stimulate other pulp cells producing a tissue similar to dentine. Depending on the cells that are stimulated, one or another tissue will be created. There are different factors that influence the type and amount of tissue that is formed in response to these stimuli, the most important are:

- Stimulus intensity: usually they are mild or moderate stimuli.

- Periodicity of the irritating stimulus: they are always more or less chronic stimuli that are repeated over time.

- Age of the tooth: defence tissues increase with age.

- State of the pulp: it must be vital.

- Aetiology of the stimulus: bacterial, chemical, traumatic, thermal, etc.

- Depth of the lesion: proximity to the pulp.

Depending on these factors, the dentine-pulp complex will generate sclerotic dentine, tertiary dentine and/or osteoid tissue, and it can sometimes generate all types of reactive tissue in different areas of the same tooth.

The pulp can also react by calcifying itself, forming pulp calcifications or it can respond with inflammation, leading to pulp necrosis, especially if the stimulus is acute and intense.

### 4.1.1 Sclerotic dentine

It is formed as a defensive response of the dentine-pulp complex against chronic and slow irritative stimuli. The odontoblasts, when irritated, accelerate the production of dentine causing sclerosis or closure of the dentine tubules, resulting in hypermineralized dentine, especially the peritubular dentine, with acid-resistant mineral deposits inside the tubules that are partially or totally closed. This reduces the permeability of the dentine and isolates the pulp from aggression, protecting its vitality.

There are 2 types of sclerosis:

- Continuous formation of peritubular dentine. It is physiological. It occurs in healthy teeth and increases with age.
- Intratubular calcification. It is pathological. It occurs against an aggression such as tooth decay.

#### 4.1.1.1 Aetiology

A) Physiological factors. Physiological sclerosis

Tubular sclerosis is physiological in elderly people and it is frequently found due to the progressive aging of the population. It is produced by the normal aging of the tooth. With age, the odontoblastic process regresses and retracts towards the pulp due to the decrease in vascular supply due to fibrosis of the pulp tissue. At the same time, the peritubular dentine matrix gets calcified and obliterates the space occupied by the odontoblastic process, closing it from the outside in. It is peritubular sclerosis. Finally, the lumen of the tubules disappears and they are blocked by that peritubular dentine that takes on a very transparent appearance. Hydroxyapatite crystals in intertubular dentine are smaller in size in the sclerotic dentine of the old tooth than in the healthy dentine of the young tooth.

B) Pathological factors. Pathological sclerosis

It is an intratubular sclerosis due to the precipitation of mineral salts within the dentinal tubules. It consists of intratubular mineralization combined with hypermineralization of the peritubular dentine and greater resistance to demineralization of the intertubular dentine. In order for this dentine to be produced, it is necessary for the odontoblast to be alive, so it is only created against chronic stimuli of mild or moderate intensity and in teeth with vital pulp. It is found in the translucent area under the caries, in the exposed dentine of teeth with attrition, abrasion and in patients with bruxism. It may be due to:

- Traumatic agents, microtraumas that act repeatedly, continuously and for a long time on the tooth. They may be:

    - o Premature contacts due to dental malposition, overhanging fillings, etc.
    - o Frequent interposition of a foreign body (pen, pencil, pipes, etc.).
    - o Destructive processes such as erosion, attrition or abrasion.
    - o Bruxism.

- Infectious agents, caries is the most important, even in shallow caries the response of the pulp is immediate. Sclerotic dentine usually appears under slowly evolving caries.

- Iatrogenic agents, direct pulp coverings and cavity bases stimulate the formation of sclerotic dentine.

4.1.1.2 Histopathology

Healthy dentine is opaque but sclerotic dentine is translucent and shiny, very similar to amber. Areas of tubules that have not been filled with dentine can be seen and these are called dead tracts. This seems to be due to the fact that before the odontoblast extension fully retracts, the peritubular dentine closes the tubule above and below, and inside remains of the degenerating odontoblastic process are trapped, leaving the tubule empty. These tubules can allow caries a faster progression and allow the arrival of chemical agents to the pulp.

In physiological sclerosis, sclerotic dentine deposition begins in the tubules on the apical third of the root and ascends towards the crown, sometimes reaching the middle. This gives the apex a characteristic translucent appearance that is used to identify an old tooth.

In pathological sclerosis, only the tubules whose extensions are in the area of injury are affected. In very advanced stages, all the tubules can be closed. The layer of hypermineralized dentine is shaped like a cone with the base facing the lesion.

4.1.1.3 Clinical manifestations

We can find sclerotic dentine under a caries, a filling, etc.… or see it exposed on the occlusal or cervical surfaces in cases of erosion, attrition, bruxism and also in the apexes of old teeth extracted.

This sclerotic dentine should be maintained whenever possible as it is a protective tissue. It has an appearance similar to amber, translucent or transparent orange or brown. It is smooth, shiny and hard when trying to remove it with an excavator. It shows increased radiopacity and it is impermeable to dyes so it is not stained by caries detectors.

The degree of sensitivity is variable and depends on the number of tubules affected and the degree of obturation of these. In exposed dentine the sensitivity is high but when sclerotic dentine begins to form the sensitivity decreases until the tubules close completely and becomes very insensitive to cold, heat, sweets, etc.

4.1.2 Tertiary dentine (reactionary dentine)

It is a mineralized tissue similar to physiological sclerotic dentine but with different histological characteristics. It appears to defend and protect the pulp from chronic aggressions in an attempt to compensate for the lost dentine. Both odontoblasts and other differentiated cells called odontoblast-like or odontoblastoid cells are able to form tertiary dentine.

It is generally less permeable than physiological dentine. This is because the number of tubules is usually smaller or non-existent and if there are, they are usually clogged at its pulpal end by the newly formed tissue.

The shape of the tubules may vary, some are regular similar to normal dentine and others are very irregular, twisted, with no communication with normal dentinal tubules or newly formed ones.

The rate of deposition and structure of the dentine depends on the degree of injury, intensity, and duration. The more severe the damage, the more irregular and scattered the tissue formed and the faster the deposit. In the tertiary dentine, that is formed rapidly, odontoblasts, other cells, vessels or elements of pulp tissue can be found and it is called osteodentine. It also shows almost no interlaced tubules. The separation between the physiological dentine and the formed tertiary dentine is called the calciotraumatic line (CTL).

Some authors differentiate 2 types of tertiary dentine:

- Reactive dentine, produced by odontoblasts that survive external aggressions of moderate intensity.

- Reparative dentine, produced by odontoblast-like cells that react to more severe lesions that have destroyed odontoblasts.

Both reactive and reparative tertiary dentine are a way of defence, so any aggression that exposes dentine or makes contact with it has the potential to stimulate its formation. Some authors define a third type of tertiary dentine:

- Physiological tertiary dentine, which occurs throughout life but we prefer to call this dentine, secondary dentine, because it is the one that creates the tooth from the end of the formation of the root throughout its life, and tertiary dentine the one formed due to an aggression to the pulp.

When an injury occurs, the odontoblastic extensions are exposed or sectioned and the odontoblast usually degenerates and dies although some may remain alive and form tertiary dentine. New cells are formed, the odontoblast-like or odontoblastoid cells, below the odontoblast layer, in the subodontoblastic layer and in the cell-rich layer. These cells have no intratubular extension and acquire a cuboidal or flattened appearance, having no nervous control. Their activity is not stopped by dentinogenesis and generates tertiary dentine continuously until the pulp is almost completely obliterated.

Much has been speculated about the defensive or non-defensive role of this dentine on the pulp. It can be deposited even if the pulp is irreversibly damaged or undergoes a chronic inflammatory process.

This dentine is less permeable but encompasses areas of necrotic tissue where it is more permeable. It seems clear that it delays the entry of the harmful agent into the pulp but the structure it will adopt cannot be predicted and the barrier may be incomplete and not fully protective.

4.1.2.1 Aetiology

A) Traumatic agents

- Attrition, abrasion, erosion, bruxism.

- Crown-root fractures.

- Iatrogeny, preparations with poor refrigeration, direct and indirect pulp coatings.

B) Infectious agents

- Caries: causes tertiary dentine at the pulp end of the affected tubules.

- Periodontal disease.

C) Dentine anomalies

- Dental dystrophies and dysplasias.

- Dentinogenesis imperfecta (DI).

In the same tooth, some odontoblasts are irritated and produce sclerotic and/or tertiary dentine, others are destroyed and replaced by odontoblastoid cells, that also generate tertiary dentine, and others remain unaltered occupying the lumen of the dentinal tubules.

4.1.2.2 Histopathology

Tertiary dentine can have a very varied structure, it has no phosphoproteins and it is produced by cells directly involved with the stimulus. It has histological differences according to the cells that generate it and the number of tubules present. Based on that, the following histopathological classification can be established:

A) According to the cells that generates it

- Reactive dentine: produced by the odontoblasts that survive. It appears to face external aggressions of moderate intensity and slow progression. It is a more organized and more regular dentine with more tubules.

- Reparative dentine: produced by neodontoblasts (newly formed odontoblasts) or odontoblast-like cells. It is a response to more intense external aggressions that appear quickly. It is a more irregular, disorganized dentine with fewer tubules than reactive tertiary dentine.

B) According to the number of tubules (These 3 types of dentine can be found in the same tooth)

- Tubular dentine: with tubules

- Oligotubular dentine: few tubules

- Non-tubular dentine: without tubules

4.1.2.3 Clinical manifestations

Tertiary dentine is usually located in the pulp area next to the lesion. It is more common in the chamber walls at the level of the pulpal horns in molars and premolars and/or in the root canals, deforming their contours.

Radiographically, it is possible to see it modifying the contour in a localized area, for example:

- On the ceiling of the chamber, reducing its size. This is seen when the occlusal face of the tooth is affected, as in bruxism.

- In the pulp horn underlying the lesion, as in caries or a filling.

- In the root canals and apexes, as a result of chronic inflammatory or traumatic periapical injuries.

4.1.3 Osteoid Tissue

It is the pulp's immediate response to chronic aggression. It is produced by pulpal fibroblasts when the aggression destroyed the odontoblasts and cells of the subodontoblastic layer but is not so intense as to destroy the fibroblasts.

Suffering the tooth an aggression, the differentiation of the new odontoblasts takes 4-6 weeks. During this time the fibroblasts are producing osteoid tissue: it is the urgent response to that aggression.

When tertiary dentine begins to be generated, a mixed tissue called osteodentine (a mixture of tertiary dentine and osteoid tissue) quickly forms. It may have cell inclusions, vessels, and pulpal tissue with an irregular tubular pattern.

4.1.4 Pulp calcifications

Their aetiology is unknown and they are clinically asymptomatic. They are only detectable radiographically when they are large, which usually occurs in 10% of cases. There are two types:

- Diffuse, it is a calcification without a defined shape in the chamber or in the pulp canal.

- Nodular, it is also called pulp calculus because it is located in the pulp chamber. It can be formed by dentine (true denticles) or be amorphous (false denticles)

Depending on their position, the nodular calcifications may be:

- Free, surrounded by pulp tissue, without contact with dentine.

- Attached, fastened by a small dentine bridge to it.

- Embedded, completely coated with dentine, immersed in it.

#### 4.1.5 False pulp stones

They are calcifications formed by concentric layers of calcified material that have nothing to do with dentine. They develop in a healthy pulp and they are the most common calcifications. They appear mainly in the pulp chamber but are not uncommon in the root canals. It seems that there is a genetic predisposition to form pulp calculus. We must differentiate them from the calcium degeneration that is formed in a pathological pulp and generally in the radicular pulp.

##### 4.1.5.1 Aetiology

They appear mainly due to the physiological aging of the tooth and their frequency increases with age. They also occur in response to chronic irritative stimuli such as tooth decay or periodontal disease. Other times they appear spontaneously.

##### 4.1.5.2 Etiopathogenesis

Pulp stones are produced by the initial calcification of one of the components of the pulp tissue that serves as a nest so that later calcification material is deposited on it in concentric or radial layers. Vascular damage following trauma or metabolic dysfunction could be the trigger for nest development.

##### 4.1.5.3 Histopathology

They are usually more or less rounded, well defined and generally small so they are not seen on x-rays. Other times they are large and occupy almost the entire pulp chamber. They generally develop near vascular structures and are more common in the pulp chamber. There may be several calcifications that fuse as they grow, making endodontic treatment difficult. You can find them:

- Free, surrounded by healthy pulp tissue. At first all calculus is free.

- Attached: free stones that by apposition of secondary or tertiary dentine, are united to the rest of dentine by a bridge.

- Embedded: adhered stones that in advanced stages are completely surrounded by dentine.

##### 4.1.5.4 Clinical manifestations

They are usually asymptomatic and are usually radiological findings. Authors found a higher frequency of pulpal stones in patients with calcification-producing diseases such as arteriosclerosis.

Radiologically they depend on the density and the size. If they are very small, they are not seen but with time they increase in size and density and they can be observed deforming the contours of the pulp chamber, with undefined edges. If they are very large, they can occupy almost completely the pulp chamber.

#### 4.1.6 Diffuse pulp calcifications

They are small, irregular, calcified deposits scattered throughout the pulp tissue. They are also called diffuse linear calcifications because of their orientation. They are

frequent, increase with age, and usually appear in the radicular pulp and then spread to the pulp chamber.

4.1.6.1 Histopathology

They have an irregular matrix of collagen fibres and electron-dense interfibrillar material in which the hydroxyapatite crystals are deposited.

4.1.6.2 Clinical manifestations

They are not visible clinically or radiographically and can only be observed in histological sections due to their dispersion and size and lack of clinical significance.

4.1.7. Calcific metamorphosis (CM)

It is the pulp response to acute or chronic trauma that stimulates the formation of hard tissue within the root canal space and it is characterized by a yellow discoloration of the clinical crown (Gutmann, 2001). Most often it is accompanied by tertiary dentine which causes narrowing of the root canal and probably the change in coloration of the clinical crown. There is a decrease in tooth translucency due to an increase in dentine thickness under the enamel.

4.1.7.1 Aetiology

It may be due to trauma to a young tooth that usually reoccurs over time, although it may also appear as a side effect in temporary teeth treated with pulpotomies or in teeth with orthodontic treatment. It is more common in young teeth with incomplete root development and crown fractures or subluxation.

4.1.7.2 Histopathology

The mechanism of formation is not clear and the authors do not agree. It can be a response of the pulpal tissue to a not too intense trauma, especially in young incisors, which is repeated over the years. Depending on the cells of the pulp that respond, calcified formations of tertiary dentine, osteoid tissue or osteodentine will be found. Most of the time it is a mixture of these tissues.

4.1.7.3 Clinical manifestations

These teeth have a fairly opaque orange-yellow crown. The pulp can be vital, the vitality can be diminished or even be negative, according to the degree of obliteration of the canals. Pulp necrosis can occur.

The consequence is the obliteration, usually partial, of the pulp cavity and especially of the canals, which can greatly hinder endodontic treatment, making it sometimes impossible.

Radiographically, a partial or total obliteration of the pulp cavity can be seen, although this does not mean the absence of pulp or pulp space. Periodontal ligament enlargement or periapical radiolucency with or without subjective symptoms may be observed.

### 4.2 Reactive cementum processes

They are those cementum tissues that appear due to the physiological aging of the tooth or as a defence of the cementum against a chronic pathology.

4.2.1 Cementum growth by age: passive eruption

Tooth cementum has the ability to be formed by cementoblasts throughout the life of the tooth. There is a slow and continuous physiological formation of cementum that facilitates tooth movement and provides Sharpey's fibres with a surface to join the tooth.

Over the years, the tooth loses length due to physiological occlusal attrition, shortening the crown. As a mechanism of compensation for coronal wear, a cementum deposit is generated continuously to keep the tooth in position with respect to antagonist and adjacent teeth, thus keeping the tooth in the plane of occlusion. This is called passive eruption.

There are 2 types of cementum tissues:

A) Apical and interradicular: by concentric apposition of successive layers of cementum to maintain the dental intraosseous portion. This can vary the position of the apical foramen or the path of the accessory canals and it must be taken into account when performing an endodontic treatment.

B) Lateral: the apposition of new layers slowly and continuously allows the insertion of the fibres of the alveolar-dental ligament to be modified and renewed continuously. This results in a reduction in the root concavities.

4.2.2 Hypercementosis

This is an abnormal thickening of the cementum, usually in the middle or apical third of the root. The exact amount of cementum to consider that hypercementosis exists is not defined in terms of measurable amounts. The diagnosis is made based on clinical experience, by comparison with other teeth or by radiological findings.

It occurs in devitalized teeth as the metabolism in these teeth continues and facilitates the biological closure of the apex in endodontic treatments.

4.2.2.1 Clinical manifestations

- Diffuse or circumscribed.

- Affecting a single tooth, lower teeth are more affected, especially the premolars, followed by the first and second molars.

- Affecting one, several or all roots in the multiradicular teeth.

- It can affect all teeth: generalized idiopathic hypercementosis.

- It can affect part or all of the root of the tooth.

- When a certain degree is exceeded, the cement-forming hyperactivity stops and the opposite phenomenon occurs: cementolysis.

4.2.2.2 Aetiology

- Local factors, more or less chronic and not very intense pathological stimuli such as anocclusion, chronic periodontal disease, orthodontic treatments and low-intensity but constant trauma (overload).

- General or systemic factors, such as Paget's disease, acromegaly, rheumatic fever, etc. Systemic and hereditary diseases are considered the most important etiological factor in generalized cases.

- Idiopathic factors.

4.2.2.3 Etiopathogenesis

- Cementum hypertrophy, related to the improvement of the functional properties of the cementum.

- Cementum hyperplasia, the cementum deposit is not a response to excessive function or are non-functional teeth or is caused by local or systemic pathological factors. When cementum hyperplasia occurs in non-functional teeth it is characterized by a reduction in the number of Sharpey's fibres and when the hyperplasia is secondary to apical inflammation or high occlusal pressures, ankylosis of the roots can occur, which may require removal of bone to make possible the exodontia of those teeth.

4.2.2.4 Histopathology

Irritant lesions will stimulate undifferentiated periodontal ligament cells that differentiate into cementoblasts. These produce a cementoid apposition (tissue similar to cement but without the apatite mineral component) on the outer surface of the cementum, with abundant cementoblasts, which is then calcified and the cementoblasts disappear.

Hypercementosis can occur in 2 ways:

- Located: in areas subject to greater traction. Spike-like cementum protrusions form that provide a larger surface area for the insertion fibres of the periodontal ligament and ensure a firmer anchorage of the tooth in the surrounding bone.

- Diffuse: The most common cause is chronic periapical inflammation and overload. It occurs mainly in the apical area producing a root in "drumstick".

Diffuse and generalized forms often appear due to systemic, idiopathic diseases or after orthodontic treatments and are quite rare. They are seen mainly in older patients. In young patients, the localized form as spicules is more common.

4.2.2.5 Clinical manifestations

There are no signs or symptoms, the vitality and percussion tests are not altered, there are no signs in the mucosa that covers the hypercementosis and there is no expansion of the bone cortex.

Hypercementosis in young people can delay the eruption and it may affect the mobility of the teeth during the eruption. Mobility is also affected in orthodontic treatments.

The shape of the root is altered due to the apposition of cementum but it will be surrounded by a continuous and uninterrupted periodontal space and a normal alveolar cortex.

Radiographically, it is a by chance finding. This cementum sometimes has the same density as dentine so hypercementosis is manifested as a localized or generalized radiographic increase in the root. Sometimes the newly formed cementum has a lower density than dentine, so its edges are clearly visible.

In devitalized teeth, hypercementosis can be stimulated by chronic inflammation of the periodontal membrane. In this case, areas without periodontal or alveolar cortical ligament are observed radiographically. An area of bone destruction can also be seen in the periapical area.

4.2.2.6 Treatment

These teeth do not require any treatment, just keep in mind that exodontias can be more difficult due to ankylosis from root to bone and require surgery and that in young people and children with hypercementosis orthodontic treatment will have special considerations.

4.2.3 Cementicles

They are small mineralized bodies in the thickness of the periodontal ligament, at any level, but especially in the apical third.

They are usually rounded or ovoid and very small (usually no more than 0.2 mm) so it is very difficult to see them on an x-ray. They can be presented alone or in groups near the cementum surface. If they are very numerous, they produce a calcification of the periodontal ligament.

4.2.3.1 Etiopathogenesis

They increase with age and are more common in periodontal disease and trauma. Depending on their origin can be of 2 types:

- Formed by cementum tissue: the participation of cementoblasts is required, which is why they are often associated with hypercementosis.

- Formed by calcium precipitation: they are caused by vascular alterations, capillary or venous thrombosis, or slowing of the circulation that facilitates calcium deposits.

4.2.3.2 Histopathology

They have a structure of concentric laminar cementum layers. Those formed by calcium precipitate are amorphous or osteoid-looking formations. They can be of 3 types:

- Free: they are surrounded by soft connective tissue.

- Attached: when growing the nearest cementum is fused with it

- Embedded: they are fully incorporated and covered by the cementum.

#### 4.2.3.3 Clinical manifestations

They are asymptomatic although periodontal pain due to nerve compressions has been described. They can be confused with false cementicles that are fragments of cementum that come off in those teeth with mobility, where strong and/or inadequate tractions are done.

# 5

# TEETH CONSUMPTION PROCESSES WITH PHYSICAL AND CHEMICAL ORIGIN

5.1 Attrition.

5.2 Abfraction.

5.4 Abrasion.

5.3. Erosion.

**Introduction**

The definition of consumptive in the dictionary says that "consumptive is understood as one that has the capacity, virtue and effectiveness to consume or to be consumed". There are several such processes at the dental level that require our attention, attrition, abfraction, abrasion and erosion.

**5.1. Attrition.**

Attrition is the gradual wear and tear of dental hard tissues due to chewing by tooth-to-tooth contact. This can be physiological, which is what occurs normally during chewing food and derived from a natural use over time or pathological, due to overuse in both time and intensity. Establishing a dividing line between the physiological and the pathological is quite complicated by the variability of factors, intensities and uses of each individual. The factors that can influence the degree of attrition in each individual are:

- Structure and occlusal relationship of the teeth.
- Calcification of the enamel.
- Development of chewing muscles.
- Chewing habits.
- Ability of food abrasion.

5.1.1 Etiopathogenesis

Attrition increases with age, either physiologically due to the natural wear of the parts or pathological due to an increase in wear and tear due to vital complications, medication use, greater muscle power, etc.

Attrition is greater in men than in women, due to increased hypertrophy and power of male muscles, although the differences are not overly significant.

Occlusal conditions also influence, as a fewer teeth in the arch causes them to suffer a greater masticatory workload.

Hyperfunction (bruxism) is the most common cause of attrition. Its prevalence rate is 5 to 20% in the adult population. The physiological loss at the vertical level of the tooth is 65µm / year and that in bruxists this figure is multiplied by three or even four.

The estimated contact time between dental pieces of both archs is estimated at about 17.5 min / day, considering the processes of chewing food and the contacts derived from the swallowing. All the extra time of contact between the pieces would suppose an excess of work.

With regard to nutrition, abrasive particles in meals, chewing gum, tobacco, betel nut, produce a pathological attrition.

In addition, the lack of lubrication of the saliva (xerostomia) for various reasons, it increases the wear capacity of the dental pieces. It would be something similar to what happens when the pistons of a combustion engine are not lubricated, there is an excess of friction and the durability of the same is quite affected.

5.1.2 Clinical manifestations

Broca's classification establishes the different degrees of affectation:

0: No attrition.
1: Enamel, the cusps are still identified.
2: Exposed dentine.
3: Occlusal morphology is lost.
4: Inverted cusps.

The clinical manifestations of attrition are like their degrees of affectation, being a slow process, it makes the response mechanisms of the tooth have time to adapt to the loss of material, not manifesting in many cases dental hypersensitivity and except in extreme cases, common pulpal exposure processes are not often seen. What we can observe are discolouration processes, due to the porosity of the dentine, tissue remineralization processes in response to aggression, observing hypermineralised tissues with 8% more than normal.

Moderate occlusal attrition is usually associated with a lower incidence of caries, due to the elimination of grooves and occlusal fissures that represent retentive elements, disappear and allow greater ease of washing. In the same way, periodontal problems have a lower incidence, since when cusp interference is eliminated, the disclusions are performed in a freer way. At the proximal level, it creates large contact surfaces due to the loss of dental material at the vertical level, affecting the contact points. So mesial migrations of the teeth can appear and a shortening of the dental arch of up to one centimetre can also occur. At the level of the necks of the teeth it causes a phenomenon called abfraction. We will see this further on. There can also be facial defects in patients with Angle´s class III malocclusion (prognathism) and anterior crossbite.

In a physiological attrition the wear follows a constant pattern, which initially affects the incisal edges, as the process progresses it affects the occlusal surfaces of molars, maxillary lingual cusps and mandibular facial cusps. Most cases are presented as concave defects surrounded by enamel, having a higher incidence in men and aggravated by the association with erosion and / or abrasion.

A pathological attrition can be caused by a pathological occlusion either by alterations in development or by processes of partial edentulism caused by different reasons. Habits such as bruxism or chewing gum or tobacco increase attrition. Similarly, the processes of structural alterations in the formation of dental tissues such as amelogenesis and dentinogenesis imperfecta will make the wear of these tissues much faster than if they had adequate structure and mineralization.

Radiographically we can observe a reduction in the size of the pulp chamber, increase in periodontal space, loss of alveolar bone, osteoporosis and osteosclerosis.

5.1.3 Prevention and Treatment

For preventive purposes, we will focus efforts on patient observation and follow-up. On action on the aetiological factors that cause pathological attrition, dietary modification, both in factors that enhance attrition and in those that synergistically weaken the structures exposed by this process and control of parafunction.

Restorative treatment is focusing on returning the patient to functionality by recovering the vertical dimension, improving the aesthetics of those injuries that involve visual defects that the patient wants to correct, performing these procedures through composites, onlays, inlays or crowns of total coating.

**5.2 Abfraction**

Abfraction (ab, "far"; fractio, "fracture") is the loss of hard dental tissue caused by biomechanical loading forces.

5.2.1 Etiopathogenesis

Physiologically, the enamel layer thins towards the neck of the tooth, being a very thin enamel and with a greater breaking capacity than other surfaces of the tooth. In attrition, the tooth is subjected to constant flexion and shear forces by occlusal movements. The union of these two factors causes the enamel closest to the cementoenamel junction to come off and this abfraction phenomenon to occur. We must be careful because after the breakage of these surfaces of the enamel the exposed dentine is at the mercy of processes such as abrasion by brushing and the action of cariogenic agents.

5.2.2 Clinical manifestations

Clinically we will find signs of loss of dental structure, fracture and repeated detachment of restorations, being the classic symptom the dentinal hypersensitivity of diffuse location. The differential diagnosis is simple, brushing injuries affect several teeth and in abfraction the surrounding teeth are not affected. Only the tooth involved in the process and sometimes its antagonist of the opposite arch.

A) Clinical forms in enamel:

- Filiform cracks, visible and accentuated with transmitted light.
- Striations, irregular horizontal bands of enamel due to molecular fractures and called Luder's lines.
- In the shape of a plate, located entirely in the enamel.
- Semilunar, located entirely in the enamel.
- Invagination at the end of a cusp.

B) Clinical forms in dentine:

- Gingival, deep and acute angular groove that appears in the gingival margin of the facial surface, denominated groove of McCoy.
- Circumferential, completely surrounds the cervical region of the tooth.
- Multiple, two or more grooves appear on a surface.
- Subgingival, below the gingival margin and can be single or multiple.
- Lingual, on the lingual surface at the cement-adamantine junction.
- Interproximal, it is observed on rotated teeth, outside its normal position in the arch.
- Alternating, the abfraction appears in a tooth, but is absent in the adjacent tooth.
- Angular, the abfraction lesion is at an angle of 45º.
- Crown margin, the lesion extends below the finish line of the prosthetic crown.
- Restoration margin, a gingival caries-free abfraction appears to all types of restoration (composite, amalgam) without a sign of abrasion by brushing.

5.2.3 Treatment

Abrasion treatment begins with an early occlusal adjustment to reduce the workloads of the parts to reduce compression and flexion on the affected teeth. Most defect repairs of these lesions are performed with composite resins. The decrease in the existence of habits such as bruxism through relaxation techniques, control and the use of splints would be in the long run a fairly effective tool to reduce the possible complications of such processes.

**5.3 Abrasion**

Abrasion is defined as the dental pathology caused by the friction of a foreign body on the dental structure, independently of the occlusion between the teeth. It can be located in any area where friction occurs.

5.3.1. Aetiology

A) Individual factors:

- Brushing: technique, strength, time.
- Dominant side.
- Protruding teeth: canines.

B) Materials:

- Abrasives: calcium carbonate, aluminum oxide, silica, sodium bicarbonate.
- Beveled bristles and of lower concentration.

C) Factors associated with therapy:

- Periodontal treatment.
- Prosthetic treatment.
- Orthodontic treatment.

D) Labour factors:

- Construction.
- Shoemakers.
- Tailors.
- Musicians.
- Work with abrasives in suspension.
- Piercings.

5.3.2 Clinical manifestations

Its location is usually established in the facial surfaces at the level of the cementoenamel joint, being more frequent from canine to first mandibular molar, in maxillary premolars and incisal edges.

On exploration we find:

- Indefinite contour.
- Smooth and hard surface.
- There is no plaque.
- There is no discolouration.
- Smooth and shiny enamel.
- Very polished dentine.
- Plate-shaped injury.
- Gingival recession.

The symptoms of the abrasion will depend on the degree of wear or loss of the dental material suffered. A slight abrasion may not trigger any symptoms or cause occasional hypersensitivity.

5.3.3 Histopathology

Histologically, we can observe an obliteration of the tubules and the formation of reactive dentine. Usually, there is no pulpal exposure, except in cases of tissue loss due to abrasion.

5.3.4 Prevention and Treatment

At the preventive level, we will focus on the triggers of the process, change the brush to medium hardness and use kinds of toothpaste with lower abrasion volume, improve the brushing technique and reduce its strength. We will also have to correct harmful habits such as holding objects with teeth.

At the restorative level, as we have seen in the abfraction we will use adhesive restorations based on composite resins, to solve aesthetic defects. We will try to reduce dentinal sensitivity with specific kinds of toothpaste and mouthwashes. Sometimes we will need to use varnishes or even resorting to restorations with adhesive to reduce it.

**5.4 Erosion**

Erosion is the loss of hard tissue from the tooth by dissolution due to acids that do not come from bacterial metabolism. The risk of erosion is increased by current dietary habits, as well as by psychological alterations resulting from inadequate dietary patterns.

5.4.1 Aetiology

We can find two types of agents that can cause erosion:

A) Extrinsic agents:

- Diet.
- Environmental factors.
- Medications.

B) Intrinsic agents:

- Perimolysis.
- Salivary deficits: Sjögren, Mikulicz

With regard to diet, we know that the consumption of foods with a low pH such as citrus fruits, vinegar, carbonated drinks and acidulants, as well as juices tend to cause erosive processes. In this table, we see the pH of the most traditional drinks consumed compared to gastric juice with a pH close to 2 (table 2).

| SUBSTANCE | pH | SUBSTANCE | pH |
|---|---|---|---|
| Gastric juice | 2 | Rum | 3.97 |
| Cola drink | 2.5 - 3 | Energy drinks | 4 |
| Beer | 3.5 - 4 | Lemon-orange soda | 2.8 - 3 |
| Wine | 4 | Coffee | 5 |

Table 2. The pH of the most consumed drinks.

At the environmental level, we know that airborne acids in various chemical industries can be the source of dental erosion. Exposure to extrinsic acids can also be associated with activities such as swimming due to the high concentration of chlorine in the water, with painters, with laboratory workers who use pipettes with acids and professional wine tasters are also at greater risk of erosion.

The drugs directly involved in erosion are:

- Chewable aspirins.
- Medicines for the treatment of achlorhydria.
- Tablets with iron supplements or vitamin C (chewable and effervescent).
- Artificial saliva and salivary flow stimulants as they contain maleic and citric acids.
- Inhalers for asthma, as they cause a greater decrease in salivary flow.

In most of them, it will depend on the frequency of use and the duration of the treatment.

Among the intrinsic factors we have the derivatives of gastrointestinal problems, we must remember that gastric juice has a pH of 2 so that all processes that cause reflux or regurgitation will be likely to cause tooth erosion. Such as hiatal hernia, pregnancy or alcoholism. Being able to be enhanced or favoured by spicy foods, onions or tobacco.

We must not forget that the mouth has control systems and neutralization of acidic pH so at the oral level there are a number of biological factors that modulate erosion such as:

- Saliva. The more saliva the more acid neutralization.
- Dental structure. Teeth with higher mineralization will be less susceptible to erosion.
- Dental anatomy and occlusion. A correct occlusion and dental surfaces with non-retentive grooves and pits will reduce the effect of erosion on the teeth.

The anatomy and physiological movements of the soft tissues of the oral cavity are aimed at preventing the acid from being suspended on the tooth long enough to cause injury to its structure, dragging and causing the washing effect.

5.4.2 Clinical manifestations

The location will be variable according to the aetiology, finding injuries in the facial surface at the incisal and cervical level, in the occlusal surfaces of molars and premolars, in the lingual surfaces and in the proximal surfaces. The injuries usually have discoid areas of wear, where we can see an over-raised enamel and previous restorations if the tooth had them.

Depending on the evolution of the process, we will find that the active areas are clean and without colour changes and the inactive ones may have discolourations caused by the porosity of the dentine. In most cases, they are usually bilateral with no predominance of vertical or horizontal extension. They can be associated with other consumptive processes and coexist. According to the degree of affectation, we have two classifications, one of them refers to the affected tissue and the other to the state in which the process is:

Classification of Eccles and Jenkins:

- Class 1: enamel
- Class 2: dentine (less than one third)
- Class 3: generalized dentine involvement

Manneberg classification:

- Latent injury: with gloss and prominent edges
- Active injury: simulates an engraved enamel

The evolution of the damage could have a slow or fast progression. When it is slow, it gives time to the organism to rearm in front of the acid attack being able to observe in these processes the dentine of dead tracts, the translucent one and the reactive one. In the fast progression, it can cause processes of dental hypersensitivity and sometimes cases pulpal exposure.

5.4.3 Prevention and treatment.

The elimination of the aetiological factors that produce it is the key to the prevention of new damages caused by erosion, the decrease of acidic foods, rinsing with plain water or water with baking soda after taking some inhaled medicines or after having vomiting episodes or regurgitation is a good measure to reduce heartburn.

Observing the symptoms that cause an acute process of erosion, it is likely that we will have to perform emergency clinical treatments that can range from the control of hypersensitivity, to the treatment of ducts for pulpal pathologies.

As in the rest of the consumptive processes, the repair of the injuries will be performed with composite resins in moderate buccal defects, being able to use porcelain or resin facets. When the injuries are severe, we will have to resort to crowns of total coating.

# 6

# TEETH CONSUMPTION PROCESSES WITH CELLULAR ORIGIN

6.1. Internal root reabsorption.

6.2. External root reabsorption.

## Introduction

Root reabsorption is a destructive process due to a mechanism of cellular phagocytosis that leads to the loss of dental hard tissue. It affects cement and dentine but never affects enamel. Tissue phagocytosis is performed by primary cells of the connective bone tissue, cement tissue or pulpal tissue. They evolve differentiating into other cells with phagocytic function, in addition to macrophages, which may also be involved in this process.

Root reabsorption can be confused with caries, although caries is activated by bacteria and reabsorption by clastic cells. Although reabsorbed teeth can suffer bacterial contamination later. According to its location, we can differentiate two types of reabsorptions:

- Internal, begins inside the duct and goes on destroying dentine and then cement, so that if it reaches the outer surface it becomes communicating.

- External, begins in the periodontium of the tooth and can advance until reaching the root pulp.

## 6.1 Internal root reabsorption

### 6.1.1 Aetiology

It is caused by chronic inflammation of the pulp that stimulates the development of osteoclasts that phagocytose dentin. The causes of chronic pulpal inflammation can be:

- Caries and deep fillings.

- Dental trauma.

- Orthodontic movements.

- Pulp exhibitions.

- Idiopathic.

### 6.1.2 Histopathology

The innermost portion of the dentine of the root canal is the predentine, which is 25-30 μm thick and is the non-mineralized matrix of the dentine so it becomes a protective barrier of the dentine against osteoclasts that can only destroy hard tissue.

To begin the process of reabsorption there must be a chronic inflammatory process of the pulp that causes an increase in vascularity and the development of granulation tissue that transforms connective cells into osteoclasts. With the decrease in pH

caused by chronic inflammation, the solubility of hydroxyapatite increases and the mineralization of predentine occurs, thus becoming a hard tissue susceptible to osteoclastic action, leaving the dentine unprotected.

When the pulp is necrotic or removed, this process is stopped as the odontoclasts no longer have a power supply.

Dentinal resorption may be continuous or intermittent. If it is continuous it will destroy cement and there will be perforation towards the periodontal space. When it is intermittent, there are periods of destruction and periods of repair in which a poorly structured irregular dentine is formed.

### 6.1.3 Clinical manifestations

Internal resorption is uncommon and has a greater incidence in the upper incisors than in the rest of the teeth. Pulp vitality tests give normal results except in very advanced cases in which the pulp is replaced by granulation tissue and undergoes necrosis.

Internal resorptions are often discovered as accidental findings on x-rays taken for other purposes as they are usually asymptomatic in most cases. In the initial phase, it usually presents as a rounded radiolucent lesion on the duct and the pain appears when there is perforation towards the periodontal space. It can start in the duct or in the pulp chamber thus affecting the crown of the tooth. This differentiates them from external reabsorptions. When it affects the pulp chamber and the reabsorption becomes large enough, the granulation tissue is reflected through the enamel giving the tooth a characteristic appearance called a "pink tooth".

Teeth that have suffered trauma should be monitored for years to see if reabsorption occurs. For the first 6 months, radiographic checks and vitality tests are performed every 4-6 weeks, followed by annual check-ups.

### 6.1.4 Treatment

When internal reabsorption is diagnosed, we must act immediately as waiting only leads to a worsening of the situation. Duct treatment is mandatory as the pulp tissue is inflamed. If the reabsorption did not cause perforation, the prognosis is more favourable.

When treating ducts in the reabsorption zone we must remove all the pulp residues very well. This is complicated because the files do not reach the lacunar areas of the injury well. The use of ultrasound and its cavitation effect on hypochlorite helps to remove all granulation tissue from the damaged area, as the duct should not be widened to the size of the defect.

Some authors are in favour of leaving calcium hydroxide in the ducts and not doing endodontics in a single session as the alkaline pH of calcium hydroxide combats the decrease in pH produced in the duct. This favours the mineralization of predentine and its bactericidal effect destroys osteoclasts and macrophages that are causing reabsorption. The problem with the use of calcium hydroxide is the difficulty in

removing it completely from the reabsorption zone of the duct. When sealing the duct in endodontic treatment we will have more difficulties to make a good three-dimensional sealing if we use lateral condensation technique than if we use a thermoplastic gutta-percha technique. Because with hot gutta-percha it is easier and better to fill the defect caused by reabsorption.

### 6.2. External root reabsorption

It begins in the periodontium, in the lateral or external wall of the tooth. It affects the cement and in advanced stages, the dentine can reach the pulp thus producing a perforation that communicates the pulp and the periodontium. External reabsorption is unique to the tooth´s root as the enamel does not undergo reabsorption processes.

6.2.1 Aetiology

Harmful stimuli on the periodontal ligament can trigger external reabsorption.

In the case of pressure reabsorptions, the elements that exert pressure on the root can trigger external reabsorption. These can be:

- Inflammatory, as a result of trauma, orthodontics, periodontal disease or apical extension of a pulpal inflammation.

- Occlusal trauma
- Cysts or tumours
- Rash of impacted teeth

We can also find an endosteal or replacement origin as a result of:

- Avulsion
- Intrusion
- Orthodontics

Invasive cervical resorption, as a result of:

- Internal or external tooth whitening
- Trauma
- Orthodontics
- Systemic diseases

Unknown origin but there are different hypotheses to justify the appearance. The possible causes are:

- Trauma.
- Local factors.
- Systemic diseases with altered phosphocalcic metabolism.
- Endocrine disorders (hyper or hypoparathyroidism).
- Paget´s disease
- Kidney diseases
- Narcotics

### 6.2.2 Histopathology

As with internal reabsorption, there must be a chronic inflammatory process in the periodontium that causes the mineralization of the precement. Precement is the outermost part of the cement and fulfils a protective function just like predentine. If it is mineralized it allows the action of osteoclasts and reabsorption begins.

Replacement reabsorption is a bit special as it destroys cement and dentine like other reabsorptions but the difference between them is that bone is deposited directly within the reabsorbed structures. When the periodontal ligament is injured, it is replaced by bone, causing ankylosis of the tooth (immobility). Replacement resorption is a chronic and slow process that can last for years, although in young patients it evolves faster than in older patients and usually ends in complete tooth loss.

Invasive cervical reabsorption was so named by Frank in 1983, evolving very rapidly and located in the cervical 1/3 of the root which makes the prognosis worse by having direct contact with the oral environment.

### 6.2.3 Clinical manifestations

They are usually asymptomatic and are random radiological findings. Increased tooth mobility, which is often the only symptom until communication with the root pulp occurs and pain and pulpal necrosis are triggered.

A clinical history of previous traumas should make us suspect that due to the few symptoms they trigger, early diagnosis is very important. Because the more advanced the reabsorption the more difficult the treatment and the worse its prognosis.

### 6.2.4 Treatment

Due to the varied and sometimes unknown aetiology, different types of therapeutic treatments have been generated over time that are not always effective, making the prognosis of resorptions unpredictable.

#### 6.2.4.1 Treatment of pressure reabsorption

Eliminating the cause of the pressure causes the reabsorption to stop. The impacted teeth that generate that pressure must be removed. Depending on the degree of involvement in the root of the adjacent tooth we can wait and control if they are small injuries and see if there is spontaneous healing. But in cases of impacted third molars, the involvement of the distal root of the second molar is so large that endodontics, root amputation, or even molar extraction must be performed.

If the cause of the pressure is orthodontic treatment, this treatment should be discontinued immediately, as in other types of resorption caused by orthodontics.

#### 6.2.4.2 Treatment of inflammatory reabsorption

Elimination or decrease of the cause that causes that infection. If it is of pulp origin, the endodontic treatment will slow down the apical reabsorption and if the origin is

periodontal, specific periodontal treatment will be required. In external apical reabsorption, the root apex is destroyed so we have no tactile reference of apical constriction or clear radiological image. The reabsorption will stop months after the treatment, not immediately, therefore when doing the treatment of ducts, we will tend to create the new apical stop at about two millimetres from the apex. Sometimes apical surgery is needed to fix the case.

In the lateral inflammatory reabsorption caused by traumas, when they are diagnosed, there is usually pulpal involvement, so you must start by treating ducts and then have surgery to clean the granulation tissue and seal the area with different materials.

6.2.4.3 Replacement resorption treatment

There is no treatment for this type of resorption. Once started it will continue until the tooth is completely lost.

6.2.4.4 Treatment of invasive cervical resorption

The location of this type of resorption makes the surgical approach to the injury easier in order to make the affected area curettage and sealed. If there is no pulpal involvement, no endodontic treatment is done, but periodic vitality checks must be done because if it becomes affected, endodontics will have to be done.

This type of reabsorption is usually very aggressive and with a bad prognosis that often causes the teeth involved to end up being removed.

The problem that exists when sealing these reabsorptions is that on the one hand, they are usually in contact with the bone, but they are also in contact with the oral environment. Sealing materials that are biocompatible with the bone such as MTA (mineral trioxide aggregate) do not do so well in contact with the oral environment, which is why in many cases two types of materials must be used.

6.2.4.5 Treatment of idiopathic resorptions

Surgical approach of the injury is performed to make curettage and sealing of the affected area. If the pulp is not affected, no endodontics is performed as doing so does not decrease or eliminate the action of osteoclasts in reabsorption.

### 6.3 Differential diagnosis between external and internal root resorption

When in an X-ray we discover an image compatible with a reabsorption, we must know if it is an external or internal reabsorption. It is not easy to distinguish them at first, but an early diagnosis will improve the prognosis and treatment will vary from one to another. X-rays are two-dimensional images so it is difficult to distinguish one from another and in more advanced stages it will be more difficult as both can become communicating. To make the differential diagnosis we will be based on three aspects:

A) Morphology of the lesions

The internal reabsorption is rounded with symmetrical radiolucency while the external has an irregular and asymmetrical shape. Internal resorption is seen as an expansion of the root canal or pulp chamber and the boundary between the lesion and the pulpal structures is indistinguishable.

On the external one, a clear delimitation is seen between the structures and the lesion, and on radiographs, the lesion seems to be superimposed on the root canal.

B) Variation on the angle of the ray tube

We will do x-rays with different angles of the ray beam, from mesial or distal. In internal resorptions, although we vary the angle of focus, the lesion will be integrated with the duct but in the external ones, the lesion will be separated from it.

C) Tomographs

In cases where it is impossible for us to diagnose what type of resorption it is, we can do more complex radiological studies such as conical beam computed tomography (CBCT). This type of study gives us clearer and more accurate images to be able to diagnose the type of reabsorption that is.

# Part III

## DENTAL PATHOLOGY OF INFECTIOUS ORIGIN

# 7

# INTRODUCTION TO CARIOLOGY

7.1 History.

7.2 Current concept and definition.

7.3 Epidemiology.

7.4 Etiological factors.

7.5 Pathogenesis.

7.6 Prevention and treatment.

## Introduction

Dental caries is a complex and multifactorial disease that is a fundamental axis of dental pathology. Knowing the starting points of its historical evolution, the factors that trigger it and the different approaches to it are the backbone of this issue. All of this will allow us to become aware of caries as a disease and it will help us to give diagnostic and therapeutic approaches from prevention to the treatment of the signs and manifestations of this disease.

## 7.1 History

Dental caries is already described in ancient civilizations in Asia, Africa and pre-Columbian America where individuals with cariogenic processes are observed.

Already in the 5th century BC theories about its aetiology were postulated. They were differentiated into two aspects, the so-called endogenous theories, which attributed the cause of caries to a process of internal imbalance of the body or the tooth, and exogenous theories, who attributed the disease to an external cause of the body.

Among the most important endogenous theories we have the humoral theory of Hippocrates, where it was postulated that caries was caused by the accumulation of harmful humour within the tooth (456 BC). Galen in 130 BC states that cephalic disorders are caused by a "corruption of humours that can pass into the mouth and cause cavities." Jordain, in the 18th century, postulates the vital theory, where he establishes that it is an inflammation of the odontoblast that causes caries due to some systemic process or metabolic alteration that he could not determine.

With regard to exogenous theories, one of the most enduring over time was the "legend of worm or tooth-worm theory" whose first reference dates from 5000 BC in an Assyrian board, which states that it is a worm that causes the carious lesion. In the Egyptian Papyrus of Ebers, dating from 1500 BC and that combines the medical knowledge of many centuries, one of its sections is dedicated to the diseases of the teeth and their treatment by means of enchantments, fumigations and the local application of vegetal and mineral substances. In Spanish, this worm was named as "neguijón", referring to the Latin term (nigellus, diminutive of niger: black) since in most cases this blackish colour was observed in the lesions caused by caries. On many occasions, the neurovascular bundle inside the canals was confused with the worms that caused the disease. This theory took time to disappear, until well into the 17th century, with the publication of *Le chirurgien dentiste ou traité des dents* (1728) by the French surgeon Pierre Fauchard, who marked the birth of modern dentistry.

In the 19th century Robertson and Regnart performed experiments with inorganic acids and observed that they had the ability to destroy enamel. Later in 1983 Erdl observed under the microscope the deposits on the tooth and he found some filamentous parasites. Finicius, a few years later, and after observing these parasites in a sample of a caries, gave them the name of denticolae.

In 1890 Miller proposed the acidogenic or chemoparasitic theory, combining the studies of Pasteur, who had described how microorganisms decomposed sugars into lactic acid, and the studies of Magiot, who showed that the fermentation of sugars caused the dissolution of enamel. The in vitro study concluded that:

- The microorganisms in the mouth ferment the carbohydrates on the dental surfaces and produce acids (especially lactic acid).
- That these acids first decalcify the tissues, which in the case of enamel means its destruction.
- Later on, the dentine is also decalcified, dissolved or digested by proteolytic bacteria that penetrate inside.

This study led to many others such as Williams (1897) and Black (1898) which described dental plaque and how it makes possible the mechanisms of adhesion and enzymatic activity.

In 1922, Mcintoch gave more importance to the microbial factor in the disease by achieving the production of caries in vitro by inoculating Lactobacillus acidophilus in a glucose broth on a healthy extracted tooth.

### 7.2 Current concept and definition

It is currently accepted that caries is a disease of infectious and multifactorial origin that conditions the demineralization of the tooth starting with the most mineralized tissues and continuing with the destruction of the organic matrix. Therefore, the dental caries is a complex infectious disease of a chronic and multifactorial nature in which environmental, social, behavioural and biological factors interacting cause the destruction of the hard tissues of the tooth and a possible pulpal involvement.

For tooth decay to occur, we necessarily need the presence of bacteria, but it is not enough on its own, as we also need a number of additional factors for it to occur, such as carbohydrate intake, host susceptibility, alterations in brushing, etc.

It is important to establish the approach to dental caries. Since historically caries was considered as a cavity that occurred in the tooth, all efforts were made to repair the damage that occurred in it. Thanks to the advancement of science and the studies, this approach has now changed, and caries is established as a disease or process (such as diabetes or coronary processes). This allows us to act at various levels, and to stop the effects of the caries before the lesion occurs.

Etymologically, the word caries comes from Latin and means rot. Aulo Cornelio Celso uses it in his medical treatise alluding to the destruction of any bone tissue in the human body. The term caries appeared in our language in 1450 and from the 17$^{th}$ century began to be used specifically for the destruction of dental tissues.

The World Health Organization (WHO) has defined tooth decay as a localized process of multifactorial origin, that begins after the tooth eruption, causing the hard tissue of the tooth to soften and evolve into the formation of a cavity.

### 7.3 Epidemiology

Historically, it has always been related to the consumption of refined sugars. The arrival of sugar cane in the European continent has meant a significant increase in the incidence of caries. Later on, hygiene techniques and prevention measures have been changing the trend of incidence of caries in the last years of the 20$^{th}$ century. In developing countries, the arrival of refined sugars and dietary changes combined with the few hygienic and preventive measures have led to increases in the incidence of caries.

Dental caries is the most common chronic disease in the world from childhood and affecting all ages. According to the WHO, the overall prevalence (averaging all ages) of untreated caries in permanent teeth is 40% and is the most common condition of the 291 processes analysed in the Global Burden of Disease Study.

The consequences of untreated caries are chewing difficulties that can have a negative impact on overall health and growth, sleep disturbance and problems with infections, both chronic and acute, being the main cause of school and work absenteeism.

Dental caries, as the most common chronic disease in humans, is present in all races and in all age groups. Attending the periods of incidence of caries, we find that:

- Between the ages of 2 and 8, caries occurs in the temporary dentition and the first permanent molars. In this period the temporary dentition is already complete and fully functional. The phases of crushed food have already passed and dietary habits closer to those of an adult are established. The appearance of the first permanent molars, their marked anatomy and the condition of an enamel more susceptible to demineralization make this a period with a higher incidence of dental caries.
- Between the ages of 11 and 18 there is another rise in caries, in this case with all the final dentition. Caries occurs on the occlusal surfaces of permanent molars and the presence of caries begins to appear in interproximal areas of premolars and permanent molars.
- From the age of 55 with the retraction of gums and the alveolar bone loss associated with the processes of periodontitis, the accumulation of food in the interdental spaces is facilitated and the alterations of the salivary flow due to hormonal changes or pharmacological treatments complicate the ability to drag and rinse of the saliva, causing the appearance of root caries.

It is currently said that 20% of the population accumulates 60% of the disease. This means that the population most susceptible to caries has a very high incidence of caries and that we must focus efforts to establish good risk indicators, detect caries in the early stages and improve treatments in the early stages.

## 7.4 Aetiological factors

In 1960, Keyes proposed that the aetiology of caries is due to three agents that must interact with each other: host, microorganisms, and diet. It's called the Keyes Triad.

In 1978, Newbrun added to the etiological factors the influence of time, since for caries to start, the cariogenic plaque must be in contact with the carbohydrates of the diet on the surface of the tooth for a certain period of time to provoke the biochemical imbalance that leads to dental cavitation. These are the ones that today we consider primary, basic or main aetiological factors, indispensable for the development of the disease, because it is necessary an interaction over a period of time of a susceptible host, cariogenic oral flora and an appropriate substrate.

As knowledge about caries advances, it is observed that not only does it depend on these primary factors, but there are other modulating etiological factors involved in the appearance and evolution of caries lesions, although they do not always appear with the same intensity in all cases of caries. Secondary factors have also been added that are as well involved in the production of this disease.

### 7.4.1 Microbiological factors

The localized decrease in pH at the biofilm-tooth interface is caused by the metabolism of dental plaque microorganisms, but only plaque with high concentrations of certain bacteria, such as *Streptococcus mutans*, *Streptococcus sobrinus*, lactobacilli and some others, can produce a pH low enough to demineralize the tooth.

*S. mutans* is the first microorganism to colonize the tooth surface after the eruption. It is highly cariogenic, and it is present in 90% of patients with multiple caries. The characteristics that make *Streptococcus mutans* especially virulent and that directly relate it to dental caries are the following:

- Acidogenic.
- Aciduric.
- Acidophile.
- Synthesis of glucans and fructans.
- Synthesis of intracellular polysaccharides.
- Dextranase production.
- Adhesion of *S. mutans* to the surface of the tooth.
- Synthesis of bacteriocins, which are protein toxins that inhibit the growth of similar bacteria or nearby strains.

However, Kleinberg, in 2002, observed that individuals with high counts of *S. mutans* did not always have caries lesions. In turn, Okada et al. and Linquist et al. observed that *S. sobrinus* is more acidogenic and more aciduric than *S. mutans*, being the coexistence of both species a determining factor in the development of caries lesions.

Lactobacillus has long been blamed for tooth decay. They are gram-positive, facultative anaerobes, acidogenic, acidophiles and aciduric bacillus. A pH close to 5

favours their growth and the beginning of their proteolytic activity. From sucrose they synthesize intra and extracellular polysaccharides, although they have little ability to adhere to smooth surfaces and must make use of other mechanisms to adhere to the dental surface. They are found mainly in cavitated caries and they have coaggregation with other bacteria species. They are able to produce caries in gnotobiotic rats, especially root caries, and also periodontitis.

Actinomyces is the microorganism most often isolated in the subgingival plaque and in root caries. *A. naeslundii* predominates in the plaque of children and *L. viscosus* in the adults one. The presence of fimbriae in microorganisms determines their virulence, as they contribute to the phenomena of adhesion, aggregation and coaggregation and the production of proteolytic enzymes such as neuraminidase of great importance as caries lesions progress to the inner dentine.

7.4.2 Diet related factors

- The more refined carbohydrates we eat in the diet, the greater the likelihood of tooth decay.
- Food acidity. Currently, the consumption of carbonated drinks, citric acid and vinegar, promote the appearance of caries.
- Consistency (retention capacity), the presence of "sticky" foods makes them stick to the tooth for longer so there is more contact time between the carbohydrate and the bacterial plaque enhancing the appearance of dental caries.
- Frequency and consumption habits. The higher the frequency of consumption of potentially cariogenic foods, the greater the pH drops below the critical level and therefore the greater the number of chances of caries.
- Temporary distribution of consumption. If you have for example 5 candies, it is preferable, from the oral health point of view, to ingest them all at once than space them in time, since this would take several curves of descent of pH against an only curve that would be produced with the ingestion of all at once.

7.4.3 Host related factors

- Previous experience of caries.
- Morphology of the tooth.
- Presence of "microcracks".
- Morphology of the dental arch.
- Enamel maturity.
- Post-eruption age of the tooth.
- Salivary factors.
- Oral environment rich in fluorides.
- Oral hygiene habits.

And the factors related to tooth morphology:

- Occlusal surfaces of molars.
- Vestibular surfaces of the first lower molars.

- Palatal surfaces of the first upper molars.
- Interproximal surfaces.

### 7.4.4 Saliva related factors

Oral clearance is the function of dragging or sweeping that occurs due to the presence of a fluid (saliva), added to the muscular action of the tongue, cheeks and lips and which determines a mechanical dragging action that makes possible continuous cleaning of bacteria, bacterial products and food debris that are free in the mouth and in accessible areas of the oral mucosa and teeth.

The clearance time of a sugar is, for example, the time required to reduce it to levels before it was ingested or to leave it at a concentration of less than 0.1%. It is more effective during chewing or oral stimulation, because at that time more saliva is secreted. Clearance times may be prolonged by decreased saliva flow, increased saliva viscosity, increased food viscosity, decreased muscle activity, and also the existence of retentive factors in dentition.

The presence of fluoride in saliva increases the rate of neutralization of acidity by 4 to 8 times. Therefore, the intake of fluoride, either systemically or topically, by rinsing and from the toothpaste, will obviously improve the prevention of progression of caries.

In the same way, oral hygiene has a direct relationship between the contact time of microorganisms and carbohydrates with dental surfaces, which is why the frequency of brushing will significantly reduce the appearance of caries on equal terms.

### 7.4.5 Socio-demographic factors

The socioeconomic level does play a very important role. Currently, access to dental care is quite complex, thanks to child dental care programs, attempts are being made to reverse this situation, but even so, in adulthood access to these programs is conditioned by economic aspects and there are deficits in oral health education. Similarly, immigration and family breakdown are other factors that increase the likelihood of caries.

Geographical location has seen a declining trend in the incidence of caries in developed countries in the last years of the 20$^{th}$ and early 21$^{st}$ century. A slight upward trend in developing countries due to access to sugary products and changes in eating habits has been observed.

### 7.4.6 Fisher-Owens diagram (2007)

We must consider the large number of factors that influence the appearance, development and severity of dental caries and that these act over time at both community and family or individual level. Fisher-Owens expanded the original Keyes diagram in 2007 to include influencing factors at different levels.

| COMMUNITY- LEVEL INFLUENCES | FAMILY- LEVEL INFLUENCES | INDIVIDUAL - LEVEL INFLUENCES | ORAL HEALTH |
|---|---|---|---|
| - Culture<br>- Social capital<br>- Oral health<br>- Physical environment<br>- Environmental safety<br>- Health system<br>- Oral care system | - Healthy habits<br>- Culture<br>- Family functioning<br>- Parent's health<br>- Physical security<br>- Social support<br>- Socioeconomic level | - Use of dental services<br>- Dental insurance<br>- Healthy habits<br>- Physical and demographic characteristics<br>- Biological legacy<br>- Genetic legacy<br>- Development | - Diet<br>- Host/tooth<br>- Biofilm<br>- Saliva |
| TIME | | | |

Table 3. Fisher-Owens diagram.

### 7.5 Pathogenesis

As we have mentioned in previous sections, it is important to know the different approaches that have been given to caries disease, both as a cavity injury or as a process or disease. The physiological dynamics of the cavity involves periods of demineralization and remineralization of tooth enamel. When the balance of this process is tilted towards demineralization the first signs of the disease begin to appear, producing subsurface demineralization of the enamel, progressing later to the lesion of white spot and finally to the cavitated lesion. This evolution of caries will be studied in more detail in later chapters.

### 7.6 Prevention and treatment

The combination of the primary etiological factors with the modulators of cariogenic processes lead to clinical manifestations that evolve as the severity of the lesion progresses. The observation of these manifestations helps us to find an appropriate diagnosis aimed at prevention, recurrence and treatment of injuries that have already occurred.

The treatment of dental caries will vary according to the approach given to caries. Until recently, dental caries was only the cavity lesion, as well as pulpal alterations that the cariogenic process produced. Nowadays and after understanding caries as an evolutionary process, it is possible to act on all those aetiological factors that are likely to cause the disease.

Regarding microbiological factors, we can control the bacterial plaque by trying to eliminate focus of infection, reduce the bacterial plaque by correct brushing techniques, toothpastes, mouthwashes with antimicrobial agents, etc.

We can also act on the diet, regulating eating habits both in frequency and in its composition, reducing the consumption of refined sugars, carbonated drinks, etc. And trying to establish balanced diets, reducing retentive foods, improving dietary balances, etc.

At the host level we can improve the remineralization processes by providing fluoride either systemically, by water fluoridation or taking fluoride supplements for the neoformation of fluorapatite crystals. We can also promote the buffering effect of saliva to neutralize acids and thus reduce the possibility of demineralization of dental pieces. And likewise, the increase in salivary flow will provide a better washing effect, as well as a dilution of plaque acids and therefore reduce the likelihood of suffering from manifestations of the disease.

With regard to the treatment of the signs and clinical manifestations of caries, we will perform the least invasive that will be aimed at remineralizing superficial lesions and without cavitation, through the use of sealants or fluoridations. With cavitated lesions we will resort to the removal of decayed tissue, remineralization of uninfected softened dentin and the restoration of lesions using composite resins, ceramic or resin coatings to return functionality and aesthetics to the parts.

Finally, when there are infectious processes that affect the dental pulp we will resort to the treatment of the dental canals (endodontics), with the subsequent functional and aesthetic rehabilitation of the tooth.

# 8

# BIOFILMS RELATED TO THE AETIOLOGY OF CARIES

8.1 Biofilm.

8.2 Dental plaque.

8.3 Dental plaque microbiology.

8.4 Dental plaque formation.

8.5 Ecological determinants in dental plaque formation.

## Introduction

One of the aetiological factors of caries represented in the Keyes triad are the cariogenic microorganisms. These are present in dental plaque, which is a type of biofilm with more than 500 different species of bacteria coexisting, and only some of them are associated with the aetiology of dental caries.

## 8.1 Biofilm

A biofilm is a bacterial community that attaches to a surface, whether alive, inert, soft or hard, through a liquid-solid interface. This means that it can be formed in multiple places as long as they have a surface in contact with a liquid, such as pipes, ponds, etc. It is organized in a three-dimensional structure formed by an extracellular polymeric substance or glycocalyx that surrounds large sessile bacterial colonies. In living organisms, it can develop in many tissues, such as the tooth surface, where it causes various pathologies other than caries.

As early as the 17$^{th}$ century, Van Leeuwenhoek was the first to describe the existence of microorganisms attached to the tooth surface, but it was not until 2002 that Donlan gave a widely accepted description of today´s biofilm: "sessile microbial community, characterized by bacteria that are adhered irreversibly to a substrate or interface, or with each other, enclosed in an extracellular polymeric substance that they have produced, and exhibit an altered phenotype in relation to the rate of growth and gene transcription ".

The extracellular polymeric substance or glycocalyx of the biofilm far exceeds the bacterial mass (15-20% of the total volume) and is formed by extracellular polymers (exopolysaccharides) produced by the same bacteria, which in turn are protected by this matrix. Bacteria, to bind to the surface and form the biofilm, need chemical signals that allow them to communicate with each other and coordinate. There is an interrelationship between bacteria by small molecules that act as messengers called quorum sensing. The signaling molecules that make bacteria “communicate” with quorum sensing are called autoinducers.

In the biofilm coexist bacteria species that would be incompatible in other conditions, but as bacterial metabolism produces microenvironments with variations in pH, oxygen concentration, nutrients and metabolic products, survival is possible, although in the deeper areas of the biofilm bacteria must be adapted to a lower availability of oxygen.

This organization of the bacteria in a biofilm makes them more resistant to antibiotics than the bacteria themselves in planktonic form. This resistance may be due to different factors:

A) Physical barrier, the extracellular matrix or glycocalyx that surrounds the bacterial colonies reduces and slows the penetration of the antibiotic.

B) Metabolic causes:

- Limitation of oxygen and nutrients in the deep areas, slows down the metabolism of bacteria and slows down or prevents their division, making them more resistant.

- Decrease in pH in the biofilm, also interferes with the action of antimicrobials.

C) Genetic changes, biofilms favour genetic changes that increase resistance to many antibiotics.

D) Spore formation, this is a hypothesis to explain the resistance in newly formed biofilm with little thickness in which the above hypotheses do not work.

8.1.1 Biofilm formation

The biology of the biofilm is a dynamic process. There is a succession of several phases:

- Phase 1: Formation of an acellular layer:

  This acellular layer is required for subsequent microbial adhesion and it is composed of polymers that cover all liquid-coated surfaces. This film alters surface energy and surface charges and it provides specific receptors for bacterial adhesion. This pellicle is essential for the formation of the biofilm as it promotes the adhesion of bacteria and it serves as a nutrient.

  In dental biofilm, the acellular layer is called the acquired pellicle which is always formed in the enamel in contact with saliva. It is made of albumin, glycoproteins, phosphoproteins and lipids. Once the acquired pellicle is formed, colonization by the bacteria begins.

- Phase 2: Primary bacterial adhesion: initial attachment:

  Planktonic bacteria approach the surface by a flow current, by chemotaxis or by mobility of the bacterium itself. When it is less than 1nm from the surface, the electrostatic forces between the bacteria and the surface determine the attraction or repulsion between them. It is a reversible adhesion phase.

- Phase 3: Secondary bacterial adhesion: irreversible attachment:

  The binding is consolidated by the production of exopolysaccharides that promote the binding of bacteria to the surface and because of the bacteria piles, fimbriae and fibrils. A more specific and firmer bond is established between components of the bacterial wall called adhesins and their complementary receptors in the acquired pellicle. Bacteria can bind to each other of the same species (coaggregation) and to bacteria of different species that are already bound to the surface forming substrate microcolonies (coadhesion).

- Phase 4: Growth or maturation:

  Bacteria begin to divide, the extracellular polysaccharides they produce interact with the organic and inorganic molecules already present in the oral environment and the glycocalyx of the biofilm is formed. In this way, biofilm grows three-dimensionally.

- Phase 5: Separation or detachment phase: dispersion

Once the maturity of the biofilm is reached, some bacteria are released from the matrix and colonize new surfaces, beginning a new life cycle of the biofilm. Bacteria can be

released in isolation (by erosion) or in the form of bacterial conglomerates (by migration). This release can be favoured by external forces to the biofilm or by active mechanisms of the biofilm itself.

The formation of the biofilm therefore involves an orderly sequence of communities that occupy the surface on which it is formed. The pioneering microorganisms are those that initiate the colonization of a habitat. This established community is the one that modifies the local environment and in turn determines the new community that can be incorporated. This process of mutual change in the community and its environment is called "ecological succession." The final phase of succession is a stable biological community called the "climax community."

The development of a biological community where there was previously none is called a "primary succession." "Secondary succession" is the process of restoring the climax community after an interruption of the community structure. Interruptions of this type occur continuously on the surface of the tooth each time we brush our teeth.

## 8.2 Dental plaque

Dental plaque is defined as the microbial community that is on the dental surface in the form of biofilm. It has all the characteristics that we have mentioned of biofilms, it is attached to a surface (tooth) with a liquid-solid interface (saliva-tooth), it has glycocalyx and colonies of sessile bacteria. It occurs in the mouths of all individuals and it is involved in the aetiology of the most prevalent oral diseases: dental caries and periodontal disease.

Dental plaque is a necessary factor in the formation of caries. It is more easily formed in pits and fissures of the occlusal faces, in proximal gingival faces up to the contact point and in the gingival margin. The process of plaque formation is constant and inevitable. If it is removed from the surface of the enamel it is re-formed. The only thing we can do is to control it so that the clinical caries lesion does not form.

For caries to begin to form, the presence of plaque or biofilm is not enough. It requires that the metabolic activity of the biofilm generates fluctuations in the pH that cause alterations in the mineral composition on the surface of the enamel: the onset of the lesion. In the mouth there are approximately 200 to 300 species of bacteria, yeasts and protozoa and the metabolic activity of this complex bacterial community is what causes pathology in hard and soft tissues.

## 8.3 Dental plaque microbiology

The microbiology of caries is complex and it would be too simplistic today to say that caries in its early stages is caused by *streptococcus mutans* and advanced caries in dentine by lactobacillus.

### 8.3.1 Streptococci

Different species of streptococci have been isolated in the oral cavity: *S. mutans, S. sobrinus, S. salivarius, S. parasanguinis, S. sanguinis* (sanguis), *S. cistatus, S. oralis,*

*S. mitis, S. constellatus, S. gordonii, S. anginosus,* and *S. oligofermentans*. Of all of them, *S. mutans* has been the most studied, but *S. sobrinus* and *S. salivarius* are also known to be actively involved in the development of caries.

*S. mutans* was first described in 1924 by Clarke but it was not until the 1960s that it was given importance in bacterial plaque thanks to the studies of Fizt-gerald and Keyes. It can produce caries in cracks and smooth surfaces of all experimental animals as it is very cariogenic, but there are 10-20% of patients with many caries in which it is not detected. It is the first to colonize the surface of the tooth after the eruption and its name was given due to its tendency to change shape. It can be found as a coccus or as a bacillus. It grows in chains or in pairs. It is unable to move. It does not form spores. It is gram-negative, facultative anaerobic and produces lactic acid. It is part of the oral and upper respiratory tract flora and it is an opportunistic pathogen also in diseases such as caries and infective endocarditis. When colonizing teeth, it tends to concentrate on fissures and interproximal smooth surfaces and it also colonizes prosthetic teeth. It has a low speed of transmission, so it does not spread quickly over all surfaces and areas of high concentration can coexist with other non-colonized next to them. It can be inoculated iatrogenically with a dental explorer or dental floss in non-colonized areas.

The virulence factors that make it the protagonist in the onset of enamel caries are the following:

- Acidogenic, it can ferment sugars in the diet producing lactic acid that lowers the pH and demineralizes tooth enamel.

- Aciduric, it is able to produce acid in low pH conditions.

- Acidophilic, it can resist acidity by pumping protons out of the cell. They are bacteria with the "lactate shuttle" mechanism.

- It synthesizes glucans and fructans from sucrose. These extracellular polymers help the bacteria adhere to the tooth and they are used as a nutrient reserve.

- The synthesis of intracellular polysaccharides, such as glycogen, serves as a food reserve and its metabolism maintains the production of acids for long periods even in the absence of sugar.

- It produces dextranase, it converts glucan into glucose and an energy source.

- The adhesion to the surface of the tooth is mediated by the interaction between a protein of this bacterium and some of the saliva that form the acquired pellicle. It has adhesins that are Ag I/II and Spa P that bind to salivary glycoproteins.

- Synthesis of bacteriocins. These are protein toxins that inhibit the growth of similar bacteria or strains.

The *S. mutans* count can be used as a diagnostic aid to identify patients at risk for caries If it is greater than 100,000 CFU (Colony Forming Units)/ml in saliva it is considered an indicator of caries risk. This test will be used to assess the need for preventive dental treatment.

Despite all this evidence, studies have shown that in the absence of *S. mutans* caries lesions can form and also that in individuals with high counts of *S. mutans* caries lesions do not necessarily develop. It was also observed that *S. sobrinus* is more acidogenic and more aciduric than *S. mutans*. The coexistence of both is a determining factor in the development of caries.

*S. mutans* has been isolated in some cases from subacute bacterial endocarditis in patients with valvular heart diseases undergoing dental treatment. This is why penicillin antibiotic prophylaxis is performed in patients who require dental treatment and are at risk for endocarditis.

8.3.2 Lactobacillus

Ever since Miller enunciated the "chemoparasitic theory", in the late 19$^{th}$ century, the existence of this germ in the oral cavity is known. For a long time was imputed as the most responsible for caries.

They are gram-positive, facultative anaerobic, acidogenic, acidophilic and aciduric bacilli. A pH close to 5 favours their growth and the beginning of their proteolytic activity. They adhere very little to smooth surfaces on their own, so they must use other mechanisms to adhere to dental surfaces. They are found mainly in cavitated caries, especially in infected deep dentine. They coaggregate with other bacterial species.

The most common species of bacilli in the oral cavity are: *L. casei, L. acidophilus, L. plantarum, L. salivarius, L.fermentum, L. brevis, L. buchneri* and *L. cellobiosus.*

In humans they can be isolated in the saliva, back of the tongue, tooth surface and mucosa. *L. acidofilus* predominates in saliva and *L. casei* in dental plaque and caries lesions. The number of lactobacilli in saliva is correlated with existing caries so it can be used as a measure of the prevalence of active caries. The number of lactobacilli is affected by the diet, being higher in patients who consume a lot of carbohydrates. The restriction of sugars and the treatment of caries considerably decrease the number of these germs in the mouth.

8.3.3 Actinomyces

It is a gram-positive, facultative anaerobic filamentous bacillus, capable of synthesizing extracellular polysaccharides and acidogenic. Actinomyces is the most frequently isolated microorganism in subgingival plaque and root caries and it is capable of producing root caries and periodontitis.

The 2 most important in relation to oral pathology are: *A. viscosus* that predominates in the plaque of adults, and *A. naeslundii* that predominates in the plaque of children.

Its virulence is determined by the presence of fimbriae that contributes to the phenomena of adhesion, aggregation and coaggregation and the production of proteolytic enzymes, such as neuraminidase, of great importance in caries lesions that have advanced to the deep dentine.

8.3.4 Bifidobacterium

They are gram-positive, anaerobic bacilli, which are often grouped in branches and are generally present in the healthy gastrointestinal tract of humans and animals.

In 1974 they described *B. dentium* species isolated in human dental caries and in 2002 a high concentration of Bifidobacterium was found in shallow cavity lesions and deep decayed dentine.

8.3.5 Prevotella

They are strict anaerobic bacilli, gram-negative, non-sporulated, immobile, with a marked proteolytic and haemolysing activity.

The most common in the oral cavity are: *P. melaninogenica, P. oralis, P. intermedia, P. buccae, P. nigrescens, P. denticola* and *P. loeschii.*

They are associated with periodontal disease and infections of endodontic origin, but in the case of caries it is not clear the role that this bacterium plays.

8.3.6 Veillonella

They are gram-negative diplococci, strict anaerobes, which are part of the resistant flora in the oral cavity and upper respiratory tract.

Veillonella has insufficient direct adhesion to oral tissues, but it is found in large quantities in subgingival dental plaque, supragingival dental plaque and on oral surfaces due to bacteria coaggregation mechanisms.

It plays an important role in its ability to neutralize acids produced by cariogenic microorganisms as it metabolizes lactic acid produced by other bacteria and it forms propionic acid and acetic acid which are weaker.

8.3.7 Considerations in the microbiology of the dental plaque

- In healthy mouths without caries, *S. sanguinis* and *S. mitis* predominate.

- *S. mutans* is related to the onset and progression of caries, although its presence is not essential for the progression of the disease, nor in advanced caries.

- Lactobacillus, Bifidobacterium and Prevotella are secondary opportunistic microorganisms that colonize the biofilm in advanced lesions contributing to the progression of the lesion.

- Actinomyces is present in dental plaque in children and it is associated with the onset of caries lesions in primary dentition, and in adults with the onset of root caries lesions.

## 8.4 Dental plaque formation

The pioneering colonization of the oral cavity begins a few hours after birth. The first species are transmitted to the child by parents, siblings and caregivers. These are usually *S. salivarius, S. mitis, Veillonella* and *Neisseiria*. When the teeth erupt the flora

changes and begins to have characteristics of the adult, *S. sanguinis* appears and with the following teeth does *S. mutans*. The earlier *S. mutans* colonization happens in the child, the more likely it is to develop caries in the future. In the oral environment, as in other environments where biofilms are formed, microorganisms with the ability to adhere to a surface survive better than those that do not adhere, which are eliminated by salivary flow and swallowing.

Streptococci, due to their ability to produce the extracellular substance that allows them to bind together, in addition to coadhesion and coaggregation, can adhere to the oral mucosa and tooth surface. This allows them to successfully colonize the tooth surface being the pioneer communities. The metabolism of these alters the conditions of the environment favouring the growth of other bacteria. Actinomyces and other gram-positive bacilli adhere to the Streptococcus, which have a lower capacity to adhere to the acquired pellicle but do have the ability to coaggregate and co-adhere. As the plaque develops and grows, the medium becomes unfavourable to aerobic bacteria and facultative anaerobes and anaerobes begin to predominate. The metabolites of the pioneer communities also serve as nutrients for other species. Microbial succession occurs, which increases the diversity of bacterial species in the plaque maturation process. It continues to grow until reaching the climax community that will be different in the different ecological niches that occur on the tooth surface. In the fissures and grooves predominate streptococci while in the root surface and gingival sulcus predominate filamentous and spiral bacteria.

A dental plaque in which *S. sanguis* predominates is desirable for its ability to inhibit the establishment of more pathogenic microorganisms. When all niches are saturated, only highly competitive microorganisms can displace endogenous bacteria from that niche. This happens due to a homeostatic mechanism called "colonization resistance" (which can be quantified by measuring the threshold dose or number of microorganisms needed to establish the new resident population). The threshold dose (total number of bacteria inoculated in the mouth) is an essential factor for a new microorganism to be part of the plaque. On this concept are based some bacteriological tests of saliva to assess the risk of caries. Therefore *S. mutans* spread to the dental surfaces must exist in sufficient numbers in the saliva to overcome the resistance to colonization of the non-pathogenic microflora previously existing in the mouth. An active caries can be a reservoir of *S. mutans* and Lactobacilli, so it is essential to eliminate the active caries as soon as possible to prevent the colonization of other dental surfaces.

Professional cleaning involves the removal of almost all organic material and bacteria from the dental surface. In the formation of dental plaque after each brushing the following steps are taken:

A) After about 2 hours of cleaning, the acquired pellicle accumulates on the surface of the tooth, which is essential for microbial adhesion. The functions of this acquired pellicle are:

- Protecting the enamel from acid attack and reducing friction between the teeth.

- Providing elements for the remineralization of the enamel. It is a reservoir of ions such as fluorine.

- Allowing the initial adhesion of bacteria on the surface of the enamel.

- Serving as substrate for bacteria adhering to the enamel surface.

B) Two hours later (about 4 hours after brushing), the bacteria in the mouth adhere to the acquired pellicle. This colonization will depend on the microorganisms floating in the saliva or on bacteria or conglomerates released from the biofilm in other niches. *S. sanguis, A. viscosus, A.naeslundii* and *Peptostreptococcus* are the first to bind to the acquired pellicle due to their greater ability to adhere to hydroxyapatite and because they have specific receptors to bind to salivary proteins precipitated in such pellicle. *S. mutans* has an enzyme that, in the presence of sucrose, forms the extracellular matrix of the biofilm, so the presence of sucrose in the oral environment enhances the binding of *S. mutans* to the acquired pellicle.

C) In the first phases, aerobic bacteria with fewer harmful metabolites predominate, but when the plaque matures, the predominance of anaerobic bacteria begins and the metabolism produces acidic metabolites, mainly lactic acid. It is at this time of development of the plaque when it is able to generate caries.

## 8.5 Ecological determinants in the formation of dental plaque

These are environmental factors that determine the characteristics of dental plaque.

### 8.5.1 Nutrients available for dental plaque

There are nutrients that promote plaque growth. Nutritional needs are different in different microorganisms, so pioneers in colonization have simpler nutritional requirements. *S. sanguis* and *S. mutans* can meet their needs from salivary protein metabolism. In supragingival areas where there is a lot of oxygen, carbohydrates and are bathed in saliva, facultative streptococci abound. A diet rich in sucrose makes *S. mutans* the main component of the supragingival bacterial plaque.

In the subgingival area oxygen saturation is lower, there are fewer carbohydrates and fewer salivary nutrients. Bleeding and exudate from the sulcus provide protein and nutrients. Bacteroides melaninogenicus are obligate anaerobe and require albumin and hemins so it is closely related to periodontal disease.

### 8.5.2 Oral hygiene

It involves an interruption of the bacterial plaque. The dental surface will be re-colonized in a process of secondary succession. This process is fast because there are always residents of the climax community. Since *S. sanguis* and *S. mutans* compete to colonize the tooth, anything that promotes colonization by *S. sanguis* will reduce the risk of tooth decay, so a diet low in sucrose will promote the growth of *S. sanguis* versus *S. mutans.*

### 8.5.3 Shelter factor

The smooth vestibular and lingual surfaces of the teeth are in continuous friction with the cheek and tongue and subjected to the action of saliva. The points where caries usually begin, coincide with the refuge areas such as:

- Pits and fissures. Pit and fissures caries are the most prevalent of all caries lesions. *S. mutans* and other streptococci predominate. The appearance of caries occurs between 6 to 24 months after continuous colonization by *S. mutans,* so the sealing of pits and fissures is an important measure in the fight against caries.

- Smooth surfaces of proximal surfaces gingival to the contact point. Is the second most common location in the appearance of caries because they are protected areas from salivary flow, tongue and cheeks action. In this area there are other ecological determinants such as the shape and size of the papilla (depending on gingival health and age), oral hygiene (use or not of interdental brushes or dental floss) and irregularity of the dental surface (overhanging restorations, structural defects, etc.). Patients with gingival health have the papilla filling the interproximal space, leaving the enamel subgingivally, making colonization by *S. mutans* less likely. Periodontal disease or age leaves those spaces without papilla so they become refuge areas where colonization by *S. mutans* is more likely and, consequently, caries can appear at that level. The gingival third of the vestibular and lingual surfaces, that is more apical to the line of maximum contour of the tooth, is also an area that becomes a habitat for a mature cariogenic plaque as it is not subjected to food friction or meticulous brushing.

- Root surface. At the gingival level, on the proximal surface of the tooth, it usually has a concave shape. The enamel-cement transition is usually rough and, if there is also gingival retraction, the formation of mature cariogenic plaque is facilitated. *A. viscosus* and *A. naeslundii* predominate. They are areas of difficult hygiene and in the elderly with gingival retractions, less skill in hygiene manoeuvres and lower salivary flow increases the risk of caries. These cavities progress faster, the dental pulp is close and sometimes difficult to restore. These factors make an early diagnosis important.

- Subgingival areas. The gingival sulcus is a peculiar habitat where filamentous bacteria and spirochetes predominate. Initially the subgingival plaque is an extension of the supragingival one. The metabolites of this initial plaque produce an inflammatory reaction with the presence of leukocytes, immunoglobulins, albumin, hemins, etc. that eliminate some microorganisms. Bacteroides melaninogenicus survive thanks to the iron contained in hemins. This bacterium produces enzymes that destroy the gingival epithelium and cause periodontal disease.

# 9

# ELEMENTS OF THE DIET RELATED TO THE AETIOLOGY AND PATHOGENY OF CARIES

9.1 Nutritional and dietary aspects.

9.2 Cariogenic factors of the diet.

9.3 Cariogenicity of carbohydrates.

9.4 Other elements related to the development of caries.

9.5 Conclusions and recommendations.

**Introduction**

Food is one of the basic pillars in the development of dental caries because it involves the supply of nutrients necessary for the microorganisms involved in its appearance, whose metabolism is directly related to the appearance and frequency of caries. The relationship between diet and the appearance of caries is sufficiently demonstrated, on the other hand, there is no direct relationship between malnutrition and dental caries, although the impact it has on general health could influence the appearance of caries.

**9.1 Nutritional and dietary aspects.**

We consider food as the process of food intake that allows us to develop and obtain energy. This intake can be considered under two aspects: nutritional and dietary.

From a nutritional point of view, ingested food undergoes metabolic processes in the body in order to be used as a source of energy and nutrients to form body structures, maintain them, regulate metabolic processes and perform all vital functions. The content of foods is divided into five groups according to their composition: carbohydrates, fats, proteins, vitamins and minerals.

It is clear that the oral cavity is part of an organism and therefore depends on its general health and this, in turn, is not possible without proper nutrition.

The diet would be the amounts and types of food that an individual eats daily, its knowledge is recommended to understand the patient as a whole. Foods can be divided into seven groups that allow us to classify them according to their nutritional value:

- Group 1: milk and derivatives.
- Group 2: meat, fish and eggs.
- Group 3: legumes, nuts and potatoes.
- Group 4: vegetables.
- Group 5: fruits.
- Group 6: cereals.
- Group 7: butter and oils

Depending on the quantity and distribution in the daily intake of these food groups, an individual's diet can be established. The "Mediterranean diet" has been enacted as the most appropriate and balanced for humans. This diet is based on a pyramidal organisation of food based on the amount in which they should be eaten. According to the Mediterranean diet, we must take:

- At every meal: tubers, cereals or their derivatives and fruit.

- Every day: oil, dairy products, vegetables.

- From 3 to 5 times a week: legumes, white meats or fish and eggs.

- From 1 to 3 times a week: red meats.

- Occasionally: sweets and fats

- It is also advisable as a healthy lifestyle to exercise regularly.

**9.2 Cariogenic factors in the diet.**

As we already know, diet influences the appearance and evolution of dental caries, but not only from a quantitative point of view, as there are other modulating factors that are related to the characteristics of food and are as important or more than the amount of carbohydrates containing. These factors are:

9.2.1 Consistency and adhesiveness.

Cariogenic foods increase their danger as their consistency and adhesiveness increase, as this slows their removal from the tooth surface and increasing the production of acids that damage the enamel. Foods rich in starch, given their high consistency and adhesiveness, are more cariogenic than others rich in pure sucrose.

9.2.2 Time of intake

The grouped consumption of the food facilitates that the own mechanisms of self-cleaning clear food debris from the mouth. This is because during the meals the increase of the production of saliva, the movements of tongue and cheeks and the own movements of the mastication exert an action sweep on the surface of the teeth. Therefore, the consumption of cariogenic foods during meals is less dangerous than their consumption between meals, being the worst time for consumption before going to bed, since during sleep the volume of salivary secretion decreases physiologically.

9.2.3 Frequency

Frequent and short-spaced intakes of cariogenic foods generate continuous drops in pH below the critical point and prolong the oral rinsing time, which increases the risk of enamel demineralization. (fig 1)

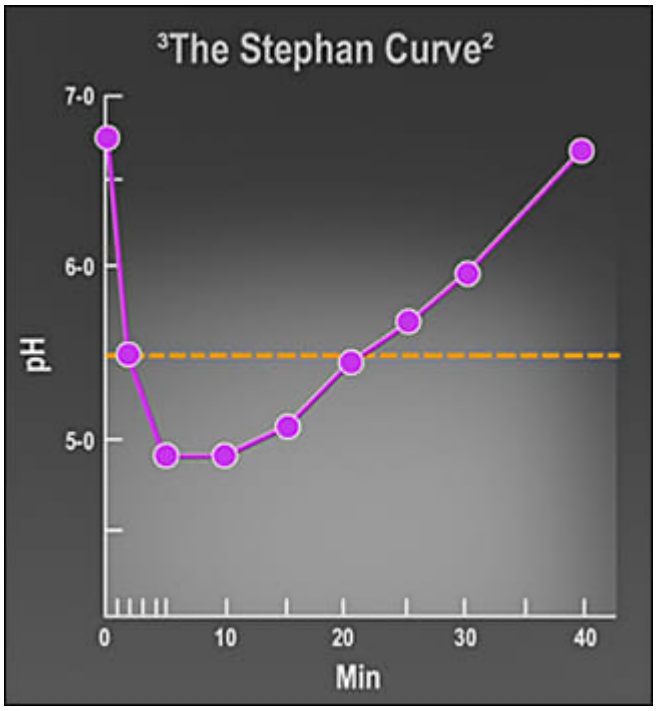


Figure 1. Stephan´s curve

*Adapted from: Stephan RM, Miller BF. A quantitative method for evaluating physical and chemical agents which modify production of acids in bacterial plaques on human teeth. J Dent Res. 1943;22;45-51. the-stephan-curve.

9.2.4 Food composition.

The cariogenicity of carbohydrates has been widely demonstrated. The protective effect of some food components against caries is also known today. Phosphates and some components of cocoa have been shown to reduce tooth decay.

9.2.5 Identification of risk groups.

The combination of these factors allows us to establish high-risk groups in developing caries lesions, including identifying age-associated risk groups.

During infancy and after the rash, breast milk alone is not cariogenic. However, the excess carbohydrates caused by the continued use of the bottle with juices, porridge or sugary drinks are associated with early caries in the child, which appear on smooth surfaces and are rapidly evolving and very destructive.

In adolescents, there has been an increase in the development of caries associated with the abuse of sugary and carbonated beverages.

In medically compromised patients, a decrease in salivary flow is frequent, and therefore a decrease in oral clearance. This situation worsens in cases of eating with a soft diet with greater adherence to the tooth surface. Workers with monotonous jobs,

as well as catering or bakery workers, generally have a higher number of intakes (pecking) separated by short periods of time.

### 9.3 Cariogenicity of Carbohydrates

The pathogenic action of carbohydrates is due to the fact that these, and especially sucrose (fructose and glucose) when metabolized by microorganisms, produce acids that demineralize in the enamel. In addition to this acidogenic action, *Streptococcus Mutans* produces glucan, extracellular polysaccharide, by which the bacterium can adhere firmly to the tooth.

Various experimental studies have linked caries to carbohydrate intake. The best-known being, perhaps for its lack of ethics, the Vipeholm study (Gustafsson, 1954). This was a 5-year longitudinal study that used 436 mentally ill people with the aim of relating carbohydrates to caries and assessing how they influence the frequency and characteristics of the foods that contain them.

Patients from a psychiatrist were taken as a sample and divided into groups that were fed carbohydrates of different consistency and with different frequency, obtaining the following result:

- The control group, with a low-carbohydrate diet, barely had caries.

-Groups who consumed sugary liquids or bread with meals showed a slight increase in caries activity.

- The group that consumed chocolates between meals four times a day showed a moderate increase in caries activity.

-The groups that consumed 8 and 24 toffee-type candies between meals showed a dramatic increase in caries.

In view of these results, Vipeholm's study came to the following conclusions:

- Sugar has a caries-promoting effect on the surface of the tooth.

- Foods rich in starch (bread) are not as cariogenic as those rich in sucrose.

- The amount of sugar is not of paramount importance in the formation of caries.

- The form of presentation and composition of sugars can be decisive; thus, sugars in adherent form are more cariogenic.

- Sugary foods are more cariogenic if consumed between meals than if consumed with them.

- The more frequent the intake of cariogenic foods, the greater the cariogenic activity.

- The increase in caries activity in uniform experimental conditions varies from person to person.

- The increase in cariogenic activity disappears with the withdrawal of foods rich in sugar.

- Caries appear despite removing sugar from the diet.

9.3.1 Miller's acidophilic theory

Miller's acidophilic theory is the best known for explaining the formation of dental caries, it is based on four basic pillars:

- Some bacteria in the oral cavity are able to metabolize carbohydrates through the anaerobic glycolytic pathway, which produces acids. Mainly lactic acid.

- The enamel is formed by hydroxyapatite crystals ($[Ca^{+2}]_{10}\ [PO_4^{-3}]_6\ [OH^-]_2$ [8]) in more than 90% and these in turn by calcium salts, which can be dissolved by action of acids.

- The formation of organic acids by bacteria is detectable in the mouth after ingestion of carbohydrates.

- The decrease in pH caused by acids begins the process of decalcification of the enamel when it drops from 5.5 (critical pH for enamel). These acids in high concentrations solubilize hydroxyapatite and consequently demineralize the enamel, which is known as acid solubilization.

$$[Ca^{+2}]_{10}\ [PO_4^{-3}]_6\ [OH^-]_2\ +\ H^+\ 6\ PO_4\ H^-\ +\ H_2O\ +\ 10\ Ca^{+2}$$

$$[Ca^{+2}]_{10}\ [PO_4^{-3}]_6\ [OH^-]_2\ +\ H^+\ 6\ PO_4\ H^{-2}\ +\ H_2O\ +\ 10\ Ca^{+2}\ [8]$$

We can summarize this theory that carbohydrates in the oral cavity are metabolized by bacteria producing acids that release $H^+$, lowering the pH below the critical point, which initiates the decalcification of the enamel.

9.3.2 Danger of carbohydrates.

Carbohydrates have different cariogenic potential depending on their composition and structure. The most cariogenic of all is sucrose (common sugar), followed by monosaccharides such as glucose and fructose (fruits, honey) and disaccharides such as lactose (milk). Lastly are the large starch-type polysaccharides, although cooked is more cariogenic than raw.

Knowing the conclusions of the Vipeholm study and the carbohydrate hazard scale, it is possible to establish a food hazard scale that allows us to guide patients towards a less cariogenic diet. Thus, the hazard scale is in descending order:

- Solid and retentive foods consumed between main meals, or before bedtime.

- Solid and retentive foods consumed during meals.

- Liquid foods rich in carbohydrates, non-retentive, consumed between meals, or before bedtime.

- Liquid foods rich in carbohydrates, non-retentive consumed during meals.

As we see on this hazard scale of food, we should lower the frequency of carbohydrate intake by avoiding pecking between meals of carbohydrate-rich products, as salivary flow is lower and the ability to rinse worsens, especially with sticky and insoluble food.

## 9.4 Other elements related to the development of caries

### 9.4.1 Sugar substitutes

There are sweeteners that have a similar taste to sugars, but as they are not carbohydrates, they are not metabolized by plaque bacteria and no acids are produced. Or if any acid is produced in their metabolism it is in very small amounts, so the number of caries is reduced. There are two types of non-cariogenic sweeteners: caloric and non-caloric.

Caloric sweeteners are sugar alcohols and their use are widespread. The most common being xylitol, sorbitol, mannitol, lactitol and maltitol. Of these, xylitol has remarkable characteristics, is a pentitol with a sweetness similar to that of sucrose. It also has the peculiarity of exerting a cariostatic action by interfering with the metabolism of *Streptococcus mutans*, so that their saliva and plaque levels are reduced. In addition, if the xylitol is in chewing gum the continuous mastication increases the salivary flow and increases the pH.

Non-caloric sweeteners also provide intense sweetness, but do not provide energy, or their contribution is so low that it does not matter. The most used are saccharin and aspartame.

### 9.4.2 Protective factors of the diet

On the other hand, there are different foods that can have protective effects against caries.

Food fats coat the surface of the tooth, preventing the plaque from adhering and retaining carbohydrates, and they can also be toxic to bacteria.

Proteins prevent enamel erosion because they increase the buffering capacity of saliva.

The associated consumption of fats and proteins after carbohydrate intake increases the pH.

### 9.4.3 Influence of fluoride use

It has been shown that the use of fluorides prevents the appearance of caries, remineralizes the areas where the demineralization of the enamel has begun and decreases the rate of evolution in case of existing injuries.

Its main preventive effect against caries is based on its ability to bind to the phosphate and calcium of enamel hydroxyapatite and dentine, decomposing into calcium fluoride and sodium phosphate. Calcium fluoride precipitates on the enamel to form fluorapatite, which is more resistant to acid attack than hydroxyapatite. If calcium fluoride is left unresolved, it is entrained by saliva and can be deposited in a second reaction. Another of the anti-caries effects of fluoride is based on the ability to modify the surface load of the tooth, preventing the formation of the acquired pellicle and the consequent adhesion of bacteria.

In incipient caries lesions fluorides interact with hard tissues promoting their remineralization and slowing demineralization. The remineralizing effect of the lesions is achieved by precipitation in the enamel of calcium and phosphate present in saliva, replacing manganese and carbonate salts lost by the demineralization of acids produced by bacteria.

The most important protective function of fluorides in the aqueous phase against caries is based on their ability to alter the saturation of the saliva and plaque surrounding the tooth, decreasing the dissolution of the enamel and promoting its remineralization.

At the bacterial level, fluorides in low concentrations prevent the formation of extracellular polysaccharides from glucose by inhibiting glucosyl transferase, thereby decreasing bacterial adhesion. It also inhibits the formation of intracellular polysaccharides because it prevents bacteria from storing carbohydrates, thus limiting their metabolism when no carbohydrates are present. Fluorides in high concentrations have a bactericidal effect on *Streptococcus Mutans*.

## 9.5 Conclusions and recommendations

Diet is a determining factor in the environment of the oral cavity for the development of caries, so it is necessary to make patients aware of its importance. On the other hand, we must not forget that a diet with an excessive intake of carbohydrates will not only suppose a higher risk of caries, but also increases the risk of obesity, metabolic disorders (diabetes), cardiovascular disease (hypertension), respiratory (apnoea) or fractures.

From a cariogenic point of view, we must analyse the diet to determine the possible correlation existing in our patients, with the aim to convey to them the importance of following some recommendations as a general rule:

- Reduce sugar consumption <40 g / day.

- Reduce the frequency of consumption.

- Avoid snacking between meals.

- Reduce the consumption of sticky foods (propose substitutes).

- Advocate the use of other sweeteners.

# 10

# STRUCTURAL ALTERATIONS IN CARIES INJURIES

10.1 Enamel caries.

10.2 Dentine caries.

10.3 Cement caries.

## Introduction

The substrate where caries develop is represented by the hard tissues of the tooth, which have significant differences in its composition and structural organization. This will condition the structural alterations that will produce caries, as well as the way it evolves.

As we already know, caries is an infectious, multifactorial and universal disease that affects the mineralized matrices of dental tissues by acid attack. It also determines their demineralization, dissolution and degradation, and given the evolutionary nature of the disease structural damage to the teeth´s hard tissues can only be made of enamel or cement, even affecting the dentine.

## 10.1 Enamel caries

The enamel is a tissue with low permeability and quite resistant to dilution by biofilm acids. Prism sheaths, striae of Retzius, interprismatic spaces, perikymata, enamel lamellae, and fusiform bodies or enamel´s spindles are the main routes of water diffusion and small ions such as $H^+$ since they are the elements of the enamel with lower mineralization and higher organic content.

The critical pH at which enamel demineralization processes begin to appear is below 5.5. In most cases the demineralization processes are followed by remineralization processes with which there is an apparent balance that does not affect the integrity of the tooth. Only when the balance tilts to the side of demineralization, the first clinical manifestations of dental caries begin to appear. The curious thing is that the outermost 10-30μm of the enamel remain intact, these correspond to the aprismatic enamel as we have already mentioned, more resistant to acidic pH and where the remineralization processes based on fluorapatite are established.

### 10.1.1 Evolution of enamel caries.

The onset of the damage begins in the subsurface enamel causing the first clinical manifestation also called white spot. Apparently and from a clinical point of view, the surface looks healthy, but when dried, an opaque calcareous whitish surface appears in the enamel. Moisture lesion is not detected because the wet porous area is translucent.

Temporal evolution has shown that at least two weeks must pass without altering the plaque to see the appearance of this stain on the tooth. After 3 or 4 weeks the white spot lesion is visible even without drying.

The shape of the lesion will depend on its location:

- Pits and grooves, will have a conical shape, with the base towards the dentinoenamel junction by the direction of the prisms at this level.

- Proximal surfaces of the tooth, kidney-shaped, elongated in buccal-lingual sense, located between the contact point and the gingival margin.
- Smooth surfaces: conical shape with the base of the cone on the surface.

At the structural level, the following changes occur:

- Dissolution of hydroxyapatite crystals.
- The crystals come loose.
- The prisms separate.
- There is an increase in interprismatic spaces.
- The central area of the prisms.

This white spot injury is reversible as long as the enamel retains its mineral structure. The calcium and phosphate ions in saliva, usually hypersaturated from these ions, as well as the fluorine inputs from water, mouthwashes and toothpaste, penetrate the enamel and remineralize these crystals. The presence of fluorine allows fluorapatite to form during remineralization, which is much more resistant to acid attack than hydroxyapatite as its critical pH is lower.

Remineralized lesions are sometimes pigmented brown because when the enamel was demineralized, it allowed organic molecules and metal ions to become trapped, resulting in the so-called brown stain.

When the demineralization of the subsurface is so extensive that the enamel collapses, cavitation occurs. This process is now irreversible, as a result of a combination of demineralization and remineralization cycles with a predominance of the former.

When enamel cavitation occurs, the exposed surface is rougher and this allows bacteria that do not easily adhere to the surface to now colonize it, such as filamentous bacteria. When the plaque becomes thick enough, it becomes anaerobic on the tooth surface.

The shape of the lesion is conical, extending through the prisms of the enamel with the base of the cone at the dentinoenamel boundary. Microbiologically, we will see how *Streptococcus mutans* have the main role of the initial caries lesion. Then Lactobacillus is a prominent bacteria in cavitary lesions and their progress in the dentine.

From the third or fourth week of evolution of the bacterial plaque on the tooth, this sub-superficial demineralization is seen, and four histological layers are already clearly observed in the white spot lesion:

- Translucent area. It is the deepest area of the lesion. It has 1% porosity, which represents 10 times more porosity than healthy enamel. It is called translucent because by filling the pores with quinoline and observing the lesion with polarized light, it has the same refractive index as healthy enamel.
- Dark area. It is the layer immediately above the translucent one. It has 2 to 4% porosity, although it has smaller pores, it is believed that this phenomenon has to do with the apposition of ions in the remineralization phases produced

recently. It does not transmit polarized light as the translucent layer does, hence its name. It has 5 to 8% less mineral tissue, compared to healthy enamel.
- Body of the lesion. It is the largest area of enamel caries. It is located between the surface area and the dark area. It has a higher percentage of pores, 5-25% of the total volume and the porosity is higher in the centre of the area. The mineral loss is 10 to 15% compared to healthy enamel.
- Surface area. It is only present in lesions in which the enamel has not yet cavitated. It has a thickness of 20 to 50 µm. The mineral content is 5-10% lower than that of healthy enamel. The surface of the enamel is remineralized with fluorides and as the lesion progresses conical defects can be observed in it which make the surface more retentive. The remineralization of this area always gives harder enamels, more or less retentive depending on the degree of superficial affectation.

Once we know the histological layers that exist in a caries lesion, we can understand the six phases of the development of the enamel lesion described by Darling in 1959:

- Phase 1: only the surface area and the translucent area are observed.
- Phase 2: the dark area appears between the surface layer and the translucent one.
- Phase 3: at this time the dark area is wider, and the body of the lesion appears.
- Phase 4: this phase is only a progression of phase 3. The surface layer is still intact, but the lesion appears clinically as a white spot and can be detected radiographically on the interproximal surfaces. In the last part of this phase, caries already reaches the dentinoenamel junction.
- Phase 5: the surface enamel acquires a chalky appearance and can break with an exploration material.
- Phase 6: the surface layer collapses and the enamel cavitation occurs.

**10.2 Dentine caries**

Dentine is a less mineralized and more permeable tissue than enamel. It includes tubules that allow water, bacteria and ions to penetrate. It also contains cytoplasmic extensions of odontoblasts; whose cell bodies are in the outermost layer of the pulp. Because of this, it has mechanisms of reaction against the advance of caries, which determines its progression.

The caries process usually reaches the dentinoenamel junction long before the enamel cavitation occurs. That is, the dentine-pulp complex involvement in the caries process is very early. When caries reaches the dentinoenamel limit, there is a lateral extension of the lesion due, precisely, to the lower mineralization and the higher organic component of the dentine, continuing the progression following the direction of the tubules. Therefore, the lesion in the dentine has a cone shape with the base at the dentinoenamel junction.

10.2.1 Evolution of dentine caries.

In the dentine affected by caries, before the cavity occurs in the enamel and especially in the slowly advancing caries, the following histological layers are distinguished from the surface to the deepest area of the lesion:

- Body of the lesion or demineralization area. It is the most superficial area in the dentine, adjacent to the amelodentinal limit. The lesion follows the normal curvature of the tubules. It appears when bacterial products reach the dentine. It concentrates the highest demineralization and the highest percentage of bacteria.
- Translucent area, transparent area or sclerotic reaction. It is between the body of the lesion and the dentine not affected by caries. There is an area with tubular sclerosis that histologically is observed as a translucent region because the contrast medium does not penetrate the tubules. In this area the intertubular dentine is demineralized, but the collagen network is preserved, which serves as a template for remineralization. Inside the tubules, crystals are formed and partially or completely occlude their lumen. There are no bacteria in the tubules, but there is already an alteration in the odontoblastic processes.
- Normal dentine. The translucent area may extend to the pulp, although it is possible to find an unaffected dentine area between the sclerotic dentine and the pulp.
- Reactive dentine, as a defensive reaction.

As we have seen in the enamel, the progression of the caries lesion in the dentine also follows an established chronological pattern that we can observe in the following phases:

- Phase 1: The biochemical changes in the environment, produce the partial or total obliteration of the tubular lumen by the deposit of crystals in the tubules and the creation of peritubular dentine by the extensions of the odontoblasts. This process begins between the dentine where caries and pulp progress.
- Phase 2: Plaque acids initiate the process of dentinal demineralization by dissolving the peritubular dentine and permeabilizing the tubules.
- Phase 3: When the enamel cavities, the germs invade the tubules, exacerbating the acid attack. The tubules are abnormally widened discontinuously.
- Phase 4: the injury continues with the involvement of the intertubular dentine, which undergoes demineralization and alteration of its organic matrix.
- Phase 5: Intratubular and peritubular lesions join to form rounded microcavities in the dentine that contain germs and debris from tissue destruction.
- Phase 6: The microcavities converge causing dentinal softening.

We must not forget that this process does not always develop in the same way, since the dentine-pulp complex is able to develop defence mechanisms to respond to the progress of caries.

10.2.2 Defensive reactions of the dentine-pulp complex.

The dentine-pulp complex reacts to aggression in three different ways: by trying to obliterate (block) the lumen of the tubule, by remineralizing the affected tissue, and by trying to protect the pulp from the advance of caries.

The intensity of the defensive response will depend on the previous state of the pulp, the presence of a good vascular supply, the dentinal structure and the intensity of the injury attack.

- The intensity of the attack. Caries is an intermittent disease that alternates periods of rapid evolution with others chronic over time. When progress is slow, the defensive response is more favourable.
- Dentine structure and permeability. Dentin with narrow tubules and reduced in number, makes it difficult for caries to progress. This is why the progression of caries is faster in the young than in the elderly.
- The previous state of the pulp. The defensive response of the dentine-pulp complex is more favourable if the pulp is young and has no previous pathological or restorative involvement.

The mechanisms of the defensive response of the dentine-pulp complex are:

- Cellular changes. Morphologically there is a reduction in the number and size of the cell bodies of odontoblasts adopting a flat or cuboid shape compared to their natural cylindrical morphology. In cases where there are dead primary odontoblasts in adjacent areas to the lesion, it is observed how undifferentiated mesenchymal cells of the pulp assume the role of these odontoblasts.
- Sclerotic dentine is darker in colour than normal dentine, bright and hard to the touch with the spoon excavator or explorer. It is much less permeable than normal dentine, which makes the progression of caries slower. Radiographically it can sometimes be observed as a more radiopaque area in front of the more radiolucent area of caries given its greater mineral component, existing often under the old restorations. It is not necessary to eliminate it during the treatment since this tissue is not infected. For sclerotic dentine to form, it is essential that the odontoblasts are alive.
- Tertiary Dentine. The acidic products, hydrolytic enzymes, bacterial toxins, cause necrosis of the odontoblast and leave dead tracts. As a reaction to the progress of caries, the dentine-pulp complex forms dentine which we call tertiary. There are two types of this tertiary dentine: reactive dentine, which is created by odontoblasts surviving aggression and reparative dentine produced by secondary odontoblasts or new odontoblasts generated from undifferentiated mesenchymal cells. Tertiary dentine forms in the pulpal wall, adjacent to the caries advancement area. This dentine has a more irregular structure than normal dentine.
- Intrapulpal calculations (pulpolites), which are dentine not attached to the natural dentine and which is established as a stone in the dental pulp.

10.2.3 Pulp reactions

It is important to know that the dental structure is irrigated by small apical foramen, and there is no collateral vascularization. It is a rigid structure, which cannot expand or dilate. This means that when there is an aggression in the dentine-pulp complex, and there is inflammation, which requires a greater arrival of blood to the tooth, there is an increase in the size of the artery at the level of the apical foramen. But just as it produces an increase in volume at the entrance, the same volume must come out of the tooth through the venous system. Being a tissue without elastic capacity there is a collapse at the height of the foramen, with an increase in pressure inside the pulp that in many cases and depending on factors such as the duration time, previous state of the piece and intensity of the process, they make the dental pulp unable to withstand the pressure and eventually degeneration and necrosis occur more quickly. Elementally in teeth that are still in formation and have the apexes open the ability to react to inflammation will be greater. Depending on the nature of the progress we can see how in acute lesions, inflammation occurs with vasodilation and increased vascular permeability and accumulation of leukocytes. Compared to chronic processes where the cell infiltration is composed of macrophages, lymphocytes and plasma cells with a proliferation of vessels, fibroblasts and deposits of collagen fibres, i.e. with the ability to repair connective tissue.

**10.3 Cement caries.**

Cement is a thicker, rougher tissue than enamel. In this case, caries lesions follow a similar pattern to that described above in structural enamel alterations, but with faster bacterial penetration. They are usually more extensive lesions than those of the enamel, but shallow with less defined edges and a "u" shape in cross-section. A good brushing technique and the use of fluoride toothpaste and mouthwashes can stop the progression of the injury.

# 11

# INTERACTIONS BETWEEN DENTAL TISSUES AND ORAL FLUIDS

11.1 Characteristics of the enamel.

11.2 Characteristics of saliva.

11.3 Demineralization.

11.4 Remineralization.

11.5 Role of saliva in demineralization-remineralization processes.

11.6 Anticariogenic role of saliva.

## Introduction

Saliva protects the general oral health and provides an important regulatory mechanism in the processes of demineralization and remineralization of the tooth. This interaction that occurs between oral fluids and dental tissues is conditioned by several factors. The study of these factors will allow us to know the involvement of saliva in regulatory processes, the dynamism of enamel remineralization processes, and identify the circumstances or pathologies in which this regulatory mechanism does not work properly and the imbalance that causes the onset of dental caries.

Understanding the pathophysiology of tooth demineralization processes, it is easier to understand the importance in implementing protocols that protect the enamel against acids and favour the remineralization processes against demineralization.

### 11.1 Characteristics of the enamel.

Enamel is the hardest substance in the human body. It contains no cells and is made of hydroxyapatite structural units arranged in prisms. Its mineralization is higher than in dentine or cement because it is formed in more than 90% by an inorganic matrix of long and fine hydroxyapatite crystals arranged in prisms that can have inclusions of carbonate, sodium and fluorine. The inorganic matrix is formed by the following compounds:

- Phosphate and carbonate mineral salts (forming hydroxyapatite crystals), calcium, sulphates and trace elements (potassium, magnesium, iron, fluorine, manganese, copper, etc.).

- Fluoride ions, which can replace hydroxyl groups in a proportion of one in forty crystals, converting hydroxyapatite to fluorohydroxyapatite, more resistant and less soluble in acids.

The rest, which would be the space between crystals, is occupied by water and organic matrix. This organic matrix is formed by the following organic components:

- Amelogenins (90%), located throughout the enamel.

- Enamelins (2-3%), located as a coating on the periphery of hydroxyapatite crystals.

- Ameloblastins (5%), located at the periphery of enamel prisms.

- Tuftelin, 1-2% located in the amelodentinal junction.

- Serum Proteins, chondroitin 4-sulfate, chondroitin 6-sulfates plus lipids.

The water content of the enamel creates a layer on the hydroxyapatite crystals that moisturizes its surface. It is very low, about 3% and its percentage decreases with age.

### 11.1.1 Physiology of enamel

The minerals present in hydroxyapatite have a certain solubility in water and when dissolved the calcium, phosphate and hydroxyl ions are released into the solution that coats the tooth. This hydroxyapatite solution slows down and comes to a stop when the solution is saturated with these ions.

The pH of the environment influences its saturation. The presence of $H^+$ (hydron, positive hydrogen ions) lowers the pH of the oral environment and decreases the saturation of phosphates. This causes the hydroxyapatite to continue to dissolve to equalize the concentrations. The pH from which, if it continues to fall, the dissolution of the mineral is favoured is called "critical pH". The critical pH of hydroxyapatite is 5.5.

We must keep in mind that the enamel of a tooth is not homogeneous, with crystals close to the surface having more fluorine and less carbonate than deeper crystals. This makes the outer area less soluble. The hydroxyapatite crystals of the enamel may further contain other inorganic ions in their structure apart from the calcium, phosphate and hydroxyl ions. It is common to incorporate the carbonate ion instead of the phosphate ion, or the calcium ion by sodium ion, or the hydroxyl ion by fluorine ion. The apatite of adult enamel is often carbonated fluorohydroxyapatite. Other ions that can also be incorporated in small proportions are chlorine and magnesium. The incorporation of these ions into the enamel produces changes in its solubility: fluorine makes it less soluble while carbonate makes it more soluble. The critical pH of fluorapatite is 4.5 while for hydroxyapatite is 5.5

The composition of the plaque fluid is very important as it covers the enamel in some areas avoiding direct contact with saliva. The plaque has a higher content of calcium and phosphate ions. This is good because it reduces the dissolution and facilitates the remineralization of the enamel, but also favours the calcification of the plaque itself forming the tartar or calcified plaque. With the contributions of sucrose to the plaque there is a decrease in pH due to the release of acids from the bacterial metabolism of that sucrose. When the pH drops below 5.5, the demineralization of the enamel is facilitated as there is a decrease in hydroxyl and phosphate ions in the fluid.

## 11.2 Characteristics of saliva

Saliva is a complex natural and physiological fluid, which lubricates the oral cavity. It is secreted by the major (93%) and minor (7%) salivary glands. It is composed of 99% water and organic and inorganic molecules. It is transparent, of variable viscosity and odourless. When it leaves the salivary glands, it is a sterile fluid, but it ceases to be so immediately, as it mixes with gingival crevicular fluid (contains plasma products), food and cellular debris and oral bacteria.

The volume of physiological secretion varies between individuals and with age. Usually, the total daily volume ranges between 500 and 700 ml, remaining constantly around 1.1 ml in the oral cavity.

Its production is controlled by the autonomic nervous system and in the same person varies according to the time of day and the presence or not of stimuli. The basal secretion is between 0.25 and 0.35 ml/min, being able to increase before stimuli up to 1.5 ml/min. The highest peak of production takes place before, during and after meals and the lowest peak of production takes place at night, during sleep.

Saliva plays a key role in caries protection, so an alteration in its quantity or quality is another of the etiological factors of the appearance of caries. We differentiate between the pathological and objective decrease of saliva, called hyposalivation or hyposialia, from the subjective sensation of dry mouth, called xerostomia. In contrast, overproduction is called sialorrhea. The measurement of salivary production volume is called sialometry.

Saliva is the main regulatory mechanism of the host's own plaque. The nutrients in saliva allow the resident flora to survive, which is beneficial, and its antimicrobial components often inhibit non-resident microorganisms. Patients receiving head and neck radiotherapy, with decreased salivary secretion and changes in the oral microflora, increase the number of cavities.

### 11.2.1 Functions of saliva

A) Humidification and lubrication of the oral mucosa and lips. Saliva is one of the best lubricants of natural origin. Provides adequate lubrication for diction, chewing and swallowing. Salivary mucins, with their low solubility and high viscosity, elasticity and adhesion, act by coating the oral tissues and the food bolus, contributing to the protective function of the oral and labial mucosa.

B) Control of the oral microbiota. Saliva has a defence mechanism with antimicrobial function. It acts as a regulator keeping a balance of the different species of microorganisms present in the oral cavity. Saliva performs this function by acting as a protective barrier that interferes with the process of adhesion of pathogens, essential for their survival.

C) Cleaning. The salivary flow and the movement of the tongue and the lips generate a mechanical action of washing, dragging and sweeping food and cell debris and microorganisms in suspension.

D) Conservation of oral tissues. Saliva intervenes in the mechanisms of demineralization-remineralization of dental hard tissues, maintaining and protecting their integrity. It provides the minerals in suspension necessary for the tooth surface to be harder and less permeable to the oral environment.

E) Digestion. This is one of the most important functions of saliva. It does it through two processes, one of them is the presence of salivary amylase, that acts by degrading starch, and the other by mixing with food to form the food bolus so that digestion begins.

F) Neutralization. Saliva has a buffer capacity which neutralizes acids in the mouth. This capacity comes mainly from the bicarbonate and phosphate systems. Bicarbonate is able to diffuse into oral biofilms and neutralize the acid formed by microbial metabolism. The buffer capacity depends on the secretion rate. Stimulated saliva contains higher bicarbonate concentration increasing its neutralizing capacity.

G) Taste. Saliva is essential for normal taste perception, as it dilutes food and washes the papillae, leaving them in a position to recognize different flavours.

H) Dilution and temperature adjustment. Saliva flow increases sharply after the penetration of acidic substances in order to dilute them and maintain the pH. In the same way, it cools down hot food or heats up the cold food.

I) Salivary excretion. Saliva is a route by which some organic products and some products introduced into the body will be eliminated. It eliminates urea, uric acid, certain hormones and viruses responsible for diseases, such as mumps, are also eliminated.

J) Diagnostic function. The use of saliva as a diagnostic tool has gained considerable attention and it has become a well-accepted method. Saliva offers superiority over serum as it is a non-invasive and cost-effective procedure to study in large populations. Sample acquisition is painless, reducing the discomfort for those patients who have to undergo repeated blood extractions.

11.2.2 Hyposialia

Salivary secretion decreases physiologically during sleep and pathologically due to different causes:

A) Drugs. This is the most common cause. There are many drugs that have this effect being the most common tricyclic antidepressants, antiparkinsonians, phenothiazines and benzodiazepines, as well as anticholinergics, antihistamines, antihypertensives, antipsychotics and diuretics.

B) Systemic diseases such as Sjögren's syndrome, rheumatoid arthritis, systemic lupus erythematosus, scleroderma. Diabetes causes hyposialia due to dehydration and alteration of the salivary glands. Dehydration in febrile processes, diarrhoea, polyuria, haemorrhage and gastroenteritis also produce hyposialia. It can be observed as well in cases of hypertension, polyneuropathy, and some deficiencies of vitamins, riboflavin and nicotinic acid.

C) Radiotherapy of head and neck. It produces irreversible hyposialia with doses higher than 40 Gy, bilaterally, in the major salivary glands.

D) Psychiatric disorders, especially anxiety and depression.

E) Marijuana use.

F) Aging. It is associated with the consumption of drugs and the presence of diseases that affect the salivary glands.

### 11.3 Demineralization

Demineralization of the hard tissues of the tooth occurs when the saturation of mineral ions in the oral environment is low in relation to the mineral content of the tooth. When the pH drops to 5.5 and begins the process of demineralization of the tooth. This process depends on the existence of an acidic medium and a mineralized substrate. This process of demineralization of enamel by acids can give two types of injury depending on the origin of the acid: caries or erosion.

In caries, the lactic and acetic organic acids that cause the demineralization of enamel come from the metabolism of carbohydrates by microorganisms. For the caries injury to begin it is necessary to break the balance between the processes of demineralization and remineralization in favour of demineralization produced by the drop in pH. The acid initially produces the surface demineralization of the enamel. As the demineralization progresses in depth, the surface initiates remineralization processes, forming a remineralized layer 20-50 μm thick and a sub-surface layer with a demineralization up to 30- 50% advancing in enamel and dentine. In this way, as the caries lesion progresses the internal demineralization increases while the superficial layer stays intact until it collapses originating an open cavity later on.

In erosion there is a supply of exogenous (beverages, drugs, food, etc.) or endogenous acids (vomiting, regurgitation, etc.) without the participation of bacteria. Progressive layer-by-layer demineralization occurs without surface remineralization as these lesions occur at a lower pH than in caries preventing surface remineralization.

### 11.4 Remineralization

In the hydroxyapatite the reverse process of demineralization also occurs. Under certain circumstances in the environment, new crystals can precipitate on the surface of the mineral. This occurs when the oral environment is saturated with calcium and phosphate ions with respect to hydroxyapatite, when the pH increases (concentration of phosphate and hydroxyl ions increases). There are substances such as pyrophosphate and some salivary proteins that inhibit the new formation of hydroxyapatite. For this reason, there is no indefinite growth of crystals on the surface of the enamel.

This mechanism of formation of new hydroxyapatite crystals is called remineralization and it is a very important repair mechanism of the enamel. It consists in the repair by replacement of the minerals that the tooth has lost. The phosphate ions, calcium and other lost minerals are replaced by ions from saliva, which may be the same or similar, or include fluorine, resulting in the formation of fluorapatite crystals.

In case of incipient caries lesions, the remineralization processes act by decreasing the size of the lesion and increasing the resistance of the remineralized tooth. The incorporation of fluorides in the remineralized areas decreases the pH at which demineralization begins. This is because fluorapatite crystals are larger and more resistant to acid dissolution than the hydroxyapatite crystals of the original enamel, so that the strength of the exposed surfaces of the tooth increases.

This balance between the demineralization and remineralization phases is influenced by the cariogenic bacteria present in the oral environment, the supply of nutrients to the bacteria and the substrate on which they are found, and regulated by saliva, as it exerts a modulating factor on the above factors and transport in suspension the ions for the remineralization process. Thus, we can consider as the beginning of the caries destruction of the hard tissues of the tooth the imbalance between the processes of demineralization and remineralization in favour of the processes of demineralization.

### 11.5 Saliva in demineralization-remineralization processes

As long as the processes of remineralization predominate in the oral cavity over those of demineralization, caries will not appear, so the aim of prevention methods will be to favour this situation. And this is where saliva plays an important role because of its physical and chemical characteristics providing a natural defence system against demineralization processes and promoting the repair of damaged structures. In addition, saliva decreases the solubility of enamel by different mechanisms:

A) It forms the acquired pellicle that acts as an organic barrier for the diffusion of acids and offers protection to the enamel against demineralization.

B) Contains fluorine. Fluoride is incorporated into the structure of the enamel in the remineralization phases together with calcium and phosphate ions, quickly forming fluorapatite crystals, larger and more resistant to acids than hydroxyapatite. The ability of fluoride to be incorporated into the enamel in remineralization processes, resulting in a more resistant enamel along with its important antimicrobial effect, have made the topical use of fluorides the most important measure of caries prevention in public health. To do this, it is necessary that its presence, although it is sufficient at low concentrations, is continuous as it is easily eliminated.

C) Contains phosphate and calcium ions. A high concentration of these ions in saliva with respect to enamel prevents the dissolution of hydroxyapatite crystals. At normal pH the enamel does not dissolve in the mouth, but the decreases in pH caused by the metabolism of some foods and other acid intakes are frequent. However, despite containing calcium and phosphate ions, it also acts as a regulator to prevent excessive deposition of these in the teeth.

D) Sweeping. It also exerts an indirect function due to its ability to sweep both bacteria and acids on the tooth surface.
Mineral compounds of calcium, phosphate, sodium and silica have been developed and, when incorporated into saliva, they bind to the tooth providing a continuous deposit of crystalline carbonated hydroxyapatite. These compounds can also act by binding to calcium carbonate, causing a slow solution that releases ions of calcium and phosphate. It has been observed that these contributions of calcium and phosphate ions to saliva that aim to increase their repair capacity in areas of erosion and sensitivity also favour remineralization processes.

There are also compounds that contain casein as an active ingredient, a milk derivative that has calcium and phosphate that, when it binds to the tooth, can be released during

pH drops.

Both compounds were developed in principle for the treatment of dental sensitivity caused by erosion, however they have shown the ability to remineralize incipient caries lesions, in addition to interfering with the adhesion of bacteria to the tooth surface.

## 11.6 Anticariogenic role of saliva

### 11.6.1 Mechanical sweeping or oral clearance

The presence of saliva, added to the muscular action of the tongue, cheeks and lips, determines a mechanical drag action that makes possible the continuous cleaning of bacteria, bacterial products and food debris that are free in the mouth and in the accessible areas of the mucous membranes and teeth. This function of dragging or mechanical sweeping is called oral clearance. The oral carbohydrate clearance is the time required to reduce it to levels before it was ingested or to leave it at a concentration of less than 0.1%. It is more effective during chewing or oral stimulation as more saliva is secreted.

Clearance times may be prolonged by decreased saliva flow, increased saliva viscosity, increased food viscosity, decreased muscle activity and the existence of retentive factors in dentition (crowding, brackets, misaligned prostheses, overhanging restorations, etc.). Oral clearance time shows large individual variations and there is a clear relationship between slow clearance and increased risk of caries.

### 11.6.2 Buffer action

Saliva has several components that act as a buffer trying to maintain a constant pH. It is more effective on the surface of the enamel and in the middle of the biofilm. These components are:

#### 11.6.2.1 The carbonic acid/bicarbonate buffer

The bicarbonate in saliva is found as bicarbonate ions. When an acid faces this buffer system, the hydrogen ions ($H^+$) are captured by the bicarbonate ions and are neutralized keeping the pH stable. The hydration of carbon dioxide into carbonic acid, and vice versa, is a reaction catalysed by the enzyme carbonic anhydrase. This system is the most important of the salivary buffer systems. Its concentration increases when the rate of salivary secretion increases. It has been shown that when carbonic anhydrase is removed from saliva its buffer capacity is significantly reduced.

#### 11.6.2.2 Phosphate-phosphoric buffer

It works similarly to the previous one but does not release $CO_2$ in the reaction. Its concentration is lower than the bicarbonate buffer, therefore its total power as a buffer is lower. In addition, this concentration does not increase with increasing salivary flow.

11.6.2.3 Salivary urea

Urea is found in saliva at a concentration of 20 mg/100ml. Its metabolism by plaque microorganisms produces the formation of ammonia, which increases the pH level and neutralizes acids.

11.6.2.4 Salivary proteins

Salivary proteins act as a buffer when the pH of the oral environment is below its isoelectric point. Many proteins have their isoelectric point at a pH between 5 and 9 so when the pH is below 5 these proteins have the ability to capture protons and behave like a good buffer.

In addition, when plaque microorganisms use proteins as nutrients, ammonia is released from their metabolism. This also neutralizes the hydrogen ions in the acids.

11.6.2.5 pH-raising peptide or sialin

It is an arginine peptide. Individuals with active caries have low levels of this peptide compared to caries-resistant groups. Ammonia is also a metabolite of arginine which also reduces the drop in pH.

11.6.3 Antibacterial function

Saliva contains a number of antibacterial substances that regulate the oral ecological system. The antibacterial components of saliva are:

A) Lysozyme. It is not able to cause lysis or stop the growth of bacteria but, in the presence of a detergent found in some toothpastes (lauryl sulphate), can cause lysis in many streptococci. Its role would be to maintain ecological balance.

B) Lactoperoxidase inhibits bacterial growth as it prevents bacteria from taking advantage of some nutrients. It acts in the presence of hydrogen peroxide so it is very active against the lactobacillus as they accumulate peroxide inside.

C) Lactoferrin has a high affinity for iron which is an essential nutrient for bacteria. It competes with them for that nutrient.

D) Phosphoproteins exert detergent action taking part in the dissolution of microbial membranes. They delay the formation of bacterial plaque and hinder a direct bacterial binding on the enamel covered with saliva. They also prevent extracellular polysaccharides from binding to hydroxyapatite. These actions do not last long as the bacterial proteases quickly digest the phosphoproteins and use them as a source of nutrition.

E) Glycoproteins agglutinate microorganisms forming bacterial masses that are swallowed with saliva. Therefore, they help with the bacteria clearance.

F) Immunoglobulins. The main one existing in saliva is IgA and its functions are:

- Anti-inflammatory activity in the mucosa
- Inhibition of bacterial adhesion by:

- blocking of the particles determining adhesion
- reducing the hydrophobicity of bacteria
- agglutinating bacteria

- Inhibition of bacterial enzymes.

IgG and IgM from the crevicular fluid also exist in the oral fluid. They are of serum origin and they are in a lower proportion than IgA. They decrease bacterial adhesion and inhibit bacterial enzymes. They are also involved in bacterial opsonization so that the bacteria can be phagocytosed by polymorphonuclear leukocytes. In opsonization the immunoglobulin binds to the bacterial antigen on one end and to the leukocyte on the other.

G) Complement system comes from the crevicular fluid and it is able to activate polymorphonuclear leukocytes that also come from this fluid. Both, along with immunoglobulins, contribute to the specific defence against microorganisms.

# 12

# CLINICAL MANIFESTATIONS OF DENTAL CARIES. DIAGNOSIS AND PROGNOSIS

12.1 Clinical manifestations of caries.

12.2 Classification of caries.

12.3 Determination of the degree of caries activity.

12.4 Diagnosis of caries.

## Introduction

Caries lesions reflect the evolutionary nature of the disease. The understanding of this evolutionary nature has meant that the concept of the disease, and therefore the classification systems of the lesions, have also changed. In the early 20th century, Black established a classification of caries lesions aimed at describing the preparations needed to place the restorative material used at the time, the silver amalgam. But this classification was quite deficient as it did not mention the extent of caries nor contemplated the initial lesions of demineralization or white spot as it spoke only of cavities, no lesions.

That is why, in 1997, Mount and Hume developed a new classification system called "SiSta" referring to the site and stage of the carious lesion. In 2002, Pits compares this classification with the parable of the iceberg, representing the most initial stages of the injury below the line of sight, passing, in many cases, unnoticed by clinicians, and only being visible the effects of injuries in more developed stages such as cavitated dentine and lesions involving the dentine-pulp complex.

## 12.1 Clinical manifestations of caries

The clinical form in which dental caries manifests depends on the degree of evolution in which it is found. It will progress from the white spots of the enamel to the total deterioration of the hard tissues of the tooth, being time necessary for this evolution. The coincidence of determining factors during long periods of time favour the evolution of the lesions. An incipient lesion takes to become cavitated on a smooth surface between 12 and 24 months.

In the past, non-obvious subclinical and clinical stages were considered as early stages of carious lesion. Currently they are considered to be the onset of this, so we take the Pitts Pyramid 1997 (modified by ICDAS in 2004) as a guide for the evolution of clinical manifestations. Depending on the degree of evolution of the caries lesions we can find different forms of clinical manifestation, which condition our diagnosis, prognosis and treatment.

### 12.1.1 Very incipient disease. Subclinical lesion.

At this stage, microscopic and ultrastructural changes occur in the enamel. To detect this type of injury, advanced technology is needed. It is important to emphasize the patient's medical history and background to take the necessary educational preventive measures.

### 12.1.2 Incipient disease. White spot

It is the first manifestation of caries clinically visible in enamel, but the surface is intact because the demineralization of the enamel is in the sub-surface, with superficial remineralization. This is best distinguished on smooth tooth surfaces and its appearance is accentuated when the tooth dries with air. These incipient carious

lesions are usually reversible as long as treatment is given in a timely manner.

12.1.3 Established disease. Enamel caries

If the white spot is not inactivated, it progresses to an obvious cavity. As the continuity of the enamel is broken, the dental biofilm previously located on its surface migrates to new physical spaces represented initially by the microcavities and then by the clinically visible cavity.

12.1.4 Severe illness. Dentine caries

Dentine lesion is a very complex process that involves the demineralization of the inorganic part and the degradation of the collagen of the decayed dentine by the action of host enzymes. Such enzymes belong to the matrix family - metalloproteinases (MMP) and they are activated with the fall of the pH below its critical point, exerting its maximum action during its subsequent ascent. The cavitation of the enamel is quickly colonized by bacteria and their nutrients, making it difficult to remove and facilitating the progression of the lesion much faster. Dentine permeability is a key factor in determining the pulp response to dental caries. It is worth mentioning that the dentine lesion may be non-cavitated (in which case it will be referred to as an established disease).

12.1.5 Pulp pathology

As the caries lesion progresses in depth and width, the bacteria advance towards the pulp, reacting before the bacteria arrive. In medium, deep caries lesions and/or pulp exposures (although the clinical distinction between exposed and unexposed pulp tissue may be questionable) inflammatory processes may occur whose degree confers the characteristics of reversibility or irreversibility.

**12.2 Classification of caries**

According to the point of view from which they are observed, there are different classifications of caries, being more useful those that help us establish the treatment plan based on the urgency to repair the lesion, as they allow us to prioritize the repair of active against arrested caries.

12.2.1 According to the location

- Caries of pits, furrows and fissures are coronary caries. They begin inside these anatomical structures and at first it is difficult the differential diagnosis with pigmentation. It can be translucent through the enamel with a greyish colour that is a sign of caries in dentine.

- Caries of smooth enamel surfaces. They can be in proximal surfaces or in vestibular or lingual surfaces, generally below the line of maximum contour.

- Root caries appears in exposed roots. They are frequent in interproximal surfaces, in the gingival area of badly adapted previous fillings.

12.2.2 According to the degree of activity.

It indicates the speed of evolution:

- Active. They are most caries. They can evolve slowly, quickly or stop.

- Arrested. They are frequent in adults. They can be seen as hyperpigmented hard tissue, bright and smooth.

- Rampant. They are rapidly evolving and simultaneously affect several teeth.

12.2.3 According to the stage of development

- White spot. It is the first clinical manifestation of a caries lesion. It is due to sub-superficial demineralization of the enamel. Drying with air, it dehydrates the pores of the enamel and the lesion is more easily observed.

- Dentine is affected but not cavitated. Dentine caries with intact enamel.

- Cavitated lesion. The enamel has collapsed. They are always active lesions and their presence is indicative of caries in the future.

- Lesion with pulp exposure. They are very advanced lesions. In case of remaining vital pulp there may be pulp polyps, although it is more frequent that the pulp became previously necrotic.

- Lesion with destruction of the crown. When there is practically no clinical crown left, in some cases the pulp vitality may persist, the pulp retracts and the pulp chamber is occupied by tertiary dentine.

12.2.4 According to the manner of appearance

- Primary caries. It appears in healthy tissue. It is the initial attack on the tooth surface and it is not adjacent to previous restorations.

- Secondary or recurrent caries appears around the margins and under previous restorations.

- Residual caries is not eliminated caries remaining under a restoration.

### 12.2.5 According to the location and size

#### 12.2.5.1 Black system

The Black (GV Black) system was proposed as a classification of cavities for amalgam restoration, dividing caries into five classes based on the surfaces they affect. The mesial and distal surfaces are considered proximal, as they are adjacent to the interproximal areas of the tooth. Class I comprises pits and fissures of all teeth, as well as two occlusal thirds of the teeth, except the proximal surfaces. Classes II, III and IV comprise proximal surfaces and class V are the smooth surfaces of the gingival third.

- Class I: Caries of the occlusal surface of the molars and premolars, lingual of incisors, buccal of lower molars and palatine of upper molars.

- Class II: Caries on the proximal surface of the premolars and molars.

- Class III: Caries on the proximal surface of anterior teeth.

- Class IV: Caries on the proximal surface of anterior teeth that includes loss of angle.

- Class V: Caries lesions found in the gingival third of the vestibular and lingual faces of all teeth.

- Class VI: Occasionally used to describe caries located in the occlusal cusps tips and incisal edges in anterior teeth.

Although it is still used, it has important limitations. It is a classification of cavities and not of lesions and it does not give information on the size of the lesion or on the character of its evolution.

#### 12.2.5.2 Mount and Hume system

Mount and Hume (1997) proposed a system for the classification of caries and not cavities, linking location, size and susceptibility. It recognizes three locations: pits and fissures, contact areas, and cervical areas. And it considers the size of the lesion as: initial lesion, with the possibility of professional remineralization; caries lesion beyond remineralization; cusps undermined by caries or possible cusp fracture due to caries; loss of the cusp or incisal edge.
The classification of caries meets both criteria by constructing a compound index, which was revised including the category of no cavity expressed with zero (Mount et al., 2006; Chalmers, 2006)

| Sites of caries susceptibility: | |
|---|---|
| Site 1 | Caries lesions in pits and fissures, occlusal pits and vestibular and lingual grooves of all teeth. All surfaces except proximal |
| Site 2 | Caries lesions on the proximal surfaces of all teeth |
| Site 3 | Caries lesions of the cervical surfaces of all teeth, whether coronal or root surface |

Table 4. Sites of caries susceptibility.

| Stages of caries progression | |
|---|---|
| Stage 0 | Active injury without cavitation. Restorative treatment is not required |
| Stage 1 | Injuries with alteration to the tooth enamel. Minimally invasive restorative treatment is required |
| Stage 2 | Moderate lesions with localized cavitation, has progressed inside the dentine without producing cusp weakening. Restorative treatment is required |
| Stage 3 | Advanced lesions with cavitation that has progressed in dentine causing weakening of the cusps. Restorative treatment is required |
| Stage 4 | Advanced cavitation lesions that have progressed and destroyed one or more cusps. Restorative treatment is required |

Table 5. Stages of caries progression.

| Classification of lesions on dental surfaces | | | | | |
|---|---|---|---|---|---|
| | Stage 0<br>No cavity | Stage 1<br>Minimum | Stage 2<br>Moderate | Stage 3<br>Great | Stage 4<br>Extensive |
| Site 1 Pits and fis. | 1.0 | 1.1 | 1.2 | 1.3 | 1.4 |
| Site 2 Proximal | 2.0 | 2.1 | 2.2 | 2.3 | 2.4 |
| Site 3 Cervical | 3.0 | 3.1 | 3.2 | 3.3 | 3.4 |

Table 6. Classification of lesions on dental surfaces.

This classification allows to establish treatment protocols based on the stage of caries progression:

- Stage 0: remineralization techniques.
- Stage 1: minimally invasive restorative treatment.
- Stage 2,3 and 4: complex restorative treatment

12.2.5.3 ICDAS system

ICDAS II is an international caries detection and diagnosis system, agreed in Baltimore, Maryland. USA in 2005, applicable to clinical practice, research and development of public health programs. The aim was to develop a visual method for the detection of caries, as early as possible, and also to detect its severity and its level of activity.

The nomenclature comprises two digits, the first from 0 to 8 corresponds to the "Restoration and sealant code", the number 9 corresponds to the "Missing tooth code"; and the second digit from 0 to 6 corresponds to the "Code of enamel and dentine caries"

First digit ICDAS II:

0: Not restored or sealed

1: Partial sealant

2: Complete sealant

3: Tooth coloured restoration

4: Amalgam coloured restoration

5: Steel crown

6: Crown or veneer in porcelain, metal-porcelain or gold

7: Lost or fractured restoration

8: Provisional restoration

90: Implant placed due to tooth loss due to other causes

91: Implant placed for tooth loss due to caries (90 and 91 were replaced by the letter "P" when using the ICDAS statistical software)

92: Pontic made for tooth loss due to other causes

93: Pontic made for tooth loss due to caries

96: Surface of teeth that cannot be examined. Surface excluded

97: Absent tooth, extracted by caries

98: Tooth missing for other reasons

99: Not erupted

Second digit ICDAS II

0: Absence of caries

1: Initial visual change in enamel. It is only possible to see it after drying for 5 sec or it is restricted to the boundaries of the pit or the fissure

2: Visual change in non-dried enamel

3: Locally fractured enamel, without dentine involvement

4: Dark shadow of the dentine below the enamel. Solution of continuity in the enamel when observed from the vestibular o palatine. Gray, blue or brown shade, discontinuity of the enamel surface without exposure of dentine. It is easier to see wet.

5: Visible cavitation in dentine that reaches less than half of its thickness.

6: Extensive cavity in dentine that affects half or more of its thickness. It can affect the pulp.

**12.3 Determination of the degree of caries activity**

| ACTIVITY OF A CARIES LESION | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | ICDAS CODE | | | | | | PLAQUE PRESENCE | | PROBING | |
| VARIABLES | 1 | 2 | 3 | 4 | 5 | 6 | NO | SI | SMOOTH HARD | ROUGH SOFT |
| POINTS | 1 | 3 | 4 | 4 | 4 | 4 | 1 | 3 | 2 | 4 |

Table 7. Determination of the degree of caries activity

Arrested caries: scores from 4 to 7.

Active caries: scores from 8 to 11.

In the first box of the variables, you will find the ICDAS codes from 1 to 6. Below there is a score that correlates with the ICDAS codes indicating severity. For example, if an ICDAS code 3 is diagnosed on the dental surface, a severity score of 4 corresponds. If the ICDAS code is 1 it corresponds to a score of 1 in severity.

In the second box of the variables, if there is bacterial plaque present in the lesion observed by the passage of a probe the corresponding score is 3. If no plaque is observed the score would be 1. And in the third box of variables, if probing softly the lesion is smooth and hard the corresponding score is 2 in severity. But if there is roughness and in turn it feels soft the score would be a 4.

We determine a cut-off value of 8. If it is <8, it is an arrested caries. If it is ≥ to 8, it is an active caries

**12.4 Diagnosis of caries**

The diagnosis of caries must be made globally as caries is a multifactorial disease. This will allow us to adapt the treatment in each patient based on the prognosis of the risk of caries and the evolution of existing lesions. Therefore, the diagnosis should include visual examination, radiological examination and individual risk assessment. Diagnosis should be as early as possible to prevent major tooth destruction.

The visual examination is the most used for its low cost and ease of execution. It must be performed in an organized manner, including all surfaces of all teeth. For this we will previously remove the remnants of plaque and food with our probe and dry by quadrants. The inspection should be performed only with a mirror and triple syringe. We dry the surfaces with air and evaluate the changes in the translucency of the enamel, loss of gloss, pigmentation and presence of soft tissue or changes in the texture of the enamel. The sharp-tip probe should not be used for the initial diagnosis of caries, as by applying pressure to a superficial lesion we can cavitate it, which would turn a white spot into a cavitated lesion.

Exploring pits, furrows and fissures we must keep in mind that the white spot occurs on the sides of the fissure and it is not seen directly. When drying, the matte enamel is observed. These are lesions that are difficult to diagnose in their initial stage.

In the exploration of interproximal surfaces, visual inspection has little value, it requires a radiograph. Although sometimes and depending on the degree of evolution, they can be detected by transillumination or with the help of dental floss.

In free vestibular or palatal faces, diagnosis is easier due to its easy visual inspection. They are usually opaque and oval white lesions in the mesiodistal direction below the area of maximum contour of the tooth because this is an area with less autolysis.

Root lesions are usually dark brown with a softened surface. They are more common in the margins of previous restorations and fixed prostheses and also, in the interproximal surface when the contact point has been lost.

| Black classification | |
|---|---|
| Class I | Caries of the occlusal surface of the molars and premolars. |
| Class II | Caries on the proximal surface of the premolars and molars |
| Class III | Caries on the proximal surface of anterior teeth. |
| Class IV | Caries on the proximal surface of anterior teeth that includes loss of angle. |
| Class V | Caries on the gingival third of the vestibular and lingual surfaces of all teeth. |
| Class VI | Occasionally used to describe caries located in the occlusal cusp tips and incisal edges in anterior teeth. |

| Classification of Mount and Hume | |
|---|---|
| <u>Sites of caries susceptibility</u> | |
| Site 1 | Caries lesions in pits and fissures (occlusal pits and vestibular and lingual grooves of all teeth and other structural defects) |
| Site 2 | Caries lesions on the proximal surfaces of all teeth |
| Site 3 | Caries lesions on the cervical surfaces of all teeth, whether coronal or root surface |
| <u>Stages of caries progression</u> | |

| | |
|---|---|
| Stage 0 | Active lesion without cavitation. Restorative treatment is not required |
| Stage 1 | Lesion with alterations in the enamel. Restorative treatment is required |
| Stage 2 | Moderate lesion with localized cavitation. Caries has progressed inside the dentine without producing cusp weakening. Restorative treatment is required |
| Stage 3 | Advanced lesion with cavitation that has progressed into dentine causing weakening of the cusps. Restorative treatment is required |
| Stage 4 | Advanced cavitated lesion that has progressed and destroyed one or more cusps. Restorative treatment is required |

| ICDAS Classification II | |
|---|---|
| Restoration code and sealant (first digit) | |
| 0 | Not restored or sealed |
| 1 | Partial sealant |
| 2 | Complete sealant |
| 3 | Tooth coloured restoration |
| 4 | Amalgam coloured restoration |
| 5 | Steel crown |
| 6 | Crown or veneer in porcelain, metal-porcelain or gold |
| 7 | Lost or fractured restoration |

| | |
|---|---|
| 8 | Provisional restoration |
| 96 | Surface of teeth that cannot be examined. Excluded surfaces |
| 97 | Absent tooth, extracted by caries |
| 98 | Tooth missing for other reasons |
| 99 | Not erupted |
| Enamel and dentine caries code | |
| 0 | Absence of caries |
| 1 | Initial visual change in enamel. It is only possible to see it after prolonged drying of the tooth (5 sec.) Or restricted to the confines of the pit or fissure |
| 2 | Visual change in non-dried enamel |
| 3 | Locally fractured enamel, without dentine involvement |
| 4 | Dark dentine shadow below the enamel. Solution of continuity in the enamel when observed from vestibular or palatal. Gray, blue or brown shade, discontinuity of the enamel without exposure of dentine. It can be seen better when wet |
| 5 | Visible cavitation in dentine that reaches less than half of its thickness |
| 6 | Extensive cavity in dentine that affects half or more of its thickness. It can affect pulp |

# 13

# RADIOLOGICAL DIAGNOSIS OF CARIES AND OTHER DIAGNOSTIC PROCEDURES

13.1 Radiological diagnosis.
13.2 Conical beam computed tomography. CBCT
13.3 Transillumination.
13.4 Electrical tests.
13.5 Laser fluorescence.
13.6 Comparison between diagnostic tests.

## Introduction

It is important to know that the demineralization of caries allows the passage of x-rays impressing the film, i.e. the loss of mineral material in the tooth´s structure makes it more radiolucent (black image, the material is allowed to pass through the radiant energy). Radiation photons can penetrate the tooth´s structure and impress the radiographic film. This is the fundamental concept of radiology.

The most important drawback of radiology for the diagnosis of caries is that for this to occur demineralization must exceed 40%. Therefore, in initial lesions in which the loss of mineral material does not exceed this figure, it can give us images of the absence of caries or injuries of less extent than those that we will actually find when treating them. "The radiographic image is always one step behind the actual injury"

There are different radiological alternatives that vary depending on the type of pathology we want to observe. That is, if we want to assess an injury by an abscess, we will not make the same projection as if we want to make a general assess of caries in a whole quadrant. We always have to take into account the possibility of radiographic artifacts (any optical density present in an X-ray not caused by the interposition of the anatomical structures of interest).

## 13.1 Radiological diagnosis

### 13.1.1 Advantages of radiological diagnosis

- In many occasions, it allows the diagnosis of interproximal caries before cavitation occurs. The interproximal surfaces are a difficult area to explore, since in most cases we do not have visual access to them, and they only become evident when the cavitation of the enamel takes place.

- The reaction of the dentine-pulp complex to the progress of caries can be observed. In a large part of the radiographic plates where we see lesions due to caries of moderate/severe intensity, we can find areas of sclerotic dentine, and retractions of the pulp horns due to the formation of tertiary or repair dentine. Similarly, at the periapical level, we can observe the presence of infections and necrosis processes, manifesting as radiolucent images in the periapical area.

- Assess the progress of caries and its proximity to the pulp. As we have already mentioned, the radiological diagnosis is always one step behind. But even so, it is of vital importance for the planning of treatments. It allows us to calculate more or less the extension of the injury and its proximity to the dental pulp. We are also able to study, evaluate and prevent possible complications that may arise during the treatment.

- Diagnosis of periapical pathology. The clinical diagnosis and tests of pulp vitality, sometimes do not allow us to verify a periapical pathology in a direct

way. However, in most cases, the presence of a rounded radiolucent image confirms the diagnosis. It will be necessary to perform periapical projections.

- Diagnosis of recurrent caries. When there are previous restorations, whether of composite or silver amalgam, the exploration of the dentine surfaces on which they are based is very complicated. Being able to assess them through an X-ray plate facilitates the diagnosis, being able to observe lesions "hidden under these restorations" or to be able to dismiss the presence of the same.

- It allows us to verify the gingival adaptation of our restorations. When we make fillings with subgingival preparation margins, it is very difficult to visually observe that we have done a good job. Making perfect adaptations helps us prevent future complications of treatment such as gingival irritations, food packaging and the appearance of secondary caries. Being able to have a simple tool that can corroborate that our work is correct, is quite useful in daily clinical practice.

13.1.2 Disadvantages of radiological diagnosis

- It needs at least 40% demineralization. This is the main drawback of the radiographic diagnosis. Caries injury produces progressive demineralizations, which do not always affect more than 40% of mineral tissue. In many cases we can believe that there is no injury or that its extent is smaller than it really is. This is why we must build, not only on radiological tests, but on a correct clinical examination and rely on additional tests such as vitality tests, transillumination, etc.

- The lesion is not real. Only the most demineralized areas are seen on the x-ray and the lesion is always larger than it appears on the x-ray. "X-rays are always one step behind caries injury."

13.1.3 Conventional radiographs

The most used for the detection of caries is the bitewing radiograph. With a single plate, we have information on a large group of teeth. It allows us, with lower doses of radiation obtain information on the structure of the tooth, adjacent parts, periodontal situation, etc. When caries lesions are extensive enough to suspect that pulpal pathology exists, this projection will not be useful to us, as it does not allow us to see the apical third of the root, nor the surface of alveolar bone adjacent to it. It is therefore that in the face with a suspicion of pulpal pathology processes, we must resort to periapical projections. Although these projections are focused on a smaller group of teeth and always with a single quadrant, we will get enough information to make a correct diagnosis and verify the progress in the treatment of these injuries.

The radiographic image in dental caries can often be seen as a lesion in the enamel and yet histologically there is an involvement of dentinal tissue. In moderate caries, it usually shows a triangular lesion that affects more than the outer half of the enamel with the base towards the surface. While in advanced caries, it is observed that the

radiolucent image extends laterally when reaching the dentinoenamel limit. The lesion in the dentine has a triangular shape whose base is at the dentinoenamel boundary and the apex towards the pulp chamber. In more severe cariogenic lesions, we can observe the formation of tertiary dentine that forms in the pulpal wall, adjacent to the advance of caries and as a consequence the loss of the pulpal horn closest to the lesion. In these cases where the progression of the lesion has been more severe, and if the clinical examination and vitality test makes us suspect there is nerve involvement, we should do these periapical projections to verify our suspicion.

Conventional radiography has a number of shortcomings, which we must know, so as not to fall into misinterpretations and make inaccurate decisions based on them:

A) Occlusal surfaces

- Initial lesions of occlusal caries are not seen on x-ray.

- In moderate occlusal lesions affecting the dentine, a wide radiolucent area is observed in the dentine without the enamel generally being affected. It can be interpreted that there is no apparent lesion if the radiolucency is not completely manifest.

- Sometimes a more radiopaque area between caries and the pulp is observed. This represents the sclerotic dentine that occurs as a reaction to the progress of caries. In these cases, we must check that despite the formation of this dentine the pulpal vitality remains intact.

B) Interproximal surfaces

- Sometimes there is a more radiolucent area in the cervical interproximal notch. This image is projected on the radiograph and can be confused with caries. Once again, we insist on the vital importance of the correct and exhaustive clinical diagnosis, so as not to fall into the performance of treatments on non-existent injuries.

C) Smooth surfaces

- Caries, on smooth surfaces, usually manifests as elliptical shapes in the mesiodistal direction.

- In buccal and lingual caries, it is difficult to diagnose with a radiograph. The superposition of the rest of the dental tissues on the radiographic plate may show apparent normality due to the absorption of photons by the mineral tissues before or after the injury.

- Differential diagnosis should be made with abrasions and abfractions, as they also produce a loss of material similar to the caries of smooth surfaces and radiographically the image is quite similar. The scan with an explorer to check the hardness of the lesion is crucial for this diagnosis.

D) Root surfaces

- Caries at the root level are often associated with gingival recessions. Radiographically the interproximal caries are the ones we see best, although they are not the most common, as there is less overlap of healthy dental tissue. There may be no involvement of the enamel until the lesion is already very advanced, as the progression of root caries spreads more rapidly through the dentine.

- Requires a differential diagnosis with root resorption.

E) Previously restored surfaces

- They are often hidden in the projection by the generally more radiopaque restoration material. It is more evident in metal restorations and coating crowns.

13.1.4 Digital radiographs.

The big difference with respect to conventional radiography is that we obtain digital images instead of physical radiographic plates with a considerable reduction in radiation for the same type of plates. They currently have an image quality similar to conventional radiography.

It does not need a developing process, since the processing of the image is computerized and therefore the waiting times are reduced. At clinical level this supposes a greater optimization of resources and in the dental procedures, it reduces the working times.

Being a digital system, sharing images with the patient and other professionals is a much simpler task, as the image being digitized can be duplicated and forwarded without any complications. Likewise, the computer file allows the preservation of the image not to lose quality over time.

With suitable software, it will be possible to carry out estimations of different measurements, very useful in endodontics and to realize modifications of a series of parameters of the x-ray, like for example the colour, contrast etc.

There are two types of digital radiography, which differ fundamentally in the way the image reaches the computer receiver. Addressing this issue, we have:

- Direct or RadioVisioGraphy (RVG): Uses an electronic receiver or sensor that captures the energy of X-ray photons and converts them into electronic signals that it transmits to a computer. The image arrives at the computer through a physical cable of connection type USB, or through systems without cable (Wireless) Bluetooth, WIFI, etc.

- Indirect: replaces the film with a plate covered with phosphor crystals (PSP, photostimulable phosphorus) that stores X-ray photons for a period of time. You need a scanner that converts the energy captured by the sensor into digital images and transfers them to the computer.

### 13.2 Conical beam computed tomography (CBCT)

It offers us a three-dimensional vision. There are some oral treatments, in which a two-dimensional vision, does not provide us with the necessary information to be able to make diagnoses or treatments in the correct way. To this end, the system that offers the tomography provides us with that third dimension we need.

Today, it has a lower resolution image imaging than conventional radiography, but just as digital radiograph systems were able to refine the sharpness and accuracy of their images, we must expect the same to happen with these CBCT systems.

Exposure times to radiation are greater than a conventional digital x-ray. Therefore, we must be responsible in their use and perform this type of complementary examinations as long as the benefit we hope to obtain is greater than the risk of exposure suffered by the patient.

It will be very useful in endodontics, both for the diagnosis of periapical pathology and for the planning of treatments.

### 13.3 Transillumination

Caries alters the optical properties of teeth. Demineralization of enamel alters its prisms and tissue structure (loses its translucency). When light strikes the tooth, the healthy tissue looks translucent and clear and the area of caries, darker and opaque. This effect can be assessed with the light of the dental chair reflected on the intraoral mirror or with other types of external light sources such as fibre-optic transillumination (FOTI). Daves (2001) rated the sensitivity and specificity of fibre-optic transillumination as superior to that of proximal caries radiographs. Derived from this type of technique, similar ones emerge, but with digital registration with the advantage of being able to store data and make comparisons of the same area at two different time points.

It is especially indicated in interproximal caries of anterior teeth as it sometimes allows to detect the lesions before the enamel collapses and before its vision in x-ray. Obtaining the benefit of not having to irradiate the patient, and with the certainty that transillumination does not represent any risk for the patient.

As a drawback, in the diagnosis of caries in the subgingival areas, it is not very useful.

### 13.4 Electrical tests

These systems have been in existence for more than half a century but have not had a special diffusion among clinicians. It is based on the fact that healthy enamel is a bad conductor, however, decayed tissue becomes porous and gaps are filled with saliva improving conductivity.
The mechanism of action of this system is based on two electrodes, one in the area to be diagnosed and another one on the cheek to detect the decrease in electrical resistance. It is quite useful in occlusal caries hidden in the bottoms of grooves and fissures.

### 13.5 Laser fluorescence

They emerged in the late twentieth century. Its mechanism of action is based on the fact that, when applying a laser beam on the decayed tissue, fluorescence of greater intensity is generated as the caries lesion is more advanced. The mechanism of why fluorescence increases when tissue is affected by caries is not well understood. Some studies attribute it to the fact that it is more due to the incorporation of bacterial metabolites into the lesion than to the actual disestablishment of hydroxyapatite crystals.
The best-known systems are Diagnodent ® and Diagnodent pen ® from Kavo which emit a laser beam with a wavelength of 655 nm. The system captures the wavelength and quantifies it in values ranging from enamel incipient, surface dentine and deep dentine. The main problem is that it does not distinguish between active injuries and those that are chronic or stopped.

### 13.6 Comparison between diagnostic tests

Here is a summary table of the complementary diagnostic tests and their usefulness for the different types of caries according to the surface where they are found.

| | INTERPROXIMAL SURFACE | SMOOTH SURFACE facial/ lingual | CRACKS/ GROOVES | ROOT SURFACE |
|---|---|---|---|---|
| INSPECTION | - | ++ | + | + |
| EXPLORER | - | - | - | ++ |
| PROBE | + | - | - | - |
| LASER | + | + | ++ | + |
| ELECTRICAL CONDUCTANCE | - | + | ++ | + |
| X-RAY | ++ | - | ++ | + |

Not useful: - Useful: + Choice method: ++
Table 8. Comparison table between different diagnostic tests

# PART IV

## DENTAL PATHOLOGY WITH A TRAUMATIC ORIGIN

# 14

# GENERALITIES OF DENTOALVEOLAR TRAUMA

14.1 Epidemiology.
14.2 Prevention and correction of predisposing factors.
14.3 Aetiology.
14.4 Pathogenesis.
14.5 Classification of traumatic tooth injuries.

## Introduction

Dental traumatology is the branch of dentistry that deals with the prevention, diagnosis, treatment and prognosis of injuries that affect the teeth, maxilla / mandible and soft tissues of the stomatognathic apparatus, as well as the possible sequelae that may result from these misfortunes.

Numerous studies show that the percentage of patients who come to our clinics after suffering dental trauma is increasing. In most cases, they are the result of a fortuitous and unexpected action that requires emergency treatment. The time-lapse between the incident and until the patient is treated by the dentist becomes a key factor. Reception staff must be properly trained to interrogate the patient or their interlocutor, primarily parents by telephone, and be able to identify when an accident is listed as red code and requires priority treatment. They must give relevant instructions and facilitate their urgent care even if that means changing patient planning on the agenda.

Given the importance of the clinical case to be treated, and in order to avoid or greatly minimize the occurrence of both aesthetic and functional sequelae, it is essential that the dentist remains up to date on this type of treatment. Otherwise, the dentist should choose to refer the patient to any partner who is accustomed to handling this type of clinical situation. Precisely the largest number of post-traumatic complications are due to inadequate dental care. There are several articles that corroborate the lack of information and assimilation of basic concepts for the management of these situations, both by the clinical team (dentists, hygienists and support staff), as for patients and their immediate environment (parents, coaches, activity monitors and teachers).

## 14.1 Epidemiology

The first peak of incidence occurs in the temporary dentition. It corresponds to an age in which subjects have less motor coordination when crawling, walking, running, going up and downstairs. The second occurs in permanent dentition with the development of competitive sports activities and with a higher risk of trauma, also coinciding with the first autonomous journeys by bicycle, moped and other means of transport. We can find the following epidemiological variables:

### 14.1.1 Gender

At an early age, male patients present dentoalveolar trauma more frequently than female patients, in an approximate ratio of M:F = 3:2. This could be due to the difference in the use of protective elements between both genders. However, from adolescence and especially in adulthood this difference becomes less significant, practically equalling the incidence percentages.

### 14.1.2 Location

Dentoalveolar trauma most often affects the upper arch, especially the anterior sector, and in particular the upper central incisors. The upper lateral incisors are also usually affected but to a lesser extent than the central ones. Except for those patients with class III malocclusion and/or overbite, this percentage of involvement of the dental elements could vary, also involving the lower incisors and premolars or molars, especially in cases of indirect impacts on the jaw or at older ages.

14.1.3 Malocclusion

- Dolichofacial profile.
- Mouth breathing.
- Molar and canine Class II.
- Overjet.
- Lip incompetence.
- Open bite.
- Overbite.

14.1.4 Sport practised

Especially in contact sports without the use of intraoral and extraoral protection elements.

14.1.5 Pathologies with dental origin

- Caries.
- Molar incisor hypomineralization (MIH)
- Amelogenesis imperfecta.
- Dentinogenesis imperfecta.
- Fluorosis.

14.1.6 Previous dental treatments

- Old, fractured or filtered restorations.
- Devitalized teeth with root canal treatment.
- Patients with orthodontic appliances.

14.1.7 People with different pathologies are more likely to fall down.

- Fainting.
- Dehydration.
- Myocardial infarction.
- Epilepsy.
- Stroke.

14.1.8 Other factors

- Time of year.
- Alcoholism and drug addiction.
- Socio-economic level.

## 14.2 Prevention and correction of predisposing factors

As the most common pathology is maxillary prognathism or skeletal class II, the appropriate type of prevention would be orthodontic treatment in the developmental stage prior to the peak incidence of trauma. Another type of prevention would be the use of mouthguards when exercising activities that predispose to the crown-root injury. There are numerous types of mouthguards tailored to the needs of each patient.

Correcting bad habits during dentofacial development is also an important preventative factor. For example, removing the pacifier before the age of three, avoiding thumb-sucking, controlling the use of the bottle, etc.… This prevents the appearance of the malocclusions discussed above.

## 14.3 Aetiology.

- Domestic accidents.
- Traffic accidents.
- Sports events.
- Fights.
- Abuse.
- Intubation during general anaesthesia in surgical interventions.
- Conscious and inappropriate use of teeth as a tool to open or hold objects.
- Additional elements present in the soft tissues of the mouth (i.e. piercings).

14.3.1 Etiological variables

14.3.1.1 The intensity of traumatic force

The higher the rate of impact on the crown-root structures, the greater the likelihood of injuries affecting the hard tissues (crown fractures). On the other hand, if the speed is slower, soft and periodontal tissue injuries (luxation) will be relatively more likely to occur.

14.3.1.2 The direction of traumatic force

It is important to determine the extent and trajectory of the fracture line with which the trauma manifests, both at the level of the crowns and the roots (horizontal or vertical). It can cause an intrusive, extrusive luxating movement or the complete exit of the tooth from its alveolus (avulsion).

14.3.1.3 The shape of the object

The smaller the object, the smaller the area of application of the force and, therefore, the greater the entity of the same per unit area. It will increase the probability of fracture to the detriment of dislocation phenomena.

14.3.1.4 Resilience or deformation capacity of the object

The severity of the injuries appears to be directly proportional to the modulus of elasticity of the impacting object. If this object is rather elastic, dislocations or fractures of lesser severity will be more likely to occur.

## 14.4 Pathogenesis

Depending on how the trauma occurs and its consequences, it can condition the type of treatment and its prognosis.

14.4.1 Intrinsic factors

- Infractions.
- Abfractions.
- Restorations.
- Devitalization / Duct treatment (endodontics).
- Abrasions.

14.4.2 Extrinsic factors

- Intensity.
- The direction of impact.
- Force of impact.
- Resilience.
- The shape of the object.

## 14.5 Classification of traumatic tooth injuries

Traumatic dental injuries have been variously studied. There are currently more than fifty different classifications depending on their author. According to a recent literature review, the Andreasen Classification is often the most widely used internationally.

### 14.5.1 Andreasen classification

A) Crown fractures:

a) Crown infraction. Exclusively enamel fissure and fracture.
b) Uncomplicated crown fracture (enamel and dentine without pulp exposure).
c) Complicated crown fracture (enamel and dentine with pulp exposure).

B) Crown-root fractures:

**a)** Complicated crown root fracture (with pulp exposure).
**b)** Uncomplicated crown root fracture (no pulp exposure).
**c)** Root fractures.

C) Injuries to the periodontal tissues:

**a)** Concussion.
**b)** Subluxation.
**c)** Intrusive luxation (central dislocation)
**d)** Extrusive luxation (peripheral dislocation or partial avulsion).
**e)** Lateral luxation.
**f)** Avulsion.

D) Injuries of the supporting bone:

**a)** Comminution of the alveolar socket.
**b)** Fracture of the alveolar socket wall.
**c)** Fracture of the alveolar process.
**d)** Fracture of the mandible and maxilla.

E) Injuries to gingiva or oral mucosa:

**a)** Laceration.
**b)** Contusion.
**c)** Abrasion.

# 15

# DENTOALVEOLAR FRACTURES IN PERMANENT TEETH

## Introduction

Dentoalveolar traumas in permanent dentition are a fairly high percentage of emergency dental care. The most common types are those that affect the crown and supporting tissue, causing the rupture and/or displacement of the tooth. It is essential to establish an accurate diagnosis, an appropriate treatment plan and to perform follow-up visits in order to monitor the evolution of the affected teeth.
The International Association of Dental Traumatology (IADT) is composed of a group of experts in the field who, periodically, publish protocols to be followed for each of the traumatic entities. They are the result of a review of the most current literature, combined with consensus meetings in those cases where the literature does not provide conclusive data on how to proceed in certain situations.
However, since a good number of cases occur in children and adolescents, it is essential to maintain the vitality of the pulp, especially in the immature permanent teeth to ensure proper root development. Otherwise, the consequences of trauma can be lifelong. The immature permanent tooth has a great ability to heal in cases of fracture with pulp exposure, luxation and root fracture. Therefore, techniques are used to promote revascularization and regeneration of the pulp cells, even in cases of incipient necrosis.

## 15.1 Diagnosis. Generalities.

### 15.1.1 Clinical examination

It is a priority to perform a complete clinical evaluation before performing any treatment. First, an oral history form will be completed in relation to where, when and how the trauma occurred.

#### 15.1.1.1 Extraoral examination

It consists of the observation of the lateral, frontal and superior profiles of the face. We will assess the possible presence of edema, bruises, asymmetries and areas of sinking caused by fractures. We must not forget the inspection of the mandibular angles and the zygomatic-maxillary processes.

#### 15.1.1.2 Intraoral examination

Soft tissues: we will check the integrity of the gum and other mucosa in order to locate lacerations, swellings and haemorrhages that could indicate concealed maxillary fractures.

Hard tissues: using the handle of the mirror or a probe we will analyse the integrity of the crowns, we will assess their position, mobility (both vertical and horizontal) and, through palpation of the alveolar process, we will check the presence or absence of bone fractures.

### 15.1.2 Complementary tests

#### 15.1.2.1 Radiographs

It is essential to take x-rays of traumatized teeth, adjacent and antagonistic teeth. The radiographic examination provides information regarding the state of root development of the traumatized teeth, something that undoubtedly determines the type of treatment

to be performed. In many cases, an x-ray of the lip and cheek is also indicated to rule out the inclusion of dental fragments or foreign bodies.
It is recommended to use different projections and angles, being the clinician the one that must decide the type of x-ray that needs for each particular case. In all cases, we will perform the following intraoral radiographs with the help of a positioner:

- Periapical with front projection at 90º angle.
- Periapical with mesial and distal angles.
- Occlusal.

Extraoral radiographs are very useful to rule out mandibular fractures:

- Orthopantomography (OPG)
- Conical beam computed tomography (CBCT). Its availability is limited and it is not considered a routine test but, undoubtedly, it provides a better visualization, especially in cases of:
    - Root fractures.
    - Lateral luxation.
    - Healing monitoring.
    - Addressing complications.

15.1.2.2 Photographs

We must take pictures both before and after treatment. They serve as documentation of the case (also to critically analyse our work in order to improve in future treatments) and they are legal evidence in the assessment of the consequences of the trauma by insurance companies. In these cases, it is always better to have as many photographs as possible.

- Extraoral: Face. Front, profile and profile angled (45º left and right). Both at rest and smiling.
- Intraoral: Frontal (in occlusion and dissoclude), lateral right and left in occlusion (with the help of a mirror) and occlusal (also with a mirror).

15.1.2.3 Vitality tests

The pulp response is analysed by applying temperature (cold test) or electrical stimuli (pulpometer or vitalometer). It is important to remember that after a trauma the tooth is “in shock” and it can remain that way for several days or weeks so it is advisable to repeat these tests in subsequent visits.

## 15.2 Treatment. Generalities.

### 15.2.1 Splinting.

The current literature advises the use of flexible splints, especially in cases of luxations, avulsions and root fractures, as they provide the patient with adequate aesthetics and function. However, there is no consensus on the specific type of splint and the time that should be placed in cases of fractures and luxations as the scientific literature does not provide truly significant data on the best or fastest healing of the affected elements.

### 15.2.2 Antibiotic therapy.

Scientific evidence is limited with respect to the use of systemic antibiotics for the management of luxations. In fractures, there is no evidence that justifies their use, although the dentist may consider that the administration of antibiotics is necessary to treat other injuries associated with the trauma, as well as possible complications arising from it.

## 15.3 Enamel infraction.

### 15.3.1 Clinical findings

It is an incomplete fracture of the enamel without loss of dental structure. In many cases they go unnoticed and the patient does not seek treatment. However, they can mask a root dislocation or fracture.

### 15.3.2 Radiographic findings

Radiographically there are no significant findings. It is recommended at least one periapical, in case there are no other associated signs.

### 15.3.3 Treatment

In most cases they do not usually require treatment. Depending on their size, they may require a small polishing of the incisal edge or, to a greater degree, engraving of the enamel with orthophosphoric acid, application of adhesive and restitution with composite resin in order to prevent discoloration of the fractured incisal edge.

### 15.3.4 Control

They usually do not require follow-up visits unless it is to check other associated injuries.

### 15.3.5 Evolution

Evolution can be positive and asymptomatic. There is pulp vitality and normal root development in cases of immature teeth. However, there are also cases where the tooth becomes symptomatic, it does not respond to vitality tests, radiographically it shows apical injury and/or root development in immature teeth is not performed properly. In these situations, it would be necessary to perform endodontic treatment.

## 15.4 Enamel fracture

15.4.1 Clinical findings

It is a fracture that affects the enamel, with loss of tooth structure, but without pulp involvement. It is not normally tender to percussion or mobile and pulp vitality tests are positive.

15.4.2 Radiographic findings

It is recommended to take periapical radiographs (frontal and with mesial and distal angles) and an occlusal radiograph in order to rule out luxation and/or root fracture. Remember that if the tooth fragment has not been located, it is necessary to x-ray the lip and/or the cheeks to rule out their impact on the soft tissue.

15.4.3 Treatment

If the fragment is available, in whole or in part, the fastest and most predictable treatment is to relocate it by applying orthophosphoric acid, adhesive and composite (usually heated or fluid), removing the excess before curing. If this is not the case, we can always reconstruct the fragment directly in the mouth with composite (with or without previous waxing) or indirectly working on a plaster model resulting from the impression of the patient's arches.

15.4.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed after 6-8 weeks and after 1 year.

15.4.5 Evolution

They can evolve asymptomatically with positive pulp vitality tests and a correct root development in cases of immature teeth. If symptoms appear, the sensitivity tests are negative, there are signs of apical periodontitis or the root of immature teeth is not developing properly, it will be necessary to proceed with endodontics.

## 15.5 Fracture of enamel and dentine without pulp exposure

15.5.1 Clinical findings

It is a fracture that affects the enamel and dentine, with loss of tooth structure, but without pulp involvement. It is not tender to percussion nor usually presents mobility but it is necessary to verify it to discard luxation or root fracture. Pulp vitality is usually positive.

15.5.2 Radiographic findings

It is recommended to take periapical radiographs (frontal and with mesial and distal angles) and an occlusal radiograph in order to rule out luxation and/or root fracture. Remember that if the tooth fragment has not been located, it is necessary to x-ray the lip and/or the cheeks to rule out their impact on the soft tissue.

15.5.3 Treatment

If the fragment is available, in whole or in part, the fastest and most predictable treatment is to relocate it by applying orthophosphoric acid, adhesive and composite (usually heated or fluid), removing the excess before curing. If not, we can opt for a temporary treatment that consists of disinfecting the dentine surface and performing an indirect pulp coating with glass ionomer, or reconstruct the fragment directly in the mouth with composite (with or without previous waxing) or indirectly working on a plaster model resulting from the impression of the patient's arches. In cases where the pulp becomes transparent (without the presence of bleeding), a baseliner of calcium hydroxide and a glass ionomer over it will be applied.

15.5.4 Follow up
Follow up visits (clinical and radiographic evaluations) are usually performed at 6-8 weeks and 1 year.

15.5.5 Evolution

They can evolve asymptomatically, with positive pulp vitality tests and a correct root development in immature teeth. If symptoms appear, the sensitivity tests are negative, there are signs of apical periodontitis or the root of immature teeth is not developing properly, it will be necessary to proceed with endodontic treatment.

**15.6 Fracture of enamel and dentine with pulp exposure.**

15.6.1 Clinical findings

It is a fracture that affects the enamel and dentine, with loss of tooth structure and pulp involvement. It is not tender to percussion nor usually presents mobility but it is necessary to verify it to discard luxation or root fracture. Pulp vitality is usually positive to stimuli, such as the direct application of air through the chair syringe.

15.6.2 Radiographic findings

It is recommended to perform periapical radiographs (frontal and with mesial and distal angles) and an occlusal radiograph in order to rule out luxation and/or root fracture. Remember that if the tooth fragment has not been located, it is necessary to x-ray the lip and/or the cheeks to rule out their impact on the soft tissue.

15.6.3 Treatment

In young patients or those with immature teeth, it is important to preserve pulp vitality by direct pulp coating or by performing a partial pulpotomy. Non-setting calcium hydroxide is a suitable material for treating such cases. In teeth with closed apex, the same treatment can be chosen or endodontic treatment depending on the clinical judgment.

If the fragment is available, in whole or in part, the quickest and most predictable treatment is, over the pulp coating or partial pulpotomy, to proceed with its relocation by applying orthophosphoric acid, adhesive and composite (usually heated or fluid), removing the excesses before light curing. Either reconstruct the fragment directly in the mouth with composite (with or without previous waxing) or indirectly working on a plaster model resulting from the impression of the patient's arches. These are probably cases that end up being rehabilitated later with a prosthetic crown.

15.6.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 6-8 weeks, 3 months, 6 months and 1 year.

15.6.5 Evolution

They can evolve asymptomatically with positive pulp sensitivity tests and proper root development in immature teeth. In case of symptomatology, negative vitality tests, signs of apical periodontitis or the root of immature teeth does not develop properly, it will be necessary to proceed with endodontic treatment.

**15.7 Crown and root fracture without pulp exposure.**

15.7.1 Clinical findings

It is a fracture that, although it affects enamel, dentine and cementum, has no pulp exposure. The trajectory covers part of the crown and extends below the gingival margin. This fact can be seen clinically with a slight mobility of the coronal fragment and discomfort to the percussion. The pulp usually responds positively to vitality tests.

15.7.2 Radiographic findings

It is recommended to take periapical radiographs (frontal and with mesial and distal angles) and an occlusal radiograph in order to be able to intuit, as long as there is no displacement of the fragments, the presence of fracture lines.

15.7.3 Treatment

As an emergency treatment, temporary splinting can be performed on adjacent teeth until the final treatment plan is executed. To proceed with the restoration, the fractured coronary root fragment is usually removed, exposing the margin of rupture by gingivectomy and ostectomy depending on the subgingival depth of the case.

Sometimes it is necessary to perform endodontic treatment, reconstruction using a post and reinforcement with a prosthetic crown. These procedures are usually preceded by a gingivectomy, accompanied in many cases by ostectomy, osteoplasty and orthodontic or surgical extrusion of the root fragment. It is essential to have enough ferrule around the entire contour of the restoration where the prosthetic crown is subsequently cemented.

If the prognosis is uncertain and the patient is an adult, we can consider exodontia to rehabilitate by means of an immediate or delayed implant, or a bridge, since extraction is usually inevitable in very extensive vertical fractures.

15.7.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 1 week, 6-8 weeks, 3 months, 6 months and 1 year. Then yearly for at least 5 years.

15.7.5 Evolution

They can evolve asymptomatically, with positive pulp vitality tests and a correct root development in immature teeth. If symptoms appear, the sensitivity tests are negative,

there are signs of apical periodontitis or the root of immature teeth is not developing properly, it will be necessary to proceed with endodontic treatment.

## 15.8 Crown and root fracture with pulp exposure

### 15.8.1 Clinical findings

It is a fracture that in addition to affecting the enamel, dentine and cementum, exposes the pulp cavity. This fact can be seen clinically with a slight mobility of the coronal fragment and discomfort to the percussion.

### 15.8.2 Radiographic findings

Periapical and occlusal radiographs are recommended.

### 15.8.3 Treatment

As an emergency treatment, a temporary splinting of the coronal fragment to the adjacent teeth can be performed.
In young patients or those with immature teeth, it is important to preserve pulp vitality by performing a partial pulpotomy. Non-setting calcium hydroxide is a suitable material for treating such cases. To proceed with the restoration, the fractured coronary root fragment is usually removed, exposing the margin of rupture by gingivectomy and ostectomy depending on the subgingival depth of the case.

In adult patients or teeth with a closed apex, endodontics, post-reconstruction and reinforcement with a prosthetic crown are usually performed. These procedures are usually preceded by a gingivectomy, accompanied in many cases by ostectomy, osteoplasty and orthodontic or surgical extrusion of the root fragment. It is essential to have enough ferrule around the entire contour of the restoration where the prosthetic crown is subsequently cemented.

If the prognosis is uncertain and the patient is an adult, we can consider exodontia to rehabilitate by means of an immediate or delayed implant, or a bridge, since extraction is usually inevitable in very extensive vertical fractures.

### 15.8.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 1 week, 6-8 weeks, 3 months, 6 months and 1 year. Then yearly for at least 5 years.

### 15.8.5 Evolution

They can evolve asymptomatically, with positive pulp vitality tests and a correct root development in immature teeth. If symptoms appear, the sensitivity tests are negative, there are signs of apical periodontitis or the root of immature teeth is not developing properly, it will be necessary to proceed with endodontic treatment.

## 15.9 Root fracture

### 15.9.1 Clinical findings

The coronal fragment may be mobile or displaced from its original position, experiencing discomfort to percussion. Due to the coronal mobility we can observe the presence of bleeding in the gingival sulcus. Vitality tests may be negative but time will determine whether the pulp damage is transient or permanent. It can happen that over time the crown changes colour towards reddish or greyish tones.

### 15.9.2 Radiographic findings

The fracture affects exclusively the root with a horizontal or oblique trajectory at any level of it: cervical, middle or apical third. Horizontal fractures are usually detected by a periapical x-ray with an angle of 90 ° from a frontal projection, something that is easy to perform using a positioner and very common in fractures of the cervical third. If the fracture plane is more oblique, being at the level of the middle and/or apical thirds, occlusal and/or periapical radiographs with different angles will be chosen.

### 15.9.3 Treatment

We must proceed to the replacement of the fragment urgently. This position must be checked radiographically and place a flexible splint for 4 weeks. If the fracture is close to the cervical third of the root, it would be advisable to increase the period of splinting up to 4 months. In any case, it must be monitored for at least one year to check the evolution of the pulp state. If signs of necrosis appear, endodontics of the coronal fragment will be performed to the fracture line to try to preserve the tooth in the arch.

### 15.9.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 4 weeks (removal of the splint for mid-root and apical third fractures), 6-8 weeks, 4 months (removal of the splint for cervical third fractures), 6 months, 1 year and then yearly for at least 5 years (clinic).

### 15.9.5 Evolution

They can evolve positively by responding to vitality tests, although it is common to get false negatives during the first 3 months. Radiographically we can see signs of repair between the fragments of the crown and the root. In case the tooth becomes symptomatic, responds negatively to vitality tests, extrusion of the coronal fragment occurs, a radiographic lesion appears in the fracture line and/or apical periodontitis with abscesses, endodontic treatment will be performed.

## 15.10 Alveolar bone fracture

### 15.10.1 Clinical findings

The fracture affects the alveolar bone and sometimes the underlying bone structures. It is common to find several teeth with block mobility and displacement with respect to the original position, something that the patient describes as an abnormality when occluding. Vitality tests, depending on each case, can be positive or negative.

### 15.10.2 Radiographic findings

Since fracture lines can be found at any level, it is recommended to take periapical, occlusal and panoramic radiographs.

### 15.10.3 Treatment

This involves repositioning the bone segment with subsequent splinting for 4 weeks. Sometimes it is necessary to suture the associated lesions in the soft tissues of the lips and/or cheeks.

### 15.10.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 4 weeks (splint removal), 6-8 weeks, 4 months, 6 months, 1 year and then yearly for at least 5 years.

### 15.10.5 Evolution

They can evolve positively by responding to vitality tests, although it is common to get false negatives during the first 3 months. If the tooth becomes symptomatic, responds negatively to vitality tests, apical periodontitis or inflammatory process of external root resorption appears, endodontic treatment will be performed.

# 16

# TRAUMATIC INJURIES WITH PERIODONTAL AFFECTION IN PERMANENT TEETH

## 16.1 Concussion

### 16.1.1 Clinical findings

Although the tooth is tender to percussion, it does not show displacement or increased mobility. Even vitality tests are usually positive.

### 16.1.2 Radiographic findings

There are no abnormal signs on the x-rays.

### 16.1.3 Treatment

It does not require treatment, simply a control of the pulp condition for at least 1 year.

### 16.1.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 4 weeks and 1 year.

### 16.1.5 Evolution

They can evolve asymptomatically by responding positively to vitality tests, although false negatives can be observed during the first 3 months. Immature teeth sometimes continue normally with root development. In case the tooth becomes symptomatic, responds negatively to vitality tests, apical periodontitis appears or the development of the root is not completed, endodontic treatment will be performed.

## 16.2 Subluxation

### 16.2.1 Clinical findings

Although the tooth is tender to percussion and has no displacement, there is an increased mobility that can be accompanied with the presence of bleeding in the gingival sulcus. Vitality tests are likely to be initially negative, indicating transient pulp damage that should be monitored until pulp diagnosis is certain.

### 16.2.2 Radiographic findings

There are no abnormal signs on the x-rays.

### 16.2.3 Treatment

They do not normally require treatment but flexible splinting for 2 weeks can improve the patient's comfort.

### 16.2.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually carried out at 2 weeks (splint removal), 12 weeks, 6 months and 1 year.

16.2.5 Evolution

They can evolve asymptomatically by responding positively to vitality tests, although it is probable to get false negatives during the first 3 months. Immature teeth sometimes continue normally with root development. If the tooth becomes symptomatic, responds negatively to vitality tests, apical periodontitis appears, external root resorption occurs or root development is not completed, endodontic treatment will be performed.

## 16.3 Extrusive luxation

16.3.1 Clinical findings

The tooth appears elongated and excessively mobile. The pulp response to stimuli is probably negative.

16.3.2 Radiographic findings

Periodontal ligament widening is observed at the apical level.

16.3.3 Treatment

It consists of trying to reposition the tooth by reinserting it in its alveolus and then placing a flexible splint for 2 weeks. In teeth where the presence of pulp necrosis is observed, endodontic treatment will be performed.

16.3.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 2 weeks (splint removal), 4 weeks, 8 weeks, 12 weeks, 6 months, 1 year (clinical) and then yearly for at least 5 years.

16.3.5 Evolution

They can evolve asymptomatically by responding positively to vitality tests, although it is probable to get false negatives during the first 3 months. Immature teeth sometimes continue normally with root development and a cure of the periodontium is visible radiographically. If the tooth becomes symptomatic, responds negatively to vitality tests, apical periodontitis appears, external root resorption occurs or root development is not completed, endodontic treatment will be performed. If the marginal bone does not recover, it is advisable to splint the tooth for another 3-4 weeks.

## 16.4 Lateral luxation

16.4.1 Clinical findings

The tooth is visibly displaced in the palatal/lingual or vestibular direction with no mobility. A characteristic percussive metallic sound can be perceived that describes a possible ankylosis. It is common to be accompanied by fracture of the alveolar bone in the opposite direction to the inclination of the crown, where the root impacts.

16.4.2 Radiographic findings

Eccentric periapical or occlusal radiographs are performed in order to visualize a widening of the periodontal ligament.

16.4.3 Treatment

It consists of repositioning the tooth with the fingers, or with a forceps, to eliminate the entrapment of the root in the fractured alveolar bone. It must then be flexibly splinted for 4 weeks and the pulp vitality monitored. In case of necrosis it is necessary to perform endodontics to prevent root resorption.

16.4.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 2 weeks, 4 weeks (splint removal), 8 weeks, 12 weeks, 6 months, 1 year (clinical) and then yearly for at least 5 years.

16.4.5 Evolution

They can evolve asymptomatically by responding positively to vitality tests, although it is probable to get false negatives the first 3 months. Immature teeth sometimes continue normally with root development and a cure of the periodontium is visible radiographically. If the tooth becomes symptomatic, responds negatively to sensitivity tests, apical periodontitis appears, external root resorption occurs or root development is not completed, endodontic treatment will be performed. If the marginal bone does not recover, it is advisable to splint the tooth for another 3-4 weeks.

**16.5 Intrusive luxation**

16.5.1 Clinical findings

The tooth is displaced within the alveolus in the axial direction and remains with no mobility. A characteristic metallic sound can be perceived on the percussion that identifies a possible ankylosis.

16.5.2 Radiographic findings

The space corresponding to the periodontal ligament is practically not visible due to the intrusive movement. The amelocementary line is more apical compared to other uninjured teeth.

16.5.3 Treatment

It focuses on the degree of root development. With immature apexes it is convenient to wait for the spontaneous eruption but in case of possible ankylosis it is chosen to perform an orthodontic or surgical extrusion, especially when the intrusion is greater than 7 mm.

With close apexes spontaneous eruption is expected as long as the intrusion is less than 3 mm. If after 2-4 weeks there are no eruptive changes, an orthodontic or surgical extrusion will be chosen before the tooth becomes ankylosed. If the intrusion is larger than 7 mm, surgical extrusion is valued as the only option. It is common for the pulp tissue to turn necrotic and in these cases, it is recommended to treat the root canals with a temporary dressing of calcium hydroxide 2-3 weeks after surgery.

After repositioning the tooth, extruding it to the original position, it must be flexibly splinted for a minimum of 4-8 weeks.

16.5.4 Follow up

Follow up visits (clinical and radiographic evaluations) are usually performed at 2 weeks, 4 weeks (splint removal), 8 weeks, 12 weeks, 6 months, 1 year and then yearly for at least 5 years.

16.5.5 Evolution

They can evolve asymptomatically, erupting spontaneously and without signs of resorption. Immature teeth sometimes continue normally with root development. In case the tooth becomes ankylosed, an apical periodontitis radiographically appears or external root resorption occurs, endodontic treatment will be performed.

## 16.6 Avulsion

The avulsion of permanent teeth is one of the most serious clinical conditions that currently occur during clinical practice. They require emergency intervention in order to evolve favourably and minimize the consequences, permanent in many cases.

The International Association of Dental Traumatology (IADT) has developed treatment protocols resulting from a thorough review of the current scientific literature and group meetings to share the various experiences of specialists in the field. In cases where the data provided by the literature do not seem too conclusive, it has been decided to agree on guidelines based on the clinical evidence of experts and researchers.

16.6.1 Intervention at the scene of the accident

When an avulsion of a permanent tooth occurs, the first thing to try is to reimplant it. If this is not possible, there are various means of storage and preservation until you reach the dental practice urgently. We recommend:

- Keep the patient calm, or their interlocutor (parents/guardians), whether by telephone or in person.
- Locate the avulsed tooth and take it by the crown avoiding touching the root.
- If the tooth is dirty, it will be washed with cold water and gently for a maximum of 10 seconds before trying to reimplant it. Once it is back in its original position, we will urge the patient to bite a gauze or handkerchief to keep it under pressure in a stable position.

- If it cannot be reimplanted, the tooth will be immersed in a glass of milk for the time it takes to reach the dental practice. It can also be transported into the patient's mouth if he is conscious. The ideal storage medium is HBSS or Hank's Balanced Salt Solution but its availability is very limited, especially in Spain.
- Go to the dentist urgently.

16.6.2 Treatment of avulsions of permanent teeth with closed apex.

16.6.2.1 Teeth reimplanted before arriving at the dental practice

- Leave the tooth in place.
- Clean the area with water spray, saline or chlorhexidine.
- Suture soft tissues if necessary.
- Check clinically and radiographically that the tooth is positioned correctly.
- Stabilize the tooth with a passive flexible splint for 2 weeks. In cases of associated alveolar or jawbone fracture, a more rigid splint is indicated and should be left in place for about 4 weeks.
- Administer systemic antibiotics.
- Check tetanus coverage.
- Give instructions to the patient.
- Perform endodontics within 7-10 days after reimplantation and after removing the splint.
- Follow-up.

16.6.2.2 Properly stored teeth or dry time less than 60 minutes

Suitable storage media are HBSS or Hank's Balanced Salt Solution, milk, saline or saliva.

- Clean the root and apical foramen with a stream of saline.
- Immerse the entire tooth in saline to remove necrotic cell tissue and possible contamination.
- Administer local anaesthesia (preferably without vasoconstrictor) and wash the alveolus with sterile saline.
- Examine the alveolar socket. If there is a fracture of the socket wall, reposition the fractured fragment into its original position with a suitable instrument to facilitate reimplantation.

- Reimplant the tooth with your fingers without excessive pressure.
- Suture soft tissues if necessary.
- Check clinically and radiographically that the tooth is positioned correctly.
- Stabilize the tooth for 2 weeks using a passive, flexible splint. In cases of associated alveolar or jawbone fracture, a more rigid splint is indicated and should be left in place for about 4 weeks.
- Administer systemic antibiotics.
- Check tetanus coverage.
- Provide post-operative instructions.
- Perform endodontics within 7-10 days after reimplantation and after removing the splint.
- Follow-up.

16.6.2.3 Teeth without viable cells or dry time greater than 60 minutes

The fibres of the periodontal ligament experience necrosis due to excessive dry time so the long-term prognosis will not be good. However, it is chosen to perform the reimplantation for several reasons, including recovering the patient's aesthetics and preserving the bone for subsequent rehabilitation with implants. The predictable evolution is that tooth ends with ankylosis and/or with root resorption and it will require extraction.

- Remove the cell tissue very carefully, for example with a gauze. There is no consensus on how best to do this.
- You can choose to perform endodontics of the avulsed tooth before proceeding with reimplantation or treat the tooth once reimplanted.
- Administer local anaesthesia and wash the alveolus with saline.
- Reimplant with your fingers without exerting excessive pressure. Some authors suggest immersing the root in 2% sodium fluoride for 20 'to delay reabsorption but there is no consensus.
- Suture the wounds if necessary.
- Check clinically and radiographically that the tooth is positioned correctly.
- Splint flexibly for 4 weeks.
- Administer systemic antibiotics.
- Check tetanus coverage.
- Provide post-operative instructions
- Follow-up.

16.6.3 Treatment of avulsions of permanent teeth with open apex

16.6.3.1 The tooth has been reimplanted before patient's arrival at the clinic

- Leave the tooth in place.
- Clean the area with water spray, saline or chlorhexidine.
- Administer local anaesthesia, if necessary, and preferably with no vasoconstrictor.
- Suture soft tissues if necessary.
- Check clinically and radiographically that the tooth is positioned correctly. If the tooth or teeth were replanted in the wrong socket or rotated, consider repositioning the tooth/teeth into the proper location for up to 48 hours after the trauma.
- Stabilize the tooth for 2 weeks using a passive and flexible splint. In cases of associated alveolar or jawbone fracture, a more rigid splint is indicated and should be left in place for 4 weeks.
- Administer systemic antibiotics.
- Check tetanus coverage.
- Provide post-operative instructions.
- The goal is for revascularization to occur but if this does not happen it is recommended to perform endodontics.
- Follow up.

16.6.3.2 Properly stored teeth or dry time less than 60 minutes

Suitable storage media are HBSS or Hank's Balanced Salt Solution, milk, saline and saliva.

- Clean the root and apical foramen with a stream of saline or by gently agitating it in the storage medium.
- Administer local anaesthesia, preferably without vasoconstrictor.
- Irrigate the socket with sterile saline.
- Examine the alveolus and, if you find a fracture in the walls, try to reposition with a suitable instrument to facilitate reimplantation.
- Reimplant the tooth with your fingers without excessive pressure.
- Suture soft tissues if necessary.

- Check clinically and radiographically that the tooth is positioned correctly.
- Stabilize the tooth for 2 weeks using a passive and flexible splint. In cases of associated alveolar or jawbone fracture, a more rigid splint is indicated and should be left for about 4 weeks.
- Administer systemic antibiotics.
- Check tetanus coverage.
- Provide post-operative instructions.
- The goal is for revascularization to occur, but the risk of an infection that triggers root resorption is quite high and rapidly evolving, especially in children. If revascularization does not occur, endodontics is recommended.
- Follow-up.

16.6.3.3 Teeth without viable cells or dry time greater than 60 minutes

The fibres of the periodontal ligament experience necrosis due to excessive dry time so the long-term prognosis will not be good. However, it is chosen to perform the reimplantation for several reasons, including recovering the patient's aesthetics and preserving the bone for subsequent rehabilitation with implants. The predictable evolution is that tooth ends with ankylosis and/or with root resorption and it will require extraction.

- Remove debris from its surface by gently agitating it in the storage medium. Alternatively, a stream of saline can be used to rinse its surface.
- You can choose to perform endodontics of the avulsed tooth before proceeding with reimplantation or treat the tooth once reimplanted. (not in the 2020 guidelines)
- Administer local anaesthesia, preferably without vasoconstrictor
- Irrigate the socket with sterile saline.
- Examine the alveolar socket. If there is a fracture of the socket wall, reposition the fractured segment with a suitable instrument.
- Reimplant the tooth with your fingers without excessive pressure. Suture soft tissues if necessary.
- Check clinically and radiographically that the tooth is positioned correctly.
- Stabilize the tooth for 2 weeks using a passive and flexible splint. In cases of associated alveolar or jawbone fracture, a more rigid splint is indicated and should be left for about 4 weeks.
- Administer systemic antibiotics.

- Revascularization of the pulp space, which can lead to further root development and maturation, is the goal when replanting immature teeth in children. The risk of external infection-related (inflammatory) root resorption should be weighed against the chances of revascularization. Such resorption is very rapid in children. If spontaneous revascularization does not occur, apexification, pulp revitalization/revascularization, or root canal treatment should be initiated as soon as pulp necrosis and infection is identified
- Check tetanus coverage.
- Provide post-operative instructions.
- Follow up.

16.6.4 Antibiotic therapy

The use of antibiotics after the reimplantation of avulsed teeth is recommended because, although their performance is still clinically questioned, experimental studies conclude that they have beneficial effects for periodontal and pulp recovery, especially when administered topically. However, given the situation of the traumatic event and the possible presence of associated injuries, its use may be justified.

Tetracycline is usually the systemic antibiotic of first choice at a dose appropriate to the patient's age and weight during the first week after reimplantation. However, in young patients there is a risk of permanent discoloration of the teeth and this aspect must be highly considered by the dentist. In many countries the use of tetracyclines is discouraged in children under 12 years of age. Alternatively, an age-appropriate dose of amoxicillin may be administered to the patient.

Regarding the use of topical antibiotics, it is chosen to immerse the avulsed tooth in a solution composed of 1 mg of minocycline or doxycycline diluted in 20 ml of saline, as it seems to have beneficial effects on pulp revascularization and periodontal recovery of immature teeth.

16.6.5 Splinting

Scientific studies have shown that flexible splinting is best suited for permanent avulsed teeth as a certain degree of movement, for a not very long time, is beneficial for periodontal and pulp recovery.

Obviously, the splint must be placed on the vestibular surfaces in order to avoid occlusal interferences and also to have the palatal or lingual surfaces free in case any root canal treatment needs to be performed. The splinting time of the avulsed and reimplanted permanent teeth is 2 - 4 weeks.

16.6.6 Post-operative instructions for the patient

Patient’s care at home, as well as attendance at regular follow-up visits, are critical to recover from trauma. It is important to instruct on wearing protection during sports in order to avoid further mishaps or aggravation of previous injuries. Hygiene guidelines must be very strict, reinforcing for the first 2 weeks with a 0.12% chlorhexidine alcohol-free mouthwash.

# 17

# DENTOALVEOLAR FRACTURES IN PRIMARY DENTITION

## Introduction

The management of dentoalveolar trauma in primary dentition has a different approach compared to permanent dentition. The International Association of Dental Traumatology (IADT) is composed of a group of experts in the field who, periodically, publish protocols to be followed before each of the traumatic entities. They are the result of a review of the most current scientific literature, combined with consensus meetings in those cases where the literature does not provide conclusive data on how to proceed in certain situations. The goal is to establish clear and predictable treatment protocols that, while not guaranteed success in all situations, can contribute to positive results in dental therapy.

## 17.1 Special considerations

We must keep in mind that trauma to a temporary tooth, especially if it is an intrusive dislocation and/or avulsion, can greatly affect the germ of the permanent tooth. It can produce in its malformations, modification of its eruptive trajectory, colour change in the crown (whitish, yellowish or brown spots) and hypoplasia.

The choice of the appropriate therapy logically depends on many factors but mainly on the collaboration of the patient and the opinion of his parents on the different treatment options available explained by the dentist. There is no consensus as to which type of treatment is best, as some experts opt for exodontia, while others advocate conservation to keep the temporary tooth in the mouth for as long as possible. In any case, those temporary teeth that are avulsed, given the presence of the germ of the final tooth in the same alveolus, are not reimplanted.

Luxations usually resolve spontaneously without having to resort to any type of orthodontic or surgical traction, thus avoiding exposure of the patient, usually young, to a treatment, in a way, invasive. If you choose to perform endodontics to preserve the temporary element in the mouth, zinc oxide-eugenol, calcium hydroxide or iodoform paste is used when proceeding with the pulpectomy.

Although traumas with pulpal exposure have a low incidence in temporary dentition because the ratio of the dental element´s proportion crown / root is close to 1:1 (different from permanent teeth). Periodontium and alveolar bone are the tissues that absorb most of the impact force. Partial pulpotomy will be the treatment of choice in these situations.

## 17.2 Radiographs

It is essential to perform periapical radiographs with 90º frontal and occlusal angulation. Using plates adapted to the patient's size (in the case of a child) and minimizing the risk of radiation with the appropriate protective elements as they serve to determine the scope of periodontal injury, degree of root development, and position of permanent replacement teeth. In any case, it is the dentist who will determine which projections are necessary also depending on the degree of collaboration of the patient.

## 17.3 Dental splinting

It is used only in cases of fracture of the alveolar bone or root within the alveolus.

## 17.4 Antibiotic therapy

Scientific evidence is limited with respect to the use of systemic antibiotics for the management of luxations in the temporary dentition. However, its use may be justified for the treatment of associated injuries. It is advisable for the dentist to consult with the paediatrician in order to know the general health of the patient and determine the need for antibiotics.

## 17.5 Sensitivity tests

Pulp sensitivity tests are not performed because their results are inconclusive in the temporary dentition.

## 17.6 Discolourations

It is a very consulted and relatively frequent question in the case of luxations. After discolouration of the temporary tooth has established, it may fade over time or darken further. In the latter situation, it is necessary to monitor the patient clinically and radiographically in order to determine the presence of pulpal necrosis that would involve the treatment of ducts or exodontia, depending on aspects such as the patient's age, degree of cooperation and opinion of their parents.

## 17.7 Enamel fracture

### 17.7.1 Clinical findings

It is a fracture that affects the enamel, with loss of part of the dental element.

### 17.7.2 Radiographic findings

No pathological situations are observed.

### 17.7.3 Treatment

Smooth the edge of the fracture line with a bur to prevent rubbing and cuts on the soft tissues or contact with the tongue.

### 17.7.4 Control

No monitoring required.

### 17.7.5 Evolution

There are usually no symptoms.

## 17.8 Fracture of enamel and dentin without pulpal exposure

### 17.8.1 Clinical findings

It is a fracture that affects the enamel and dentine, with loss of tooth structure, but without pulpal involvement.

### 17.8.2 Radiographic findings

No pathological situations are observed.

### 17.8.3 Treatment

Treatment depends on the patient's maturity and ability to cooperate. It would be convenient to seal the exposed dentine with glass ionomer to prevent microfiltration and, in case the structural loss is greater, to repair it by a direct restoration with composite.

### 17.8.4 Control

A follow-up visit is usually performed at 3-4 weeks (clinic).

### 17.8.5 Evolution

There are usually no symptoms.

## 17.9 Fracture of enamel and dentin with pulpal exposure

### 17.9.1 Clinical findings

It is a fracture that affects the enamel and dentine, with loss of tooth structure and pulpal involvement.

### 17.9.2 Radiographic findings

It is convenient to determine the degree of root development with a periapical radiograph.

### 17.9.3Treatment

Treatment depends on the patient's maturity and ability to cooperate. In order to preserve the pulpal vitality, a partial pulpotomy is chosen, compacting a small amount of pure calcium hydroxide in the pulp chamber. Then applying a coating with glass ionomer and composite until the restoration is completed. In case of not having the collaboration of the patient, the alternative is the exodontia.

17.9.4 Control

Control visits are usually performed at 1 week (clinical), 6-8 weeks (clinical and radiographic) and 1 year (clinical and radiographic).

17.9.5 Evolution

They can evolve asymptomatically continuing with root development in cases of immature teeth. If signs of apical periodontitis appear or the root does not develop properly, it will be necessary to proceed with endodontics or removal.

**17.10 Crown and root fracture**

17.10.1 Clinical findings

It is a fracture that affects the enamel, dentine and root cementum, where the pulp may or may not be exposed. The trajectory covers part of the crown and extends below the gingival margin, presenting a small or moderate displacement as the tooth is loose but still remains in the arch by the gingival tissue fibres.

17.10.2 Radiographic findings

The presence of several fragments is observed on a periapical radiograph.

17.10.3 Treatment

Depending on the clinical situation, it is possible to opt for the removal of the coronal fragment in case it affects a small part of the root and has sufficient stability to allow the restoration of the crown. If not, the exodontia will be performed.

17.10.4 Control

If the removal of the coronal fragment is chosen, control visits are performed that take place at 1 week (clinical), 6-8 weeks (clinical and radiographic) and 1 year (clinical).

17.10.5 Evolution

They can evolve asymptomatically continuing with root development in cases of immature teeth. If signs of apical periodontitis appear or the root does not develop properly, it will be necessary to proceed with the exodontia.

**17.11 Root fracture**

17.11.1 Clinical findings

The coronal fragment may be mobile or displaced from its original position.

17.11.2 Radiographic findings

With a periapical radiograph, it is possible to locate at what level of the root the fracture is, being frequent in the middle or apical third.

17.11.3 Treatment

If the coronal fragment is not displaced, no treatment is required. In case of displacement it is necessary to reposition it and splint it. Another option is to extract the fragment and leave the root so that it is reabsorbed.

17.11.4 Control

When the fragment is not displaced, several control visits are usually performed that take place at 1 week (clinical), 6-8 weeks (clinical) and 1 year (clinical and radiographic). If only the coronal fragment is removed, the root condition will be checked additionally once a year until it is exfoliated.

17.11.5 Evolution

They can evolve in a positive way being able to observe radiographically signs of repair between both fragments. If only the apical fragment is preserved, it is reabsorbed naturally.

**17.12 Alveolar bone fracture**

17.12.1 Clinical findings

The fracture affects the alveolar bone and, as it can extend to the underlying socket. It is common for the patient to have mobility and displacement of an entire segment (including one or more teeth), which contributes to occlusal interference.

17.12.2 Radiographic findings

A periapical x-ray can locate the level of the alveolar bone where the fracture is, as well as its relationship to the permanent replacement teeth.

17.12.3 Treatment

It usually requires general anaesthesia to reposition the fractured bone fragment and splint it for 4 weeks.

17.12.4 Control

Several follow-up visits are usually performed which take place at 1 week (clinical), 3-4 weeks (removal of the splint, clinical and radiographic), 6-8 weeks (clinical and radiographic), 1 year (clinical and radiographic) and once a year until exfoliated.

17.12.5 Evolution

They can evolve positively by recovering the usual occlusion, without developing apical periodontitis or complications in the permanent replacement teeth. Or negatively with the presence of periodontitis and / or inflammatory reabsorption that affects both temporary and permanent teeth.

# 18

# TRAUMATIC INJURIES WITH PERIODONTAL INVOLVEMENT IN TEMPORARY DENTITION

18.1 Concussion.

18.2 Subluxation.

18.3 Extrusive luxation.

18.4 Lateral luxation.

18.5 Intrusive luxation.

18.6 Avulsion.

## Introduction

The International Association of Dental Traumatology (IADT) is composed of a group of experts in the field who, periodically, publish protocols to be followed before each of the traumatic entities. They are the result of a review of the most current scientific literature, combined with consensus meetings in those cases where the literature does not provide conclusive data on how to proceed in certain situations.

## 18.1 Concussion

18.1.1 Clinical findings

Although the tooth is sensitive to percussion, there is no increase in mobility or bleeding in the gingival sulcus (also gingival groove).

18.1. Radiographic findings

There are no out-of-the-ordinary signs on x-rays as the periodontal area is usually normal.

18.1.3 Treatment

No treatment required, just stay under observation.

18.1.4 Control

Control visits are usually performed at 1 week (clinic) and 6-8 weeks (clinic).

18.1.5 Evolution

Immature teeth may continue normally with root development or experience a pause in growth. In cases of darkening of the crown, treatment will only be performed when an apical problem develops.

## 18.2 Subluxation

18.2.1 Clinical findings

Although the tooth has increased mobility and bleeding in the gingival sulcus, there is no evidence of displacement.

18.2.2 Radiographic findings

There are no abnormal signs on the x-rays as the periodontal area is normal. The occlusal projection is useful to detect possible root fractures and also serves as a reference in case of complications.

18.2.3 Treatment

No treatment required, just stay under observation. In hygiene guidelines, it is recommended to use a soft brush and apply a 0.12% chlorhexidine gel with a cotton ball to the affected area at least twice a day for a week.

18.2.4 Control

Control visits are usually performed at 1 week (clinic) and 6-8 weeks (clinic). In the case of darkening of the crown, it is important to monitor the tooth to detect the presence of signs of infection or the appearance of a fistula.

18.2.5 Evolution

Immature teeth can continue normally with root development even if there is discolouration of the crown. The shades can be reddish or greyish. When there is yellowing it indicates an obliteration of the pulp chamber and is a sign of good prognosis. Sometimes they experience a negative break in root growth and crown darkening, but treatment will only be performed when an apical problem develops.

**18.3 Extrusive luxation**

18.3.1 Clinical findings

The tooth appears elongated and excessively mobile as it is partially displaced outside the alveolus.

18.3.2 Radiographic findings

An increase in the space relative to the periodontal ligament is observed at the apical level.

18.3.3 Treatment

Treatment options are determined by the degree of mobility, root development, and the patient's ability to cooperate. When the extrusion of an immature temporary tooth is less than 3 mm, you can choose to try to reposition it carefully or leave it to its free evolution. While exodontia is performed directly when it is a severe extrusion in a temporary tooth completely formed at the root level.

18.3.4 Control

Control visits are usually performed at 1 week (clinical), 6-8 weeks (clinical and radiographic), 6 months (clinical and radiographic) and 1 year (clinical and radiographic). In the case of darkening of the crown it is important to monitor the tooth to detect the presence of signs of infection or the appearance of a fistula.

18.3.5 Evolution

Immature teeth can continue normally with root development even if there is discolouration of the crown. The shades can be reddish or greyish. When there is yellowing it indicates an obliteration of the pulp chamber and is a sign of good prognosis. Sometimes they experience a negative break in root growth and crown darkening, but treatment will only be performed when an apical problem develops.

**18.4 Lateral luxation.**

18.4.1 Clinical findings

The tooth is visibly displaced in the lingual or facial direction but remains motionless.

18.4.2 Radiographic findings

Occlusal radiographs are performed, where a widening of the periodontal ligament is visualized and in order to determine the relationship of the apex of the temporal tooth with the germ of the permanent one.

18.4.3 Treatment

When the patient has a previous open bite without occlusal interference, the temporary tooth itself continues to erupt spontaneously, obviously in a different position from the original. If there is occlusal interference due to displacement, you can opt for selective carving, a replacement under local anaesthesia or exodontia in severe cases.

18.4.4 Control

Control visits are usually performed at 1 week (clinical), 2-3 weeks (clinical), 6-8 weeks (clinical and radiographic) and 1 year (clinical and radiographic).

18.4.5 Evolution

They can evolve asymptomatically with radiographic signs of normality and healing in the area of the periodontal ligament, even with a transitional discolouration of the crown. Sometimes they experience a negative break in root growth and crown darkening, but treatment will only be performed when an apical problem develops.

**18.5 Intrusive luxation.**

18.5.1 Clinical findings

The tooth is displaced inside the alveolus in the axial direction, focused towards the buccal plate or towards the permanent germ hindering its eruption.

18.5.2 Radiographic findings

When the apex of the temporal tooth is focused towards the buccal plate, it is perceived radiographically shorter than the contralateral one. However, when the root is towards the permanent germ it is the other way around, it looks longer on an x-ray.

18.5.3 Treatment

In case of having an apex of the temporary tooth towards the buccal plate, the spontaneous eruption is chosen. But when the disposition of the root is oriented towards the germ of the permanent, the treatment of choice is exodontia.

18.5.4 Control

Control visits are usually performed at 1 week (clinical), 3-4 weeks (clinical and radiographic), 6-8 weeks (clinical), 6 months (clinical and radiographic), 1 year (clinical and radiographic) and annually until the eruption of the permanent tooth.

18.5.5 Evolution

They may erupt naturally with or without discolouration of the crown. However, some temporary teeth remain ankylosed, with persistent discolouration, signs of apical periodontitis, and damage to the permanent replacement tooth.

**18.6 Avulsion**

18.6.1 Clinical findings

The tooth is completely displaced outside the alveolus.

18.6.2 Radiographic findings

When the dental element is not located outside the patient's mouth, which is common if the parents have not witnessed the time of the accident, it is necessary to perform a periapical x-ray to dismiss that it is completely intruded.

18.6.3 Treatment

Reimplantation is not recommended in primary teeth.

18.6.4 Control

Several control visits are usually performed that take place at 1 week (clinical), 6 months (clinical and radiographic), 1 year (clinical and radiographic) and annually until the eruption of the permanent.

18.6.5 Evolution

They can affect the permanent tooth.

# 19

# MOUTH PROTECTORS

## Introduction

A mouthguard or dental protector is an intraoral device made of flexible materials that ensures maximum protection of the teeth, jaws, temporomandibular joint and underlying soft tissues It can attenuate brain damage caused by the force of an impact on the facial bone structure.

## 19.1 Preliminary considerations

When choosing a mouthguard, some considerations must be taken into account. The collection of these data is done through a questionnaire prior to dental examination:

- Age of the patient.
- Sports discipline.
- Amateur, semi-professional and professional level.
- Duration of the competition depending on the sport practiced.
- Number of weekly workouts.
- Need to communicate with teammates.

## 19.2 Features

- They must be made of a resilient and tear-resistant elastic material.
- Rounded margins covering up to half of the last molar.
- Odourless, tasteless, non-porous and easily washable.
- Non-toxic and antiallergic.
- Do not have secondary orthodontic effects.
- Do not hold the jaw in a forced position.
- Comfortable and retentive.
- Easy to put on and take off.
- Resistant to saliva.
- Do not suffer deformations.
- Do not prevent the patient from breathing or ingesting fluids.
- Variable thickness depending on the sport practiced.
- Possibility of adapting to a patient with fixed orthodontic appliances.

## 19.3 Types of mouthguards

### 19.3.1 Type I. Non-individualized standard mouthguard

It is a plastic device that can be purchased in specialty stores. It is very cheap but cannot be adapted to the patient's mouth. It has a horseshoe shape and it is available in various sizes and colours, being made of polyvinyl chloride, polyurethane or copolymer of vinyl acetate or ethylene. The external surface that contacts the antagonistic arch is flat, which makes it difficult to support the jaw and fix it in case of direct impacts on it.
They hardly protect against any type of trauma. The fact of being standard can cause friction and pressure ulcers in the soft tissue and often it falls when the patient does not remain with his mouth closed putting constant pressure on the device. For this reason, it is totally incompatible with being able to communicate with teammates in team sports and makes it difficult to breathe and ingest liquids/gels.

### 19.3.2 Type II. Individualized standard mouthguard (boil & bite)

They are made of a thermoplastic material that must be immersed in boiling water to adapt it to the patient's mouth, which can cause burns during preparation. It is designed so that it is the patient himself who adapts it to his mouth, which means discarding several units and not achieving a perfect fit that preserves the appropriate thickness of the material to protect the dentoalveolar structures from possible trauma. Although its outer surface shows the logical indentations, it does not solve the problem of fixing the jaw. Their shape and size are also standard and, although they are not the ideal protectors for all the reasons described above, growing patients are often the main users.

### 19.3.3 Type III. Individualized mouthguard (custom made)

#### 19.3.3.1 Single laminate mouthguard

The biggest advantage of the tailor-made protector is that the design can be individualized according to the individual’s oral anatomy and the type of sport the athlete practices. This device is manufactured in the laboratory from impressions taken with alginate or intraoral scanner, plus a constructive bite. It is a single laminate device used in low-impact sports or by growing teenage athletes who need to frequently renew the device at a not very high cost.

#### 19.3.3.2 Laminated mouthguard

It is an optimal product made by joining two or more sheets of different thicknesses, being a perfect answer for the specific needs of the athlete and his mouth. Highly recommended for every type of sport and perfectly stable to allow the mouth to open without falling. There are indentation marks of the lower occlusal surfaces that work as a gear assembly. They are more comfortable and safer, allowing talking, breathing and drinking fluids/gels. They are also the most expensive and difficult to make.

## 19.4 Clinical protocol

- Medical and dental history.
- Complementary tests: photographs, panoramic radiograph and impressions.

- Occlusion records.
- Determination of the necessary thickness depending on the sport practiced.
- Preparation by suction or pressure.
- Adjustments.
- Evaluation of comfort at fit appointment.
- Test for 15 days for possible adjustments.

**19.5 Maintenance**

- Wash before and after each use.
- Store in a plastic box with several holes.
- Do not expose it to direct sunlight or heat sources.
- Do not bend.
- Do not bite.
- Do not handle or use another person's mouth guard.
- Take it to check-up appointments.
- Replace it at least once a year.

**19.6 Advanced procedures**

- Record taking with intraoral scanner.
- Obtaining models using a 3D printer.
- Virtual articulation.
- Surface electromyography.
- Stabilometric and/or baropodometric platform.
- Accelerometer.
- Digital analysis of occlusion.
- Biosensors.

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