preservation and restoration of tooth structure

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Salivary flow

Both the quality and the quantity of saliva being secreted will vary throughout the day while awake and will be depressed during sleep. Unstimulated saliva contains little bicarbonate buffer, with fewer Ca^{2+} ions and more PO_4^{3-} ions than found in plasma. Reflex stimulation of salivary flow by chewing (such as chewing gum) or through the presence of acidic foods (such as citric acid) can increase the flow by a factor of more than ten (**Fig. 2.4**). Bicarbonate buffer concentrations can increase sixty times upon stimulation. Ca^{2+} ion levels also increase slightly, but PO_4^{3-} ions do not increase in proportion to flow rate.

Saliva provides the major source of natural protection of and repair to teeth following acid challenge. Reduction of maximum salivary flow to less than 0.7 ml/min may increase caries risk, although this depends on many other interacting factors.

Occasionally there may be a marked reduction in the resting pH of saliva not necessarily associated with a reduced flow. The cause is often not clear and the patient will then be susceptible to an increased caries rate.

Fluoride

Effect on enamel

Fluoride plays a highly significant role in the demineralisation–remineralisation process. In an acid environment, the fluoride ion reacts strongly with free Ca^{2+} and HPO_4^{2-} ions, forming fluorapatite crystals $Ca_{10}(PO_4)_6(OH.F)_2$, in which fluoride substitutes for some hydroxyl ions. Fluorapatite is less soluble than pure hydroxyapatite because of better subunit stacking. Fluorapatite crystals are unable to be dissolved by acid ions above pH 4.5 (the critical pH for fluorapatite), with the result that the mineral is more resistant to acid dissolution.

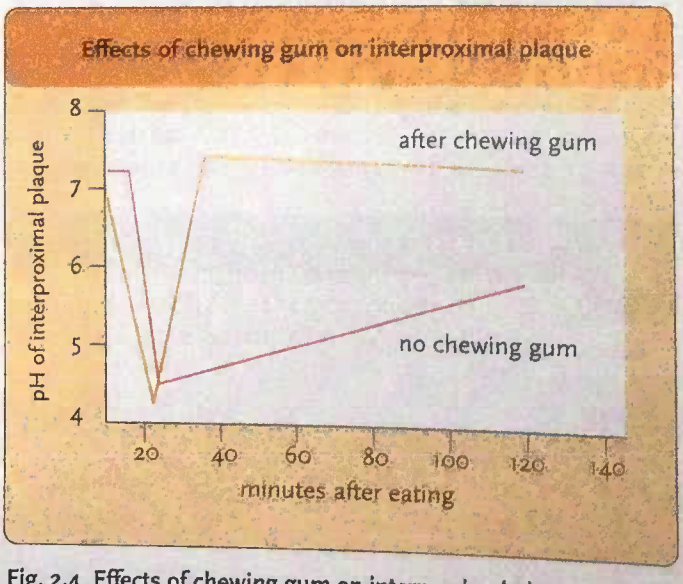

Fig. 2.4 Effects of chewing gum on interproximal plaque. Two hours after a meal, not followed by cleaning teeth, the interproximal plaque still shows a low pH. Chewing two pellets of sugared chewing gum for 20 minutes after eating is sufficient to raise the pH back to normal.

Fluoride ions are present within tooth structure in concentrations as high as 2500–4000 p.p.m (132–210 μmol/l), at the surface of enamel but the concentration in saliva may be as low as 0.03 p.p.m (1.6 μmol/l). The incorporation of fluorides into teeth during development, or the use of topical fluoride after eruption, enhances the availability of these ions, leading to increased inhibition of demineralisation and enhancement of remineralisation when acid ions interact with the tooth surface.

Daily consumption of water containing fluoride at 1 mg/l (52.6 μmol/l), for the whole of life, increases resistance to caries in all age groups from infancy to old age. Topical applications of fluoride can also help inhibit dental caries in those with a high caries rate.

Summary

Fluoride

Reacts directly with enamel and dentine and produces several effects.

- Forms fluorapatite, which is less soluble than hydroxyapatite
- Inhibits demineralisation
- Enhances remineralisation
- Inhibits bacterial metabolism
- Reduces 'wettability' of tooth structure
- Inhibits plaque formation

Inhibition of caries

Fluoride inhibits the development of caries by:

- inhibiting the demineralisation process and enhancing the normal remineralisation process by preferentially reacting with hydroxyapatite breakdown products to form fluorapatite or a fluoride-enriched apatite.
- inhibiting bacterial metabolism.

Methods of inhibition

The most efficient inhibition of caries occurs through frequent daily contact of low concentrations of fluoride ion with the tooth surface to inhibit demineralisation and enhance remineralisation of that surface. The optimal level necessary to achieve this will vary for each person according to the level of acid ions present in relation to the level of balancing protective agents.

High concentration fluorides store excess fluoride ion as CaF_2 around the apatite crystals. This may lead to heavy remineralisation at the surface of enamel lesions and the fluoride ion may not be able initially to penetrate more deeply into the subsurface body of the lesion. Subsequent acid challenge will progressively ionise this layer to permit free fluoride ions to penetrate more deeply. However, even this additional CaF_2 is quickly lost in the highly acid environment found in the patient with active caries and needs to be replenished more frequently to be effective.

Effect on established lesions

The fluoride ion will not only prevent initial lesions developing, but will also stabilise established lesions. That is, it can:

- contribute to remineralisation of incipient enamel caries
- partly remineralise carious dentine and thus slow down or arrest the caries process in the cavitated coronal lesion
- remineralise root surface lesions to the extent that they may not need restoration.

Topical fluoride is more effective in inhibiting smooth surface caries and in aiding remineralisation of enamel or cementum–dentine. It is less effective in fissure or interproximal caries because of the difficulty of removing stubborn or mature plaque. Daily application of topical fluoride to demineralised root surfaces over a period of 2–4 months will lead to significant hardening of the exposed dentine, indicating that a remineralising balance has been established. Deep and extensive root caries can be hardened within the same period of time but requires the use of higher concentrations of fluoride. The surfaces of such remineralised lesions can become glass-like in texture because of hypermineralisation.

NATURE OF THE ACID ION INTERACTION WITH APATITE AT THE TOOTH SURFACE

To understand the mechanism of the carious process it is necessary to understand the basic nature of the chemical reactions that occur at the tooth surface.

Demineralisation

The mineral component of enamel, dentine and cementum is hydroxyapatite, $Ca_{10}(PO_4)_6(OH)_2$. In a neutral environment, hydroxyapatite is in equilibrium with the local aqueous environment, which is saturated with Ca^{2+} and PO_4^{3-} ions.

Hydroxyapatite is reactive to hydrogen ions at pH 5.5 (the critical pH for hydroxyapatite) and below. H^+ reacts preferentially with the phosphate groups in the aqueous environment immediately adjacent to the crystal surface. The process can be thought of as conversion of PO_4^{3-} to HPO_4^{2-} by the addition of H^+ and the H^+ being buffered at the same time. The HPO_4^{2-} is then not able to contribute to the normal hydroxyapatite equilibrium because it contains PO_4, not HPO_4, and the hydroxyapatite crystal therefore dissolves. This is termed demineralisation.

Remineralisation

The demineralisation process can be reversed if the pH is neutral and there are sufficient Ca^{2+} and PO_4^{3-} ions in the immediate environment. Either the apatite dissolution products can reach neutrality by buffering or the Ca^{2+} and PO_4^{3-} ions in saliva can inhibit the process of dissolution through the common ion effect. This enables rebuilding of partly dissolved apatite crystals and is termed remineralisation.

This interaction can be greatly enhanced by the presence of fluoride ion at the reaction site. The overall reaction, which may be characterised as the 'demineralisation–remineralisation' process, can be symbolised in general terms as shown in **Figure 2.5**.

The chemical basis of the demineralisation–remineralisation process is similar for enamel, dentine and root cementum. However, the different structures and relative quantity of the mineral and organic tissue content of each of these materials causes significant differences in the nature and progress of the carious lesion. These differences will be described later.

Acid reaction with apatite at the tooth surface

Following eruption there is a process of continuing mineralisation of enamel from salivary calcium and phosphate (see Chapter 1). Initially, enamel apatite contains many carbonate and magnesium ions, which are highly soluble in even mild acidic conditions. However, there is a rapid and extensive exchange of hydroxyl and fluoride ions as the magnesium and carbonate are dissolved, leading to a more 'mature' enamel with a greater resistance to acid ion challenge. This level of maturity, or acid resistance, can be greatly enhanced by the presence of fluoride.

When a pulse of acid ions is generated at the tooth surface, regardless of the level of maturity, the general reaction can be symbolised by the diagram in **Figure 2.6.**

As the pH decreases the acid ions react, principally with the phosphates in saliva and plaque (or calculus), until the critical pH for dissociation of hydroxyapatite is reached at approximately pH 5.5–5.2. Further decrease in pH results in progressive interaction of the acid ions with the phosphate groups of hydroxyapatite, causing partial or full dissolution of the surface crystallite. Stored fluoride released in this process reacts with the Ca^{2+} and HPO_4^{2-} ion breakdown products, forming fluorapatite, or fluoride-enriched apatite. If the pH decreases further below 4.5, which is the critical pH for fluorapatite dissolution, even fluorapatite will then dissolve. If acid ions are neutralised, and the Ca^{2+} and HPO_4^{2-} ions are retained in this hypothetical model, the reverse processes of remineralisation are able to occur as shown in Figure 2.6.

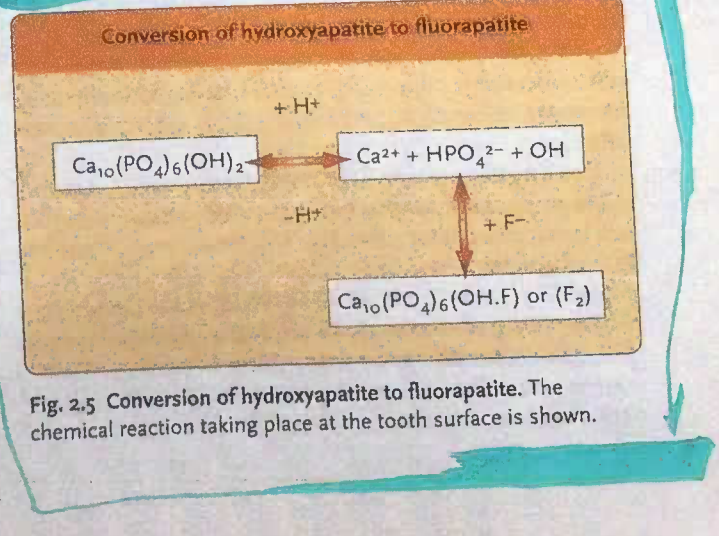

Fig. 2.5 Conversion of hydroxyapatite to fluorapatite. The chemical reaction taking place at the tooth surface is shown.

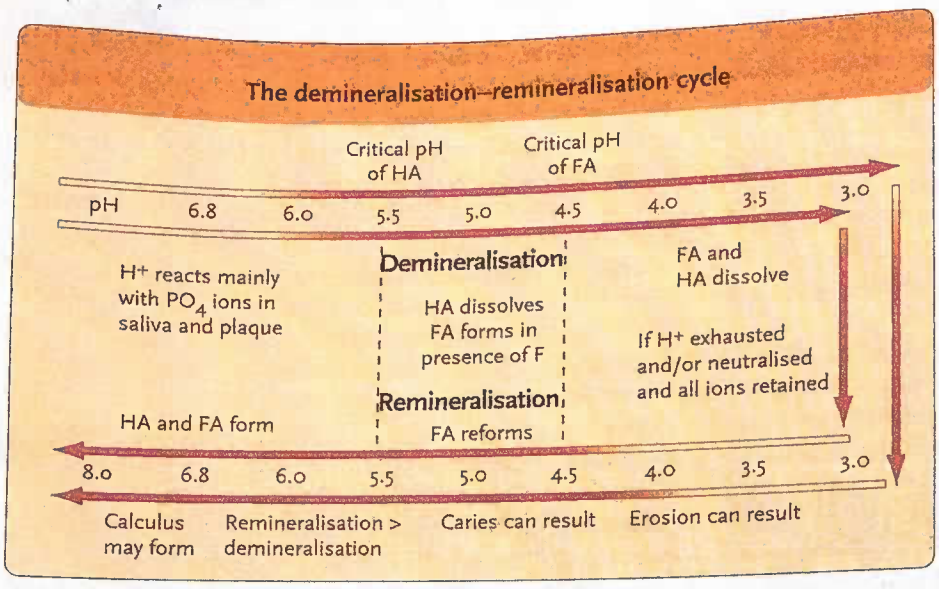

Fig. 2.6 The demineralisation–remineralisation cycle. A conceptual chart to demonstrate the levels of pH at which the stages of the demineralisation–remineralisation cycle occur.
F, fluoride; FA, fluorapatite; HA, hydroxyapatite.

In terms of the cycle proposed, in reality, there will be variation in both the level of acid ion production as well as neutralisation under differing situations in the oral cavity. Furthermore, Ca^{2+} and HPO_4^{2-} ions usually diffuse to the tooth surface and may be lost, particularly in the presence of more severe demineralisation. Partial replacement by salivary ions may result in remineralisation occurring in the surface layers, and over time, even in the deeper regions of demineralisation within the lesion (**Fig. 2.7**).

Possible sequelae

It is apparent from the pH cycle diagram that, depending on the strength of the acid that is present, the frequency and duration of its production and the remineralisation potential in each particular situation, any one of the following sequelae can occur:

- the enamel may continue to mature
- chronic caries may develop – slow demineralisation with active remineralisation
- rapid (rampant) caries may arise – rapid demineralisation with inadequate remineralisation
- erosion may occur – very rapid demineralisation with no remineralisation at all.

At every food intake there will be an acid-induced demineralisation in areas of tooth surface beneath mature plaque.

If eating frequency of sugar is low, local fluoride concentration is high and salivary buffering is good, then the mineral loss will be reversible (that is, remineralisation will occur).

If eating frequency is high, local fluoride concentration is low and salivary buffering is poor, then demineralisation outweighs remineralisation. This situation is conducive to dental caries.

It is important for the clinician to identify whether the carious process is chronic or rapidly active as this will determine the degree of urgency and intensity of the control phase. The characteristics of chronic and rampant caries will be discussed.

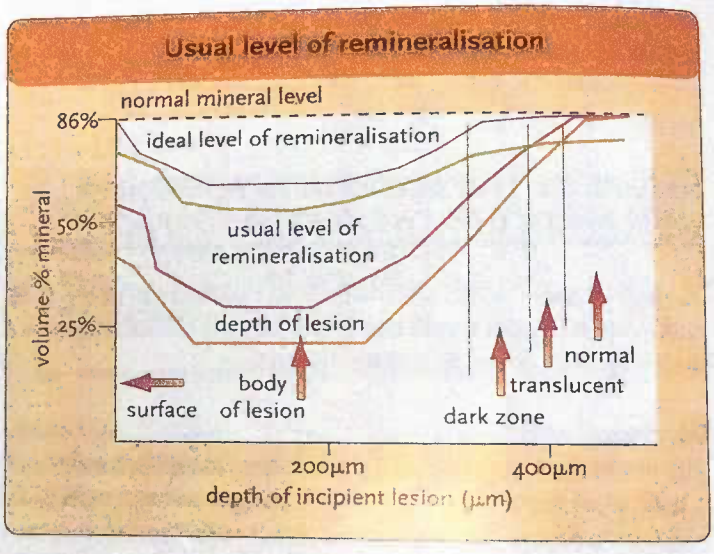

Fig. 2.7 Usual level of remineralisation. A schematic concept of the amount of remineralisation which may take place in enamel following demineralisation. The level will not achieve the theoretical normal but will be adequate to enhance the physical properties of the enamel.

THE PROGRESSING CARIOUS LESION

Early enamel lesion

The initial enamel lesion results when the pH level at the tooth surface exceeds that which can be counterbalanced by remineralisation, but is not low enough to inhibit surface remineralisation. The acid ions penetrate deeply into the prism sheath porosities, leading to subsurface demineralisation. The tooth surface may remain intact through remineralisation, which occurs preferentially at the surface due to increased levels of calcium, phosphate, fluoride ions, and buffering by salivary products (**Fig. 2.8**).

CD 24–37

Fluoride safety factors for adults

Be aware

Fluoride safety for adults

Maximum dose for adults

- 5 mg of fluoride per kilogram of body weight per day

The probable toxic dosage of fluoride ion is 5 mg of fluoride ion per kilogram of body weight per day. For the frail, chronically ill adult, this dosage should be considered high and prescribed doses kept well below this level.

Steps should be taken to minimise ingestion during application. In the office, use adequate suction. During home application, allow the patient to drool liberally over a sink. Have the patient spit out the excess for 1 minute after each application. The amount swallowed will then be well below those levels considered necessary to raise total blood levels to those considered likely to cause chronic toxicity.

The fluoride ion may be retained in the oral cavity for prolonged periods with a relatively slow rate of clearance by ingestion and absorption. It is therefore essential for dentists to prescribe the minimal dose necessary to gain the required result and instruct patients very clearly in the correct means of self-application.

Fluoride safety factors for children

Be aware

Fluoride safety for children

Maximum dose for children

- 5.0 mg of fluoride per kilogram of body weight per day

NOTE: A daily dose greater than 0.07 mg of fluoride per kilogram of body weight per day for children with developing teeth may result in fluorosis. Use topical fluorides with caution

The probable toxic dose of fluoride for children is 5 mg of fluoride ion per kilogram of body weight per day. Containers of fluoride tablets or drops used to supplement systemic fluoride intake should not contain more than 100 mg fluoride ion in total. They should be kept well out of the reach of young children to avoid accidental overdose.

Careful supervision of the amounts of fluoride toothpaste used daily is important because ingestion of more than 1 mg fluoride ion daily may lead to fluorosis. Regular fluoride-containing dentifrice holds up to 0.1% fluoride ion by weight, so a full brush head of paste contains approximately 1.5 mg of fluoride ion.

Particularly for small children, use a 'junior paste' which contains only 0.05% of fluoride ion by weight. Children under 3 years of age are likely to swallow all unused paste unless carefully watched, and those up to 6 years of age may regularly ingest approximately 30% of paste used.

Concentrated gels, and mouth rinses containing 0.2% NaF, should not be prescribed for young children even when infant caries has occurred. Localised application of varnish, or a more concentrated solution, may be undertaken by a dental professional.

Protective sealants

Fissure sealants are a proven protective measure against the development of carious lesions in pits and fissures in children. For the high-risk teenager or young adult patient there is increasing evidence for the benefits of sealing the pits and fissures on the occlusal surfaces of newly erupted teeth using either resin or glass–ionomer cement. Even if the seal is applied subsequent to some degree of demineralisation, the process will be arrested and bacterial activity is likely to become dormant (Chapter 11).

CD 38–48

CARIES RISK ASSESSMENT

Caries risk estimation for each patient leads to the development of a thorough picture of the patient's present and future dental health, with an understanding of the need for preventive or surgical intervention. Options for future dental management, and the anticipated cost in time and money of the various options, will be clearer.

The essential components of a proper caries risk-assessment programme include a careful oral examination, a detailed medical and dental history, diagnostic testing of the oral environment and an understanding of the patient's attitude and behaviour.

Examination

A thorough oral examination should assess the extent and location of incipient lesions, cavitated lesions and restorations. It should include an assessment of the activity status of the lesions and the condition of the restorations.

The following evidence should be carefully gathered and recorded:

- Coronal surfaces should be examined for change in colour and translucency and for the presence of cavitation. The use of a fine, sharp explorer is valuable in detecting surface roughness and cavitation within fissures but should be used with caution to avoid damage to a demineralised surface. Bite-wing radiography will reveal the presence of incipient or more advanced lesions under fissures or on proximal surfaces. Occasionally, transillumination may suggest early caries but this should be confirmed by other diagnostic means. Techniques using electronic fissure testing, lasers, ultrasound techniques and fibre-optic probes are being developed to enhance clinical ability to detect the early lesion.
- On the exposed root surface, incipient caries may be