



# Prevention and Interventions in Oral Health Care in Children

# 3

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## Learning Outcomes

By the end of this chapter, readers will:

- Be able to describe early childhood caries as well as common oral soft tissue pathologies and dental hard tissue defects and anomalies presenting in 0–3 year olds
- Understand preventive strategies for early childhood caries
- Understand management strategies for soft tissue pathologies in the 0–3 year old children

## 3.1 Early Childhood Caries

### 3.1.1 Definition and Aetiology

Early childhood caries (ECC) is defined as caries in any tooth in those aged under 6 years and so any caries seen in the 0–3 age group would be classified as ECC. The term ECC has replaced terms such as nursing caries or bottle caries in recognition of the complexity of the aetiology of the disease. Earlier definitions of ECC, which may still be seen, limited the term to caries in incisors, but this has now been superseded.

The disease is very prevalent worldwide with figures ranging from 12 to 98%, although a lack of standardisation around measurement of caries complicates this picture. It is however clear that the disease emerges from an early age, with one review finding that the mean prevalence globally at 1 year of age was 17%. In England, a study of 3 year olds in 2013 found that 12% had visible decay into dentine.

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**Table 3.1** Risk factors for early childhood caries

|  |
|--|
| Free sugar consumption   |
| Bottle feeding   |
| Extended breastfeeding beyond 2 years  |
| Developmental defects of enamel  |
| Socioeconomic factors such as deprivation, parental education levels and ethnicity |

ECC has multiple impacts on the child and family including the direct effects of pain and infection, disturbed sleeping and impacts on quality of life. The management of the disease may incur further impact on quality of life as well as costs both directly related to the cost of treatment, where children's dentistry is not covered by public funding and indirectly relating to the costs of accessing dental services including time off work for parents and carers. ECC has also been linked to poorer nutrition, development and growth of the child.

The aetiology of ECC is a complex interaction of biological, behavioural and socioeconomic factors changing the risk of caries development. The mechanism of caries is not specific to ECC but as with all caries relies on the demineralisation of tooth tissue from acids produced by bacteria fermenting carbohydrates outweighing the remineralisation that occurs in the oral cavity. The aetiological factors therefore tip the balance towards demineralisation and away from remineralisation.

Specific factors that are linked to increased risk are shown in Table 3.1.

Free sugars are defined as those that are added to foods as well as those naturally present in syrups, honey and fruit juices. The link between both amount and frequency of free sugar consumption and caries is well proven. In ECC, free sugars in both drinks and food play an important role in increasing caries risk and the method of intake is also important. The use of baby feeding bottles to consume drinks with free sugars has been linked with increased caries risk.

The link between extended breastfeeding and dental caries remains controversial, given the many benefits of breastfeeding and the global effort to increase breastfeeding rates. Studies to look at links between the two have been complicated by the potential confounding effects of other influences, most notably other food and drink intake. However, low quality evidence points to there being protection against caries with breastfeeding up to 1 year, no increased risk of caries with breastfeeding up to the age of 2 but an increased risk if breastfeeding continues beyond the age of 2, particularly when feeding is on demand and during the night.

### 3.1.2 Detection and Diagnosis

The mainstay of detection of ECC is visual examination and a typical appearance is shown in Fig. 3.1. Ideal detection conditions include the cleaning and drying of teeth with good lighting. Caries in enamel only is particularly likely to be missed if conditions are not ideal and this is unfortunate as this is a key stage for reversing caries. Sometimes achieving these ideal detection conditions may be difficult in the

**Fig. 3.1** Clinical appearance of early childhood caries



0–3 age group. The importance of early dental visits and approaches to examination are covered in Chap. 1.

Whilst visual examination will detect some caries, much caries remains undetected using this method alone. Most adjuncts to detection are unlikely to be feasible in this age group due to cooperation, but for some children it will be possible to consider radiographs, ideally bitewings and other caries detection adjuncts. Where bitewings are not possible, a dental panoramic tomogram (ideally on bitewing or reduced exposure setting) or lateral oblique views (Fig. 3.2a, b) may be considered.

Although detection of caries is an important element of diagnosis, full diagnosis of caries also relies on staging of caries, evaluation of the activity of a lesion (at present limited to whether the lesion appears to be arrested or not) and assessment of caries risk, especially in relation to risk of progression. Much of this diagnostic activity is no different in ECC than in caries in other age groups, although the caries risk assessment will need to take into account the factors listed in Table 3.1.

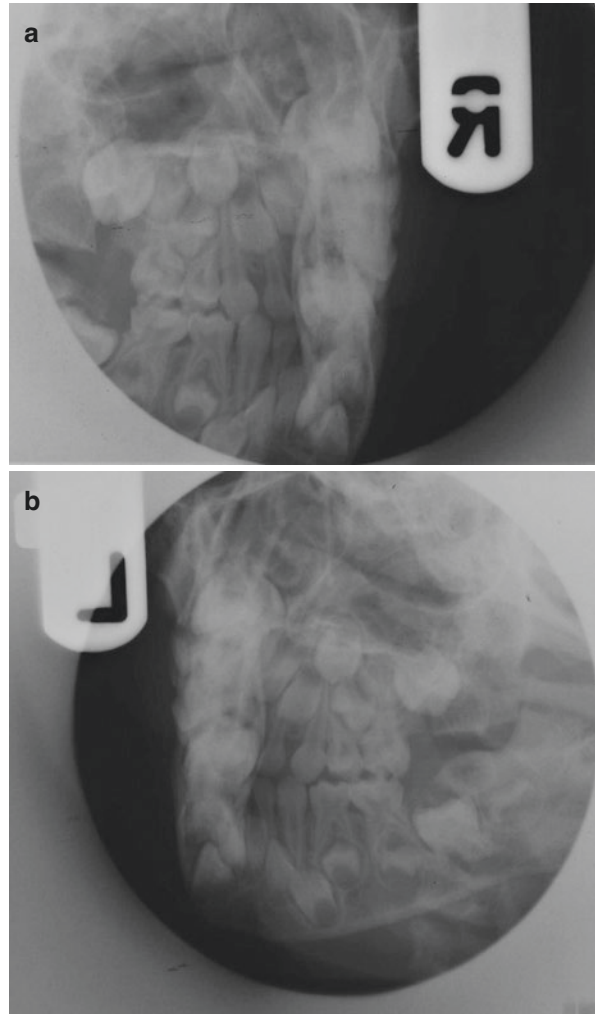
### 3.1.3 Management: Prevention, Remineralisation and Arrest

Prevention taken in its fullest sense and applied to ECC can mean maintenance of sound teeth (primary prevention), stopping or reversing progression of caries (secondary prevention) or repair of carious lesions (tertiary prevention). This section will concentrate on primary and secondary prevention.

The prevention of ECC depends on good risk assessment. This then allows more intensive prevention to be targeted at those at higher risk, whilst recognising there is a need for universal prevention for all. Prevention can be undertaken at the individual and family level or at a population level.

Population level interventions include:

**Fig. 3.2** (a, b)  
Radiographic appearance  
of early childhood caries



- Fluoride access through fluoridated water, milk or other foods and drinks such as juice and salt.
- Supervised toothbrushing schemes, especially in schools and nurseries and/or provision of free toothbrushes and toothpaste.
- Community based fluoride varnish application, which can often be undertaken by members of the dental team other than dentists.
- Actions to reduce sugar intake including healthier, tooth friendly choices being made available, disincentives for high free sugar drinks and foods (such as sugar taxes) and action by commercial manufacturers to reformulate foods to reduce free sugar content.

**Table 3.2** The pillars of caries prevention

|  |
|--|
| Dietary intake                           |
| Oral hygiene practices to control plaque |
| Access to fluoride                       |
| Fissure sealants                         |
| Routine dental examination               |

Although individual dentists and dental teams are unlikely to be involved in the planning or execution of many of these schemes directly, every member of the dental team has an important role in advocating for such schemes. In addition, other professionals involved with the care of children, especially health care workers, may have a role delivering individual level prevention and again dental teams should be involved in advocating for this, forming networks with those other professionals and potentially delivering training. The focus of this chapter, however, is on the role of dental teams in the direct care of patients and so we will now turn to this specifically in relation to prevention.

Individual level prevention is classically focussed on four areas, known as the ‘Pillars of Prevention’ but a fifth area is now also recognised. These pillars are shown in Table 3.2.

The first two of these pillars rely on achieving behaviour change. There are many theoretical models of how behaviour change could occur in individuals and much research is still ongoing about the best ways of achieving this practically. It is generally accepted that advice should increase knowledge, which could in turn influence attitudes which could then lead to behaviour change. However, in reality the links between these steps are complicated by other influences both internal and external to the individual. Many interventions seem to be able to change knowledge but then it is more difficult to influence attitudes and in turn behaviour.

Guidance from NICE in the UK has suggested that effective advice intending to create individual behaviour change should have several facets shown in Table 3.3.

### 3.1.3.1 Dietary Intake

The aim of any caries preventive intervention in relation to diet should be to reduce the amount and frequency of free sugars consumed. Despite the central nature of free sugar intake to the aetiology of ECC, there is little evidence to support the best ways of facilitating positive dietary changes in relation to caries, beyond the generic behaviour change advice already outlined.

It would seem sensible that if advice is to be personal it will be necessary to ascertain current dietary practices. A brief verbal discussion may highlight obvious areas for change, but this is unlikely to give a reliable and in depth assessment of dietary intake. Carers of children at higher risk of caries should therefore be encouraged to complete a more formal dietary assessment, usually in the form of a 3 day diet diary including all intakes, times and quantities. It can be useful to consider different environments that the child may encounter, so for example if the child spends different days with different carers or some days in nursery care, it will be useful to have diary days covering each of these settings. Given the importance of

**Table 3.3** Facets of individual behaviour change

|   |
|---|
| Be personalised to the individual   |
| Be positively framed (focus on the positive consequences of changing behaviour, rather than the negative consequences of failing to change behaviour) |
| Should be non-judgmental and ensure that individuals feel valued and empowered  |
| Should involve setting achievable and specific goals with the dental team member  |
| These goals should be shared and reviewed   |
| Take into consideration the social support available to individuals   |
| Involve provision of feedback on individuals' progress (e.g. update them on their risk status)  |

**Table 3.4** Dietary model for 1–3 year old

|  |
|--|
| The 5532 dietary model for 1–3 year olds   |
| Five portions per day of starchy foods (e.g. one portion = ½ medium potato, one slice of bread, three tablespoons of rice)   |
| Five portions per day of fruit and vegetables (e.g. one portion = ½ apple, five strawberries, two cherry tomatoes, one tablespoon of cauliflower)  |
| Three portions per day of dairy (e.g. one portion = 100 ml milk (full fat until 2 years, then semi-skimmed can be considered if child is eating well), two tablespoons of grated cheese) |
| Two portions per day of protein (e.g. three tablespoons of minced meat, two tablespoons of kidney beans, one slice of cooked meat)   |

nocturnal intakes in ECC, it will also be useful to remind the carers to include these and to indicate when the child is going to bed. Paper-based diaries are most commonly used, but many carers will now find electronic recording systems more convenient and it may be useful to explore the local availability of these.

Although the focus of dietary analysis and advice in the dental setting will be on free sugars, as health professionals, the dental team have a duty to look at the diet holistically.

Under 1 year old should ideally be exclusively breastfed until 6 months, from which point complementary foods should be introduced. The only drinks from 6 months to 1 year should be breastmilk, formula milk or preboiled water and an open top or non-valve free flowing cup should be introduced in this period.

For the 1–3 year old group, the British Nutrition Society has developed a 5532 model which satisfies the UK and WHO dietary guidelines (See Table 3.4). From the age of 2, children can begin to transition from this approach towards the normal dietary guidelines, which they should meet by the age of 5. UK guidelines also recommend supplementation of the diet with Vitamin A and D for those under 5 year olds (unless under 1 and consuming formula milk). Specific advice relating to free sugars should be offered (see Table 3.5).

### 3.1.3.2 Oral Hygiene Practices

The importance of good oral hygiene practices in relation to ECC mainly relates to the delivery of fluoride through toothpaste. However, there are benefits to disrupting plaque in its own right. The brushing of teeth is the key behaviour in terms of oral hygiene in this age group and other oral hygiene aids such as flossing are not

**Table 3.5** Dietary guidelines for 1–3 year olds

|   |
|---|
| Free sugars advice  |
| Limit number of intakes containing free sugars to 3 per day (ideally consumed at mealtimes)   |
| Keep the hour before bed and overnight sugar free   |
| During introduction of complementary foods, foods should be varied in taste and texture, low in free sugars and have no sugar added |
| Good complementary foods include fruit and vegetables as well as unsweetened cereals and yoghurt                                    |
| Less than 5% of energy intake should be from free sugars (three cubes in 1–3 year olds)   |
| Encourage the use of sugar free medicines where possible  |

**Fig. 3.3** Smear of toothpaste

recommended for young children. Brushing should begin as soon as the first tooth erupts and should be done with a small soft brush. Some experts advocate wiping the gums of babies before their teeth erupt with a soft damp cloth which may aid in forming good habits. However, there is little evidence to support this practice. Brushing should be twice a day, with one episode before bedtime and the other at another time.

A smear of toothpaste containing at least 1000 ppm fluoride should be used (as illustrated in Fig. 3.3) and carers of children at higher risk should be advised to use 1350–1500 ppm fluoride pastes, unless there are concerns about the child eating or licking the toothpaste. There is no evidence for any particular technique and so carers should be encouraged to ensure all teeth are brushed, concentrating especially on molars as they erupt. Children should not be allowed to rinse their mouths after toothbrushing, to ensure that toothpaste residue is not washed away.

In this age group, children should have their teeth brushed by an adult but achieving good brushing can be difficult due to cooperation in some children. Advice to help with difficult brushing is shown in Table 3.6.

**Table 3.6** Brushing advice

|   |
|---|
| Novelty brushes may be motivational for a young child   |
| Parents should praise good behaviour with brushing and dental teams can reinforce this  |
| Simple rewards such as a star chart, where children receive star stickers for brushing are often used for older children, but might be useful towards the older end of the 0–3 age group  |
| Modelling may be helpful where older children or carers are also brushing at the same time  |
| Allowing the child to brush their own teeth first, followed by the carer then repeating the brushing for the child may be helpful in motivating the child.  |
| Different carers will find that different positions may work better or worse in their individual circumstances but some ideas would include kneeling in front of the child, from behind the child whilst holding the child (this mimics the same actions the carer will be used to making in their own mouth and allows the child to be held comfortably) or with the carer sitting on the floor, positioning the child lying down in the carer's lap |

### 3.1.3.3 Access to Fluoride

As described earlier, there are several ways of delivering fluoride at a population or community level but this section will concentrate on delivery of fluoride at an individual level. In this age group, topical fluoride will come from two main sources, 'self' applied via toothpaste and professionally applied via fluoride varnish.

As described in the oral hygiene section, 1000 ppm fluoride toothpaste is recommended for all 0–3 year olds with 1350–1500 ppm fluoride toothpastes advised for high risk children. Historically children's toothpastes contained around 400–500 ppm fluoride and some concern has been raised that using these higher concentrations will lead to an increased risk of fluorosis. However, increased ingestion of fluoride is much more related to the quantity of toothpaste rather than its fluoride concentration and so provided the advice of using a smear of toothpaste is followed, the increased risk of fluorosis is minimal and outweighed by the caries preventive benefits. Other important factors in maximising fluoride delivery via toothpaste already mentioned include using toothpaste twice daily and spitting out and not rinsing out after brushing.

The use of fluoride varnish is a very important aspect of a preventive plan for any 0–3 year old child. There is strong evidence for a preventive effect in the primary dentition and a Cochrane Review shows a 37% reduction in caries increment when applied two or more times per year. Many countries therefore advocate applying fluoride varnish universally in children (i.e. for those at both low and high caries risk) with an increased frequency beyond twice yearly for those at higher risk.

There are several different formulations of fluoride varnish available on the market, although not all are licensed for caries prevention. Many of the formulations are based on a concentration of 22,600 ppm fluoride and it is this concentration on which most of the evidence is based. In addition, many of the formulations contain colophony as one of the constituents, which there is an increased risk of reaction to in those who have severe allergies or asthma. In addition, all fluoride varnishes are contraindicated in patients who have active oral ulceration.

Application should be undertaken with a microbrush after the teeth have been dried with cotton wool or air syringe. Only a small volume should be used and some manufactures provide dispensing pads to ensure that too large a volume is not



dispensed. Different manufacturers vary in their post-placement instructions and these should be followed but in general, 30 minutes avoidance of food, drink and rinsing should be advised.

With children in the 0–3 age bracket it is important to adopt a detailed behaviour management strategy (described in Chap. 4), even for this quite simple treatment and in particular tell-show-do should be used, especially for the drying aspect. The flavour of the varnish can vary by manufacturer and this may be an important consideration for some children.

### 3.1.3.4 Fissure Sealants

Whilst fissure sealants are an important aspect of prevention for older children, their use in 0–3 year olds is very limited. There is evidence, perhaps not surprisingly, to suggest that fissure sealants in under 6 year olds can be effective in caries prevention, but many under 3 years olds are unlikely to have sufficient cooperation to allow placement of effective sealants. Nonetheless, where a child does have sufficient cooperation and a high risk of caries, fissure sealants can certainly be included in the preventive plan. The technique is described in Chap. 5.

#### Clinical Tip

Prevention of early childhood caries relies heavily on behaviour change to ensure good diet and good oral hygiene supplemented by fluoride access

### 3.1.3.5 Routine Dental Examination

Regular attendance in a dental setting is important both for early detection of disease and for providing an opportunity to deliver and reinforce preventive messages and interventions. Dental examination of the paediatric patient is covered in Chap. 1.

### 3.1.3.6 Secondary Prevention

Where early caries is detected, there is an opportunity to try to arrest the process and also to remineralise. Many of the primary preventive measures will be of value in secondary prevention too as changes to diet, good oral hygiene and increased fluoride availability will all favour remineralisation. Fissure sealants also have their place and there is now good evidence that sealants can be placed over caries and this will halt the progression of caries. This is discussed in more detail in Chap. 5.

Finally, the use of silver diamine fluoride (SDF) has a growing evidence base in the secondary prevention of cavitated caries. This intervention is a liquid varnish, applied in much the same way as fluoride varnish. The varnish will stain readily and so application of petroleum jelly to the lips and extensive use of protective equipment for the patient and operator are recommended. The British Society of Paediatric Dentistry has published resources to support the use of SDF and recommends six monthly application of 38% SDF. If successful, the caries being treated will turn a very dark black colour (See Fig. 3.4). Carers should be warned of this colour change ideally with the aid of photographs and this should be included in the consent

**Fig. 3.4** Teeth with caries 3 months after silver diamine fluoride with potassium iodide application (by kind permission of Mr. Oliver Sumner)



process. Some preparations of SDF now also include potassium iodide which reduces the amount of discoloration of the caries, but this has been reported to lower the effectiveness of SDF in arresting the caries.

### **3.1.4 Management: Operative Interventions Including Extractions**

Where caries has cavitated and a secondary preventive process is either unsuccessful or is not thought to be appropriate by dentist or carers, operative intervention may be required. Where this is necessary, often in 0–3 year olds cooperation will be insufficient to allow treatment to be completed successfully in the chair. In these cases, children at the upper end of this age scale may only rarely be considered for sedation, which in the UK would usually be inhalation sedation with nitrous oxide, although oral sedation would be considered in other countries. The majority of cases will need to be completed under general anaesthetic. Pharmacological behaviour management is considered in more detail in Chap. 4.

Treatment options for vital primary teeth include both direct plastic restorations and preformed metal crowns. Non-vital teeth may be treated using pulpotomies or pulpectomies, but in many cases extractions will be warranted as well as in cases where vital teeth are unrestorable. In some general anaesthesia services, restorations of primary teeth may not be offered due to service pressures. All of these operative interventions are described in Chap. 5.

Where extractions are required, extraction patterns will necessitate careful planning to minimise future risk of problems with the developing dentition and malocclusion. In particular balancing extractions. The evidence for balancing approaches is however limited. Early extraction of primary molars may well lead to crowding problems in the permanent dentition. Extraction patterns in primary teeth are considered further in Chap. 5.

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## 3.2 Soft Tissue Pathology

### 3.2.1 Viral

#### 3.2.1.1 Primary Herpetic Gingivostomatitis

Primary herpetic gingivostomatitis is one of the commonest oral mucosal related presentations in this age group and can be quite distressing for both the child and parents. This infection, usually resulting from Herpes Simplex Virus I, often presents in the 0–5 year age range. Its incubation period is around 1 week and symptoms typically last up to another 10 days. Diagnosis is usually possible based on the clinical signs and symptoms alone, which may include:

- Widespread oral ulcers with inflamed margins (preceded briefly by grey blisters) which can occur in most oral sites (see Fig. 3.5)
- Pain from the oral mucosa which may limit oral intake of both solids and fluids
- Excessive salivation and drooling
- Malaise and fever
- Cervical lymphadenopathy

Management is usually conservative and includes analgesic advice (ideally using paracetamol at an appropriate dose), advice to ensure good hydration, a soft diet and bed rest. Chlorhexidine is sometimes recommended to assist with oral hygiene and a gel or swab dipped in mouthwash may be appropriate delivery methods. There is only very limited evidence to support the use of antivirals, with acyclovir shown to be effective in reducing the duration of signs and symptoms if it is employed during the first 2–3 days following symptoms. Given the limited evidence, it is suggested that this therapy is reserved only for severe cases. It is prudent to review suspected cases after a short interval of around 1 week to check for resolution. If resolution is not seen, referral to a specialist should be made.

#### 3.2.1.2 Mumps

Although children with mumps are unlikely to present undiagnosed to the dental practice, the most recognisable sign of this paramyxovirus infection is swollen parotid glands. The disease can occur in under 4s although incidence increases throughout childhood. The disease is highly contagious and is also notifiable in many countries. Vaccination programmes in most countries have reduced the prevalence, although increasingly prevalent attitudes against vaccination may have an impact on vaccination rates.

**Fig. 3.5** Primary herpetic gingivostomatitis



### **3.2.1.3 Measles**

Measles is another highly contagious paramyxovirus infection. The classic sign is an erythematous rash spreading to the whole of the body. However, just before the rash appears, many cases will exhibit Koplick's Spots, small white papules on an erythematous base occurring mainly on the buccal mucosa. The incidence of measles is increasing as vaccination rates decline, although the commonest age group affected are 5–10 year olds.

### **3.2.1.4 Hand Foot and Mouth Disease**

This infection is usually caused by Coxsackie A and the commonest age group to be affected are young children. The symptoms can start with fever and malaise, a cough, sore mouth and abdominal pains. Intra-orally vesicles quickly rupture to form ulcers which can occur anywhere on the oral mucosa and may vary in size (See Fig. 3.6). Shortly afterwards, blisters, which may rupture, occur on the hands and feet. As with primary herpetic gingivostomatitis, diagnosis is based on clinical signs and symptoms and management is conservative with appropriate analgesic and fluid advice. There is no benefit from antiviral treatments.

**Fig. 3.6** Oral ulceration in hand foot and mouth disease



### 3.2.1.5 Papilloma

The squamous cell papilloma has been associated with the human papilloma virus. The usual manifestation is as a multi-leafed exophytic growth that appears the same colour as the surrounding mucosa or whiter. The lesion is benign and slow growing but can be irritating, especially if traumatised. Examination of the fingers will sometimes reveal warts there too. Although perhaps more common in adults, they are frequently seen in under 10 year olds also. Papillomas are usually excised but in the under 4s this will probably require a referral and a general anaesthetic and the risks will need to be weighed against the benefits in each case.

### 3.2.2 Bacterial

Children with bacterial infections rarely present to the dental team, either because they present elsewhere or the infections are very rare. Perhaps the commonest presentation is impetigo, caused by staphylococci or streptococci. This is a skin infection but can present on the lips and angles of the mouth with vesicles and crusting lesions. Active treatment is not usually indicated although severe cases sometimes warrant antibiotic prescription. Streptococci can also cause scarlet fever, which is known for its skin rash but also manifests with a 'strawberry' tongue with swollen papillae and a white coating. Antibiotics are usually indicated for the management of scarlet fever.

### 3.2.3 Fungal

The main fungal infection intra-orally, as in all age groups, is *Candida* with the commonest presentation in the under 3s being acute candidiasis (thrush) usually caused by *Candida albicans*. Acute candidiasis appears as a white plaque which can easily be wiped off to leave erythematous mucosa. In addition, angular cheilitis with reddening of the angles of the mouth is often seen simultaneously. Neonatal acute candidiasis is thought to be transferred during birth. Other infants may develop candidiasis if they are immunocompromised or post-antibiotic use. Topical treatment with miconazole gel is usually sufficient but the dose should be checked carefully with appropriate formularies. Further details on fungal infection in the paediatric patient are described in Chap. 15.

#### Clinical Tip

Many soft tissue infections will be self-limiting and management will consist of reassurance and symptomatic relief

### 3.2.4 Developmental

#### 3.2.4.1 Bohn's Nodules and Epstein Pearls

Many parents will bring a baby for their first dental visit concerned about white lumps on the gums, often thinking these are teeth which are erupting too early. Examination reveals hard white nodules on the alveolar ridge or in the midline of the palate (See Fig. 3.7). These are cysts resulting from proliferation of the developmental epithelium. Some literature will separate gingival cysts of newborn/dental lamina cysts (deriving from Rests of Serres) from Bohn's nodules (ectopic mucous salivary glands) but many will describe both as Bohn's nodules. Epstein pearls arise at the junction of the soft and hard palate in the midline and derive from non-odontogenic epithelium. They are hard, raised nodules, formed from keratinising cysts along developmental lines of fusion occurring in approximately 80% of all newborn

**Fig. 3.7** Bohn's nodules



infants. All of these resolve spontaneously a few months later and parents simply need to be reassured.

#### **3.2.4.2 Eruption Cysts**

Eruption cysts can occur with both primary or permanent teeth that are erupting and so are seen in the 0–3 age group as the primary teeth erupt. The cyst is characteristic in its blue appearance and will appear on the gingival crest overlying an erupting tooth. Nearly all of these cysts will burst spontaneously as the tooth erupts and do not require any intervention.

#### **3.2.4.3 Congenital Epulis**

Congenital epulis is rarely encountered and these tend to present in the maxilla and in females. The appearance is of a swelling on the alveolus, usually in the midline. Although the lesions can resolve spontaneously, they are often excised, particularly if they are interfering with feeding.

#### **3.2.4.4 Haemangioma**

Haemangiomas are vascular tumours, mainly occurring in the head and neck, although intra-oral presentation is rare. The appearance can vary considerably with both smooth and lobulated forms described although all will appear dark red and will usually blanch. The size can also vary considerably. Spontaneous resolution usually occurs and is often the preferred management option due to the risks of surgery on such vascular lesions. However, in some cases where the lesion interferes with function surgery may be considered by specialist teams.

#### **3.2.4.5 Other Developmental Cysts**

There are a wide variety of rarer developmental cysts, which may be noticed in the course of an oral examination. It is beyond the scope of this book to describe all of these in detail and onward referral of any children with suspected or unknown diagnoses is recommended. These cysts may include:

- Dermoid and Epidermoid cysts
- Thyroglossal duct cysts
- Lymphoepithelial cysts
- Nasopharyngeal cysts

### **3.2.5 Ulcers**

Most oral ulceration in 0–3 year olds is traumatic in origin, although this is only likely to occur in the upper end of this age range, given the diet and lack of teeth in younger children. Traumatic ulcers can vary in size depending on the nature of the trauma but will tend to initially have an erythematous base followed later by a yellow appearance. Traumatic ulcers should heal within 2 weeks and if there is any doubt as to the diagnosis, a review at this stage can be useful to ascertain healing

and rule out other causes. Topical anaesthetic gels tend to be poorly accepted and difficult to use in this age group but may provide some symptomatic relief.

Recurrent aphthous ulceration (RAU) usually presents at later ages but it is possible that it may present in younger children. This topic is covered in detail in Chap. 15 and the principles described there apply equally to the 0–3 year old.

### 3.2.6 Other Soft Tissue Conditions

A large number of other soft tissue conditions may present in the 0–3 year old, but they are very rare in this age group and so are not considered here. These may include epidermolysis bullosa, erythema multiforme, geographic tongue, pyogenic granuloma, giant cell granuloma and tumours including malignancies. Some of these will be considered in more detail under older age groups in Chap. 15.

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## 3.3 Hard Tissue Pathology Mainly Developmental

Whilst most of the hard tissue pathologies are described in detail in Chap. 13 and many of the observations in that chapter apply in the younger age group, a few differences for 0–3 year olds and the primary dentition are described in this section.

### 3.3.1 Tooth Number

#### 3.3.1.1 Hypodontia

Congenital absence of primary teeth is rare with prevalences usually reported at less than 1% although some higher prevalences have been found in specific populations. The most commonly absent primary teeth are incisors. Definitive diagnosis usually relies on radiographs but in the 0–3 age group, where cooperation does not allow satisfactory radiographs, a definitive diagnosis of hypodontia may not be possible. Although permanent hypodontia is associated with many syndromes (see Chap. 13), syndromic primary tooth hypodontia is often not reported with ectodermal dysplasia being the only notable exception.

Management of primary tooth hypodontia is often simply to monitor development with no active intervention. In more extreme cases, such as in ectodermal dysplasia, where several teeth are missing, a removable prosthesis can be considered but usually children in the primary dentition find tolerating these very difficult. If a denture is made, frequent replacements will be required as growth progresses and especially once the child enters the mixed dentition phase.

It is useful to remember that if there is hypodontia of a primary tooth, there is a greater risk that the permanent tooth will also be missing. In 50% of these cases, the permanent successor will be absent. It can be useful to warn parents of this when hypodontia of a primary tooth is noted.



### 3.3.1.2 Supernumerary Teeth

Supernumeraries in the primary dentition are very rare, being present in less than 1% of the population. Those that do occur are often supplemental in nature, appearing the same in form and size as adjacent teeth although conical mesiodens forms are also seen. Both forms often erupt within the normal arch. In reality, assigning non-supplemental supernumeraries to primary and permanent dentitions may be artificial, given that they may be present but unerupted whilst the primary dentition is present, but then manifest when permanent teeth fail to erupt. Delay to permanent eruption and ‘permanent’ supernumeraries are covered in depth in Chaps. 9 and 13.

Nonetheless, primary supplemental teeth are very often simply left for monitoring. They can occasionally cause problems with eruption of underlying permanent teeth and extraction may be indicated in these circumstances.

### 3.3.1.3 Gemination/Fusion

In contrast to hypodontia and supernumeraries, gemination and fusion are more common in primary teeth than permanent teeth. The prevalence has been reported at between 0.1 and 3%. Whilst both gemination and fusion will result in the same clinical endpoint, a double tooth, the aetiology is a splitting of a single tooth germ for gemination, whilst it is a joining of two tooth germs in fusion. The diagnosis is therefore usually to determine if there are a normal number of other teeth in the arch (suggesting gemination) or one less than expected (suggesting fusion). This diagnostic aid is however complicated if it is fusion with a supernumerary tooth.

Clinically the teeth can vary from one large tooth, through various depths of groove to two almost separate teeth joined only through a thin section of enamel (See Fig. 3.8). In the primary dentition, management usually starts with monitoring, although if possible grooves will often be sealed as they are sites for potential caries

**Fig. 3.8** Geminated primary tooth



development. Deep grooves may be restored with composite providing cooperation is sufficient. Double teeth may delay the eruption of permanent teeth and so this should be closely monitored and extraction of the double tooth may be necessary.

### 3.3.2 Tooth Structure

#### 3.3.2.1 Developmental Defects of Enamel

Developmental defects of enamel (DDE) in primary teeth have been underinvestigated compared to permanent teeth but are being increasingly recognised as a problem. Reported prevalence rates have varied widely between 4 and 82% but the prevalence may be masked by caries developing in these teeth, making diagnosis of enamel defects impossible.

Given that amelogenesis for primary teeth occurs intra-utero until 12 months postnatally, a number of factors may play a role in the development of DDE. The factors can include:

- Maternal Vitamin D deficiency in pregnancy
- Maternal infections in pregnancy
- Maternal smoking, alcohol use and certain medications
- Preterm birth
- Trauma at birth
- Coeliac disease
- Chronic renal and liver disease
- Infections and fever
- Nutritional deficiencies (especially vitamin D)
- Certain medication use
- Amelogenesis imperfecta (see below)

Amelogenesis imperfecta (AI) is a group of genetic conditions which result in DDE. The details of epidemiology, aetiology and classification are described in Chap. 13 but it is useful to remember that the primary dentition is usually much less severely affected than the permanent dentition. The generic management strategies for DDE described below equally apply to AI.

The influence of all of the above factors on amelogenesis can result in either hypomineralisation, where the quality of enamel is reduced and the enamel may be softer, often with a yellow/brown appearance or hypoplasia, a lack of enamel which may appear as pits, grooves or thin and missing enamel (see Fig. 3.9). The link between DDE and caries is now well established with increased caries risk where DDE are present.

Management of these teeth will depend on cooperation and initially in this age group monitoring and fluoride varnish may be all that is possible. Intensive prevention, treating the patient as high risk for caries is vital. Ideally, when cooperation allows, mildly affected molar teeth should be fissure sealed whereas molar teeth

**Fig. 3.9** Hypoplastic primary molar teeth



with more significant defects will benefit from a preformed metal crown. Anterior teeth may require composite additions, or possibly strip crowns where more of the crown is affected.

**Clinical Tip**

Primary teeth with enamel or dentine defects should be protected as early as possible with sealants and preformed metal crowns

### 3.3.2.2 Developmental Defects of Dentine

Whilst defects of dentine are less common than defects of enamel, the clinical severity is usually greater in the primary dentition.

Dentinogenesis imperfecta is a hereditary condition, with autosomal dominant inheritance and a prevalence of between 1:6000 and 1:8000. Three genes appear to be involved: COL1A1, COL1A2 and DSPP. Using the Shield's classification, three types of dentinogenesis imperfecta are recognised with two further conditions affecting the roots only, dentine dysplasia I and II which are part of the same group. The features of the different types are shown in Table 3.7. Clinically, the appearance of the teeth is translucent with blue/grey or brown shades. The crowns are usually bulbous (See Fig. 3.10). Radiographically pulp chambers are small and roots narrow with the possibility of obliteration of the pulp chamber and root canal.

Management relies on protecting affected molar teeth as soon as cooperation allows with preformed metal crowns. Milder forms may be treated with fissure sealants. Teeth will often exhibit significant tooth surface loss to the extent that extractions may be the only viable option and other teeth will develop abscesses, in which case extraction is again the only treatment possible. Usually the permanent dentition is much less affected and treatment is covered in Chap. 13.

**Clinical Tip**

Children with DI will usually present to practice once the primary teeth starts to erupt. Early referral to a specialist service is advised

**Table 3.7** Features of different types of dentinogenesis imperfecta and dentine dysplasia

| Shield's classification       | Clinical features  | Other notes   |
|-------------------------------|--|---|
| Dentinogenesis imperfecta I   | Amber translucent crowns<br>Short roots often with pulp canal obliteration | Occurs alongside osteogenesis imperfecta<br>Variable expression with some teeth hardly affected |
| Dentinogenesis imperfecta II  | As per type I but crowns also bulbous                                      | No osteogenesis imperfecta<br>All teeth affected  |
| Dentinogenesis imperfecta III | Variable including features of type I but also 'shell like' teeth          | One specific tri-racial population originating from Maryland/Washington DC                      |
| Dentine dysplasia I           | Tapering conical roots, pulp canal obliteration, multiple abscesses        |   |
| Dentine dysplasia II          | As per type I but permanent dentition not affected                         |   |

**Fig. 3.10** Dentinogenesis imperfecta in the primary dentition

X-linked hypophosphatemia (vitamin D resistant rickets) is another condition that exhibits developmental dentine defects. The clinical presentation is usually with spontaneous dental abscesses (see Fig. 3.11) and in these cases extractions are required. Early protection of the teeth with preformed metal crowns, as with dentinogenesis imperfecta, is advised.

### 3.3.2.3 Abnormal Morphology

Although other morphological abnormalities such as invaginations, evaginations, macrodont teeth and taurodontism are all possible in the primary dentition, they are exceedingly rare and the management options are usually to monitor until exfoliation or extract. These anomalies described in more detail in Chap. 13.

## 3.3.3 Eruption

### 3.3.3.1 Neonatal and Natal Teeth

The first presentation to a dentist for some children may occur within the first weeks and months of life if natal or neonatal are present. Natal teeth are those that are

**Fig. 3.11** Spontaneous abscess in patient with X-linked hypophosphatemia



**Fig. 3.12** Natal teeth in a baby with cleft lip and palate



already present at birth, whereas neonatal teeth erupt in the first month after birth (See Fig. 3.12). The teeth are almost always lower incisors and have very little root development. Although the erupting teeth can be supernumeraries this is rare and most teeth will be part of the normal series, simply with early eruption. Radiographs are not usually taken in this very young group of patients and so the differentiation is unlikely to become clear until a much later age.

A decision needs to be made as to whether to extract or leave the premature tooth. This depends on how mobile the tooth is (and therefore whether there is a risk to the airway), if the tooth is causing any trauma and if the tooth is interfering with breastfeeding or other feeding. If extraction is indicated, this is usually a simple procedure undertaken without anaesthesia, either with forceps, a clip or simply by holding the tooth with gauze.

### 3.3.3.2 Delayed Eruption

Although obstructions are the commonest cause of delayed eruption in the permanent dentition, these are rare in the primary dentition. Delayed eruption of primary teeth has been linked with premature birth, low birth weight, malnutrition and is a feature of several conditions such as Down syndrome, Turner's syndrome, hypopituitarism and hypophosphataemia. Primary failure of eruption, where a cause is not evident is also a possibility in the primary dentition. Most of these conditions simply cause a delay and reassurance is all that is required. However, where there is an obstruction, such as a supernumerary, this may require removal. Where a tooth completely fails to erupt, surgical removal may be required, although this may be delayed to allow further development of the dentition and increased cooperation. Any surgical interventions will carry the risk of damage to developing teeth and these risks must be carefully weighed against the benefits.

### 3.3.3.3 Premature Exfoliation

Premature exfoliation of primary teeth is always a cause for concern as there are a number of underlying systemic causes which need to be excluded. In some cases, the premature exfoliation may be the presenting feature of the condition and prompt onward referral is a key aspect of these cases. Conditions which may involve premature exfoliation include neutropenias, hypophosphatasia, hypophosphatemia, Langerhans cell histiocytosis Ehlers-Danlos syndrome, Papillon-Lefevre syndrome and Chediak Higashi syndrome.

### 3.3.4 Bony Pathology

Many bony pathologies of the jaw may be present in 0–3 year olds. Some of these are listed below but the details are included in Chap. 15 alongside considerations for older children. Perhaps the commonest is the dentigerous cyst, arising from the reduced enamel epithelium. Other cysts include radicular cysts, keratocysts, nasopalatine duct cysts and haemorrhagic bone cysts.

Tumours of the jaw include ameloblastomas (rare in children), ameloblastic fibromas, sarcomas, ossifying fibromas, giant cell granulomas and Langerhans cell histiocytosis. Finally, the osteodystrophies include fibrous dysplasia and cherubism.

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