Edited by
Angus C Cameron
Richard P Widmer

Handbook of PEDIATRIC DENTISTRY

Fourth Edition

Foreword by
Roger K Hall

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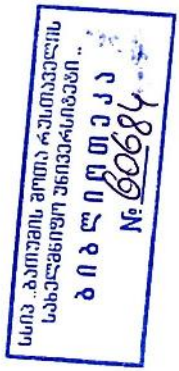
Handbook of PEDIATRIC DENTISTRY



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Handbook of PEDIATRIC DENTISTRY

4TH EDITION

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Foreword

Handbooks on paediatrics and more recently, paediatric dentistry, have been produced for some years now, by paediatric hospitals and departments of paediatric dentistry in Australia, to assist trainee staff with ready-reference access to advice on common paediatric problems.

This new more comprehensive publication is the outcome of an enthusiastic response to the first *Handbook of Pediatric Dentistry* from the Department of Paediatric Dentistry at Westmead Hospital Dental Clinical School and the University of Sydney, edited by Angus C Cameron and Richard P Widmer. In this new publication, there are additional contributions from members of the Australasian Academy of Paediatric Dentistry and the Australian and New Zealand Society of Paediatric Dentistry, and the authors have included many tables and colour plates. The addition of these good quality colour illustrations of dental abnormalities is an important adjunct to the written descriptions of disorders and facilitates diagnosis.

Most orofacial disorders in children have a developmental basis. Lesions or conditions may be present at birth, or become evident soon after. They may appear, change character or arrest and regress (or disappear) as growth proceeds. Certain diseases are inherited and others may be acquired from parents, siblings or other children, but for many, the actual aetiology is still unknown. While we may not yet know the precise cause of many conditions, we do know how to manage them – often in close cooperation with our paediatric medical and surgical colleagues. This handbook sets out, in concise form, the essentials of management of children with, e.g. oral and dental trauma, dental caries, oral infections, cardiac disease, endocrine, haematological and oncological disorders, and those who have received organ transplants.

Dental practitioners and students need information on all of these areas of dental care for children, on a daily basis. This handbook provides the basic information necessary in a clear and readily retrievable form, at the same time providing guidance on the most appropriate texts or journals, where more detailed information may be found.

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Preface

After 15 years, our undergraduate student notes have expanded to become a contemporary handbook, now in its 4th edition and translated into eight other languages. We are extremely grateful to all the members of the Australasian Academy of Paediatric Dentistry and our international colleagues for their support in the publication of this book. As we complete the final preparation for this 4th edition, it is time to reflect upon what the contemporary goals for paediatric oral health care are for clinicians, patients and the wider community.

For the clinician, it is striving to practice with a mindset that is tempered and improved by life-long learning and practice that brings with it the immense, spiritual satisfaction of helping others. For the child patient who has a "good" experience of dentistry, there is an everlasting positive memory that hopefully brings about a desire for maintaining their oral health as they become adults.

As far as the community is concerned, it is now recognised, more than ever, that oral health is part of total health and cannot be ignored. A recent declaration from the United Nations provides a new impetus for this integration to be actively promoted by dental clinicians.

The United Nations High-lever Meeting on Prevention and Control of Non-communicable Disease (NCD), held in New York in September 2011, was a truly historic occasion as it was the first time that oral diseases were recognised as a public health problem. The summit called for sustained action to address the rising burden of non-communicable disease such as diabetes, cancer, cardiovascular and respiratory diseases, with oral diseases as an integral part. Oral diseases share many of the same determinants and risk factors as the four main targeted non-communicable diseases, that is, high sugar and salt intake, tobacco and alcohol use, lack of physical activity, and also trauma and violence. They are part and parcel of the global silent tsunami of chronic disease. It is widely accepted that there is strong evidence that the general health status of young children directly influences their health, development and wellbeing throughout life. This is also true for oral health. Dental problems in early childhood have been shown not only to be predictive of future dental problems but also to influence general growth and cognitive development, by interfering with sleep, appetite and eating patterns, and leading to poor school behaviour and negative self-esteem. The impact is more significant on children from lower socioeconomic groups. Therefore, it is crucial that oral health is seen as part of total health.

Benzian *et al.*, 2012 Editorial Journal of Public Health Dentistry, 72: 91–93.

What is our role in society? Paediatric dental clinicians need to be integral in this drive to raise awareness of the problems to which poor oral health leads. There needs to be a greater involvement with our local "health community" and regular communication with all our other health colleagues, health bureaucrats and government organizations. This contact may be in the form of a "care plan" for a patient, sent to their general medical practitioners; or outlining concerns about a government programme

to bureaucrats. Whatever the activity, it will help raise awareness of paediatric dental health. While there is a perception that we are only paediatric dental *surgeons*, we should also act as paediatric oral *physicians*. In some ways, this concept is understood better by our fellow paediatric health practitioners. It embraces the aspiration of total patient care, a model expressing the broad scope of child development and oral health.

It is our hope that this, the 4th edition of our Handbook, can go some way to providing the contemporary evidence and clinical practice necessary to address these huge challenges, and help support and inspire an improvement in oral health globally, placing the importance of oral health at the forefront of our children's total health.

Angus C Cameron and Richard P Widmer
Westmead, Sydney, Australia
October 2012

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Our families and close friends must not go unmentioned for their quiet support and encouragement and finally, we would like to thank our child patients, the responsibility of whose care we are entrusted. They give us wonder as we watch them grow, the joy in our daily work and the motivation for our endeavours.

1 The philosophy of paediatric dentistry

Richard P Widmer, Angus C Cameron

What is paediatric dentistry?

Paediatric dentistry is a specialty based not on a particular skill set, but encompassing all of dentistry's technical skills against a philosophical background of understanding child development in health and disease. This new edition of the handbook emphasizes again the broader picture in treating children. A dental visit is no longer just a dental visit – it should be regarded as a 'health visit'. We are part of the team of health professionals who contribute to the well-being of children, both in an individual context and at the wider community level. Children often slip through childhood to adolescence in the blink of an eye and family life is more pressured and demanding. Commonly, children spend more time on social media than interacting directly with family and friends and, more than ever, the major influences on their lives, come from outside the family.

The pattern of childhood illness has changed and with it, clinical practice. Children presenting for treatment may have survived cancer, may have a well-managed chronic disease or may have significant behavioural and learning disorders. There are increasing, sometimes unrealistic, expectations, among parents/carers that the care of their children should be easily and readily accessible and pain-free and result in flawless aesthetics.

Caries and dental disease should be seen as reflective of the family's social condition and the dental team should be part of the community.

> Your [patients] don't have to become your friends, but they are part of your social context and that gives them a unique status in your life. Treat them with respect and take them seriously and your practice will become to feel part of the neighbourhood, part of the community.
>
> *(Hugh MacKay, psychologist, social researcher and novelist)*

In the evolving dynamics of dental practice, we feel that it is important to change, philosophically, the traditional 'adversarial nature' of the dental experience. It is well recognized that for too many, the dental experience has been traumatic. This has resulted in a significant proportion of the adult population accessing dental care only episodically, for the relief of pain. Thus, it is vital to see a community, and consumer, perspective in the provision of paediatric dental services. The successful practice of paediatric dentistry is not merely the completion of any operative procedure but also ensuring a positive dental outcome for the future oral health behaviour of that individual and family. To this end, an understanding of child development – physical, cognitive and psychosocial – is paramount. The clinician must be comfortable and skilled in talking to children, and interpersonal skills are essential. It will not usually be the child's fault if the clinician cannot work with the child.

Patient assessment

History

A clinical history should be taken in a logical and systematic way for each patient and should be updated regularly. Thorough history-taking is time consuming and requires practice. However, it is an opportunity to get to know the child and family. Furthermore, the history facilitates the diagnosis of many conditions, even before the hands-on examination. Because there are often specific questions pertinent to a child's medical history that will be relevant to their management, it is desirable that parents be present. The understanding of medical conditions that can compromise treatment is essential.

The purpose of the examination is not merely to check for caries or periodontal disease, as paediatric dentistry encompasses all areas of growth and development. Having the opportunity to see the child regularly, the dentist can often be the first to recognize significant disease and anomalies.

Current complaints

The history of any current problems should be carefully documented. This includes the nature, onset or type of pain if present, relieving and exacerbating factors or lack of eruption of permanent teeth.

Dental history

- Previous treatment – how the child has coped with other forms of treatment.
- Eruption times and dental development.
- What preventive treatment has been undertaken previously.
- Methods of pain control used previously.

Medical history

Medical history should be taken in a systematic fashion, covering all system areas of the body. The major areas include:

- Cardiovascular system (e.g. cardiac lesions, blood pressure, rheumatic fever).
- Central nervous system (e.g. seizures, cognitive delay).
- Endocrine system (e.g. diabetes).
- Gastrointestinal tract (e.g. hepatitis).
- Respiratory tract (e.g. asthma, bronchitis, upper respiratory tract infections).
- Bleeding tendencies (include family history of bleeding problems).
- Urogenital system (renal disease, ureteric reflux).
- Allergies.
- Past operations or hospital admissions.
- Current treatment and medications.

Pregnancy history

- Length of confinement.
- Birth weight.
- Apgar scores.
- Antenatal and perinatal problems, especially during delivery.
- Prematurity and treatment in a special or neonatal intensive care nursery.

Growth and development

In many countries, an infant record book is issued to parents to record postnatal growth and development, childhood illness and visits to health providers. Areas of questioning should include:

- Developmental milestones.
- Speech and language development.
- Motor skills.
- Socialization.

Current medical treatment

- Medications, including complementary medications.
- Current treatments.
- Immunizations.

Family and social history

- Family history of serious illness.
- Family pedigree tree.
- Schooling, performance in class.
- Speech and language problems.
- Pets/hobbies or other interests.

This last area is useful in beginning to establish a common interest and a rapport with the child. When asking questions and collecting information, it is important to use lay terminology. The distinction between rheumatic fever and rheumatism is often not understood and more specific questioning may be required. Furthermore, questions regarding family and social history must be neither offensive nor intrusive. An explanation of the need for this information is helpful and appropriate. It is important to recognize the changing patterns of family units and the carer accompanying the child may not always have a full knowledge of the past medical history.

Examination

Extra-oral examination

The extra-oral examination should be one of general appraisal of the child's well-being. The dentist should observe the child's gait and the general interaction with the parents or peers in the surgery. An assessment of height and weight is useful, and dentists should routinely measure both height and weight, and plot these measurements on a growth chart.

A general physical examination should be conducted. In some circumstances, this may require examination of the chest, abdomen and extremities. Although this is often not common practice in a general surgery setting, there may be situations where this is required (e.g. checking for other injuries after trauma, assessing manifestations of syndromes or medical conditions). Speech and language are also assessed at this stage (see Chapter 16).

The clinician should assess:

- Facial symmetry, dimensions and the basic orthodontic facial type.
- Eyes, including appearance of the globe, sclera, pupils and conjunctiva.
- Movements of the globe that may indicate squints or palsy.

- Skin colour and appearance.
- Temporomandibular joints.
- Cervical, submandibular and occipital lymph nodes.

Intra-oral examination
- Soft tissues including oropharynx, tonsils and uvula.
- Oral hygiene and periodontal status.
- Dental hard tissues.
- Occlusion and orthodontic relations.
- Quantity and quality of saliva.

Charting
Charting should be thorough and detail the current state of the dentition and the plan for future treatment.

Provisional diagnosis
A provisional diagnosis should be formulated for every patient. Whether this is caries, periodontal disease or, e.g. aphthous stomatitis, it is important to make an assessment of the current conditions that are present. This will influence the ordering of special examinations and the final diagnosis and treatment planning.

Special examinations

Radiography
The guidelines for prescribing radiographs in dental practice are shown in Table 1.1. The overriding principle in taking radiographs of children must be to minimize exposure to ionizing radiation consistent with the provision of the most appropriate treatment. Radiographs are essential for accurate diagnosis. If, however, the information gained from such an investigation does not influence treatment decisions, both the timing and the need for the radiograph should be questioned. The following radiographs may be used:
- Bitewing radiographs.
- Periapical radiographs.
- Panoramic radiographs.
- Occlusal films.
- Extra-oral facial films.

Note that digital radiography, or the use of intensifying screens in extra-oral films, significantly reduces radiation dosage. As such, the use of a panoramic film in children is often more valuable than a full-mouth series.

Other imaging
Many modern technologies are available to the clinician today, and their applications can be a most valuable adjunct not only in the diagnosis of orofacial pathology but also in the treatment of many conditions. These modalities include:
- Computed axial tomography (CAT) and cone-beam CT with 3-dimensional reconstruction.
- Magnetic resonance imaging (MRI).

Table 1.1 Guidelines for prescribing radiographs[a]

Patient	Child		Adolescent
	Primary dentition	Mixed dentition	
New patients			
All new patients to assess disease and growth and development	Bitewings if closed contacts between posterior teeth Panoramic film to assess other pathology or for growth and development	Bitewings and individualized examinations such as panoramic film to assess development and eruption of permanent teeth	Individualized radiographic examinations with bitewings and panoramic film
Recall patients			
No clinical caries and low risk	If contacts can be visualized or probed, then bitewings may not be required, otherwise bitewings at 12–24-month intervals	One set of bitewings once the first permanent molars have erupted	Bitewings every 18–36 months after the eruption of the second permanent molars up to age 20
Clinical caries or high risk of disease	Bitewings at 6–12-month intervals or until no new caries is evident over 12 months		
Growth and development	Usually not required	Individualized examination based on anomaly or disease presence, with periapicals or panoramic film	Panoramic or periapical films to assess position of third molars and other orthodontic considerations

[a]Based on recommendations from the American Academy of Pediatric Dentistry.

- Nuclear medicine.
- Ultrasonography.

Pulp sensibility (vitality) testing
- Thermal (i.e. carbon dioxide pencil).
- Electrical stimulation.
- Percussion.
- Mobility.
- Transillumination.

Blood investigations (see Appendix A)
- Full blood count with differential white cell count.
- Clinical chemistry.

Microbiological investigations
- Culture of microorganisms and antibiotic sensitivity.
- Cytology.
- Serology.
- Direct and indirect immunofluorescence.

Anatomical pathology
- Histological examination of biopsy specimens.
- Hard-tissue sectioning (e.g. diagnosis of enamel anomalies, see Figure 9.2C).
- Scanning and transmission electron microscopy (e.g. hair from children with ectodermal dysplasia, see Figure 9.2B).

Photography
Extra-oral and intra-oral photography provides an invaluable record of growing children. It is important as a legal document in cases of abuse or trauma, or as an aid in the diagnosis of anomalies or syndromes. Consent will need to be obtained for photography.

Diagnostic casts
Casts are essential in orthodontic or complex restorative treatment planning, and for general record keeping.

Caries activity tests
Although these are not definitive for individuals, they may be useful as an indicator of caries risk. Furthermore, identification of defects in salivation in children with medical conditions may point to significant caries susceptibility. Such tests include assessment of:
- Diet history.
- Salivary flow rates.
- Salivary buffering capacity.
- *Streptococcus mutans* and *Lactobacillus* colony counts.

Definitive diagnosis

The final diagnosis is based on examination and history and determines the final treatment plan.

Assessment of disease risk (see Chapters 4 and 5)

All children should have an 'assessment of disease risk' before the final treatment plan is determined. This is particularly important in the planning of preventive care for children with caries. This assessment should be based on:
- Past disease experience.
- Current dental status.

- Family history and carer status.
- Diet considerations.
- Oral hygiene.
- Concomitant medical conditions.
- Future expectations of disease activity.

Social factors including recent migration, language barriers, and ethnic and cultural diversities, can impact on access to dental care and will therefore influence caries risk.

Low risk of disease
- No caries present.
- Favourable family history (appropriate diet, dentally healthy siblings, motivated parents and caregivers).
- Good oral hygiene.
- Access to community water fluoridation.

Moderate risk
- One or two new lesions per year.

High risk or future high risk
- Three or more new lesions per year.
- Orthodontic treatment.
- Chronic illness or hospitalization.
- Medically compromised children.
- Social risk factors.

Treatment plan

1. Emergency care and relief of pain.
2. Preventive care.
3. Surgical treatment.
4. Restorative treatment.
5. Orthodontic treatment.
6. Extensive restorative or further surgical management.
7. Recall and review.

Clinical conduct

Infection control

It is now considered that 'universal precautions' are the expected standard of care in current paediatric dental practice. The principles of universal precautions are:

- Prevention of contamination by strictly limiting and clearly identifying a 'zone of contamination'.

- The need for elimination of contamination should be minimal if this zone of contamination is observed.

Universal precautions regard every patient as being potentially infectious. Although it is possible to identify some patients who are known to be infectious, there are many others who have an unknown infectious state. It is impossible to totally eliminate infection; thus, observing universal precautions is a sensible approach to minimizing the risk of cross-infection.

All children must be protected with safety glasses and clinicians must also wear protective clothing, eyewear, masks and gloves when treating patients.

Recording of clinical notes

Care must be taken when recording clinical information. Notes are legal documents and must be legible. Clinical notes should be succinct. The treatment plan should be reassessed at each session so that at each subsequent appointment the clinician knows what work is planned. Furthermore, at the completion of the treatment for the day, a note should be made regarding the work to be done at the next visit.

Use of rubber dam (see Chapter 8)

Wherever possible, rubber dam should be used for children. This may necessitate the use of local anaesthesia for the gingival tissues. When topical anaesthetics are used they must be given adequate time to work (i.e. at least 3 min). All rubber-dam clamps must have a tie of dental floss around the arch of the clamp to prevent accidental ingestion or aspiration.

Consent for treatment (see Chapter 3)

There is often little provision in a dental file for a signed consent for dental treatment. The consent for a dentist to carry out treatment, be it cleaning of teeth or surgical extraction, is implied when the parent or guardian and child attend the surgery. It is incumbent on the practitioner, however, to provide all the necessary information and detail in such a way as to enable 'informed consent'. This includes explaining the treatment using appropriate language to facilitate a complete understanding of proposed treatment plans.

It is important to record that the treatment plan has been discussed and that consent has been given for treatment. This consent would cover the period required to complete the work outlined. If there is any significant alteration to the original treatment plan (e.g. an extraction that was not previously anticipated), then consent should be obtained again from the parent or guardian and recorded in the file.

Generally, when undertaking clinical work on a child patient, it is good practice to advise the parent or guardian briefly at the commencement of the appointment what is proposed for that appointment. Also it is helpful to give the parent or guardian and child some idea of the treatment anticipated for the next appointment. This is especially relevant if a more invasive procedure such as the use of local anaesthesia or removal of teeth is contemplated.

2 Child development, relationships and behaviour management

Richard P Widmer, Daniel W McNeil, Cheryl B McNeil, Linda Hayes-Cameron

Promoting positive behaviour among children, adolescents and their caregivers in the dental surgery

This section is a practical guide for specific modes of interacting in the dental environment which can help produce positive and adherent behaviours in child and adolescent patients, as well as their parents and other caregivers (e.g. grandparents). These guidelines are based on principles and research findings from behavioural dentistry, as well as behavioural, developmental, child and paediatric psychology.

It is probably true for most of us that the meaning of our lives is centred on our personal relationships. These are the source of our sense of personal identity; they are the source of our emotional security or insecurity that might define us; they are the source of our greatest joys, of our deepest comforts but, of course, they are also the source of our most bitter disappointments. However, without personal relationships life for most of us would be meaningless. We might dream of escape to the proverbial 'Desert Island' but we wouldn't want to stay there for more than an hour or two or possibly a week, because we would soon realize that the lifeblood of our lives is in our relationships. That is where we get all the rich material for coping with life (Hugh MacKay, ABC radio 26 March 2010). So at home and at work, we need to nurture our relationships constantly. In dentistry, this is particularly relevant, as we are working intimately, not only with those we are caring for, but also with their carers and indeed the entire Dental Team. We spend a great deal of our waking hours 'at work', which we want to enjoy as much as possible and so our relationships become crucial and can be used to positively affect the behaviour of the child in the dental environment.

Much has been written about management of problem behaviour among children receiving oral healthcare, with a focus on the use of various techniques. This guide, however, emphasizes specific, simple methods that can be used with children and adolescents to enhance their comfort and cooperation. The general idea is to use finesse instead of trying to achieve absolute behavioural control. Since a sense of lack of control is one of the major components of anxiety and fear (along with a lack of predictability), using methods that are encouraging rather than demanding can go a long way in promoting comfort in the dental environment.

Dentists, dental hygienists and dental therapists are integral members of the healthcare team for children and adolescents and must have an awareness of practical methods of behaviour management that are based on knowledge of psychological principles and stages of growth and development. The adage that 'children are not small adults' promotes the idea of special knowledge and behaviours that are important in caring for younger dental patients. Oral health professionals must have

a knowledge base in child and adolescent medicine, as well as in social, emotional and cultural factors affecting the health and behaviour of this age group.

It is imperative that dental appointments in infancy, childhood and adolescence are positive, as research clearly shows that early experiences have strong effects on whether dental advice and treatment is sought in adulthood. Having a rapport with the parents/caregivers (e.g. grandparents) is essential, as they typically are the most influential people in the child's life.

Child behaviour and development

Working with children is, of course, different from working with adults, therefore, it is essential to be familiar with age-appropriate skills and functioning, and development. Infants, children and adolescents are undergoing progressive changes in cognitive, receptive and expressive language, fine and gross motor ability, and social/emotional development. Each child is unique and may develop at varying rates relative to their same-aged peers, For example, one child may present with strong motor skills but less well-developed language, while this may be the opposite for another same-age peer.

Developmental milestones and issues

There are two essential needs that remain constant from birth to adulthood: the desire to feel important and having an emotional connection with others. Oral health professionals who are aware of their patient's age-appropriate development and needs can use that information to develop a rapport with the child and have appropriate expectations of the behaviour of that particular child in the dental setting.

General developmental milestones and child behaviour

Age 3–4 months

- In their first 3–4 months, babies become extremely interested in their world of people, places and objects.

Age 6–8 months

- By 6–8 months, infants are discovering new ways to share and express their curiosity, joy, frustration and fear within their world. Babies can shift their attention while keeping in mind the object on which they were focusing. They can look at a 'teddy bear' and be delighted by it, then turn to look at the parents to share those feelings.
- By 8 months, babies are beginning to crawl and discover their surroundings, learning to distinguish differences in their world and people.
- Mobility sets the stage for the first significant appearance of fear. Stranger awareness begins at this time.
- Understanding of spoken words and non-verbal communication (receptive language) develops at a much greater rate than expressive language.
- The infant learns to 'social reference', where he/she shows interest in an object or person and then turns to the parents for emotional feedback. The infant is able to read the parent's/caregiver's facial expression, tone of voice and words, to understand the concept of a particular danger or safety.

Dental implications

- Advice regarding tooth eruption, initial oral hygiene measures and teething.

It is generally accepted that teething has the potential to cause local irritation, however, there is no accepted evidence connecting the systemic symptoms, such as diarrhoea, flushed cheeks and fever, to teething. It is important to seek medical advice if an infant has persistent febrile illness.

Age 9–12 months

- By 9 months, two-way conversations about feelings are now possible. Infants become aware of the possibility of others sharing their thoughts and feelings. Understanding and labelling the infant's feelings and experiences can help with relationship building, acceptance and trust.
- Object constancy or permanence is developing in which infants begin to realize that objects and people still exist even when out of sight (e.g. repeatedly throwing the spoon off the high chair and it magically reappearing).
- Separation anxiety is a consequence of this stage and may continue in varying degrees until 18 months.

Dental implications

Children's behaviour is a function of their learning and development, and so it is reasonable to expect that their behaviour in the dental environment will also vary.

- The child has limited ability to understand dental procedures. Nonetheless, with a sensitivity to the child's normal emotional development and play expectations, even without cooperation, an oral examination and some treatment can often be accomplished without sedation.
- Good rapport with the parents is required, as the dentist can educate the parent in the importance of sending positive and appropriate feedback to the infant/child about the dental experience.

Age 1–3 years (Toddler years – egocentric)

- Infants begin to develop a sense of self and explore their autonomy. They may become non-compliant for the first time, as they practise asserting themselves, trying to establish themselves as independent and avoiding situations that make them feel out of control and with a limited sense of self.
- Language develops and 'No' becomes a favourite in their repertoire of words.
- Sharing and cooperative play is meaningless at this stage, as the '*toddler rules of ownership*' outweigh all concepts, such as: If I see it it's mine. If it's yours and I want it, it's mine. If it's mine, it's mine and mine only!

Dental implications

- In the dental room, the clinician may identify an object of particular ownership such as a doll or another toy, and praise the child for taking good care of it rather than trying to remove it.
- Giving toddlers lots of little choices (a choice of two at any time) will assist in enhancing their sense of self and importance, resulting in greater cooperation.

- Preferences for 'boy' and 'girl' objects is common at this age: many toddler boys show interest in cars, trains, the colour blue and other boys, while many toddler girls show interest in dolls, fairy dresses, the colour pink and other young girls, for example. Play remains solitary, however, and is 'parallel play' to their peers.
- The ability to communicate varies according to the level of vocabulary development, which is expected to be limited. Thus, the difficulty in communication puts the child in a 'pre-cooperative' stage.
- These children are too young to be reached by words alone, and shyness may mean that the child must be allowed to handle and touch objects to understand their meaning.
- Children of this age typically should be accompanied by a parent.

Age 3 years

- By this age, children are less egocentric and like to please adults.
- They have very active imaginations and like stories; back-and-forth communication is possible, and children at this age typically have the capacity for some reasoning.
- In times of stress, they will turn to a parent and not accept a stranger's explanation. Typically, these children feel more secure if a parent is allowed to remain with them until they have become familiar with the dental professionals. Then a positive approach can be adopted.

Dental implications

- Liberal use of praise for adherence to requests in the surgery is indicated, given the child's desire to please adults.
- Telling stories during the course of treatment may help to capture the child's attention and to distract him/her from any unpleasant aspects of care being provided.

Age 4–5 (early childhood years)

- By this age, children are exploring new environments and relationships in their world. They prefer one-on-one friendships, as more than one is difficult to manage. Once at school, however, they have to learn to sit quietly in groups and pay attention. Further development of social skills and regulation of emotions is occurring while mixing with their peers.
- These children listen with interest and respond well to verbal directions. They have lively minds and may be great talkers who are prone to exaggeration. In addition, they will participate well in small social groups.
- 4-year-old children are extremely creative, as fantasy and imaginary play allows them to work through confounding problems, emotions and the stressors of daily life. Therefore, pretend play can open the door to a young child's thoughts and worries and provide the dentist with valuable information. Showing great interest, listening and reflecting back to the child what they just said or taking on the role of another toy in conversation with them, will encourage them to explore further.

Dental implications

- At this age, these children can be cooperative patients, but some may be defiant and try to impose their views and opinions. They are familiar with and respond well to 'thank you' and 'please'.
- Promotion of autonomy and the development of self-esteem by allowing decision-making and choices in their treatment, and encouraging them to take responsibility for tasks such as manoeuvring the dental chair, is important.
- Children at this age usually have no fear of leaving their parents for a dental appointment because they have no fear of new experiences. They take pride in their possessions, and comments about clothing can be effectively used to establish communication and develop a rapport. By this age, children usually have relinquished comfort objects such as thumbs and 'security blankets'.

Age 6–8 years

- By 6 years, children are established at school and are moving away from the security of the family.
- They are increasingly independent of parents and will play without their parents being in close proximity.
- For some children, this transition may cause considerable anxiety with outbursts of screaming, temper tantrums and even striking parents. Furthermore, some will exhibit marked increase in fear responses.

Dental implications

- This age may be an ideal time to help the child and parent/caregiver move from the parent/caregiver being in the surgery to the child being able to go back alone from the waiting room to the surgery.
- The increased tendency toward fearfulness prompts special care in working with children at this age, accepting that new fear(s) may develop, even if the child has been a prior patient who earlier was comfortable in the dental setting.

Age 8–12 years (the middle years)

- At this age, children are part of larger social groups and are strongly influenced by them. They notice who is accepted and who is excluded from groups. With this comes the growing concern of embarrassment, which they will avoid at all costs. While parents might wish for them to become leaders, they appropriately become followers, as this is perceived as healthier and safer.
- As a consequence, children learn to hide their feelings and thoughts at this time and adopt a 'cool' attitude.
- Intellect becomes more important as they develop cognitively and begin to reason. The pre-teenager becomes concerned with what is moral and just and becomes more literal (e.g., a parent asks: 'Pick up your clothes'. The child picks them up and places them back down stating, 'You didn't tell me where to put them!').

Dental implications

- Be cautious to not embarrass the child through criticism of his/her self-care (e.g. toothbrushing).

- Be patient in not expecting the child to freely share information without considerable rapport-building.
- Given the developing capacity to reason, children in this age range may respond well to explanations about the need to engage in toothbrushing and flossing on their own, without parental prompting.

Adolescence

- The adolescent is faced with solving major questions such as: Who am I? Who am I becoming? Whom should I be? With such tasks in mind, it is understandable that teenagers are often perceived as self-absorbed, excluding themselves from family and to some degree, their peers. Many interactions with the teenager tend to result in a narcissistic view of any situation.
- Adolescents are on a journey of self-discovery and, not unlike the toddler, are looking for greater autonomy, such as experimenting with new identities, realities and self-concepts, all of which are healthy. Experimentation and use of tobacco and other substances is common.
- Adolescents typically believe they are invulnerable, and that they will not encounter adverse results from their actions. They do not expect, for example, that negative health outcomes will result from tobacco use as 'other people' get addicted and only 'old people' have health problems.
- Appearance becomes increasingly important during the teenage years.
- Teenagers often feel that their experiences are unique, so listening with an open mind, providing independence as would be done with an adult and supporting them in reaching their goals (within safety limits), will earn trust and cooperation.
- Greater rapport is gained when the dentist adopts a non-judgemental, non-preaching and respectful approach towards the teenager.

Dental implications

- Treating the teenager as his or her own person, independent from the parent/caregiver, typically will be well received.
- Taking some time to talk about non-dental topics in an 'adult' way may be a good way to develop rapport.
- Emphasizing the importance of self-dental care to maintain their smile may be a way to 'reach' adolescents in terms of preventive behaviours.

Understanding child temperament

There has been a longstanding debate in the literature on child development about the degree to which a child's development is influenced by 'nature' versus 'nurture'. Studies suggest that children do indeed enter the world with a characteristic temperament or personality that stays with them to some degree, for the rest of their life. Thomas and Chess (1977) suggested that there are three basic temperaments that influence later personality:

Easy temperament

These children have a positive mood, regular bodily functions, are adaptable and flexible and have a positive approach to change or new situations.

Difficult temperament
These children are more irritable and intense. They have irregular body functions and take some time to develop feed, play, sleep patterns and routines. They have difficulty with new situations and adapting to change and tend to withdraw in social settings.

Slow to warm-up temperament
These children have a shy disposition and a low activity level. Initially, they are slower to adapt to new situations but once they are comfortable with their environment they begin to engage.

Approximately 65% of infants can fit into one of these three categories. The remainder have a mixture of traits.

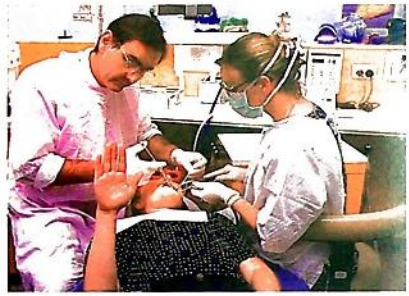
Dental implications of child temperament
Dentists working with children must use different approaches and techniques, depending on the personality type of the child. Whereas an easy temperament child may be flexible enough to handle a quick change in plan, a slow to warm-up child may need to be given a longer time to adjust. Difficult children respond best to a dentist who provides a great deal of structure in a sensitive but confident manner. The slow to warm-up child is best served by dental personnel who are calm, patient and encouraging (without being demanding).

Use of verbal and non-verbal communication to promote positive behaviour in children

The following principles are some of the important considerations in positive communication with children and their families.

- Show respect for the child and his/her feelings and interests.
- Show interest in the child as an individual. Find our his/her preferred name (e.g., nickname if any) and use it frequently in speaking with the child (and caregiver).
- Share 'free information', as much as the child/caregiver seems to want and to be able to handle.
- Give well-stated instructions (e.g., 'Please open your mouth now', instead of questions, such as, 'Would you like to open your mouth now?'). Tell the child *kindly* what he/she NEEDS TO DO, not what they should *NOT* do.
- Communicate at the child's level, both physically (Figure 2.1) and cognitively/emotionally.
- Focus on the positive aspects of a child's (and parent's) behaviours. In most situations, ignore negative behaviours.
- Avoid stereotyping and making assumptions about children (e.g. that boys are interested only in sports; that young girls are interested in dolls).
- Show ethnic, cultural and gender sensitivity.

A



B

Figure 2.1 (A) Giving children control in the dental surgery. (B) It is essential to listen to your patient. A prearranged signal of a hand raised tells the clinician that the procedure is uncomfortable. This gives the child some control over what is happening without interfering with the procedure.

Physical structuring and timing during the dental visit

Setting the stage for positive behaviour

In addition to communications from the dentist and dental staff, many aspects of the dental situation can be arranged in such a way that promote positive reactions in infants, children and adolescents. McNeil and Hembree-Kigin (2010) describe *PRIDE* skills, modified here for relevance to the dental situation, which is a conceptualization that can help prompt members of the dental team to structure their behaviour with children and teenagers. This approach is not to discourage spontaneity with youngsters, which can be so important in working positively with 'kids', but may provide a way for adults to think about including skills as part of their repertoire with children. In fact, the final point of the PRIDE skills is Enthusiasm, which speaks of communicating joy, spontaneous fun and action to youth.

PRIDE

- **Praise**: These 'social reinforcers' can be either 'labelled' or 'unlabelled'. Labelled praises (e.g. 'That's a great job keeping your mouth open, Jane!') typically are more effective at managing behaviour than unlabelled praises (e.g. 'Well done, Jane!').
- **Reflection**: Such phrases are a demonstration of the dentist listening to the child, and can involve a simple repeating of some of the child's words, perhaps with embellishment.
- **Inquire**: These questions involve asking a child for information, or otherwise prompting him or her to reply ('I'm wondering how you feel about coming to see me today?'). Open-ended questions typically produce more information and promote a more positive interview atmosphere, relative to closed-ended questions that can be answered with a Yes or No or a simple fact. Question-asking typically

is greatly over-used by adults with children, and should instead be used judiciously.

- **Describe**: These statements focus on the child's behaviour, and portraying the child's actions, typically in a positive light (e.g. 'Now you're keeping your mouth open so nicely, and letting your feet and legs be still'.).
- **Enthusiasm**: There is a time for animation and play on the part of the dentist and dental team, and a time for more reserved professionalism. Particularly with younger children in a dental environment, enthusiasm on the part of the dentist and team is often needed to combat the negative images of dental care portrayed in the media, by peers and sometimes by parents and other caregivers.

Use of these PRIDE skills will be well received by children and youth, and can help make the dental appointment reinforcing and enjoyable. PRIDE skills, however, should not be used in some automaton fashion, but rather flexibly and in concert with the dental professional's own personality and the procedures at hand. Not only are these interpersonal communication skills essential, but the physical and structural aspects of the dental appointment are also crucial.

Practical guidelines for physical and social aspects of the dental surgery

- Everyone in the surgery (dentist, auxiliary, parent) should transmit positive, comforting expectations to the patient.
- Use stimulating visual distracters in the surgery (child and adolescent-oriented posters).
- Have age-appropriate materials (safe toys, magazines) in the waiting room. Include materials for parents.
- Have toys available for younger children as distracters or tangible rewards.
- Greet the child in the waiting room without a mask and not wearing surgical garb. Use the child's preferred name. Smile at the child! Depending on the child's height and your height, you may wish to squat in greeting him/her, to be at eye level.
- Pace procedures during the appointment, based on how the patient is coping, so that they are neither rushed nor bored. Periodically ask how he or she is coping with the appointment, sometimes using closed-ended (e.g. 'Are you doing OK?') and sometimes open-ended questions.
- Inform and discuss with parent/caregiver before the appointment and at the end.
- Include children, and especially adolescents, in the decision-making and practicalities of treatment.
- Provide information in advance about the procedures to be performed at the next appointment so that the child and parent/caregiver are prepared.
- Allowing a child a visit to the 'treasure chest' to get a tangible reward at the end of an appointment, finding some positive behaviour to reinforce (even if much of the child's behaviour was challenging), can leave a child with positive memories of the dental experience.

- Structuring what is remembered about a dental appointment has been shown to be an important issue in how children perceive dental care. The oral health professional may wish to provide a short summary statement after the 'treasure chest' visit, emphasizing certain (positive) parts of the dental visit (e.g. 'Rickie, today you came in bravely and sat in the chair and kept your mouth open for a long time, even when you got a bit tired. Well done for keeping still for so long! Now, what was the best part of your visit today?').

Presence or absence of family members in the surgery

- It is appropriate that a parent be present in the surgery to support their children during treatment, particularly in their younger years. Parents/caregivers can be coached by dental professionals regarding how to be most helpful during a visit.
- If a parent is unable or unwilling to provide appropriate support, then it may be more desirable for them to wait outside the surgery. It is important to note that parental access to their children should never be denied.
- When there are other siblings, who enjoy or readily cope with dental treatment, it often is helpful to use them as a model.

Transmission of emotion to the child or adolescent

- Children acquire some of their parents' fear and anxiety about the dental treatment both in the dental environment and in the long term.
- Emotion is transferred from parents, siblings, dentist and auxiliaries to the child, whose emotional state also impacts on all of those persons. Dental staff who are calm and confident and use humour will promote positive experiences for their patients.

Physical proximity and touching

- Initially, work from the front, at eye level.
- Be aware of the child's physical distance, i.e. 'intimate zone'. This zone is approximately 45 cm, but varies in different cultures. By necessity, the dentist must 'invade' this space, but frequent stopping between procedures allows the child some time for coping.
- Touching the child can be used in non-private bodily regions, such as the lower arms and shoulders, to encourage, soothe or reward. The oral health professional should be attuned to the child's reaction to such touching, however, and whether it is well received. There are wide cultural variations in the appropriateness of such touching, both in terms of the child's background, as well as that of the oral health professional.

Timing

- It is best to introduce new procedures at an appropriate rate to avoid either rushing or boring the patient.
- Conducting less invasive procedures first will usually be more tolerable for the patient.

Figure 2.2 Involving children in their treatment. It is important for children to feel that the dental environment is non-threatening and safe, and can be a place for enjoyment.

Figure 2.3 At the first visit, it is often good to see the child and parent away from the surgery. It provides an opportunity to talk with the child and establish rapport.

Stimulating and distracting objects and situations (Figure 2.2)

- Be aware of popular culture. In some settings, it is possible to have different areas of the surgery orientated to particular patient age ranges.
- One area might include puppets and pictures of colourful cartoon characters for children up to 8 years.
- For older children, have wall posters of pop groups.
- Adolescents, like adults, are best treated in a modern, friendly environment.
- In some settings, popular electronic games and videos are appropriate.
- Fish tanks provide interesting stimulation for children of all ages, as well as adults.

Surgical clothing and instruments

- Never greet a child while wearing a face mask and gloves.
- Explain the need for protective clothing. With younger children, you can make putting on the mask and other gear a fun task that you describe to the child as you are doing it.
- Familiarize children with appropriate instruments.

Greetings in the waiting room

- It is ideal, particularly in initial meetings, for the dentist to greet the child and parent/caregiver in the waiting area.
- An interview room or non-surgical environment is useful for new patients (Figures 2.3–2.5).

Talking with parents

It is helpful for the dentist to have a positive relationship with both children and their parents. Keep parents well informed. While asking personal information, always remember to involve the child in the discussion when appropriate. Be prepared to

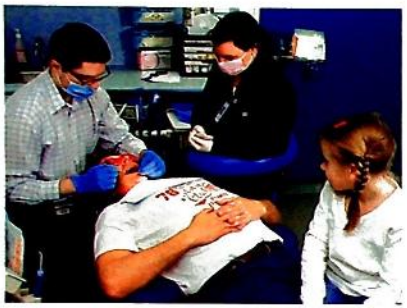


Figure 2.4 Introducing a child to the dental environment; part of the familiarization.

Figure 2.5 A dental mobile – every child should have one!

separate the child from the parent to discuss more sensitive issues if necessary. The chairside assistant can be asked to occupy the child during this discussion.

Talking with children and adolescents

Children, like adults, typically respond best if they are treated as individuals, somehow special to the provider. Consequently, using the child's name to refer to him or her, and repeating it in conversation periodically during the dental appointment, is helpful in producing a positive environment and in capturing and maintaining the child's attention. It usually helpful for the dentist and members of the dental team to speak with (not at) the child at the child's level, both physically and psychologically. Dental jargon typically is best avoided with most patients, but particularly with children. Table 2.1 suggests terminology that might be used with younger patients. Of course, use of these terms should be at a developmentally appropriate level for the child. Some mid- and older adolescents, for example, may actually respond well to learning dental jargon, as it gives them a sense of being cognitively advanced.

Special arrangements for first-time dental visits

Certain steps are appropriate for an initial visit. In general, the pace of a first appointment is much slower.

- Use pre-appointment letters giving information about the visit that also includes photographs of the rooms and what might be expected of the child and parent.
- Use an interview room for the initial contact.

The emphasis is on educating the child, promoting comfort and allowing the visit to be exciting and fun. Relatively simple and less invasive procedures are preferred. Introducing the child to the office, staff and equipment and pointing out posters and other materials of interest in the treatment room can be helpful.

Behavioural methods for reducing fear and pain sensitivity

Table 2.2 gives eight methods that can be used across a variety of situations with children and adolescents of all ages. The particular uses depend on the patient's

Table 2.1 Dental terminology and lay language equivalents for use with children (age levels are approximate and should be based on the cognitive level)

Dental terminology	Lay language
Ages 1–5	
Air syringe	Wind blower
Water syringe	Water pistol
High evacuation suction	Vacuum cleaner
Saliva ejector	Straw
Radiograph	Picture of your tooth
Prophylaxis	Electric toothbrush
Explorer	Tooth counter
Rubber dam	Raincoat for your tooth
Local anaesthesia	Putting the tooth to sleep
High-speed handpiece	Tooth whistle
Low-speed handpiece	Tooth tickler
Extraction	Wiggle your tooth
Stainless steel crown	Helmet for your tooth
Ages 6–10	
Anaesthetize	Numb
Extract	Take out or wiggle
Caries, carious lesion	Hole
Pain	Tickle or pressure
Drill	Electric motor
Dental surgery	Treatment room

developmental age and personality, as well as on a variety of other factors such as the quality and depth of the dentist's relationship with the child or adolescent.

Integrating behavioural and pharmacotherapeutic approaches

The behavioural principles and methods described above are used routinely, many in virtually every encounter with a youngster in a dental setting. When medications are needed for pain and/or anxiety control, or for sedation, sensitive behavioural approaches on the part of the oral health professional are particularly important. Using both medication alongside behavioural approaches may be the most effective way to deal

Table 2.2 Behavioural methods for reducing anxiety

Tell–show–do	Informing, then demonstrating, and finally performing part of a procedure
Playful humour	Using fun labels and suggesting use of imagination
Distraction	Ignoring and then directing attention away from a behaviour, thought or feeling, to something else
Positive reinforcement	Tangible or social reward in response to a desired behaviour
Modelling	Providing an example or demonstration about how to perform a behaviour
Shaping	Successive approximations to a desired behaviour
Fading	Providing external means to promote positive behaviour and then gradually removing the external control
Systematic desensitization	Reducing anxiety by first presenting an object or situation that evokes little fear, then progressively introducing stimuli that are more fear-provoking

with many clinical scenarios. In fact, behavioural approaches can and should be used to prepare phobia patients, for example, prior to and after pharmacotherapy, as described by Milgrom and Heaton (2007).

Referring for possible mental health evaluation and care

When to refer

It is a role of the dental professional to refer a child or family when there seem to be significant emotional or psychological issues. Even when such problems do not interfere with dental treatment, it is the dentist's role, as a member of the healthcare team, to identify possible psychopathologies and to refer for proper care. A sensitive conversation with the parents/caregivers regarding your concern for the child is essential prior to making the referral.

Common reasons for referring a child or adolescent for mental health concerns

- Evidence of abuse or neglect (e.g. bruises, broken teeth, cigarette burns, inappropriate clothing for weather, severe hygiene problems, untreated breaks or sprains).
- Extremes of behaviour, anxiety or emotion (e.g. attention deficit hyperactivity disorder, dental phobia).
- Neurological signs or symptoms (e.g., possible seizure activity, tics).
- Severe developmental or cognitive delay (e.g. possible learning disabilities, motor problems, feeding problems).
- Extremely poor parenting (e.g., sole use of excessive physical restraint and punishment).

Referral specialties

Referrals for mental health concerns should be made to psychologists, psychiatrists or social workers. In a hospital setting, it is possible to refer to one of the available departmental services. In a dentist's private surgery, referrals can be made to professionals in private practice, community agencies or hospitals. The following guidelines are suggested when selecting a specialty for referral.

Psychologists

Refer in the case of abuse or neglect, extremes of behaviour, developmental or cognitive delay or extremely poor parenting. When there is a need for sophisticated cognitive, personality, neuropsychological and/or behavioural assessment, referral to a psychologist is best as standardized psychometric tests can be used. Psychologists also can provide individual child/adolescent, parent/child and/or family therapy to address problems in the child/adolescent and family system.

Psychiatrists

Refer when there are neurological signs or symptoms. When psychoactive medications may be needed, such as when a child demonstrates signs of psychosis, referral to a psychiatrist is most appropriate; similar to cases in which there are complicating medical factors.

Social workers

Refer for social problems, abuse or neglect. Referral to social workers is appropriate when there are existing social problems in the family that require mobilization of community resources. Social workers know about, and help patients to use available services in the community.

How to refer

It is acknowledged that suggesting mental healthcare to parents can be an anxiety-provoking task for the dentist. Nevertheless, it is essential that such referrals are made, because the dentist is in a unique role as a healthcare provider. If referrals are not made in a timely fashion, then a condition can progress and worsen.

- Speak to the parents/caregivers in a private setting, informing them of the signs or symptoms that are the cause for concern, without blaming or ascribing responsibility. When the parents understand the problems and your concern, referral to a specific professional or service can be made. It is often helpful to emphasize it is for the well-being of the child and the necessity to address the problem for their proper development.
- Ensure that the parents and the child or adolescent are aware of the referral and know the specialty of the referral. (It is not appropriate merely to describe the referral as 'to a doctor who will help your child'.).
- Refer first to only one of the mental health specialties. If additional referral is necessary, it can be arranged by the first referral source. In making the referral, one can ask for feedback from the mental health professional after the appointment. If there is behavioural disruption in the surgery, the mental health professional may have recommendations for management once the child or family has been evaluated.

- Mental health concerns are considered private by many individuals. Given this desire for privacy, releases to exchange relevant information, signed by a parent or guardian and the child if of an age to understand it, are required. Such a form can be signed in the dental surgery and sent to the mental health professional, along with a request for feedback.

Further reading

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3 Pharmacological behaviour management

Eduardo A Alcaino, Jane McDonald, Michael G Cooper, Simrit Malhi

Pain management for children

The proper treatment of pain in children is often inadequate and involves misconceptions that:

- Children experience less pain than adults.
- Neonates do not feel or remember pain.
- Pain is character-building for children.
- Opioids are addictive and too dangerous in terms of respiratory distress.
- Children cannot localize or describe their pain.

Development of pain pathways

Even premature neonates have the physiological pathways and mediators to feel pain. The statement that infants and children do not experience pain, either partially or completely, is not physiologically valid.

Measurement of pain in children

There are individual circumstances for each child that affect how they respond to pain and, subsequently, how that pain will be assessed. These include:

- Age and developmental level.
- Social and medical factors.
- Previous pain experience.

Observation of non-verbal cues and behaviour is important. A quiet, withdrawn child may be in severe pain. Simple measures are there to measure pain in children of all ages.

Methods for paediatric pain assessment include

- Observer-based techniques which are useful in pre-verbal children, i.e. scales that measure blood pressure, crying, movement, agitation and verbal expression/body language.
- Self-reporting of pain is valid in children over 4–5 years of age.
- Children with severe developmental delay can be extremely difficult to assess regarding pain, even by their regular carers. Unusual changes in behaviour from normal may represent an expression of pain.

Analgesia prior to procedures (pre-emptive analgesia)

- Poor analgesia for an initial procedure in children can diminish the efficacy of analgesia for subsequent similar procedures.

- Consideration should be given to ensure adequate systemic and/or local analgesia prior to the commencement of a procedure. Appropriate time for absorption and effect should be allowed.
- A stronger analgesic may be required for the procedure with regular simple analgesics for the postoperative period.

Routes of administration

- Oral analgesia is the preferred route of administration in children. Absorption for most analgesics is generally rapid – within 30 min.
- Attention to formulation suitable to the individual child can help greatly with compliance, i.e. liquid versus tablets in younger children, pleasant taste.
- The rectal route of administration can be valuable in a child not tolerating oral fluids. Doses and time to peak levels may vary compared with oral preparations and are usually much longer. Peak levels after rectal paracetamol may take 90–120 min. Adequate explanation should be given and consent should be obtained for the rectal administration of a drug. This route is not used in an immunocompromised child due to the risk of infection or fissure formation.
- Parenteral paracetamol is now available.
- Intranasal or sublingual administration of opioids has been described as an alternative to injection, which avoids first pass metabolism by the liver.
- Repeated intramuscular injection should be avoided in children, they will often tolerate pain rather than have a painful injection. A subcutaneous cannula, inserted after using topical local anaesthetic cream (EMLA) can be used for repeated parenteral opioid analgesia.
- In obese children, the dosage given should be based on ideal body weight, which can be estimated as the 50th centile on an appropriate weight-for-age percentile chart.

Analgesics

See Table 3.1.

Paracetamol

- Dosage 20 mg/kg orally, then 15 mg/kg every 4 h.
- 30 mg/kg rectally as a single dose.
- Maximum 24-hour dosage of 90 mg/kg (or 4 g) for 2 days, then 60 mg/kg per day by any route of administration.
- Ensure adequate hydration.
- Useful as a pre-emptive analgesic.
- No effect on bleeding.
- Intravenous paracetamol is available (10 mg/mL). The same dose is used and administered over 15 min.
- Take care with dosing, as many different strengths and preparations are available.

Table 3.1 Analgesic agents for children

Drug	Oral dose	IMI, SCI, IVI dose	Notes
Paracetamol	20 mg/kg initially, then 15 mg/kg every 4 h		Maximum 90 mg/kg/day (upto 4 g) for 2 days then 60 mg/kg/day
Ibuprofen	5–10 mg/kg every 8 h		Maximum 40 mg/kg/day upto 2 g/day
Naproxen	5 mg/kg every 12 h		Maximum 10–20 mg/kg/day upto 1 g/day
Diclofenac	1 mg/kg every 8 h 1 mg/kg every 12 h (rectally)		Maximum 3 mg/kg/day upto 150 mg/day
Codeine	0.5–1 mg/kg every 4 h	0.5–1 mg/kg every 3 h Not for IV use	Maximum 3 mg/kg/day
Morphine	0.2–0.3 mg/kg every 4 h	0.1–0.15 mg/kg every 3 h	
Tramadol	1–1.5 mg/kg every 6 h	1 mg/kg every 6 h	Maximum 6 mg/kg/day upto 400 mg/day

IMI, intramuscular injection; IVI, intravenous injection; SCI, subcutaneous injection.

Non-steroidal anti-inflammatory drugs (NSAIDs)

- Effective alone after oral and dental procedures.
- May be used in conjunction with paracetamol.
- Have an opioid-sparing effect.
- Increased bleeding time due to inhibition of platelet aggregation.
- Useful analgesic once haemostasis has been achieved.
- Best given if tolerating food and drink.
- Can be used in infants over 6 months of age.

Contraindications for the use of NSAIDs in children

- Bleeding or coagulopathies.
- Renal disease.
- Haematological malignancies, in children who may have or develop thrombocytopenia.
- Severe asthma, especially if the child is sensitive to aspirin, steroid-dependent or have coexisting nasal polyps.

Aspirin

- Rarely used in children for mild pain due to the risk of Reye syndrome.
- However, aspirin is commonly used in the management of juvenile rheumatoid arthritis.

Ibuprofen

- Commonly used in children for mild pain with less gastrointestinal side-effects compared with aspirin.
- Should be avoided in patients with renal impairment.

Codeine
- Repeated administration causes constipation.
- Main action is due to metabolism to morphine (approximately 15%).
- 10% of Caucasians and up to 30% of Hong Kong Chinese cannot metabolize codeine and find it an ineffective analgesic.
- Intravenous use may cause profound hypotension.

Oxycodone
- Good oral bioavailability.
- No pharmacological differences in metabolism.
- Available as a liquid.
- Useful alternative to codeine.

Morphine
- About 30% oral bioavailability as morphine sulphate.
- May cause nausea and constipation similar to all opioids.
- There is no risk of addiction for supervised analgesic use in children.

Tramadol
- Can be used for moderate pain in children over 12 years of age.
- A weak μ-opioid agonist and has two other analgesic mechanisms (increasing neuronal synaptic 5-hydroxytryptamine and inhibition of noradrenaline uptake).
- 70% oral bioavailability.
- No effect on clotting.
- Avoid use in children with seizure disorders and those taking tricyclic or selective serotonin reuptake inhibitor (SSRI) antidepressants.

Clinical Hint

A compliant 8-year-old boy is having several teeth extracted under local infiltration and inhalation sedation with nitrous oxide. Consider:
- Preoperatively: paracetamol 20 mg/kg orally, 30 min preoperative.
- Postoperatively – ibuprofen 10 mg/kg and paracetamol 15 mg/kg every 6 h or 30 min before bedtime that night.
- Occasionally, an oral opioid may be required if analgesia is inadequate when local anaesthesia wears off, i.e. oxycodone syrup 0.1 mg/kg every 4–6 h for 2–3 doses.

Discharge criteria

Many drugs that are used for combination sedation and analgesia in children have a long half-life of several hours. Discharge criteria should be used to assess that the child is well enough prior to discharge from a free-standing facility. Criteria should include:
- Self-maintenance of airway.
- Easily rousable and able to converse.
- No ataxia, i.e. can walk properly.

- Tolerating oral fluids.
- Discharge in the care of a responsible adult with appropriate information about after-hours contact if a problem arises.

Local anaesthesia

The use of local anaesthesia in paediatric dentistry varies significantly between countries and there are also individual preferences. Every clinician must be proficient at administering painless local anaesthesia. While it is the mainstay of our pain control for operative treatment, it also represents one of the greatest fears in our patients. Use of many of the non-pharmacological techniques described in the previous chapter may enable the dentist to deliver an injection without the child being aware. There are few patients, old or young, who are not genuinely afraid of injections, and there are obvious disadvantages in the physical size of the dental cartridge syringe.

Techniques and tips

- It makes sense **NOT** to hold the syringe in front of a young child to see. While it is essential not to lie to the child, distractions such as having the dental assistant talk, or use of the low velocity suction are useful.
- The use of topical anaesthetics is essential to create the optimal experience for the child. While a multitude of agents are available with different flavours and properties, newer anaesthetics such as EMLA® (Eutectic Mixture of Local Anaesthetic) penetrate deeper through the mucosa.
- Newer products such as electronic devices for slow injection techniques may replace more conventional techniques (The Wand®).
- The use of infiltration versus block injections in the mandible is also the subject of debate, and clinicians differ in their choice of technique. The approach of the needle to the mandibular foramen differs in younger children, as the angle of the mandible is more obtuse and a shorter needle (25 mm) may be sufficient. However, even with the best technique, a mandibular block injection may still be uncomfortable.
- Infiltration injections supplemented with intra-periodontal injection may be useful.
- Palatal anaesthesia is best achieved by slowly infiltrating through the inter-dental papilla after adequate labial or buccal anaesthesia to minimize discomfort to the child (Figure 3.1).

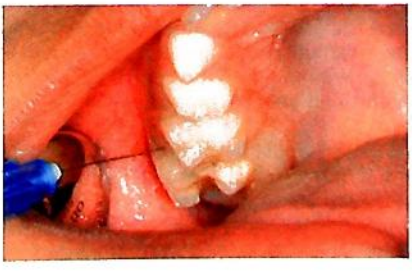


Figure 3.1 Techniques for administering palatal anaesthesia by injecting from the already anaesthetized buccal side, through the interdental papilla. Note the blanching of the palatal mucosa indicating spread of the anaesthetic solution.

Need for local anaesthesia under sedation and general anaesthesia

Some form of pain control is required when invasive procedures are performed under any form of sedation (including inhalation sedation, oral sedation, etc.). However, the need for local anaesthetic under general anaesthesia is controversial. It is well recognized that a patient's vital signs may change in response to painful stimuli (e.g. extraction), depending on the depth of anaesthesia. Local anaesthesia is not routinely used for extractions of primary teeth under general anaesthesia. Studies have observed that the child's postoperative recovery is usually independent of the procedure performed, and preschool children waking after having a general anaesthetic can be more distressed by the sensation of numbness in the mouth. However, the use of local anaesthesia is recommended when removing permanent teeth, especially first permanent molars.

Clinical Hints – Local anaesthesia

Successful local anaesthesia depends on:

- Communication with the child and parent.
- Routine use of topical anaesthesia, and leaving adequate time for it to act.
- Slow injection of warm solution.
- Avoiding direct palatal injections.
- Adequate anaesthesia for procedure being performed.

Complications with local anaesthesia

The most significant complication encountered is overdosage. Consequently, maximum doses (Table 3.2) need to be calculated according to weight and preferably written in the notes if more than just a short procedure is being performed. This clinical complication is highlighted in a paper that reviewed significant negative outcomes (death or neurological damage) in children due to local anaesthetic overdose (Goodson & Moore 1983).

Other complications include:

- Failure to adequately anaesthetize the area.
- Intravascular injection (inferior alveolar nerve blocks or, infiltration in the posterior maxillae, directly into the pterygoid venous plexus).
- Biting of the lower lip or tongue postoperatively.
- Facial nerve paralysis by injecting too far posteriorly into the parotid gland.

Consequently, adequate postoperative instructions to both children and parents are necessary to minimize these complications. In addition, inadequate local anaesthetic technique (inexperienced operator, fast delivery of solution and inadequate behaviour management) may jeopardize a successful outcome in an otherwise cooperative child. Allergic reactions to local anaesthetic solutions and needle breakage are rare in children.

The use of *articaine* with adrenaline has gained popularity recently. However, its safety and effectiveness in children under the age of 4 years has not been established. Finally, it is worth noting that there is significant evidence that inadequate local anaesthesia for initial procedures in young children may diminish the effect of adequate analgesia in subsequent procedures (Weisman et al. 1998).

Table 3.2 Maximum dosages for local anaesthetic solutions

Anaesthetic agent	Maximum dose
2% Lidocaine without vasoconstrictor	3 mg/kg
2% Lidocaine with 1:100000 adrenaline	7 mg/kg
4% Prilocaine plain	6 mg/kg
4% Prilocaine with felypressin	9 mg/kg
0.5% Bupivacaine with 1:200000 adrenaline	2 mg/kg
4% Articaine with adrenaline 1:100000 (approximately 1.5 cartridge of 2.2 mL in 20 kg child)	7 mg/kg

Calculation of local anaesthetic dosage:
2% lidocaine = 20 mg/mL
2.2 mL/carpule = 44 mg/carpule
A 20 kg child (approximately 5 years old) can tolerate a maximum dose of 2% lidocaine with vasoconstrictor of:
7 mg/kg × 20 kg = 140 mg Equivalent of 3 carpules (6.6 mL)

Sedation in paediatric dentistry

The decision to sedate a child requires careful consideration by an experienced team. The choice of a particular technique, sedative agent and route of delivery should be made at a prior *consultation appointment* to determine the suitability of the child (and their parents) to a specific technique.

The use of any form of sedation in children presents added challenges to the clinician. During sedation, a child's responses are more unpredictable than that of adults. Their proportionally smaller bodies are less tolerant to sedative agents and they may be easily over-sedated. Anatomically differences in the paediatric airways include:

- The vocal cords positioned higher and more anterior.
- The smallest portion of paediatric airway is at the level of the subglottis (below cords) at the level of the cricoid ring.
- Children have relatively larger tongue and epiglottis.
- Possible presence of large tonsillar/adenoid mass (Figure 3.2).
- Larger head to body size ratio in children.
- The mandible is less developed and retrognathic in younger children and infants.
- Children have smaller lung capacity and higher metabolic rate resulting in a smaller oxygen reserve. Hence children desaturate more quickly than adults.

Patient assessment

The preoperative assessment is among the most important factors when choosing a particular form of sedation. This assessment must include:

- A thorough medical and dental history (including current medications, previous hospitalization and past operations).
- Patient medical status (see ASA classification, below).

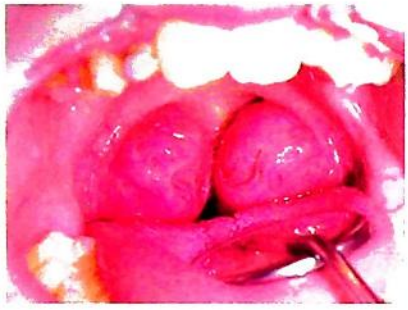


Figure 3.2 Large tonsils cause a significant risk of airway obstruction.

Table 3.3 Resting vital signs in children

Age	Heart rate (beats/min)	Blood pressure (mmHg)	Respiratory rate (breaths/min)
Neonate	120–170	75–85/45	45–60
2–4 years	110–130	90/50	40
4–6 years	100	100/60	30
10 years	90	110/60	25
15 years	80	120/65	12

- History of recent respiratory illness or current infections.
- Assessment of the airway to determine suitability for conscious sedation or general anaesthesia (GA).
- Fasting requirements and the ability of the carer to comply with instructions.
- Proposed procedures being performed.
- Patient's weight and vital signs.

The clinician should be aware that children have resting vital signs that differ according to their age (Table 3.3).

The use of monitoring devices such as pulse oximetry is desirable for lighter sedation techniques and mandatory for moderate and deep sedation. While not currently mandated during relative analgesia, it is suggested that pulse oximetry should be used in all instances when a child is sedated. Sedation and anaesthesia is a continuum and any dentist who sedates children must be capable of resuscitating the patient from any level of sedation deeper than intended (Cote & Wilson 2006). Furthermore, regulations in each country, cultural and socioeconomic factors will determine which particular approach to sedation is chosen. Parental attitudes will also determine the appropriateness of a particular sedation technique.

Pharmacological agents may be administered in a number of ways but the more common routes of delivery include:

- Inhalational sedation.
- Enteral oral sedation or rectal sedation.

- Parenteral or intravenous sedation.
- General anaesthesia.

Inhalation sedation (relative analgesia or nitrous oxide sedation)

Nitrous oxide is a weak anaesthetic agent and is extremely useful in relieving anxiety. The use of nitrous oxide (N_2O) offers the clinician a safe and relatively easy technique to use as an adjunct to clinical care. It can provide a gentle introduction to operative dentistry for the very anxious patient, or an ongoing aid for those who need assistance to accept routine operative dental care. It is effective for children who are anxious but cooperative. An uncooperative child will often not allow a mask or nasal hood to be placed over the nose. It also requires a child of sufficient maturity, age or understanding to help during the dental procedure. The acceptance of the mask is usually the biggest hurdle clinically, and often it is useful to lend the mask to the child prior to their treatment visit so they can practise and familiarize themselves with it. Alternatively, a trial appointment using inhalation sedation (IS) may be beneficial and help the clinician assess the correct concentrations to be used.

Advantages

- Very safe and relatively easy technique when only light sedation is required.
- Rapid induction and easily reversible with short recovery time.
- Can be titrated to required level.

Contraindications

The only specific contraindication to IS in children is a blocked nose. The following conditions may significantly affect the efficacy of this technique and are best avoided:

- Children with severe psychiatric disorders.
- Cystic fibrosis.
- Chronic upper airway obstruction (i.e. large adenoids).
- Communication problems.
- Unwilling patients.
- Pregnancy.
- Acute respiratory tract infections (malignant hyperthermia is not a contraindication to the use of N_2O).

Precautions in the use of nitrous oxide

Although nausea and vomiting may be a problem in some children, this is usually minimized with the routine use of rubber dam during restorative dentistry. Nausea is often brought about by fluctuating concentrations of N_2O due to alternate mouth and nose breathing.

Administration of inhalation sedation

For the safe and effective use of inhalation sedation, it is necessary to have a complete understanding of the different stages of analgesia and anaesthesia with N_2O, the delivery machine and circuits. This requires training in its administration and the careful monitoring of children. In particular, knowledge and training in emergency responses is also essential.

- The equipment must have the capacity to deliver 100% oxygen, and never less than 30% oxygen.
- Prior to commencing sedation with N_2O, always carefully inspect the apparatus and circuit for any leaks. If the reservoir bag does not inflate, examine for a tear.
- A range of fragrant nasal masks is available and useful in making the child feel more comfortable and involves them in the process by offering some choice. Offer the child the mask to take home prior to the treatment appointment, so that he/she can gain familiarity in wearing it.

Procedures

- Recline the dental chair and place the mask on the child's nose so that it fits properly. Once in place, check the mask sits comfortably on the child's face in close proximity to the skin, and secure the mask so that it covers the nostrils completely and does not move unnecessarily during the procedure.
- Determine the minute volume of the child. Variable flow IS machines are essential for use in children.
- Start the procedure with 100% oxygen with active scavenging. Monitor the reservoir bag as the child breathes – it should move at the same rate as the child's breathing with each inspiration and expiration.
- Constant monitoring is critical and the use of pulse oximetry is advised. Assess the child's eyes, general responses and level of consciousness throughout the procedure.
- When using the 'rapid induction technique', administer the N_2O quickly to 50% and then reduce to the appropriate level for that individual.
- When using a slow titration technique, the N_2O is titrated in 10% intervals.

Sensations of analgesia

Children are very open to suggestion. Their thoughts and behaviours can be guided by the dentist. Describe (suggest) the sensations that the child will feel:

- Initial 'heaviness' or sinking into the chair.
- Tingling and numbness of the extremities.
- A warm sensation and a feeling of 'lightness' or floating off the chair with increasing depth of analgesia.

Determining levels of sedation

- Once local anaesthesia has been administered successfully, the N_2O should be lowered to around 30% and maintained at this level. Repeatedly adjusting the levels can be quite disconcerting and so changes should be kept to a minimum.
- Once the procedure is complete, or near completion, the concentration of gas should be lowered, so that the child is maintained on 100% oxygen. This displaces nitrous oxide from the child's body and lessens the risk post-procedural diffusion hypoxia.
- The level at which a patient will be comfortable under IS will be different for every child. Excessive amounts of N_2O may put the child into the excitement stage of

anaesthesia (Guedel Stage II) and may induce vomiting, a feeling of fear or excessive movement.

Give clear postoperative instructions to the parent. The child should rest for the remainder of the day and only engage in sedentary activities. Physical activity should be avoided and the child should remain under continuous supervision.

- Can the child breathe through the nose? (Crying, upper respiratory tract infection, obstruction can all make breathing difficult.)
- Ensure the child is breathing through the nose and not the mouth.
- Most children will be adequately sedated between 70:30–60:40 Oxygen:N_2O.
- Rubber dam is desirable with restorative procedures and minimizes mouth breathing (Figure 3.3).
- Excessive body movement may be a sign of over-sedation.

Ideal patient:

- A cooperative 5-year-old or older (bigger nose allows better use of nasal hood).
- A child able to follow commands (e.g. nose breathing).
- 30–45-min treatment time.

Conscious sedation

The term 'conscious sedation' has been used in the past to imply a patient who is awake, responsive and able to communicate. This verbal communication with the child is an indicator of an adequate level of consciousness and maintenance of protective reflexes. In clinical practice, however, sedation (conscious sedation, deep sedation and/or general anaesthesia) is a continuum. Any technique which depresses the CNS may result in a deeper sedation state than intended, and consequently, clinicians who sedate children require a much higher level of skill with a particular technique, the relevant training and experience and the proper qualifications with the relevant regulating authority.

Sedation of children for diagnostic and therapeutic procedures remains an area of rapid change in medicine and one of considerable controversy. Publications (Cote et al. 2000) have identified several features associated with adverse sedation-related events and poor outcomes, namely:

- Occur more frequently in a non-hospital-based facility.
- Inadequate resuscitation was more often associated with a non-hospital-based setting.
- Inadequate and inconsistent physiological monitoring.
- Often associated with drug overdoses and the use of multiple agents, especially when three or more drugs were used.
- Inadequate preoperative assessment.
- Lack of an independent observer.
- Errors in medication.
- Inadequate recovery procedures.

Figure 3.3 The use of nitrous oxide with rubber dam. Placement of dam ensures that there is no mouth breathing and children are usually more settled. Note the pulse oximeter on the finger. While the use of pulse oximetry is not mandatory, it is a convenient measure of oxygen saturation and provides added safety. A disadvantage of the shape of the nasal mask is that it may make placement of protective glasses difficult

Figure 3.4 An intravenous (conscious) sedation clinic has a similar set up to a normal operating room environment with monitoring and resuscitation equipment.

Considerations for paediatric sedation in the dental setting

- Uniform, multidisciplinary-approved guidelines for monitoring children during sedation are essential.
- The same level of care should apply to hospital-based and non-hospital-based facilities (Figure 3.4).
- Pulse oximetry should be mandatory whenever a child is sedated, irrespective of the route of drug administration or the dosage.
- Age and size-appropriate equipment and medications for resuscitation should be immediately available in a designated 'crash cart', regardless of the location where the child is sedated.
- All healthcare providers who sedate children, regardless of practice venue, should have advanced airway management and resuscitation skills.
- Practitioners must carefully weigh the risks and the benefits of sedating children beyond the safety net of a hospital or hospital-like environment.
- Practitioners must understand that the absence of skilled back-up personnel could pose significant risks in the event of a medical emergency (Cote et al. 2000; Cravero & Blike 2006; Cote & Wilson 2006).

Oral sedation

Oral sedation is the most popular route used by paediatric dentists, due to the ease of administration for most children. There are a number of agents used for this technique including:

- Benzodiazepines (e.g. midazolam).
- Chloral hydrate.

- Hydroxyzine.
- Promethazine.
- Ketamine.
- Fentanyl.

Midazolam has increased in popularity in the last decade due to its safety and short-acting nature, allowing a quick recovery and discharge of the patient. Oral dosage varies from 0.3–0.7 mg/kg, however a maximum ceiling dose (e.g. 10 mg) is usually determined for the older age groups. There are a number of studies that report on the use of oral midazolam, as a successful technique for children with the following selection criteria:

- Children of ages 24 months to 6–8 years of age (depending on individual characteristics, e.g. body weight).
- ASA 1 or 2.
- Short or simple procedures (<30 min).
- Parents who are 'fit' for the technique, that is, they are able to care adequately for the child after the procedure.

Although the technique is successful in the older age groups, it may be more difficult to deal with children of larger size, once sedated. Children over 6 years may become disinhibited and there is a higher frequency of paradoxical reactions in this age group. In addition, obese children may present added airway complications and issues with pharmacokinetics of the drug. Appropriate fasting for elective procedures is preferable.

The main disadvantage of the oral route is that the drugs given cannot be titrated accurately. As most drugs undergo hepatic metabolism, only a fraction of the original dose is active. This makes titration difficult and unreliable, unlike other techniques such as IS and IV sedation. Equally, an overdose cannot be easily reversed. Oral sedation requires enough cooperation of the child to be able to take the medication orally. A child may also spit out the medication. Never re-dose, as it is impossible to accurately determine how much of the drug was ingested.

In the pre-cooperative age group, a knee-to-knee position offers good access for the delivery of oral medications. This technique is also used to treat young children as it allows good control of the patient, easy restraint by the parent/carer and good vision into the mouth by the clinician.

Clinical Hint

- Weigh the child at both the consultation and treatment visit (to minimize dosage errors).
- The dentist should administer the drug which is checked by a second person for accuracy of dose.
- Record time when drug was administered and dosage.
- Onset of effect is usually 20–30 min.
- Administer local anaesthetic.
- Use of rubber dam is recommended.
- Recover child to preoperative state (e.g. able to walk).

Rectal sedation

Although used routinely in Scandinavia and other parts of Europe, this form of sedation is less common in Australasia, Asia, the UK and the USA, because of cultural sensitivities. It is, however, an excellent route for drug administration and provides a more reliable and controllable absorption than the oral route.

Intra-nasal sedation

This implies delivery of medication directly to the nasal mucosa by spray or drops. However, due to reported complications and a poorly understood mechanism of action (there is controversy as to whether the drug is absorbed directly from the blood stream or taken up directly to the CNS), this route is considered as an IV route and consequently requires a higher level of training and monitoring.

Intravenous sedation

This technique requires a highly trained team, including an experienced and appropriately qualified sedationist or specialist anaesthetist, medical nurses trained in this technique, and also a dentist trained in and familiar with the effects of sedation in clinical dentistry. Appropriate monitoring, adequate facilities and recovery options are mandatory for the safe delivery of intravenous drugs. The relevant regulating body in each country dictates these guidelines.

Intravenous sedation has the advantage of the procedure being controllable and may be readily reversible, but as most children are frightened of needles, it might seem an inappropriate form of drug administration in extremely anxious children. Although different drug combinations may be used under IV, in Australia, a combination of midazolam and an opioid analgesic (fentanyl) is often used. These drugs are readily reversible by flumazenil and naloxone, respectively.

Patients with the following criteria ARE considered suitable for IV sedation

- Child patients 8 years of age or older.
- ASA 1 or 2.
- Must have a degree of cooperation to allow injection and have adequate venous access (dorsum of hand or antecubital fossa).
- Obese children (where resuscitation procedures may be difficult and the airway more unpredictable).
- Children with significant respiratory disease such as cystic fibrosis, poorly controlled asthma or sleep apnoea.
- Dysphagia, liquid diet or thickened fluids, history of aspiration pneumonia.
- Poorly controlled epilepsy or reflux.
- Parents who may not provide adequate care to the child postoperatively.

Suitable procedures for IV sedation

- In general, short procedures that require approximately 30 min duration.
- Primary teeth extractions or up to two permanent molars.
- 1–2 quadrants of restorative dentistry.
- Short surgical procedures with good access to surgical area.

Procedures usually NOT suitable for IV sedation

- 3–4 quadrants of dentistry (unless minor restorative).
- Extractions of permanent molars in each quadrant (invasive procedure and bleeding from all four quadrants make airway management more difficult).

- Obese children (where resuscitation procedures may be difficult and the airway more unpredictable).
- Parents who may not provide adequate care to the child postoperatively.

- The technique is highly dependent on the dentist and clinical support staff with experience working under this technique.
- Elevate the chin to minimize/avoid airway obstruction.
- Body movement may be present and this may interfere with treatment.
- Watch breathing pattern, coughing or signs of obstruction.

Intravenous sedation is usually performed in a hospital environment or in dental surgeries that have been duly accredited for the use of these more advanced sedation techniques.

Sedation protocols are strictly defined in many countries by regulations and guidelines. A comprehensive document (PS9) applies to several medical and dental colleges in Australia and New Zealand (PS9, 2010).

General anaesthesia

While it is the most expensive form of treatment, the use of general anaesthesia (GA) for dental treatment has increased globally. This is due to the increase in availability, safety, and an understanding that it is the most appropriate way in which to manage young children requiring extensive dental treatment. This is also in-line with the management of most other invasive medical procedures that are performed under anaesthesia around the world.

It is significant that mortality rates from anaesthesia have decreased around the world. In Australia, in 2005, deaths due to anaesthesia in all age groups was estimated to be 1 : 53 000. The mortality rate for children, although unable to be accurately quantified, was much lower than this, and is estimated to be 1 : 150 000. There are no available figures documenting morbidity in children arising from general anaesthesia.

Although most children will cope with dentistry in a normal setting, many may benefit from delivery of extensive dentistry in one session under GA. The decision to arrange general anaesthesia should not be taken lightly, as there are risks and although less frequent, more serious complications may arise from the anaesthetic. The clinician must make a decision balancing the need against the risk. Economic (public health access and private insurance) and cultural factors and access to anaesthetic facilities may also influence the use of GA. When deciding to place the child under general anaesthesia, the clinician must look at the whole picture.

What is the dental condition?

- Is there gross dental caries?
- Does the child have a facial swelling?
- Is the child in pain?

Is the treatment absolutely necessary?

- Could the patient be managed more conservatively?
- Has the child undergone a period of familiarization?

- Has there been a history of emotional trauma associated with the dental environment?

Certain clinical situations automatically indicate the need for general anaesthesia:
- Multiple carious and abscessed teeth in multiple quadrants in very young children.
- Severe facial cellulitis.
- Facial trauma.

Treatment planning under GA requires experience and careful consideration to avoid treatment failures or repeat GAs. Consequently, GA treatment in paediatric dentistry ideally should only be carried out by dentists with the appropriate postgraduate qualifications.

Consent for treatment

In many countries consent is dependent on the age of the child. For descriptive purposes only the age of consent has been described as it applies in Australia.

Consent for children younger than 14 years of age

In children under 14 years of age, a specific 'Consent form' is required. The parent or guardian must sign the form and a dentist must witness the signature.

Consent for children 14–16 years of age

Children aged 14–16 years must give their own consent for the treatment to be performed. Although a 'responsible informed child' can give this consent, the parent or guardian should also give consent and sign the form. The dentist should explain the procedure and witness the signatures.

Although there is no authoritative statement in statute law regarding consent for children younger than 16 years, common law (Australasia and the UK) dictates that:

> ... as a matter of law the parents' right to determine whether or not their minor child below the age of 16 will have medical treatment terminates if and when their child achieves a sufficient understanding to enable him to understand fully what is proposed.
>
> *(Gillick v West Norfolk Area Health Authority [1986] AC 112, UK)*

Consent over 16 years

A patient 16 years and over must consent for their own treatment.

Emergency treatment

In emergency situations, dental treatment may be performed without the consent of the child or parent or guardian if, in the opinion of the practitioner, the treatment is necessary and a matter of urgency in order to save the child's life, or to prevent serious damage to the child's health (Section 20B of the Children [Care and Protection] Act [1987] NSW, Australia). Fortunately, there are few situations where this will occur in the dental environment, although situations do arise for those working in hospital settings. The overriding point is to 'do no harm'.

It is important that when possible, 'informed consent' be obtained. The clinician must carefully explain all the procedures planned using lay language as appropriate.

All potential risks need to be mentioned, discussed and documented. When completing the sections on standard forms on the nature of the operation, be specific, do not use abbreviations and include all the procedures planned. Where appropriate, use simple terminology to describe the operation.

Pre-anaesthetic assessment for general anaesthesia

A medical history and examination by the anaesthetist is required prior to the procedure. If a patient has complex medical problems, a preoperative anaesthetic assessment may be required as a separate consultation prior to the day of surgery.

The anaesthetist will particularly want to be aware of:

- Behavioural issues, e.g. autism, developmental delay, extreme anxiety and needle phobia.
- Syndromes, e.g. Down syndrome, velocardiofacial syndrome.
- Cardiac disease, heart murmurs, previous surgery for congenital defects.
- Respiratory disease, e.g. asthma.
- Airway problems, e.g. history of croup, cleft palate, micrognathia, previous tracheostomy, known history of intubation difficulties, sleep apnoea.
- Neurological disease, e.g. epilepsy, previous brain injuries, cerebral palsy.
- Endocrine and metabolic disorders, e.g. diabetes, genetic metabolic disorders.
- Gastrointestinal problems, e.g. reflux, difficulty swallowing or feeding.
- Haematological, e.g. haemophilia, thrombocytopenia, haemoglobinopathies.
- Neuromuscular disorders, e.g. muscular dystrophy.

Allergies must be noted, including latex allergy.

Medications must be documented. Most medications should be continued until the time of anaesthesia unless there is a clear reason to withhold (e.g. with anticoagulants or insulin). Consultation with the original prescriber should be made before warfarin or aspirin is ceased to make an assessment of the risk or benefit of ceasing these drugs. Management of diabetic patients will require consultation with the patient's endocrinologist.

Upper respiratory tract infection

If a child presents with an upper respiratory tract infection on the day of surgery, it may be appropriate to delay elective anaesthesia for 2–3 weeks. This decision can be balanced against economic and social issues and patient factors such as the child's age, urgency of treatment, severity of the infection and any other medical problems the child may have. Ultimately, the decision to cancel or proceed is up to the anaesthetist.

Fasting

Normally, the stomach is empty of clear fluids 2 hours after ingestion. Accepted practice for fasting for anaesthesia is:

- 6 h from solids and milk.
- 4 h from breast milk.
- 2 h from clear fluids.

Keeping fasting instructions close to these guidelines will cause the least distress for the patient. Unfortunately, difficulties with organizational factors often result in longer fasting times.

There is no evidence that oral medications taken during the time of fasting increases the risk of aspiration during anaesthesia.

Operating theatre environment

There is often a misconception that everything that happens in an operating room is sterile, and unless staff are familiar with dental procedures, the experience for many children and parents can be overly bureaucratic. While clinicians must follow the protocols of the individual institution under which they operate, it is essential that auxiliary staff appreciate the anxiety that our patients feel and why they are having their treatment performed under general anaesthesia. To reduce the child's fear and anxiety, strategies should be used to help them to cope with the operating theatre environment. For example:

- Minimizing the waiting time prior to the procedure.
- Leaving them in their own clothes. It is not necessary to change into theatre attire for routine restorative procedures.
- Allowing a parent to stay with the child during induction of anaesthesia.
- Using topical local anaesthetic cream such as EMLA® if an intravenous induction is planned.
- Allowing a parent into the recovery area to be with the child as soon as they are awake and stable.
- Reassuring parents at all stages about what to expect.

Premedication

Some children may require oral premedication prior to anaesthesia. Suggested regimens are paracetamol 15 mg/kg and midazolam 0.2–0.5 mg/kg.

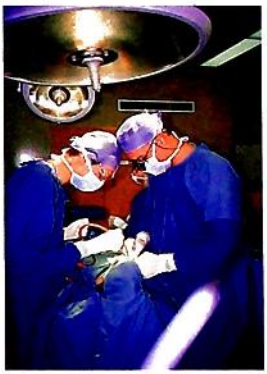
Induction

Anxiety is minimized by allowing a parent to be with the child during induction. Anaesthesia induction may be intravenous or gaseous and will be the choice of the anaesthetist. Sevoflurane with nitrous oxide and oxygen can be given for a gaseous induction. It is not too unpleasant, and with skill, can be used with little distress to the patient. The use of topical local anaesthetic cream prior to insertion of a cannula into a vein alleviates some of the pain of obtaining intravenous access. Some extremely uncooperative children may require induction with intramuscular ketamine 2–3 mg/kg. These are usually older autistic or developmentally delayed children.

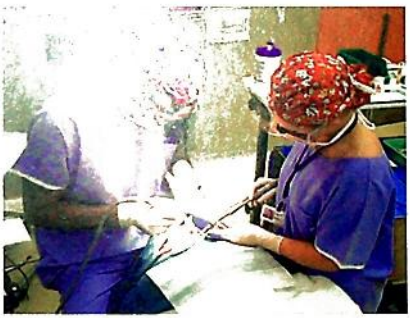
Sharing the airway (Figure 3.5)

- The anaesthetist and dentist must share the airway, so teamwork, and mutual understanding of each other's needs, is necessary.
- Nasotracheal intubation with a nasal RAE (Ring-Adair-Elwyn) tube provides good access for the dentist and a secure airway for the anaesthetist. A throat pack is usually used and it is essential to ensure the removal of a throat pack at the end of the case.
- The throat pack should not be so bulky that the tongue is forced anteriorly limiting the access to the mouth for the dentist. In young children, reduce the size of an adult-sized pack to one-third (ribbon gauze of about 30 cm moistened with saline).

A



C



B

Figure 3.5 Management under general anaesthesia. (A,B) Treatment under general anaesthesia must be conducted in a comfortable atmosphere. There must be cooperation between the anaesthetist and the operating dentist, both of whom need access to the oral cavity and the airway. Nasal intubation is invaluable. Note the anaesthetic machine in close proximity but out of the way of the operating surgeon and the dental assistant also has all the required equipment close at hand. Individual institutions' protocols vary however, and it is not usually necessary to scrub for restorative procedures, as these are considered to be 'non-sterile'. (C) Surgical procedures should be performed under sterile conditions.

- An oral laryngeal mask airway or endotracheal tube provides a satisfactory airway for the anaesthetist, but may or may not give the dentist the access they require, as it encroaches on the work area. However, this is a useful technique for less extensive dental work, such as extractions of primary teeth after trauma or when a nasal tube is contraindicated. If a laryngeal mask airway is used, a flexible one is most appropriate, but it is a less secure airway than an endotracheal tube.
- A face-mask-only technique may be used for simple extractions. The mask is removed for a short time while the extraction is performed. However, the airway must be protected. This can be done by placing a gauze swab behind the teeth being extracted.
- During anaesthesia, it is important to protect the eyes from injury by taping them shut and possibly covering them with padding.
- Before waking the patient, all foreign material such as rolls, gauze and throat packs must be removed and accounted for.

Analgesia

Analgesia, as appropriate, should be given while the patient is anaesthetized. The use of intravenous opioids may be required. As mentioned earlier, local anaesthetics may be used, but often the feeling of numbness around the mouth causes even more distress than the discomfort of the procedure. NSAIDs such as ibuprofen 10 mg/kg 6-hourly, may be prescribed. Paracetamol 15 mg/kg 4-hourly may also be used. Occasionally, oral morphine or codeine phosphate may be required.

Emergence

Ideally, parents should be able to come into the recovery area once the child is awake and in a stable condition. Distress on waking is not uncommon, and can be due to emergence delirium. The child is quite likely to be upset by the unfamiliar environment, an unpleasant taste in the mouth or because their mouth feels different because of missing teeth or new crowns.

Clinical Hints

1. Pre-operative assessment, written consent and information provided to parents at the consultation visit.
2. Dental Treatment Planning. This is an important part to reduce repeat procedures under sedation.
3. Parent contacted 24 hours prior by dentist and hospital staff confirming fasting instructions and admission protocols.
4. On day of GA/surgery
   a. Assessment by anaesthetist – confirms fitness of child for procedure (e.g. URTI, illnesses).
   b. Assessment by dentist and treatment plan discussion with parent(s). In many cases one parent will attend the consultation and the other parent presents on the day of treatment. An important step regarding informed consent.
   c. Check that all dental equipment is operational prior to commencing GA.
5. Induction. Protocol differs in each hospital but often the induction is with a parent present (current trends in paediatric GA).
6. Radiographs and Photos. In cases of dental caries and extractions, intra-oral radiographs are 'mandatory'. The absence of X-rays during dental GA may be considered negligent in some countries. Pre-operative photos are strongly recommended to record the pre-operative status.
7. Use of Rubber Dam is strongly recommended in restorative cases. (Further protection to the airway.)
8. The use of Local Anaesthesia (LA) is not constant in all cases and highly dependent on the operator's choice/experience. For instance LA may only be used for extractions of permanent teeth and surgical procedures.
9. Review Treatment Plan, account for all extracted teeth and disposable materials.
10. Dentist to discuss post-operative outcome with parents on the day of GA.
11. Arrange a post-GA follow up appointment.

Figure 3.6 A day-stay recovery ward with one-to-one nursing care after general anaesthesia. Normal day-stay recovery is a 1–2 hours after the operation.

Categories of anaesthetic risk

American Society of Anesthesiologists (ASA)

- Class 1 – Healthy patient.
- Class 2 – Mild to moderate systemic disease without significant limitations.
- Class 3 – Severe systemic disturbance without limitations.
- Class 4 – Life-threatening systemic disorder.
- Class 5 – Moribund patient not expected to survive >24 hours.
- Class E – Emergency patient.

Suitability for day-stay anaesthesia

Most children who are ASA 1 or 2 will be suitable for day-stay anaesthesia (Figure 3.6). However, children with more severe systemic disease may need peroperative and overnight hospital care to ensure that they are maintaining their airway, tolerating oral food and fluids, that any pain is satisfactorily managed and that there is no ongoing bleeding.

Ward instructions

Postoperative instructions and consultation notes in the medical file must be clear and legible. It is important for nursing staff to understand what procedures have been performed and by whom. They should also know whom to contact if complications arise.

Further reading

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4 Dental caries

David J Manton, Linda Hayes-Cameron

Factors influencing dental caries

Dentists spend most of their time treating dental caries, and yet many clinicians have a poor understanding of the mechanisms by which caries is initiated, how to identify patients at risk and how to put management plans in place to ensure that the disease does not progress. Too often, only the outcomes of the carious process are treated and not the cause of the disease itself.

Dental caries involves a complex process of enamel demineralization and remineralization that occurs due to the action of organic acids produced by microorganisms within the dental plaque. Dental caries is a multifactorial disease, resulting from the interplay between environmental, behavioural and genetic factors. The four factors that influence its progression are shown in Figure 4.1.

Dental plaque biofilm

Increasingly, dental plaque is viewed as a dynamic biofilm (Figure 4.2). This implies that plaque maintains its own microenvironment and has actions that influence oral health. While the plaque biofilm is usually viewed as undesirable, the presence of a healthy biofilm may be positive, e.g. in acting as a fluoride reservoir or as a protective barrier to erosion.

Dental plaque contains bacteria that are both acidogenic and aciduric. Although many bacterial subspecies have been shown to be associated with caries, *Streptococcus mutans* is still believed to be the most important bacterium in the initiation and progress of this disease in combination with lactobacilli. In the caries process, once the pH of the plaque drops below a critical level (around 5.5), it causes desaturation with respect to tooth mineral, the enamel demineralizes and there is net mineral loss. This desaturation will last for 20 min or longer, depending on the availability of fermentable substrate and the effect of the saliva, fluoride and calcium and phosphate.

Mutans streptococci (incl. *S. mutans* and *S. sobrinus*) is the major group of bacteria involved in the initiation of enamel demineralization. Normally, an infant is inoculated vertically with *S. mutans* by the mother/primary caregiver or horizontally by peers at a playgroup or childcare centre. The initial inoculation was thought to be dependent on the presence of a hard surface, and therefore the eruption of the first tooth, however recent research has shown the presence of this organism in newborns. In general, the earlier the detection of significant levels of mutans streptococci, the greater the caries risk of an infant. Repeated consumption of fermentable carbohydrates leads to the proportional overgrowth of mutans streptococci and other aciduric and acidogenic organisms, and the subsequent production of organic acids (lactic, formic, acetic), an increase in the extracellular polysaccharide matrix and a change in the relative components of the microflora leading to increased risk of dental caries.

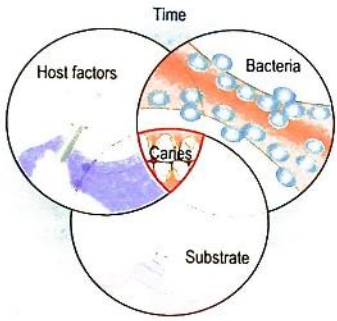


Figure 4.1 The multifactorial nature of caries involves the Host, Substrate, Bacteria and Time.

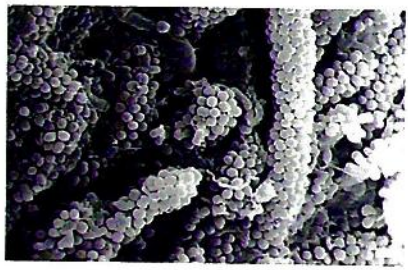


Figure 4.2 Scanning electron micrograph of dental plaque (×4555 magnification). This image shows the typical 'corncob' arrangement of streptococci held by an extracellular polysaccharide matrix on a web of central filamentous microorganisms. (Courtesy, Institute of Dental Research, SEM Unit, Westmead.)

Substrates

Bacteria can use fermentable carbohydrates as a ready source of energy and the end-products of the glycolytic pathway in bacterial metabolism are acids. Sucrose is the fermentable carbohydrate most frequently implicated, but it is important to remember that the bacteria can use all fermentable carbohydrates, including cooked starches. Although any carbohydrate may cause the production of acids, it is the availability of glucose that drives bacterial metabolism to produce lactate rather than weaker by-products such as formate, acetoacetate and alcohols. The lactate is subsequently excreted from the cell as lactic acid. Furthermore, the amount of fermentable carbohydrate is relatively unimportant, as even minute amounts of fermentable carbohydrate will be used immediately – the frequency of exposure is the important factor.

Host factors

The traditional triad of host factors – the teeth, the microbes and their diet – is a simplistic representation of the complex interrelationships in the oral cavity. With regard to the caries process, the quality of tooth structure and the saliva are the major host factors that should be considered. Poor tooth quality, such as hypomineralized enamel, is associated with increased rates of caries, and changes in salivary quantity and/or quality has a profound effect on the whole oral environment, affecting caries rates, oral comfort, periodontal health and resistance to infection.

Saliva

The importance of saliva is often over-looked, however, it has several critical roles in the caries process. Saliva is excreted from the major and minor glands at different rates and with different constituents depending on the presence or absence of stimulatory factors. Saliva stimulated by chewing has increased calcium and phosphate ion concentrations. A gustatory effect, such as that induced by some food acids, has been shown to stimulate a higher flow rate of saliva than stimulation by mechanical chewing. By removing substrate and buffering plaque acid, saliva helps to balance the caries process and has a critical role in remineralization as it provides a stabilized supersaturated solution of calcium and phosphate ions as well as fluoride ions from extrinsic sources. The major constituent of saliva is water (~99.5%), with a wide range of other inorganic and organic components, the most relevant being the salivary proteins, especially the histatins, mucins and statherins, which provide:

- antibacterial and antifungal and antiviral activity.
- lubrication, which also assists in bolus formation.
- inhibition of demineralization and stabilization of calcium and phosphate ions, which assists remineralization.

Therefore, a decrease in the amount or quality of saliva can significantly increase the caries risk.

Time

When acid challenges occur repeatedly, the eventual collapse of enough enamel crystals and subsequently rods will result in surface breakdown. This may take from months to years, depending on the intensity and frequency of the acid attack. This means that in all mouths (as most mouths will contain some cariogenic bacteria) there is continual demineralization and remineralization of enamel; therefore, an individual is never free of dental caries. The process of enamel demineralization and remineralization is constantly cycling between net loss and gain of mineral. It is only when the balance leans towards net loss that clinically identifiable signs of the process become apparent. The long-term outcome of this cycling is determined by:

- The composition and amount of plaque.
- Sugar consumption – especially sucrose (frequency and timing).
- Fluoride exposure.
- Salivary flow and quality.
- Enamel quality.
- Immune response.

Thus the term 'caries-free' often used to describe a child with no visible decay is best changed to the term 'caries-inactive' to more accurately reflect this clinical reality. For the balance to be maintained, there should be sufficient time between cariogenic challenges for the remineralization process to take place. When these challenges become too frequent, or occur when salivary flow is reduced, the rate of demineralization and subsequent tooth breakdown will increase.

The caries process

Dental enamel demineralization is a chemical process. The dissolution of hydroxyapatite can be described simply:

$$Ca_{10}(PO_4)_6(OH)_2 + 2H^+ \rightleftarrows 10Ca^{2+} + 6PO_4^{3-} + 2H_2O$$

with enamel demineralization summarized as a net loss of enamel mineral due to the action of either intrinsic or extrinsic acids, leading to dental caries or erosion. Dental caries is primarily caused by lactic and acetic acids which diffuse through the plaque and into the enamel pores between the rods as neutral ion species, where they dissociate and decrease the pH of the fluid surrounding the enamel crystals. Once dissociated, the protons dissolve the hydroxyapatite crystal surface depending on the degree of saturation of the specific apatite and the inter-rod fluid calcium and phosphate ion concentration increases.

The buffering of calcium and phosphate at the enamel surface and in the plaque biofilm leads to the development of a subsurface (or white spot lesion) with a proportionately hypermineralized surface layer. The optical changes occur due to the increased pore spaces between the thinned rods and the effect this has on the refractive qualities of the enamel. The continuation of this process eventually undermines the support for the surface layer and surface breakdown occurs – the development of a physical cavity.

Caries detection

With the significant reduction in the prevalence, incidence and severity of caries in a great proportion of Western society over the past three decades, notwithstanding some disadvantaged communities and individuals who remain at high risk, the sensitivity of many diagnostic tests for caries has been reduced. Occlusal caries detection is complicated clinically by surface morphology, fluoride exposure, anatomical fissure topography and the presence of plaque and stain. The current methods used commonly for caries detection are:

- Visual and tactile inspection.
- Radiography.
- Transillumination.
- Fluorescence.

Clinical Hint

The traditional use of a sharp probe or explorer in pits and fissures and on demineralized smooth surfaces may damage demineralized enamel, increasing the likelihood of progression of disease and restorative intervention. This invasive method provides little additional diagnostic information, therefore, the blunt probe should only be used to check the integrity of a smooth surface lesion or to clean the fissures gently before examination, and the criterion of 'sticky fissure' should be eliminated.

Newer methods of caries detection

In the past two decades, laser and light-induced fluorescence methods have been developed to detect and quantify enamel mineral content. These methods rely on the different fluorescence characteristics (loss of fluorescence) of demineralized enamel or dentine due to the scattering of light or excitation of materials in the carious lesion.

There is a strong correlation between mineral loss and fluorescence in white spot (demineralized) lesions of enamel, however these results can be confounded by stains, calculus and poor operating technique. Over-diagnosis of caries (false positive results) is the main problem.

The recent commercial development of detection systems such as Diagnodent™, QLF-D™, Canary™, Soprolife™ and CarieScan™ have the potential to increase the accuracy of detection of enamel and dentinal caries. This is because:

- Current clinical methods are limited to detecting enamel caries only at an advanced stage.
- Small changes in mineralization can be detected.
- Mineral loss can be quantified.
- Serial changes in lesion characteristics can be recorded.

Approximal caries

The detection of approximal caries at an early stage is important in paediatric dentistry due to the large proportional size of the pulp in deciduous teeth. New detection methods, such as the Diagnodent pen, have been developed for this task, however, results of research indicate that these should only be used as adjuncts to traditional methods, such as bitewing radiographs and visual and tactile examination (Chawla et al. 2012).

Preventing dental caries

Preventing, reversing or at least slowing down dental caries generally consists of altering one or more of the factors described above.

Diet modification

Although often given minimal attention by dental practitioners, diet is probably the single most important factor in caries risk. Although some dietary habits have changed, the overall consumption of sugar has increased over the past 50 years in most Western countries, especially related to increasing consumption of carbonated beverages. Many foods, although not obviously cariogenic, contain hidden sugars and fermentable carbohydrates. Dietary histories may be useful in identifying those children at high risk. Achieving changes in dietary habits is extremely difficult and therefore advice must be individual, practical and realistic.

- Frequency of intake is more important than overall quantity.
- 'Grazing' between meals should be discouraged.
- The frequent consumption of soft drinks (including fruit juices and sports drinks) should be avoided. Not only are they cariogenic but also extremely erosive and highly caloric.
- Sweets are useful rewards, but should be limited to mealtimes.
- Many foods labelled 'No added sugar' contain high levels of natural sugars.
- Dietary advice should not be all negative. Positive alternatives should be identified.

- The chewing of pH-neutral sugar-free gum increases salivary flow and assists in remineralization and the prevention of demineralization.
- Probably the best dietary advice of all is to 'give teeth a rest' for at least 2 hours between every meal or snack.

Fluorides

The principal mode of action of all fluoridated modalities (toothpastes, rinses, gels and community water fluoridation) is the topical effect at the enamel surface. Even low concentrations of fluoride in the micro-environment around the teeth inhibit demineralization and promote remineralization of the tooth surface. The incorporation of fluoride (as fluoroapatite) into the enamel will decrease its solubility (and increase its resistance to caries). However, it is now recognized that the incorporation of systemically administered fluoride into developing (unerupted) enamel has a lesser role in increasing enamel resistance (see Chapter 4).

Calcium and phosphate

The ability for net remineralization to occur is limited by available calcium and phosphate ions, intrinsically provided by saliva; therefore remineralization is 'saliva limited'. Attempts have been made over past decades to provide supersaturated ionic calcium and phosphate solutions to increase remineralization. However, these attempts have been limited by the low solubility of these ions. Recently, it has been reported that milk-derived casein phosphopeptides stabilize calcium and phosphate in an amorphous form (casein phosphopeptide-amorphous calcium phosphate; CPP-ACP), providing a supersaturated environment that drives remineralization and limits demineralization. CPP-ACP has been added to topical pastes, chewing gums and mouthrinses to increase remineralization and decrease demineralization, and to sports drinks to decrease erosivity.

Fissure sealants

Even in communities with a low incidence of caries, the pits and fissures are still susceptible to caries. The most effective way to prevent pit and fissure caries is by fissure sealing (see Chapter 5).

Plaque removal

Tooth brushing

In communities with water fluoridation, caries mostly occurs in pits and fissures and interproximally. If all plaque could be removed from the tooth surfaces, dental caries would not occur. However, this is not physically and behaviourally possible.

- Besides removing plaque, tooth brushing should be regarded also as vehicle for topical fluoride application.
- The mechanical action of tooth brushing alone will not prevent caries, as it does not effectively remove plaque from the areas mentioned above.
- Children should be encouraged to adopt good brushing habits. Brushing should commence when teeth first erupt, as a part of everyday hygiene. Gauze or a facecloth on a finger, or a small very soft toothbrush may be used to remove the plaque in infants.

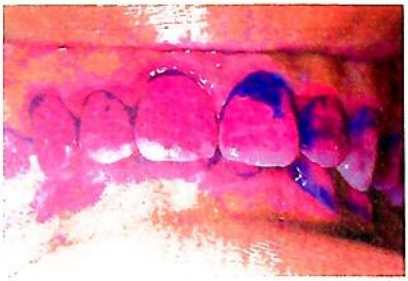


Figure 4.3 Disclosing plaque is an important part of teaching children about oral hygiene and educating parents. (Courtesy Dr Andrew McNaught.)

- It is beneficial for adults to continue to assist with tooth brushing until children are around 8–10 years old and have developed the dexterity to remove plaque effectively by themselves. Ideally, tooth brushing should be carried out twice a day with fluoridated toothpaste but parents should understand that at least once a day is essential to decrease the risk of dental caries (see Chapter 4).

Flossing

In preschool years, and in early mixed dentition, the interproximal surfaces of the primary molars become more at risk of caries. Parents can be shown how to floss these areas when the teeth are in contact and especially if there are signs of demineralization. Older children should be taught to floss themselves. They may find it easier to use one of the commercial floss holders.

Disclosing of plaque

Children, their parents and older patients find it difficult to know when they have effectively removed plaque from their teeth. Disclosing solutions and tablets are very useful for helping patients and parents to see and remove plaque more effectively (Figure 4.3).

Antimicrobials

Antibacterial mouthwashes have become part of the preventive dentistry regimen in the past few years. They do have a role for some patients in caries prevention. In particular, chlorhexidine- and triclosan-containing rinses, gels, toothpastes or varnishes may be used for patients with high risk of caries to help with plaque and microbial control. Their main role is as one part of the multifactorial management of high caries individuals and especially for those who are medically compromised. Systemic antimicrobials (antibiotics) cause significant alterations in oral microflora and have no use in caries prevention.

Determining patients at risk of dental caries

The development of a treatment strategy for a patient that is based on risk factors pertinent to that individual is the gold standard of minimally invasive treatment. That is, before deciding the appropriate methods and preventive products to recommend,

the patient's caries risk should be determined. This may be achieved by considering several aspects:

- Presence of white spot lesions and their activity status.
- Individual and familial past caries history.
- Socioeconomic status.
- Ethnicity.
- Diet.
- Total fluoride exposure.
- Salivary flow and quality.
- Oral hygiene.
- Medical history.
- Presence of developmental defects of enamel.
- Ability to comply with the recommendations.

These factors – especially the background factors – can only be a guide, but they are important to consider when deciding what preventive measures to put in place for individual patients. It has been reported that the dentist's 'hunch or gut feeling' is the most reliable predictor of future caries risk. When the risk has been determined, a preventive programme, incorporating the appropriate methods, can be used. A suggested approach is shown in Table 4.1.

Patient background

- Water fluoride levels.
- Epidemiology of caries, the caries susceptibility of the group to which the patient belongs.
- Ethnicity.
- Socioeconomic variables.
- Educational level, especially of the mother.

Individual characteristics

- Age.
- Different teeth sites are at risk at different ages.
- Teeth may be at particular risk in association with orthodontic treatment (Figure 4.4), those with developmental defects of enamel (Figure 4.5) or in those children with a medical comorbidity.

Medical history

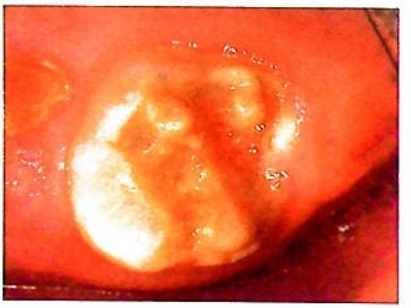
- Frequent medication?
- Does medication alter saliva?
- Is medication sweetened with a fermentable carbohydrate?
- Is oral hygiene a problem?
- Is the diet altered?
- In utero and perinatal history?

Diet

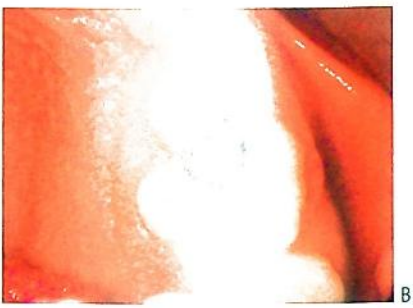
- Fermentable carbohydrate frequency?
- What is the parent's/patient's knowledge of foods with sugars?

Table 4.1 Instituting preventive programmes

	No caries	Early caries	Active caries
Risk	Low risk	High risk Clinical/radiographic enamel demineralization	Very high risk New lesions at each recall, including risk behaviours
Question	How to keep teeth caries free?	How to decrease risk and heal existing early lesions and prevent new lesions?	How to heal or restore existing lesions and prevent new lesions?
Preventive plans			
Plaque	Check what the patient is doing regarding oral hygiene. Either reinforce the behaviour or improve efficacy	Disclose; have patient remove the disclosing agent and clean as appropriate. Advise flossing after brushing with fluoridated toothpaste	Disclose; have patient remove the disclosing agent and clean as appropriate Advise flossing after brushing with fluoridated toothpaste
Diet	Reinforce good dietary habits. Check for recent changes such as use of sports drinks and give advice	Advise against frequent fermentable carbohydrate intake. Check for recent changes such as sports diets	Check the dietary habits thoroughly with a 3-day diet diary, including 1 weekend day. Advise against frequent fermentable carbohydrate intake and check that the patient can identify these
Fluoride	Check that it is being used appropriately – especially brushing with it twice per day	Check that it is being used appropriately Introduce weekly mouthwashes if appropriate for age Consider high-concentration fluoride varnish for demineralized areas and other areas of risk Provide supplementary ionic calcium and phosphate via products like Tooth Mousse/MI Paste	Check that it is being used appropriately Introduce daily mouthwashes if appropriate for age Apply concentrated fluoride treatments such as gels or varnishes Provide supplementary ionic calcium and phosphate via products like Tooth Mousse/MI Paste
Fissure sealants	Apply to deep retentive fissures only or if patient requests	Apply to molars, especially those showing demineralization	Ensure all open lesions are restored temporarily or permanently Apply fissure sealant to all molars and premolars – use GIC for semi-erupted teeth
Recall	12-monthly if there have been two 6-month periods of low caries activity	6-monthly while there are signs of caries activity or high risk remains	6-monthly or 3-monthly with medically compromised or very high-risk children



A



B

Figure 4.4 (A) Hypomineralized and/or hypoplastic teeth will be predisposed to caries, particularly if the enamel deficiencies are plaque retentive. If the enamel is completely absent and dentine is exposed, then these teeth can deteriorate rapidly. (B) Interproximal lesions that are cavitated and unable to be thoroughly cleaned do not arrest.

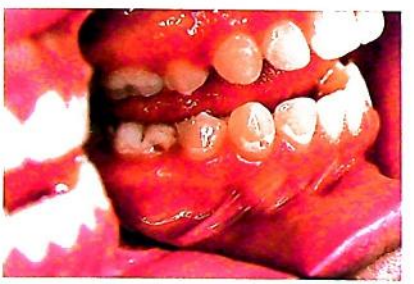


Figure 4.5 It is important to note that risk can change. A child who was previously free of caries has developed cervical lesions during orthodontic treatment.

- Protective foods (i.e. dairy foods)?
- Are there risk-associated habits?
- Nursing bottle in bed or at-will breast-feeding (esp. as it relates to other social factors)?
- Use of sipper bottles or feeding cups?
- Frequent snacking in sports training?

Family history of caries

Cariogenic bacteria are transmitted vertically from the parents and possibly horizontally from other caregivers and close associates such as siblings and playgroup members. Most cariogenic bacteria are commensal oral microorganisms, and require the appropriate environment to thrive, so trying to avoid inoculation will not prevent caries long term in an otherwise susceptible individual.

- Are the parents caries active?

Intra-oral information

Caries history

- Existing lesions (holes).
- Past restorations or caries around existing restorations.
- Signs of active demineralization ('chalky' white spot lesions).

Eruption of teeth

- The recently erupted dentition may be more at risk than the mature dentition due to immature enamel.
- It sometimes takes time for caries to progress to a visibly detectable stage. Risk may not be obvious for 3–4 years after eruption, however, high risk can occur at any stage of life.

Oral health

- Presence of plaque, esp. mature plaque?
- Is fluoridated toothpaste used?
- How frequent and effective is the oral hygiene?
- Is plaque accumulation associated with demineralization?
- Does the patient use floss?

Tooth morphology

- Deep and uncleansable pits and fissures.
- Enamel hypomineralization or hypocalcification (in utero, perinatal and infantile history).
- Have the teeth been fissure sealed?

Radiographic signs

- Are lesions increasing in size?
- How quickly are the lesions progressing?

Other diagnostic tests

- Use of light and laser fluorescence (e.g. QLF™, Sopralife™ and DiagnoDent™).
- FOTI (fibre-optic trans-illumination).
- Electrical impedance (CarieScan™ Pro).
- Photothermal radiometry (Canary™).

Saliva

- Is the resting and stimulated rate of flow and buffering capacity normal?
- Is there anything that may be affecting the composition of the saliva?

Early childhood caries

One of the causes of early childhood caries or rampant caries in young children is allowing infants and toddlers to sleep with a bottle containing fermentable carbohydrates (Figure 4.6). The reported prevalence ranges from 2.5% to 15%.

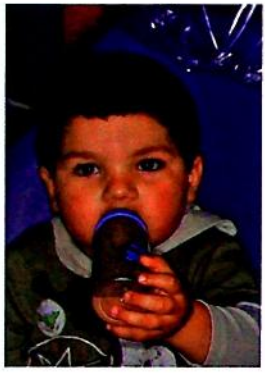


Figure 4.6 Parents should be encouraged not to use the bottle as a pacifier.

Characteristics of feeding bottle induced early childhood caries

- Rampant caries affecting the maxillary anterior teeth (Figure 4.7).
- Lesions appear as teeth erupt – later on posterior teeth, both the maxillary and mandibular first primary molars.
- Canines are affected less than first molars because of later eruption.
- Mandibular anterior teeth are usually unaffected. This is thought to be because of salivary flow and the position of the tongue. However, if they are affected, this would indicate extremely high risk.
- The bottle is often used as a pacifier to get the infant to sleep.
- Early childhood caries occurs in all socioeconomic groups and as such, often reflects the social dynamics of the family. Children who are difficult sleepers or have colic are often pacified with a bottle. The bottle can contain any liquid with fermentable carbohydrate, even milk. Commonly, drinks and juices containing vitamin C are used.
- This pattern of caries may also occur with prolonged at-will breast-feeding in conjunction with a cariogenic solids diet. The at-will breast-feeding may be an associated factor with other sociodemographic variables that influences caries risk.
- Once the habit has ceased and/or the areas of decay become self-cleansing, the carious process may arrest and the lesions will appear darker (Figure 4.8).

Aetiological factors of early childhood caries

- Long periods of exposure to cariogenic substrate. In infants, if this is from a feeding bottle, exposure to cariogenic fluids can be for up to 8 hours. However, other habits such as 'grazing' (snacking on cariogenic food constantly) also puts many children at risk, as does the use of feeding cups and sipper bottles filled with sugary fluids that toddlers walk around with.
- Low salivary flow rate at night, and therefore reduced time for remineralization and acid buffering.

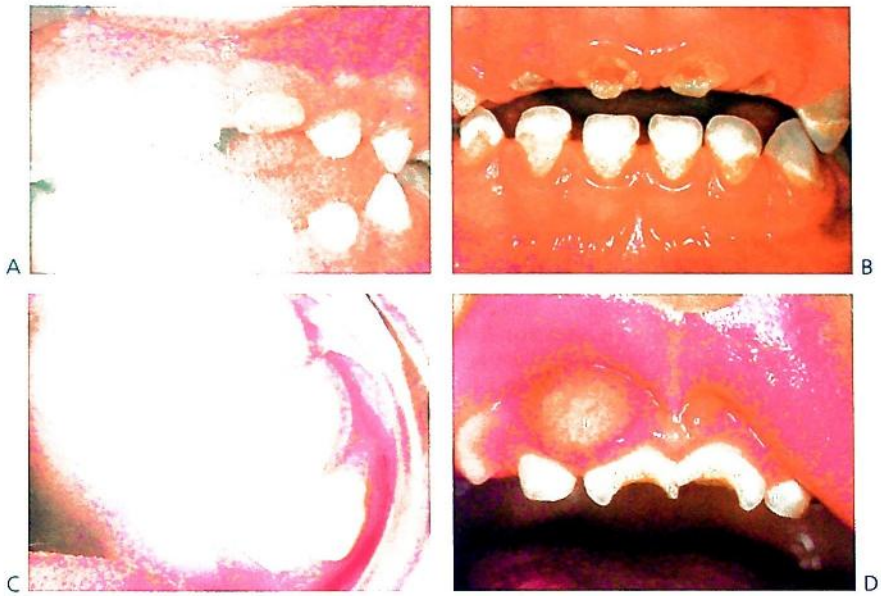


Figure 4.7 Early childhood caries (ECC). (A) ECC showing the characteristic pattern of decay. The upper anterior teeth and the molars are affected but the lower anterior teeth are spared. (B) A particularly rampant case of ECC where a pacifier had been dipped in honey (or any other cariogenic agent). Both the upper and lower anterior teeth may be affected. (C) The first primary molars are carious due to a bottle habit at night. There is no interproximal decay (due to open contact points) and the canines that have erupted later, are unaffected. (D) An abscess following pulp necrosis in carious upper incisors. Extraction is required and the abscess will resolve following removal of the tooth and drainage of the pus. It is important to note that the teeth are not fractured due to trauma, but are carious.

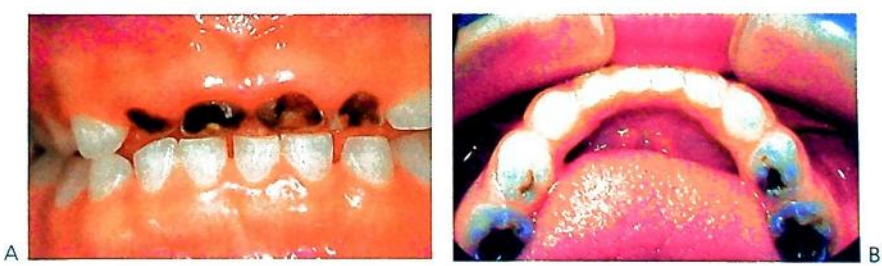


Figure 4.8 (A) ECC in a child showing arrested caries. Removal of the cause of caries has allowed the process of demineralization to slow down. (B) When the carious process has resulted in an open lesion, it is possible to slow and arrest the process. These lesions are quite different to those shown in Figure 4.4B and while they appear quite extensive, there was no pulp exposure.

- Parental history of active and untreated caries – particularly in the mother.
- Parent(s) in situations of social stress.

Social aspects of night-waking and feeding

It is important to understand normal feeding routines or when these may be associated with habits contributing to early childhood caries. Breast-feeding is encouraged, however, some mothers are unable to breast-feed and there may be circumstances when babies have medical interventions that restrict their ability to breast-feed and so take bottle-feeds (cleft palate or early days of the neonatal intensive care admission). It is NOT recommended that children fall asleep with a bottle at night.

General recommendations for infant feeding routines

• Birth to 3 months	Breast-feeding up to 10 times in 24 h or 6–8 formula bottles in 24 h.
• 3–6 months	3–4 hourly feeds, i.e. 6–8 breast-feeds or 5 formula bottles. The early morning 2 a.m. feed is ceased and usually the infant will have only one feed over night.
• 4–6 months	4–5 milk feeds. Taste and texture of solids may be introduced from 4 months, although these recommendations may differ between countries.
• 6–12 months	4 milk feeds, last one prior to midnight. 3 major solids meals – breakfast, lunch and dinner with morning and afternoon snacks.
• Over 12 months	No milk feeds required after bedtime but may have a bedtime ritual of milk feed, tooth cleaning, then a bedtime story in bed.

All infants wake at least once overnight and often resettle themselves. This may happen without the knowledge of the parents. Infants wake more often when they are unwell; there are changes in their social and physical environment or when learning a new developmental skill, such as pulling themselves up on the side of the cot to stand. If the parent offers milk or another feed to assist with resettling, then this night-waking behaviour will be reinforced and the infant will learn that the reward is the means to resettle. Unfortunately, this sets up conditioning for having feeds overnight that are not required developmentally (i.e. after 10–12 months of age). Babies that fill up with milk overnight may refuse their solid meals during the day. Children are best settled with comforts such as patting, cuddling and verbal reassurance. Unnecessary additional overnight feeds may reflect the parent(s) inability to cope with a wakeful, crying infant. Some reasons for offering feeds overnight include:

- Parental or infant illness.
- Maternal guilt, stress, anxiety or depression.
- Marital discord.
- Domestic violence.

In these cases, used as a quick fix to try to settle the infant.

It is important to give appropriate advice to the family about early childhood caries. Blame should never be attributed; in many situations, the condition may have arisen out of ignorance, misinformation or tiredness and frustration of coping with an infant with poor sleeping habits. Elimination of an 'at risk' bottle habit can be achieved by gradually reducing the amount of sugar in the bottle by diluting with water, which may be done over several weeks. Alternatively, some parents find it easier to remove the bottle, immediately offering sips of water only. The only dentally safe fluid in a feeding bottle is water.

Understanding developmentally appropriate feed, sleep and play patterns will help both the parents and the dentist come to the conclusion that the family may require extra assistance with the infant's routine. Providing empathy and support for the challenges they are experiencing with night waking is essential before offering education regarding early childhood caries. Referral to community health nurses, lactation consultants or secondary parenting services is recommended to assist with the physical and psychological support necessary to change these behavioural patterns.

Management

- Cessation of dietary habit.
- Dietary advice and modification.
- Fluoride and/or CPP-ACP application.
- Use of biofilm moderating/antimicrobial products.
- Restoration of teeth. This may consist of intracoronal tooth-coloured restorations in small lesions. For more extensive lesions, composite resin-strip crowns for anterior teeth and stainless steel crowns for posterior teeth may be required (Chapter 6).
- Extractions if required. Loss of the upper anterior teeth will not result in space loss if the canines have erupted. Speech development should not be affected. If posterior teeth have to be extracted, the parents will need to be informed about possible space-loss, and an assessment should be carried out to determine if a space maintainer is appropriate.

Treatment under general anaesthesia is often required for small children. Unfortunately, a large proportion of children continue to get new lesions even after extensive treatment, as the risk factors have not been altered. The use of motivational interviewing methodology is seen as a more effective way of gaining positive behavioural change.

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5 Fluoride and dental health

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Introduction

Fluoride has made an incredible impact on the oral health of millions of adults and children. Physiologically, fluoride is a unique member of the halogen family, in that it is termed a 'seeker of mineralized tissue'. It is this affinity with mineralized tissues which explains how fluoride can strengthen the teeth and prevent or heal dental caries.

Mechanism of action

Concepts of how fluoride prevents caries have changed markedly since the first water fluoridation schemes were introduced in the USA in the late 1940s and early 1950s. When ingested systemically, fluoride is incorporated into developing tooth enamel. The fluoride ion displaces some hydroxyl groups in hydroxyapatite to form fluorapatite. The smaller anion causes crystal stress but results in a significantly less soluble material. Initially, researchers focused on the systemic effect of fluoride as the key factor in the reduction of dental caries. However, the evidence for the systemic effect has been superseded by the realization that the reaction of the fluoride at the microenvironment of the plaque enamel interface encouraging remineralization is of major significance in terms of reducing the levels of dental caries (Centres for Disease Control and Prevention, 2001).

The important points to remember are:

- Fluoride acts topically, promoting remineralization and reducing demineralization.
- The mode of action is predominantly post-eruptive and prevention of caries requires lifelong presence of fluoride.
- When remineralization takes place in the presence of fluoride, the remineralized enamel is more resistant to caries because of increased fluorapatite within the enamel matrix.
- Only low levels of fluoride are required at the plaque enamel interface to promote effective remineralization.
- Fluoride has some effect on the glycolytic pathway of oral microorganisms reducing acid production and interfering with the enzymatic regulation of carbohydrate metabolism. This reduces the accumulation of intracellular and extracellular polysaccharides and leads to lower volumes of plaque.

Community fluoridation

There are three ways to offer fluoride on a community-wide basis: utilizing water, salt and milk.

Water fluoridation

The natural level of fluoride in drinking water is very variable. However, in reticulated community water supplies that are fluoridated, the concentration of fluoride is adjusted to approximately 0.8–1 ppm.

The majority of International Health Agencies agree with the World Health Organization in support of the continuation of community water fluoridation, as it is an effective, efficient, socially equitable and safe population approach to caries prevention.

The reduction in dental caries in fluoridated communities ranges from 20% to 40%, which is considerably less than was the case when it was first introduced in the USA because of the general increase in availability of fluoride from other sources (Downer and Blinkhorn, 2007). However, when fluoridation programmes have been discontinued, there is rapid increase in dental caries within a short time frame (Burt et al., 2000).

There are a number of points of which many members of the public are unaware when considering the value of water fluoridation:

- Fluoride benefits adults as well as children.
- There is a decreased prevalence of root-surface caries in lifelong inhabitants of areas with fluoridated water.
- The preferred source of fluoride is from community water fluoridation, as it benefits all the population and is a cost-effective intervention.
- The continuing existence of approximately 20% of children with a high caries experience indicates the need to maximize protection through the use of community water fluoridation.

The market for bottled water has grown rapidly in the last decade, and for many individuals water consumption from this source may have fully replaced reticulated water. The fluoride content of bottled water is usually very low, and consumers of bottled water in fluoridated communities will miss out on the benefits of fluoride.

Some water filters may remove fluoride, although this is mostly limited to those with reverse osmosis, bone or charcoal filters, distillation or ion exchange. Normal membrane filters will not remove a small ion such as fluoride. Ceramic and carbon filters retain fluoride in the filtered water. Filters that do not remove fluoride should be clearly labelled.

Salt fluoridation

Salt enriched with iodide has been used in many countries as an effective means of preventing goitre. It was a logical step to include fluoride in domestic table salt. It has the advantage of offering choice and does not encourage salt consumption as it is marketed as an alternative to the standard product. The amount of fluoride added is 250 mg F^-/kg salt (250 ppm).

Switzerland was the first nation to pioneer salt fluoridation and it is now available in Spain, Hungary, France and parts of Brazil. It is certainly a practical alternative to water fluoridation, but the research base is much more limited on its absolute effectiveness, especially now that fluoride toothpaste is readily available.

Milk fluoridation

Bovine milk is used as a food for babies and young children, plus in many countries free milk is offered to children at school. These positive points were noted by researchers as a potential way to supplement children's fluoride intake.

Despite its practical simplicity, milk fluoridation has not been implemented on a wide scale, mainly because of logistical difficulties and the fact that fluoride toothpaste is readily available. It may well have a place in developing countries, where the milk will improve nutrition as well as offering the benefits of fluoride.

Topical fluorides for home use

Lifetime protection against dental caries results from the continuous presence of fluoride in low concentrations, that will enhance the remineralization of white spot lesions, control initial invasive carious lesions and limit lesions occurring around existing restorations for both adults and children (Adair, 2006). An optimal concentration of fluoride each day at both the plaque/enamel interface and in saliva, will help minimize the risk of caries. Factors that should be considered when advising on a fluoride regimen include:

- The number of carious lesions or areas of demineralization.
- The frequency of consumption of sugary foods and drinks.
- Patient's age.
- Patient's compliance with oral health advice.
- Community water fluoridation levels.
- Any medical conditions.

Fluoride toothpastes

Of all the different ways of offering topical fluoride, the most common and simplest way in which to maintain elevated fluoride concentrations at the plaque/enamel interface, is the use of a toothpaste containing fluoride. Fluoride is added to toothpastes in one of the following forms:

- Sodium fluoride.
- Sodium monofluorophosphate (MFP).
- Stannous or amine fluoride.

The use of fluoride toothpastes has led to a 25% reduction in the prevalence of caries in many countries (Davies et al., 2002). It is recommended that children should brush twice a day with a toothpaste containing an appropriate concentration of fluoride, preferably last thing at night before bed and on one other occasion, ideally after breakfast. It is essential to ensure all parents are aware that vigorous rinsing after brushing will reduce the preventive effect of the toothpaste because the active agent 'Fluoride' will be washed away.

Advice on the type of toothpaste which young children should use, in terms of fluoride concentration is problematic (Franzman et al., 2006), as international guidelines differ. Members of the dental team must familiarize themselves with the guidelines appropriate for their own country and practice location. In Australia and the USA, fluoridation of public water supplies is quite common, whereas in Europe there are only a few community water fluoridation schemes (Walsh et al., 2010). However, there are a number of factors that health professionals must consider when offering advice to parents on fluoride toothpaste usage, namely:

- Low fluoride toothpastes (<1000 ppm) should not be used in areas where the water supply is not fluoridated, as they have a greatly reduced caries preventive effect.

- The age when parents are advised to begin brushing their children's teeth varies between countries and members of the dental team should be familiar with the appropriate national policy.
- General advice is that children aged 6–36 months should use only a smear of toothpaste on the brush.
- Brushing with fluoride toothpaste before 12 months of age offers larger reductions in dental caries; but parents must supervise and only a smear of toothpaste should be placed on the brush.
- Parents of children aged over 6 years should be advised to place a 'pea' sized amount of toothpaste on the brush.
- Children over 6 years of age should use a 'family' toothpaste (1000–1450 ppm). However, earlier use of a 'family' paste is indicated if children are at risk of developing dental caries.
- Young children (up to the age of 7 years) should be supervised when brushing as this monitors toothpaste usage, has been associated with greater reductions in dental caries and reduces the chances of fluorosis in the upper anterior teeth.
- Children over 10 years of age and considered to be at high risk of developing caries or have active carious lesions may be prescribed a toothpaste containing >1400 ppm fluoride. The availability of these high fluoride toothpastes varies from country to country. They may only be available on prescription in some locations.
- Safe storage of all fluoride toothpastes is important to ensure young children do not eat paste from the tube. This advice to parents should be reinforced on a regular basis.
- Brushing with a fluoride toothpaste is at the heart of any preventive programme. There is no 'right way' to brush. The important goal is to make sure the toothpaste is used twice a day and not washed away by rigorous rinsing.

Fluoride mouth rinses

In some countries, school-based daily fluoride mouthrinse (0.05% sodium fluoride) programmes have been used to offer protection from dental caries. While rinses do offer a benefit, their use as a public health measure has declined for a number of reasons:

- The widespread use of fluoride toothpaste has reduced the potential benefits for the average child.
- Rinse programmes are labour-intensive, as children need to be supervised whilst rinsing.
- Schools may not want the inconvenience of a daily rinsing programme.

Nevertheless, in some countries where people live in remote locations and toothpaste is expensive, local school-based fluoride rinsing programmes can be an effective public health measure. Members of the dental team may also offer fluoride rinses to individual patients with active caries, provided they are over 6 years of age.

Two types of rinses are available.

Daily

- 0.05% w/v neutral sodium fluoride (220 ppm F^-).
- Partly acidulated solution of sodium fluoride, phosphoric acid and sodium monobasic (200 ppm F^-).

Weekly

- 0.2% w/v neutral sodium fluoride (900 ppm F^-).

The most popular rinse is the daily one as it is simpler to rinse on a regular basis than trying to remember to use a product just once a week. Also maintaining a low level of fluoride in the mouth on a daily basis fits in with our understanding of the mode of action of fluoride on the remineralization of enamel.

It is important to use the rinse at a different time to brushing with a fluoride toothpaste as using them together does not offer an additive effect. A good time to rinse is when a child returns home from school, as there will be plaque present which incorporates the fluoride and releases it slowly over time.

There are a number of patient groups who will benefit from the prescription of a daily fluoride rinse:

- Children undergoing orthodontic treatment. The rinse can reduce demineralization around the brackets.
- Patients with hyposalivation due to medications or those with congenital absence of the major salivary glands.
- Children with medical problems for whom caries could be a serious problem, e.g. cardiac patients and individuals with bleeding disorders.
- Children with active dental caries.
- Some individuals who find toothbrushing difficult (but in many cases they will also find rinsing a problem).

Fluoride rinses are not recommended for children before the eruption of the permanent incisors because many younger patients will swallow the rinse and this may cause fluorosis.

Tooth mousse or casein phosphopeptide-amorphous calcium phosphate crèmes

CPP-ACP and CPP-ACPF are available as crèmes for topical application at home (Tooth Mousse®; Tooth Mousse Plus®, GC Corp, Japan) to be applied to surfaces at risk of caries, erosion or with white spot lesions. CPP-ACPF releases fluoride, calcium and phosphate ions for local remineralization of enamel (Reynolds, 2008).

The crème is applied to teeth after brushing by smearing across tooth surfaces with a clean finger or cotton-tipped applicator. The crème should not be rinsed out.

- Tooth Mousse Plus® contains 900 ppm F^- and should be used by people over 6 years.
- The crème should not be used by people with a milk protein allergy.

Fluoride tablets

At one time, fluoride tablets were widely recommended as a useful caries preventive measure. However, they have been superseded, as fluoride toothpastes now dominate the market and offer a better level of protection from dental caries. In addition, research has shown that compliance with tablet taking regimes is very poor and the consumption of up to 1 mg of fluoride in 1 tablet is linked to fluorosis. Thus fluoride tablets are no longer routinely recommended (Den Besten, 1999; Tubert-Jeannin et al., 2011).

Stannous fluoride gel

A stannous fluoride (SnF_2) treatment gel in a methylcellulose and glycerine carrier (marketed as Gel Kam® by Colgate Oral Care) can be used at home for the remineralization of white spot and hypomineralization lesions of enamel (e.g. molar or incisor hypomineralization). Anecdotal clinical reports support the efficacy of this product, e.g. where localized remineralization is desirable prior to the placement of definitive restorations. The 0.4% stannous fluoride gel has also proved effective in arresting root caries and has been incorporated into a synthetic saliva solution to reduce caries in post-irradiation cancer patients.

- Contains 1000 ppm F^- and 3000 ppm Sn^{2+}.
- A very small amount is placed on a cotton bud and applied to dried tooth surfaces by adult patient or for a child by the parent at home.
- The parent must fully understand the instructions given about the use of the gel.

Professionally applied fluoride products

Fluoride varnish

Varnishes were developed many years ago in order to prolong the contact time between the fluoride and dental enamel. As with fluoride rinses, varnishes have been used as both a public health prevention agent and as a specific treatment for individual patients in general dental practice (American Dental Association, 2006).

The efficacy of fluoride varnish in population health programmes to reduce dental caries has been called into question by recent research (Milsom et al., 2011), but a systematic review concluded that fluoride varnishes can offer a more than 40% reduction in dental caries (Marinho et al., 2009). However, the studies included in the review were undertaken before fluoride toothpaste had achieved total market dominance. The general presence of fluoride from toothpaste means that the use of varnish on a population basis does not offer an added advantage to the majority of participants enrolled in a varnish-based preventive programme.

The evidence on the value of varnishes for use on individual patients in primary dental care is not available but the ongoing relationship between a family and the dental team could be a positive factor in their efficacy. Varnishes can therefore be part of a prevention plan for individual patients, and indications for their use are:

- Hypersensitive areas of enamel and dentine.
- An alternative to fissure sealants on occlusal surfaces of permanent molars for apprehensive children until such time that effective sealant placement can be undertaken.
- Acclimatization for nervous children.
- Local remineralization of white spot lesions.
- As part of a preventive programme for children with active caries in the primary and/or permanent dentitions.
- A routine preventive measure for medically compromised and other special needs patients.

It must be stressed that the use of fluoride varnish for the control or prevention of dental caries is a long-term commitment (Sköld et al., 2005). It offers little value when used as a 'one off' intervention, and therefore should be applied at least three times per year for optimal effect. Varnishes are simple to apply; prophylaxis of the teeth is

not required routinely as fluoride uptake is not reduced by surface plaque. In fact, plaque can serve as a recycling reservoir for fluoride and allow prolonged exposure to enamel. Drying teeth before application facilitates adhesion and may also be beneficial for fluoride uptake.

With such highly concentrated fluoridated products, great care must be taken to avoid using liberal amounts of varnish on young children. The manufacturer of Duraphat® (Colgate Oral Care) recommends application of the following amounts of varnish, which should not be exceeded:

- Primary dentition: 0.25 mL (6 mg F^-).
- Mixed dentition: 0.40 mL (9 mg F^-).
- Permanent dentition: 0.75 mL (17 mg F^-).

Two readily available varnishes are:

- Duraphat® (Colgate Oral Care) – an alcoholic solution of natural varnishes containing 50 mg NaF/ml (5% NaF, 22 600 ppm F^-). This varnish resin remains on the teeth for up to 12–48 h after application, slowly releasing fluoride from the wax-like film.
- Fluor Protector® (Ivoclar Vivadent) is a clear varnish which contains 0.9% difluorsilane in a polyurethane varnish base with ethyl acetate and isoamyl propionate solvents. The fluoride content is equivalent to 0.1% or 1000 ppm in solution. As the solvents evaporate, the fluoride concentration will increase to much higher values.

Concentrated fluoridated gels, foams, solutions and cremes

Concentrated fluoridated gels are marketed as both caries-preventive and treatment gels. There is recent clinical evidence that concentrated fluoridated gels are more effective in the permanent dentition than the primary dentition, benefiting particularly the first permanent molars. Variable dosage during application, followed by inadvertent swallowing, can result in the ingestion of large amounts of fluoride, which may contribute to mild fluorosis of mineralizing permanent teeth. Therefore, these products need to be used with great care and should not be offered to children under 10 years of age.

High concentration fluoride gels (e.g. 9000–12 300 ppm F^-) should be limited to professional use in the dental practice and not dispensed for home use. Lower concentration gels (e.g. 1000 ppm F^-) can be used at home following careful professional instruction. The use of these products has declined as fluoride varnishes are simpler to use and can be targeted on specific teeth.

Acidulated phosphate fluoride gels

Acidulated phosphate fluoride (APF) gels, containing 12 300 ppm F^- (1.23% APF) consist of a mixture of sodium fluoride, hydrofluoric acid and orthophosphoric acid. There are also gels containing 5000 ppm F^- which contain sodium fluoride, phosphoric acid and sodium phosphate monobasic.

- Such highly concentrated fluoride gels should be limited to professional use and should not be dispensed for home use.
- The incorporation of a water-soluble polymer (sodium carboxymethyl cellulose) into aqueous APF produces a viscous solution that improves the ease of application using custom-made trays.
- Thixotropic gels in trays flow under pressure, facilitating gel penetration between teeth.

Neutral sodium fluoride gels

A neutral pH gel (e.g. 2% w/v neutral NaF gel, 9000 ppm F^-) can be used for cases of enamel erosion, exposed dentine, carious dentine or where very porous enamel surfaces (such as hypomineralization) exist.

- Sodium fluoride is chemically very stable, has an acceptable taste and is non-irritating to the gingivae. It does not discolour teeth, composite resin or porcelain restorations, in contrast to APF or stannous fluoride, which may cause discoloration.
- A neutral pH fluoridated gel or solution is preferred where restorations of glass ionomer cement, composite resin or porcelain are present as acidic preparations may etch these restorations.

Stannous fluoride solution

- 10% stannous fluoride may be used to target local 'at-risk' surfaces of teeth such as deep fissures and pits or white spot lesions on accessible proximal surfaces.
- Rapid penetration of tin and fluoride into enamel and the formation of a highly insoluble tin fluorophosphate complex coating on the enamel is the main mechanism of its action. The stannous ion may cause discolouration of teeth and staining on margins of restorations, particularly in hypocalcified areas.

Planning a preventive programme in the practice

Twice-daily tooth brushing with a fluoridated toothpaste beginning before 2 years of age together with dietary advice to reduce the frequency of consumption of sugary foods and drinks is the cornerstone of a preventive programme to ensure minimal caries activity. Reviews of epidemiological data in non-fluoridated communities indicate that the twice-daily use of a toothpaste containing fluoride will prevent new caries development in approximately 80% of children, and an estimated 60–70% of adults.

Members of the dental team offer care to many different families with varying levels of dental disease. It is important to tailor advice to maximize the benefit for each individual child. The aetiological factors leading to the development of caries are multifactorial, so risk assessment should involve all the likely key factors. Understanding the social, behavioural, microbiological, environmental and clinical factors still remain essential in the determination of caries risk during specific time periods.

Some authorities suggest that caries risk can be subdivided into moderate and high. Such definitions are somewhat arbitrary and it is more practical to focus on changing behaviour and prescribing appropriate preventive products for all children at risk of caries.

The scope of a preventive programme will be influenced by the following factors:

- white spot demineralization on cervical surfaces.
- visible deposits of dental plaque and/or gingivitis.
- new carious lesions visible on clinical examination or on radiographs.
- patients with orthodontic appliances.
- individuals who have chronic medical problems.
- children with special needs.
- frequent consumption of sugary foods and drinks.
- infrequent attendance for dental recall.
- repeated attendance for emergency treatment.

Patients with these signs will alert clinicians to the importance of concentrating on preventive advice and therapies (Threlfall et al., 2007). The failure to guide and inform families on how to control and prevent caries may lead a child to suffer pain and require more clinical intervention. The preventive program can be a team effort utilizing the skills of the dentist, hygienist and dental therapist, each playing a part in the delivery of appropriate advice.

The preventive plan will have two themes:

- prescription of preventive products.
- behaviour change.

Preventive products

It is well-known that all preventive plans must include a fluoride component in order to successfully control or prevent dental caries. The specific products will depend on the severity of the caries problem and the age of the child.

It is important to note that Early Childhood Caries in children under 3 years of age is primarily a dietary problem and fluoride products, whilst helpful, cannot overcome the constant intake of sugary foods and drinks. With that caveat in mind the preventive products at our disposal are:

- **Fluoride toothpaste** – children must brush twice a day with a family toothpaste. Parents are advised to use a smear of paste on the brush for children under 6 years of age.
- **High strength fluoride toothpastes** – these can contain 2800 to 5000 ppm fluoride and are usually only available on prescription or can only be purchased from pharmacies. These pastes offer a high level of caries protection but must be reserved for children over ten years of age and used as recommended by the dental team. May not be available in some countries.
- **Fluoride varnishes** – these are easy to apply and can be directed to specific lesions. They have a high concentration of fluoride so should be used sparingly. Varnishes need to be part of an ongoing programme and should be placed at least three times a year. Once the caries is under control the varnish applications will no longer be required.
- **Fluoride gels/solutions** – these can be used in a similar way to varnishes. They are not straightforward to apply and varnish has superseded their general use.
- **Fluoride mouthrinses** – these offer a proven benefit, but rinsing should not take place at the same time as brushing. Arriving home from school is a good time for rinsing. Not suitable for children under 6 or those individuals who cannot spit out the rinse.
- **Fissure sealants** – these are of great value especially on the vulnerable surfaces of first and second adult molars. The enamel structure of primary molars may compromise sealant adhesion as etching is not as effective. Resin based sealants are very technique sensitive so are of little value if moisture control is difficult to achieve.
- **Tooth Mousse®** – this product may have preventive benefits with ongoing use in controlling demineralization lesions, such as around orthodontic appliances.

Behaviour change

Whilst fluoride products have revolutionized the success of caries preventive programmes it is still important to explain to parents/carers the multifactorial nature of the caries process. Our focus should be on diet and toothbrushing.

- **Diet** – reducing the frequency of consumption of sugary foods and drinks is the dietary goal. How this is achieved is dependent on the age of the child and the cooperation of the parents. This chapter is essentially focusing on fluoride products, but dietary analysis is a key skill for the dental team, hence it being highlighted here.
- **Toothbrushing** – this is a behaviour which if embedded early in a child's life offers a proven benefit because it delivers fluoride toothpaste twice daily on a regular basis. In addition, as the child matures, removing plaque and controlling gingivitis becomes more important. For caries control however, the dental team needs to make sure a child brushes twice a day. There are different strategies for achieving this such as disclosing plaque, using toothbrushing charts and suggesting electric toothbrushes. The essential point is to make sure parents and carers monitor the brushing.

However, dental caries for many of our child patients will not be a major problem and our strategy should be to encourage healthy behaviour and highlight possible risk factors to parents and carers. These families can be classified as low risk, but must be given advice to maintain a healthy lifestyle.

The helpful pointers to low risk are:

- No new carious lesions within last 12 months.
- Little plaque noted on the cervical regions of the teeth.
- Parents report that child brushes twice a day with a fluoride toothpaste.
- Visits your practice once per year.
- Frequency of consumption of sugary drinks and foods is controlled.

Although a child may be at 'low risk' to caries, there are some standard actions which will be helpful in maintaining the status quo:

- Reinforce diet advice concentrating on limiting sugary snacks and drinks between meals.
- Appropriate fissure sealants on newly erupted first and second permanent molars are helpful.
- Children over 6 years of age should be advised to use a family strength fluoride toothpaste.

These simple behaviours will ensure your patients remain at very low risk of developing dental caries. Research suggests that a child with one carious lesion is five times more likely to develop further disease when compared with a child who is caries-free and as such, timely advice to parents is extremely important. This huge difference in risk highlights the importance of primary prevention and should be an essential part of the practice policy.

Techniques for toothbrushing

There are many ways advocated to clean teeth and remove plaque. In reality, the horizontal 'scrub' technique is the one usually employed by most people. It should be simply put that the plaque should be removed from:

- The 'biting surfaces' (occlusal) – using a vigorous scrubbing technique.
- All the gum margins (gingival margin) using a circular motion.
- Both sides of the teeth (labial/buccal and lingual/palatal) using a circular motion similar to above.

The one spot most poorly cleaned are the labial gingival margins of the lower anterior teeth. This is a naturally sensitive and difficult area to access, which children and parents need to be taught how to clean. It should be stressed that gingival tissues should not bleed following brushing at any age and this sign highlights the need to clean these areas more effectively. Furthermore, the current recommendation should be that children do NOT rinse following brushing. They may spit out at the end of brushing but be encouraged to swallow the residue (Figure 5.2). This is based on the qualification that the appropriate amount of fluoride toothpaste has been placed on the brush.

Dental fluorosis

Dental fluorosis is a qualitative defect of enamel resulting from an increase in fluoride concentration within the micro-environment of the ameloblasts during enamel formation (Aoba & Fejerskov, 2002). Mild fluorosis is characterized by opaque lines following the perikymata. With increasing severity, the opaque lines merge and more irregular cloudy areas become visible. More severe cases will have a totally opaque chalky appearance. In a small number of cases, there will be punched out pits and the outermost enamel will be gradually lost (Figure 5.1).

The dental profession must consider very carefully their fluoride regimes for children aged 1–6 years, as this is when the enamel of the upper anteriors is forming and the appearance can be compromised. It is interesting to note that most studies show people do not notice mild fluorosis.

Parents of children under 6 years of age should:

- Supervise brushing to check on fluoride toothpaste usage.
- Place a smear of paste on the brush.
- Store the toothpaste out of reach, so that a child will be unable to eat or suck the toothpaste.
- Avoid giving children 1 mg fluoride supplements.
- Not give baby vitamin drops containing a fluoride supplement.
- Carefully check with a member of the dental team why a high fluoride treatment is being suggested in their child's care plan.

A number of indices have been developed to record dental fluorosis. The earliest widely accepted index was developed by Trendley Dean and published in 1942 (Dean, 1942). His index of Dental Fluorosis recorded the appearance of the teeth 'wet'. The dentition was not air dried prior to assessment. This is extremely important, as it records the appearance of the teeth in their natural state. Subsequent indices dry the teeth and fluorosis will become more apparent as the enamel becomes desiccated.

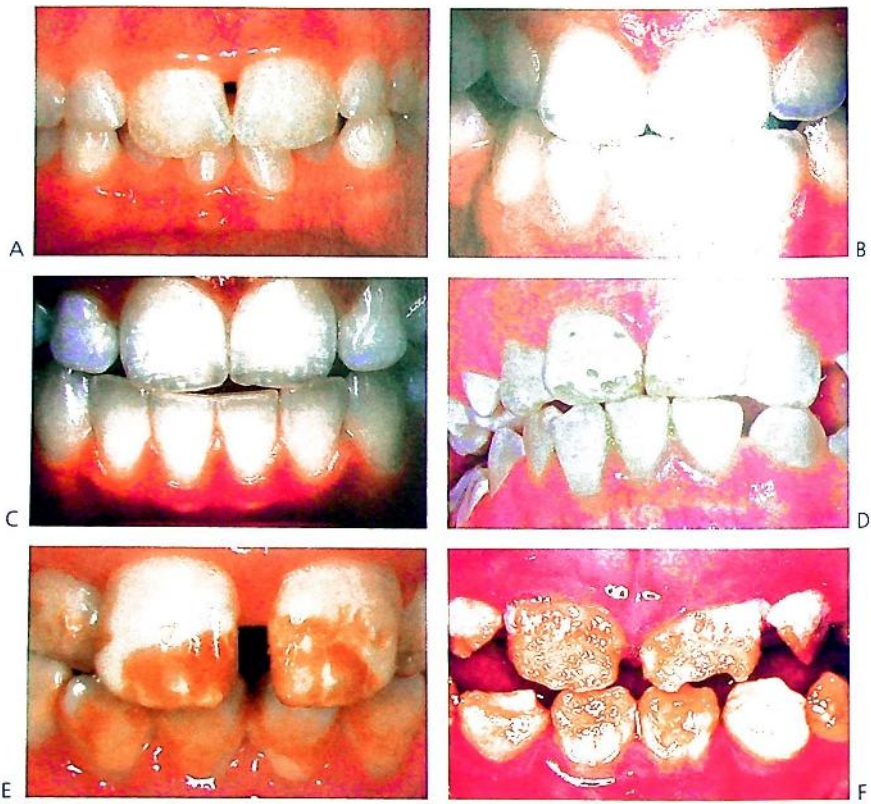

Figure 5.1 Different severities of fluorosis. (A) Very mild fluorosis with opacities following the outline of the perikymata. The primary teeth are unaffected. In younger children, the enamel is generally more opaque. (B) In an older child, the translucency of the incisal edge is evident but the opacities follow the same horizontal lines of the perikymata. (C) Mild fluorosis with white flecking through the crowns of the incisors. Note that the lower incisors are only minimally affected. This is still a surface opacity that will improve over time or may be conducive to treatment with microabrasion. (D) Moderate opacity affecting the whole crown. Note that the pits and brown mottling is secondary to tooth-surface wear and the acquisition of stains. (E) Score 6; Staining of intact enamel and discrete pitting. (F) Score 6; A more severe appearance with significant loss of enamel.

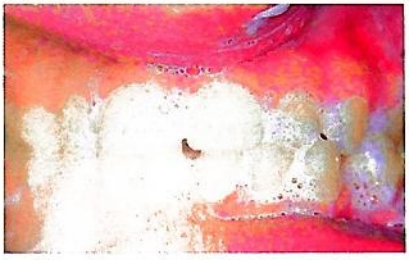

Figure 5.2 Allow children to spit out the toothpaste following brushing but try to avoid rinsing. The small amount of residue may be swallowed.

Table 5.1 Tooth Surface Index of Fluorosis (TSIF)

Score	Description (teeth not dried)
Score 0	No evidence of fluorosis
Score 1	Less than a ⅓ of visible enamel as areas showing a parchment white colour. Tips of incisors and posterior teeth showing snowcapping
Score 2	Parchment white fluorosis totals at least ⅓ but less than ⅔ of visible enamel surface
Score 3	Parchment white fluorosis totals at least ⅔ of visible surface
Score 4	Any of preceding levels of fluorosis plus staining from light to brown
Score 5	Discrete pitting which is usually discoloured when compared with surrounding enamel
Score 6	Discrete pitting, plus staining of intact enamel
Score 7	Confluent pitting, plus large areas of enamel may be missing. Dark brown stain is usually present

Dean's Index modified by Horowitz et al. 1984.

Dean's index has been modified by a number of researchers over the years, to give greater sensitivity; in particular Horowitz and others (1984) improved the index (see Table 5.1).

The other index that has found favour was developed by Thylstrup and Fejerskov in 1978. However, it is assessed when the teeth are dry and this may exaggerate the appearance of fluorosis. Nevertheless, as a research tool investigating the impact of fluoride on enamel development, it has been very useful.

Dean used his index to suggest that the 1 ppm level of fluoride in reticulated public water supplies would give the best reductions in dental caries with the least fluorosis. The appearance of fluorotic enamel changes over time with natural abrasion of the outer surface of the teeth, especially in cases of mild fluorosis.

Dental fluorosis primarily affects permanent teeth and is a dose-related condition. A diagnosis of dental fluorosis requires a detailed history of fluoride exposure. Excessive fluoride may have several detrimental actions on enamel formation including:

- Alteration of the production or composition of enamel matrix during ameloblastic secretory phase.
- Interference in the initial mineralization process caused by changes in ion-transport mechanisms.
- Disruption of ameloblast function affecting the withdrawal of protein and water from initial mineralization of enamel during the maturation phase.
- Disruption of nucleation and crystal growth in all stages of enamel formation, resulting in various degrees of enamel porosity (hypomineralization).
- Enamel mineralization appears uniquely sensitive to fluoride, and high doses of fluoride can affect breakdown and withdrawal of enamel matrix proteins (e.g. enamelins, amelogenins), resulting in permanent hypomineralization of enamel (subsurface and surface porosity). High doses of fluoride also seem to affect the activity of the ameloblasts.
- Excessive fluoride intake is of particular concern, especially during the first 36 months of life when crowns of the maxillary permanent incisors are undergoing mineralization or enamel maturation.

Clinically, dental fluorosis can be managed by remineralization, microabrasion or restorative replacement of the affected discoloured enamel and is discussed in more detail in Chapter 11.

Fluoride toxicity

Overwhelming evidence exists for the safety of fluorides at low concentration but high concentrations increase the possibility of toxic overdose. It is important to use high strength fluoride products with great care, especially in children under 4 years of age, but it is also prudent to advise parents about the safe storage of fluoride toothpastes.

Estimated probable toxic dose

- 5 mg F^-/kg of body weight.
- Gastrointestinal symptoms have been noted following ingestion of 3–5 mg F^-/kg by young children and very frail adults.
- For a 10 kg child, this corresponds to all the contents of a 45 g tube of toothpaste. Therefore, young children should not be allowed unsupervised access to fluoride toothpastes or fluoride supplements. Table 5.2 gives information on the amount of toothpaste which will cause a probable toxic fluoride dose.

Probable toxic dose (Table 5.2)

- 32–60 mg F^-/kg of body weight.
- Fatalities in children have been reported at doses of 16 mg F^-/kg of body weight. A number of concentrated topical preparations could provide such levels for young children if used in a single dose.

The inappropriate prescription of home-fluoride treatments with high concentration fluoridated gels for very young children (e.g. in the management of early childhood

Table 5.2 Amount of toothpaste ingested to receive a probable toxic fluoride dose[a]

Age of child	Average weight	Probable toxic dose F^-	Amount of 1000 ppm toothpaste (90 g tube = 90 mg F^-)		Amount of 400 ppm toothpaste (45 g tube = 18 mg F^-)	
			Weight	Tube/s	Weight	Tubes
2 years	12 kg	60 mg	60 g	66%	150 g	3
4 years	15 kg	75 mg	75 g	85%	188 g	4
6 years	20 kg	100 mg	100 g	>1 tube	250 g	5½

[a]Probable toxic dose: 5 mg F^-/kg.

caries) and inappropriate use of high fluoride products in the dental office, are of concern (Evans & Stamm, 1991). It must be emphasized that fluoride cannot control ECC in very young children without a change in diet, especially modification of the use of a night-time bottle or the use of bottle during the day as a comforter.

The management of acute fluoride toxicity consists of:

- Estimating the amount of fluoride ingested.
- Minimizing further absorption.
- Removing fluoride from the body fluids.
- Supporting the vital signs.

If vomiting has not occurred spontaneously:

- Give as much milk as can be ingested *or*
- Administer orally 5% calcium gluconate or calcium lactate or milk of magnesia.

While this immediate action is being taken, the hospital should be advised that a case of acute fluoride poisoning is in progress so that preparation for the appropriate therapeutic intervention can be made.

Note that while previous protocols advocated the use of an emetic, there has been a move away from encouraging vomiting because of the risk of aspiration of vomitus and burning the oesophagus by the hydrofluoric acid formed in the stomach, by the interaction of fluoride with hydrochloric acid. Modern emergency department protocols advocate the use of activated charcoal or gastric lavage in most poisonings.

Clinical implications

The practice of paediatric dentistry requires the dental team to be reflective and spend time carefully evaluating clinical problems prior to action. Once a diagnosis has been made, there are two key themes which must be part of practical care plans.

- The first is to consider the aetiology of oral health problems and what advice can be given to prevent or control them.
- The second is to implement appropriate clinical care.

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6 Restorative paediatric dentistry

Erin Mahoney, Nicky Kilpatrick, Sally Hibbert, Timothy Johnston

Primary teeth

Why restore primary teeth?

Our child patients deserve the best dental treatment that clinicians can provide as any treatment, preventive or restorative, will shape their dental future. Doubts have been raised about the rationale behind restoring primary teeth, amid claims that the majority of carious primary teeth exfoliate without causing symptoms. While there is ample strong evidence to contradict this view, the quality of the restorative treatment performed remains paramount. However, it is essential that there is an understanding of the caries risk of the individual patient, the developmental stage of the dentition and the status of the dental pulp prior to determining the choice of restorative material and technique. The objective of any restorative technique is to:

- Restore the damage caused by dental caries.
- Protect and preserve the remaining pulp and tooth structure; thereby managing and preventing symptoms and pain.
- Retain adequate function.
- Restore aesthetics (where applicable).
- Facilitate easy maintenance of good oral hygiene.
- Maintain arch length and space for the developing permanent dentition.

Primary versus permanent teeth

There are significant differences in the anatomy of the primary dentition in comparison with the permanent dentition that create some challenges when it comes to restoration of carious lesions (Table 6.1).

Restorative materials

There are a variety of restorative materials available to restore carious lesions in the primary dentition. Given the large number of techniques and products available on the market it is important for clinicians to understand the procedure they are using and to be aware that all approaches are operator and technique sensitive.

Table 6.2 summarizes the main advantages and disadvantages of the various dental restorative materials.

Amalgam

Historically, due to its simplicity, dental amalgam was the most popular restorative material. However today, as a result of concerns surrounding its potential toxicity and

Table 6.1 Differences in anatomy of primary teeth, compared with permanent teeth

	Anatomical features of primary teeth	Clinical significance
Crown	• Shorter • Narrower occlusal tables • Thinner enamel/dentine	• Limited room for cavity preparation – *linings are not usually required*
	• Molars have broad contact areas	• Clinical caries only detected if large – *bitewing radiographs essential for diagnosis*
	• Enamel rods in gingival 1/3 extend occlusally	• Enamel at floor of box is not undermined
	• Marked cervical constrictions	• Can be used to retain a stainless steel crown
	• Generally whiter in shade	• More opaque and whiter shades of composite resin required
Pulp	• Relatively large relative to the crown	• Limited room for cavity preparation
	• Pulp horns are closer to the surface	• Pulp exposure if clinical technique inprecise or inadequate
Roots	• Longer and more slender • More flared • Associated with lots of accessory canals	• Pulpectomy is difficult – *alternative techniques are often used*

unfavourable aesthetics, amalgam is rarely used in the primary dentition. Indeed, in some parts of the world, it has been banned in children altogether. Today, dental amalgam has been largely superseded by alternative materials and techniques in the restoration of the primary dentition.

Glass ionomer cements (GICs)

A glass ionomer consists of a basic glass and an acidic water-soluble powder that sets by an acid–base reaction between the two components. A principal benefit of GIC is that it will adhere chemically to dental hard tissues. A number of GICs are available on the market today, each having its advantages and disadvantages, however indications for the use of GICs are limited and inappropriate use is likely to lead to failure.

Conventional GICs

Conventional GICs are chemical-set glass ionomers with the weakest mechanical properties. The initial setting reaction is complete within minutes but the material continues to 'mature' over the following months. It is important to protect these materials from salivary contamination in the hours following placement or the material may shrink, crack and even debond. Adhesion of all GICs may be enhanced by the use of a dentine conditioning agent before placement. Today, chemically curing GICs are available as both restorative and protective sealant types of materials with high fluoride releasing properties.

High-viscosity GICs

High-viscosity GICs were developed for the atraumatic restorative technique (ART). These chemically cured materials have significantly better mechanical properties than

Table 6.2 Advantages and disadvantages of restorative materials used in paediatric dentistry

	Advantages	Disadvantages
Amalgam	Simple Quick Cheap Technique insensitive Durable	Not adhesive Requires mechanical retention in cavity Environmental and occupational hazards Public concerns Unaesthetic
Composite	Adhesive Aesthetic Reasonable wear properties Command set	Technique sensitive Rubber dam required Expensive
Glass ionomer cement	Adhesive Aesthetic Fluoride leaching	Brittle Susceptible to erosion and wear Technique sensitive
Resin-modified glass ionomer	Adhesive Aesthetic – better translucency than conventional GICs Command set Simple to handle Fluoride release	Water absorption Significant wear Technique sensitive
High-viscosity glass ionomer	Adhesive Aesthetic Simple to handle Fluoride release Higher compressive strength and wear resistance than conventional GICs	Water absorption Colour not as good a match as composite resins, compomers and other GICs Poorer mechanical properties than compomer and composites
Polyacid-modified composite resin (compomer)	Adhesive Aesthetic Command set Simple to handle Radiopaque	Technique sensitive Less fluoride release than GICs
Stainless steel crowns	Durable Protect and support remaining tooth structure	Extensive tooth preparation Patient cooperation required Unaesthetic

the conventional GICs and are fast setting. Research suggests that these materials have a durability comparable with amalgam, when used in occlusal (Class I) restorations in primary teeth, although the success rate is lower in inter-proximal (Class II) restorations, when other materials should be considered.

Resin-modified glass ionomer cements

Resin-modified glass ionomer cements were developed to overcome the problems of moisture sensitivity and low initial mechanical strength. They consist of a GIC along with a water-based resin system which allows photopolymerization to occur before the acid–base reaction of the glass ionomer is complete. This reaction then occurs within the light polymerized resin framework. The resin increases the fracture strength

and wear resistance of the GIC. Resin modified GICs are manufactured as restorative and lining materials for use in both primary and permanent teeth.

Composite resins

Resin-based composites (along with photopolymerization) have revolutionized clinical dentistry. In the primary dentition, composite resins are being increasingly used in combination with GICs in a 'sandwich'-style aesthetic restoration. Placement of these materials is highly technique-sensitive, as there is no doubt that patient compliance and adequate moisture isolation can prove difficult in the younger, more challenging child. There is little evidence to support this approach and yet the demand for aesthetic restorations makes this an attractive option.

Compomers (polyacid-modified composite resins)

Polyacid-modified resin composite resins or 'compomers' are materials that contain a calcium aluminium fluorosilicate glass filler and polyacid components. They contain either or both essential components of a GIC. However, they are not water-based and therefore no acid–base reaction can occur. As such, they cannot strictly be described as a glass ionomer. They set by resin photopolymerization. The acid–base reaction does occur in the moist intra-oral environment and allows fluoride release from the material. Successful adhesion requires the use of dentine-bonding primers before placement.

Stainless steel crowns

Stainless steel crowns are preformed extra-coronal restorations that are particularly useful in the restoration of large multisurface cavities and grossly broken down teeth. They cover the entire clinical crown and therefore recurrent or further caries is very unlikely. Placement of traditional stainless steel crowns is associated with considerable tooth preparation that can be challenging for patient and clinician alike. However, the introduction of minimal intervention sealed restorations (known as the 'Hall crown technique') has made the use of these restorations more realistic. They are, without doubt, the most durable restoration in the primary dentition and should be the technique of choice in the high-caries mouth.

Choice of materials

The choice of material to use in a given situation is not always simple and should not be based merely on technical considerations. Factors other than durability may be equally important in the choice of material, particularly in children.

Age

The age of a child will influence their ability to cooperate with procedures such as rubber dam application and local anaesthesia. The age of the child will also dictate for how long a restoration is required to remain satisfactory. A restoration in a first primary molar in a 9-year-old child does not require the same durability as a restoration in a second primary molar in a 4-year-old child.

Caries risk

Restorations in a child considered to be at high risk of caries may need to fulfil different objectives from restorations in a low-risk child. Although the use of a

fluoride-releasing material has obvious preventive advantages, glass ionomer cements (GICs) may not be the most appropriate choice in a mouth that is at high risk of further acid attack. Stainless steel crowns may involve a significant amount of tooth destruction, but this will be appropriate if it eliminates the need to re-treat in the future. Alternatively, GICs have a useful role in initial caries control in cases of rampant caries.

Cooperation of the child

Many young children have behaviour that is not conducive to perfect, textbook, cavity preparation and restoration. In these cases, highly technique-sensitive procedures are inappropriate. A more forgiving restoration that can tolerate some moisture contamination, without detriment to its longevity, may be suitable. The use of GICs in the management of caries in anterior primary teeth may be an excellent method of slowing the carious process and temporarily restoring aesthetics in a 2-year-old child, without recourse to general anaesthesia. By the age of 3 or 4 years, the child may be able to cope with more definitive treatment with composite resin and strip crowns.

Restorative implications of behaviour management

Unfortunately, not all children are able to cooperate with dental treatment under local anaesthesia. This may be because of their age or due to physical or intellectual disabilities necessitating the completion of treatment under sedation or general anaesthesia. When treatment is provided this way, the highest standard of dentistry possible should be provided to reduce future dental treatment for these high-need children. Use of materials and techniques that are known to have longevity, such as stainless steel crowns, are mandatory.

Restoring the primary dentition (Table 6.3)

Posterior teeth

GICs, resin-modified GICs and compomers

These materials have an increasingly important role in the management of carious lesions in primary molars because of their adhesive and fluoride-leaching properties.

Indications

Small occlusal and interproximal cavities. Because of their lack of strength, GICs should not be used in large restorations, particularly in teeth that need to be retained for 3 years or more. The use of polyacid-modified composite resins/compomers show considerable potential, particularly in terms of handling characteristics and radio-opacity. However, they have limited fluoride-leaching ability.

Success

The median survival time for conventional GICs is around 33 months. The failure rate of GICs is 33% over 5 years. High viscosity GICs demonstrate greater durability.

- Light-cured GICs have been shown to be more durable than conventionally curing materials.
- The incidence of secondary caries is reduced around fluoride-releasing materials.
- 4-year results available now suggest that compomers are adequately durable for use in the primary dentition.

A

B

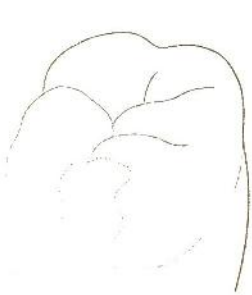
Figure 6.1 Two methods for using the rubber dam in children. (A) Traditional isolation of single teeth. (B) Split-dam technique, isolating the teeth from the canine to second primary molar with one large hole in the dam.

Table 6.3 Guide to the use of restorative materials in paediatric dentistry

Primary dentition	
Occlusal (Class I)	Glass ionomer cement (GIC) Composite resin Compomer
Proximal (Class II)	GIC Compomer Amalgam Composite resin/GIC sandwich Stainless steel crown
Gross carious breakdown or restoration after pulp therapy	Stainless steel crown
Permanent dentition	
Occlusal table	Fissure sealant
Occlusal enamel caries	Fissure sealant
Occlusal caries with minimal involvement of dentine	Preventive resin restoration
Occlusal caries with extension into dentine	Composite resin
Interproximal	Amalgam
Incisal edge	Composite resin
Cervical	GIC composite resin

Technique

1. Local anaesthesia and rubber-dam isolation should be used where needed (Figure 6.1).
2. The outline of the cavity should follow the extent of the carious lesion. There is no need for extension for prevention. A small occlusal dovetail is not usually necessary for interproximal restorations, however, additional retention form for minimal



A

B

Figure 6.2 (A,B) Class II cavity design for the placement of resin-modified glass ionomers.

proximal cavities can be achieved by placing grooves into the dentine using very small (size ½) round burs (Figure 6.2).

3. Remove all soft caries using a slow round bur or with hand instruments. Be aware of the large pulp chamber as it is easy to expose the pulp of a primary molar.
4. When placing GICs, precondition the dentine using 10% polyacrylic acid for 10 s, and wash and dry. When using a compomer, it is usually not necessary to condition the prepared tooth, however dentine-bonding primers should be used as per manufacturer's instructions.
5. When using encapsulated materials, ensure that the capsules are compressed for at least 3 s to facilitate adequate mixing of the powder and liquid components. After mixing for 10 s in the amalgamator, discard the first 3–4 mm of the mixed material, as this is often unsatisfactory. Place the remainder directly into the cavity.
6. Once the relatively thick material has been placed into the cavity, it is compressed with a ball burnisher – dipping the tip in a small amount of bonding agent or unfilled resin prevents the material sticking to the instrument.
7. The final restoration must be protected from moisture contamination. This is best achieved by the placement of a thin layer of unfilled resin over the surface and polymerizing for 20 s. In young children with behaviour management problems, Vaseline, rather than unfilled resin, may be appropriate.
8. The occlusion should be checked on removal of the rubber dam.

Composite resins

In primary molars, composite is a satisfactory restorative material provided that the child is cooperative and good moisture control is achievable.

Indications

Small to moderately sized occlusal and proximal cavities.

Success

Clinical studies suggest that Class II composite restorations in primary molars are only moderately durable, with one study reporting <40% success after 6 years. However, recent studies have shown greatly improved success rates with the newer resin-based composites.

Technique

For interproximal lesions, the cavity design needs to be modified slightly in that a bevel should be prepared around the occlusal margins for additional adhesion to enamel. The biggest problem encountered with composite restorations is the integrity of the bond at the depth of the proximal box. Placement of composite is technically difficult and highly sensitive to moisture contamination. Placement of a glass ionomer liner over the dentine not only ensures a good bond at the base of the cavity, reducing microleakage, but also provides fluoride release locally. The use of rubber dam and incremental placement of composite in the proximal box may reduce handling and polymerization contraction problems.

Increasingly, parents are requesting tooth-coloured restorations. It should be recognized, however, that use of these materials is associated with increased technical demands and expense.

Stainless steel crowns

Indications

Stainless steel crowns are preformed extra-coronal restorations that are particularly useful in the restoration of:

- Grossly broken down teeth.
- Primary molars that have undergone pulp therapy.
- Hypoplastic or hypomineralized primary or permanent teeth.
- Dentitions of children at high risk of caries, particularly children having treatment under general anaesthesia.

Success

Stainless steel crowns undoubtedly provide the most durable restoration for the primary dentition with survival times in excess of 40 months.

Relatively expensive in relation to both time and money in the short term. However, the rate of replacement of these restorations is low (3% compared with 15% for class II amalgam restorations). This makes them economically more attractive over the long term.

Can be considered unaesthetic and require a significant amount of tooth preparation, and invariably local anaesthesia – unless using the 'Hall crown technique'.

Method (Figure 6.3)

Irrespective of whether the tooth to be restored is vital or non-vital, local anaesthesia should be used when placing a stainless steel crown because of the soft-tissue manipulation. Rubber dam, although sometimes difficult to place in the broken down dentition, should be used where possible.

1. Restore the tooth using a GIC or compomer prior to preparation for the stainless steel crown.
2. Reduce the occlusal surface by about 1.5 mm using a flame-shaped or tapered diamond bur. Uniform occlusal reduction will facilitate placement of the crown without interfering with the occlusion.
3. Using a fine, long, tapered diamond bur, held slightly convergent to the long-axis of the tooth, cut interproximal slices mesially and distally. The reduction should allow a probe to be passed through the contact area (Figure 6.4).

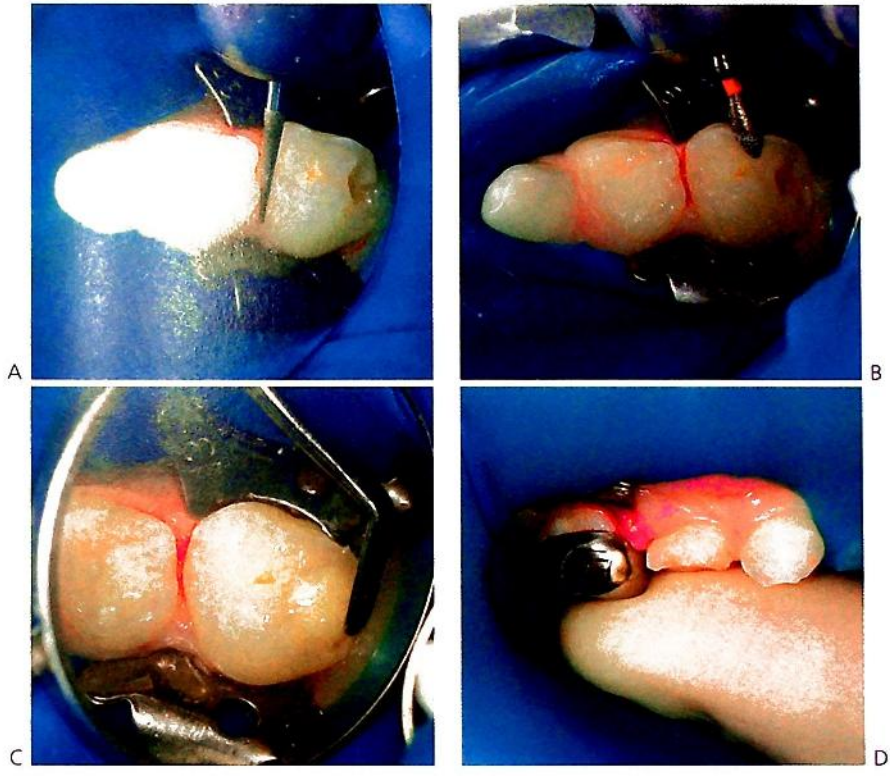


Figure 6.3 Placement of a stainless steel crown. This tooth had a large distal lesion with loss of the marginal ridge. (A) Interproximal reduction is completed with a fine tapering diamond bur taking care not to damage the adjacent tooth. (B) Occlusal reduction of up to 1.5 mm is performed with a large diamond flat fissure bur, a small wheel or in this case a flame diamond bur. (C) Glass ionomer cement is used to build up the carious distal aspect of the crown. (D) Trial fit of the crown, by seating from the lingual onto the buccal surface.

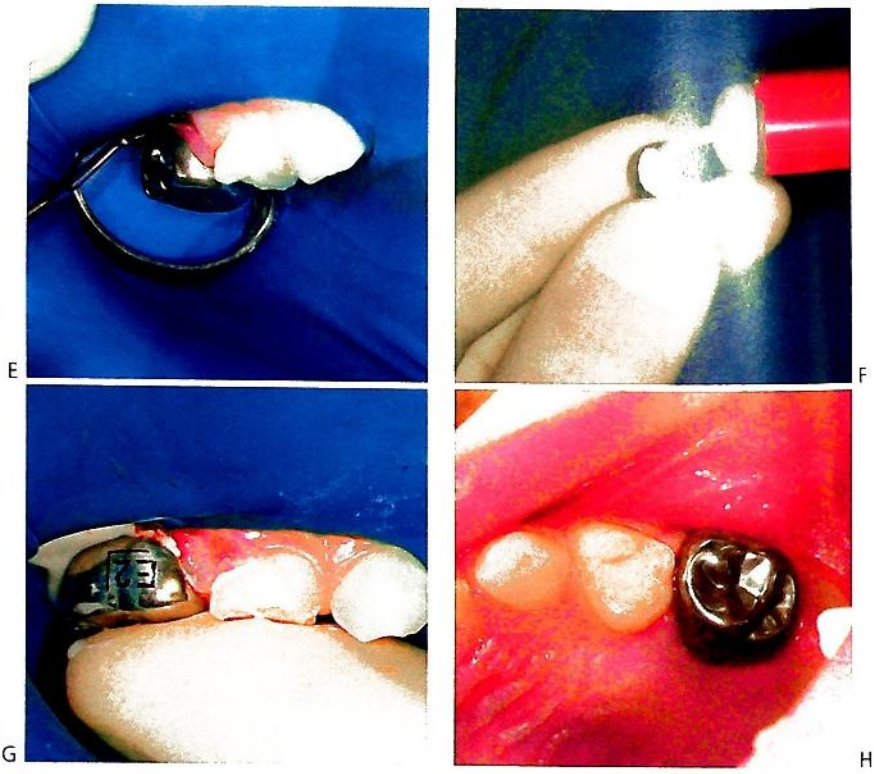


Figure 6.3 *Continued* (E) A large spoon excavator can be used to remove the crown. (F) The crown is filled with glass ionomer cement for luting and (G) the crown placed with finger pressure (H). The completed restoration should last the lifetime of the tooth.

4. Buccolingual reduction should be kept to a minimum, as these surfaces are important for retention. However, reduction may be needed when there has been significant proximal space loss or anatomical features, such as a prominent Carabelli's cusp.
5. An appropriate size of a precontoured crown is chosen by measuring the mesiodistal width.
6. A trial fit is carried out before cementation. It is important that the crown should sit no more than 1 mm subgingivally. If there is excessive blanching of the gingival tissues, the length of the crown should be reduced and the margins should be smoothed with a white stone.

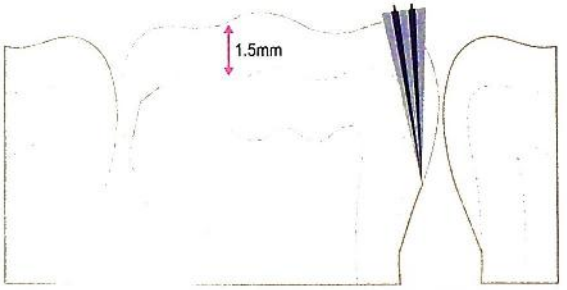


Figure 6.4 Coronal and proximal preparation required for the placement of a stainless steel crown. Note that in the proximal areas, there is a smooth contour without any ledge or step. Any such step will cause great difficulty in seating the crown.

7. Cement the crown with a GIC. If the crown has been built up before the placement of the crown, a glass ionomer luting cement may be used, otherwise a restorative GIC should be used. Care should be taken while holding the crown as it can be easily dropped during placement. Excess cement should be wiped away and a layer of Vaseline placed around the margins while the cement is setting.

Minimal intervention

The philosophy of minimal intervention

Minimal intervention is based on an increasing body of evidence that traditional approaches to cavity design involved excessive removal of tooth structure. It is undisputed that beneath the layer of *infected* dentine lies a layer of *affected* dentine. Like the infected layer, this layer is discoloured, however in contrast to the infected layer, it may have odontoblast processes present, have a relatively intact collagen network and is bacteria free. Traditionally, all discoloured dentine was removed, however unlike the 'infected zone', the 'affected' layer can be remineralized and therefore should be preserved during cavity preparation (Figure 6.5).

More recently, even this approach is being questioned as being too destructive, as researchers are beginning to query if any caries needs to be removed for a successful restoration. It has now been shown that dentists can provide long-lasting, sealed restorations, with minimal or no caries removal.

The sealed restoration

When placing any restoration, the importance of achieving a high quality coronal seal is now well recognized. Furthermore, there is growing evidence that providing a good seal is maintained, the complete removal of caries is no longer necessary. This enables us to minimize the amount of tooth tissue that is removed, which is of potential advantage in restoring primary molars with their thin enamel and dentine, and relatively large dental pulps.

When restoring occlusal lesions, adhesive materials such as GICs can usually provide an adequate seal. It can, however, be extremely difficult to achieve this seal using intracoronal techniques in primary molars with large proximal lesions. Stainless steel crowns provide an excellent coronal seal on primary molars and as such can prevent

Figure 6.5 Breakdown of GIC restorations from conservative (minimal intervention) dentistry. Note, however, that there has been a substantial slowing of the caries rate such that all the lesions are inactive and the teeth have been preserved in the mouth. While it is easy to criticize the quality of these restorations, these had been placed in a child whose behaviour was extremely difficult, without the access to general anaesthesia or other forms of sedation. Although some arch length has been lost, as the crowns have not been restored to their natural contour, the majority of the space occupied by the teeth has been preserved. This still permits the placement of stainless steel crowns in the future, when the child is better able to cope with more extensive treatment procedures in the event of an improvement in behaviour. The question should be asked whether these restorations have 'successfully' retained the teeth. Is this treatment better than having no treatment or having all these primary teeth extracted?

progression of caries. The placement of stainless steel crowns, with little or no caries removal, is known as the Hall crown technique.

The Hall crown technique

The Hall crown technique is essentially the ultimate minimally invasive restoration as it involves the placement of stainless steel crowns, directly over carious lesions, in primary molars, with little, or no, tooth preparation or caries removal. Initial trials in Scotland have shown that this simple technique can provide successful restorations in the medium term; indeed the technique not only outperformed conventional restorations but was preferred by the children and clinicians. This procedure can be a valid alternative, particularly when children are unable to accept conventional treatment with local anaesthetic. It is however, not appropriate in all cases and in particular should only be used for teeth that are symptom free and without signs or symptoms of pulpal pathology.

The question remains as to whether there is a place for this practice, when conventional techniques of proven efficiency are available. However, conventional restorations are very technique sensitive and commonly require the use of local anaesthesia and high speed handpieces, which many young children find difficult to accept. This

technique can be a useful alternative under such circumstances. It is already being extensively used in some countries, although the paucity of long-term clinical trials prevents it from being recommended generally. Further trials are currently underway in the UK and Australasia and the results of these studies may change recommendations for its use.

Indications

- Primary molar teeth with moderate decay, but no clinical signs or symptoms of pulpal pathology.
- Dentitions of children with limited cooperation, who are unable to accept conventional restorative treatment with local anaesthesia.
- Healthy children.

Success

- In a prospective, randomized control clinical trial, the Hall technique, statistically, significantly outperformed standard restorations at 5 years.
- Any disruption of the occlusion following crown placement, will usually self-correct within a few weeks.

Technique

This technique is used without local anaesthetic:

1. Pre-procedure radiograph and examination to exclude pulpal pathology.
2. Orthodontic separators may be placed at a prior appointment, to ease placement of the crown.
3. Child should be sat upright or semi-reclined, but not supine and gauze may be used to protect the airway.
4. The tooth can be cleaned with a toothbrush and if desired gross caries may be removed with a hand excavator.
5. A stainless steel crown is selected, which will fit over the tooth without any preparation.
6. A GIC cement is placed in the crown which is bitten into place by the child.
7. Excess cement may be washed or wiped away, before it has set.

Restoration of primary anterior teeth

GICs, resin-modified GICs, compomers

GICs (conventional and light-cured) and compomers all have a place as a one-surface restoration in primary anterior teeth. They provide aesthetically acceptable results and provide a degree of prevention as a result of fluoride release. However, it is important that the preparations must be caries-free for optimum results, as secondary caries is a reason for failure of all material types, even GICs. The durability of such restorations is, however, questionable and parents should be aware of this at the time of placement.

Composite resin strip crowns (Figure 6.6)

Composite is the material of choice for the restoration of primary anterior teeth. When used in conjunction with anterior strip crowns, composite resin provides an aesthetic and durable restoration.

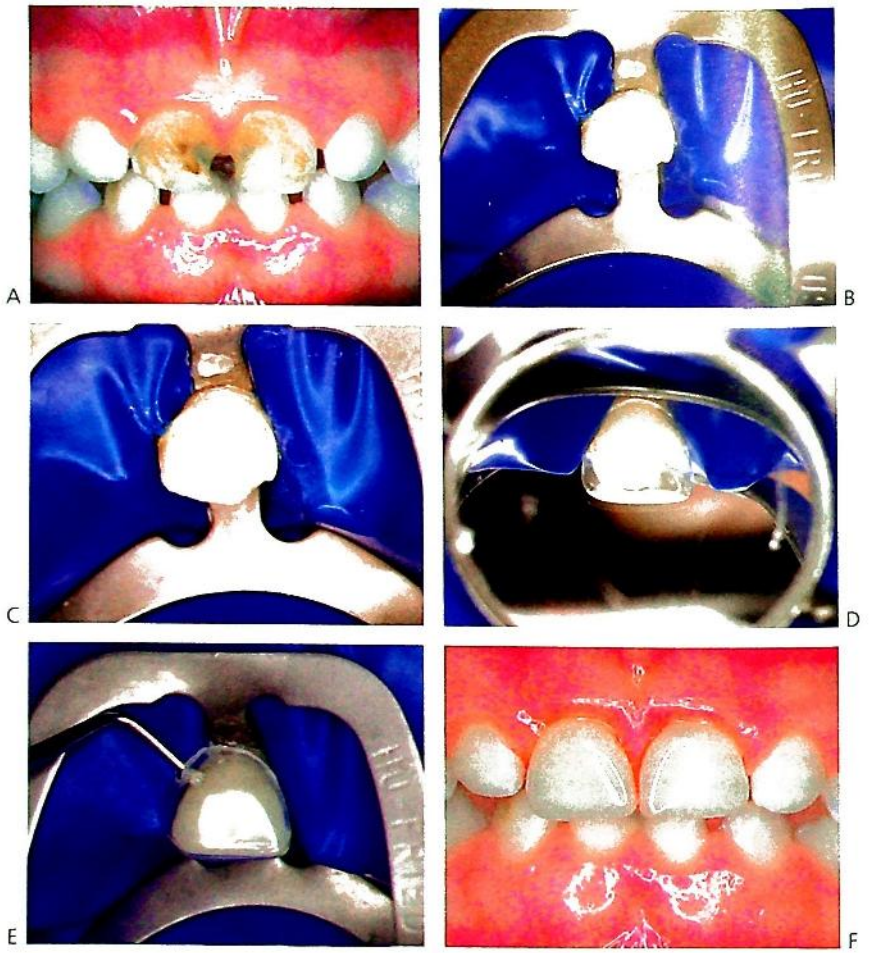


Figure 6.6 Placement of anterior strip crowns on the primary incisors. (A) Bottle caries affecting the upper anterior teeth. (B) Initial reduction of incisal edge and caries removal under the rubber dam (butterfly clamp). Proximal reduction is achieved using a high-speed tapering diamond bur. (C) Placement of a glass ionomer cement base over the dentine. (D) Trial fitting of the cellulose acetate strip crown, which is then filled with composite resin. (E) Removal of the strip crown with a small excavator. (F) Final restoration after polishing. (Courtesy of Dr E Alcaino.)

Method

1. Local anaesthesia and rubber-dam isolation should be used if possible. Alternatively, because of age and poor cooperation of younger children, the restorative work may be completed under general anaesthesia.
2. Select the correct celluloid crown form depending on the mesiodistal width of the tooth.
3. Remove the caries using a slow-speed round bur.
4. Using a high-speed tapered diamond or tungsten carbide bur, reduce the incisal height by around 2 mm, prepare interproximal slices and place a labial groove at the level of gingival and middle thirds of the crown.
5. Protect the exposed dentine with a glass ionomer lining cement.
6. Trim the crown form and make two holes in the incisal corners by piercing with a sharp explorer.
7. Etch the enamel for 20 s, wash and dry.
8. Apply a thin layer of bonding resin and cure for 20 s, ensuring all surfaces are covered equally.
9. Fill the crown form with the appropriate shade of composite and seat with gentle, even pressure, allowing the excess to exit freely. The use of small wedges may be helpful in avoiding interproximal excess.
10. Light cure each aspect (labially, incisally and palatally) equally.
11. Remove the celluloid crown gently, and adjust the form and finish with either composite finishing burs or abrasive discs.
12. Check the occlusion after removing the rubber dam.

Interproximal stripping

Stripping of interproximal enamel may be used occasionally for minimal caries in the anterior primary teeth. Opening of the contact points allows saliva and fluoride to arrest the carious process, even when the caries involves the dentine. Allowing open access for a toothbrush aids removal of cariogenic biofilm, which is difficult with a closed contact. This is often, however, an unaesthetic alternative. It goes without saying that the initiating cause, such as a nursing-bottle habit, must be eliminated.

Method

The contact points are removed with a long tapering diamond or tungsten carbide bur and a high percentage topical fluoride varnish (e.g. Duraphat® Varnish or Clinpro™ XT Varnish) is applied to the enamel and dentine. This should be repeated up to 4 times per year in high risk children.

Management of occlusal caries in permanent teeth

Dental amalgam is no longer considered the most appropriate technique for the restoration of caries lesions in the occlusal surfaces of permanent molars. The need to incorporate mechanical retention into the cavity design can lead to undermining of marginal ridges and weakens the cusps that will eventually fracture. Teeth restored in this manner often require further, even larger restorations with the risk of pulp disease, root canal treatment and finally full coverage restoration. The preventive resin

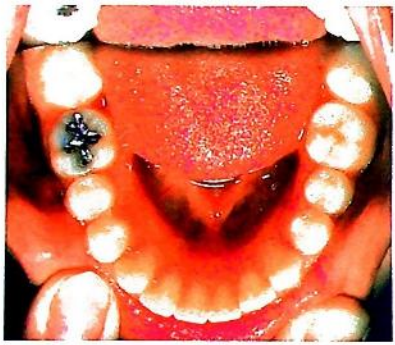


Figure 6.7 While this amalgam restoration has been well placed, it is an inappropriate restoration for a patient of 20 years, whose only caries is an incipient lesion on the occlusal surface. This amalgam will weaken the marginal ridges and supporting cusps and compromise the tooth in the long term. A preventive resin restoration would have been a much better alternative.

restoration is more appropriate, as minimal tooth structure is lost in cavity preparation and the occlusal table is protected by a fissure sealant (Figure 6.7).

Fissure sealants

In fluoridated communities throughout Australasia, where the average DMFT (decayed, missing and filled permanent teeth) is <1, the majority of caries occurs in the pits and fissures of the first permanent molar teeth. A simple and economical way of preventing pit and fissure caries is by the use of fissure sealants.

The indications for a fissure sealant are controversial. On a population basis, it has been suggested that only those children who are at moderate risk of caries should have sealants placed, but because nearly 90% of children up to 18 years have some caries (mainly in the first permanent molars) all children should be assessed for fissure sealants throughout the eruption of the permanent dentition. Treatment should be prescribed according to the individual patient's need (Figure 6.8).

All teeth being considered for a fissure sealant should be checked radiographically for the presence of occult caries. Other options to aid diagnostic accuracy before sealing of fissures include the use of miniature burs to investigate staining, laser fluorescence, electronic caries detectors and microabrasion. If caries is noted or suspected, a preventive resin restoration should be placed.

Indications

- All permanent molars in children at medium or high risk of caries. Premolars should be sealed in those children at high risk.
- In children at low risk, only the fissures that are deep and retentive need to be sealed.
- Primary posterior teeth in children at high risk of caries.

Risk assessment should continue throughout teenage years, even where caries risk was initially low. Risk status can change and fissure sealing continues to be protective into adulthood.

Sealant material

- Although some studies show differences, there seems to be no strong evidence to favour light-cured over chemically-cured sealants or either opaque, clear or coloured fissure sealants at this time.

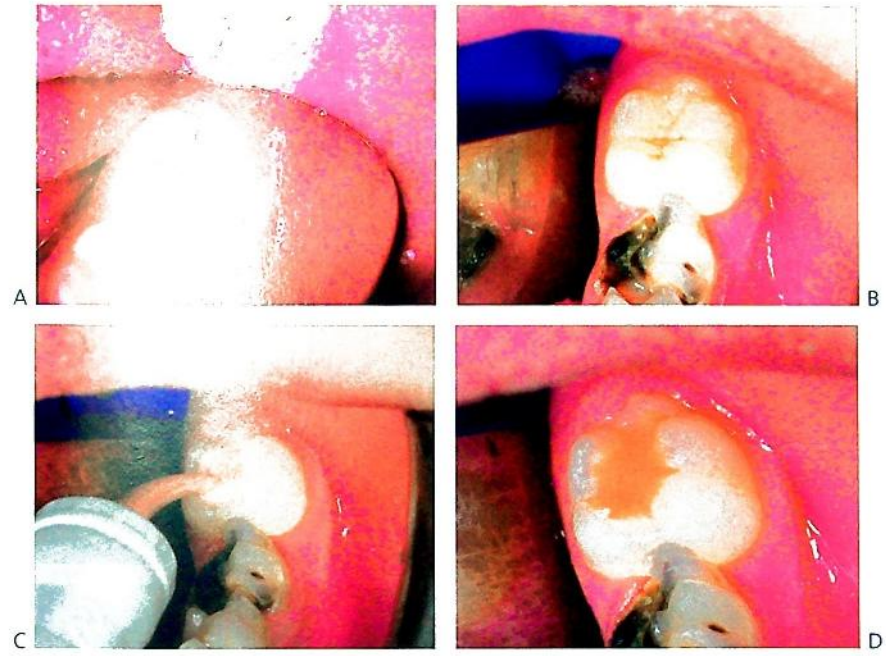


Figure 6.8 Fissure sealants. (A) An assessment must be made in the individual of caries risk. Not every tooth in every arch requires sealing, but it is important to remember that risk can change. These fissures are caries-susceptible fissures and sealing of the buccal pit in this child is essential. (B–D) Newly erupted first permanent molars can benefit from fissure protection and the placement of a high-fluoride releasing glass ionomer cement. Note how the material extends into the high-risk areas of the buccal and distal fissures.

- Sealants should be opaque so that they can be detected by other clinicians. Use of clear sealants shows stains in the fissures, which are most probably inactive caries. However, another clinician, on seeing these stains, may choose to cut a cavity into a sound tooth, defeating the whole purpose of the sealant.
- Taking into account individual caries risk, the use of resin-based sealants is appropriate for fully erupted molars or pre-molars.
- Glass ionomers are useful in high caries-active individuals, partially erupted and hypomineralized teeth that are difficult to isolate and as temporary sealants until the teeth have erupted sufficiently to allow conventional fissure sealing.

The main problem with the use of GICs as fissure sealants is the brittleness of the material when used in thin section over the occlusal surface. However, the incidence of fissure caries in these teeth is low and in the long term, similar to retained resin-based sealants. It has been suggested that either the GIC is retained in the depths of the fissures at a microscopic level or that fluoride, from the GIC, is taken up by the

surrounding enamel, so increasing the resistance of the fissure walls to demineralization.

Method (Figure 6.9)

1. Isolate the tooth with a rubber dam. If the tooth cannot be isolated, then a high-dose fluoride treatment such as a fluoride varnish or a GIC material should be applied. Review the eruption of the tooth in the following months and when the tooth has erupted sufficiently, place a fissure sealant.

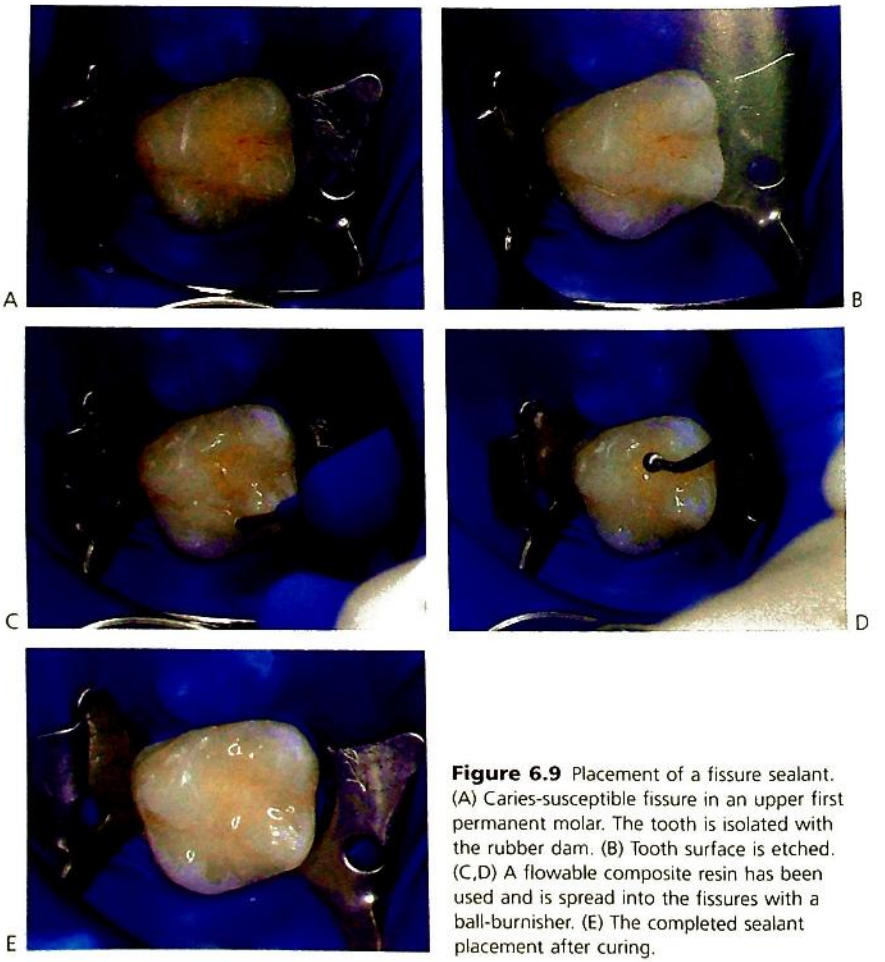


Figure 6.9 Placement of a fissure sealant. (A) Caries-susceptible fissure in an upper first permanent molar. The tooth is isolated with the rubber dam. (B) Tooth surface is etched. (C,D) A flowable composite resin has been used and is spread into the fissures with a ball-burnisher. (E) The completed sealant placement after curing.

2. Remove gross debris with a blunt probe and if necessary, clean the occlusal surface with oil-free pumice and water. In many instances, minimal widening of the occlusal fissure with a very thin, small, tapered diamond fissure bur will facilitate the penetration of sealant material into the depth of the fissure. It also removes the more acid-resistant surface layer of enamel lining the walls of the occlusal fissure. However, it is preferable to avoid any removal of tooth structure if possible.
3. Etch the tooth with a gel etchant for 20 s and wash with copious water and dry with air irrigation for 20 s.
4. If the tooth is contaminated it should be re-etched for 15 s.
5. Apply a thin coat of sealant to the pits and fissures, making sure to include the buccal extension on lower molars and the palatal groove in upper molar teeth. Apply the polymerization light for 20 s.
6. Remove the rubber dam and check the occlusion.

Preventive resin restoration (PRR)/occlusal restoration

Due to its superior wear resistance and superior mechanical properties, composite resin materials rather than glass ionomers are the material of choice for the treatment of early occlusal caries in permanent teeth. The development of preventive resin restorations has changed the management of occlusal caries dramatically in young patients. The advantage of the PRR is the use of an unfilled resin base to seal over not just the underlying restoration itself but all the residual non-carious fissures, thus acting as a preventive restoration.

Indications

- Enamel-only lesions.
- Incipient occlusal lesions just into dentine.
- Small class I lesions.

Success

The durability of preventive resin restoration has been proved to be as good as occlusal amalgam restorations and can be achieved with significantly less removal of sound tooth tissue. The proviso is that a sound hermetic seal is achieved such that there is no marginal leakage. Good technique is therefore essential.

Method for preventive resin restoration

1. Use local anaesthesia and rubber-dam isolation if caries extends into dentine.
2. With a small high-speed diamond bur obtain access into the questionable fissure.
3. Remove the carious dentine. Although it is important not to remove more enamel than necessary it is essential to have adequate access to the underlying dentine to be certain of complete caries removal. Unsupported enamel need not be removed if access and vision are clear. The cross-section most closely resembles a tear drop shape (Figures 6.10, 6.11).
4. Deeper dentinal caries should be removed using a slow-speed round bur.
5. Place a glass ionomer liner over the dentine extending it up to the amelodentinal junction and light cure for 40 s.

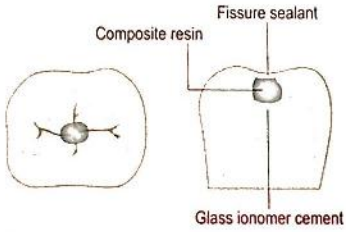


Figure 6.10 Preventive resin restoration.

6. Gel etchant is placed for 20 s on the enamel margins and occlusal surface, and washed and dried. It is not necessary to etch the liner; sufficient roughening of the surface of the GIC will result from the washing process.
7. Place a thin layer of bonding resin into the cavity and cure for 20 s. An excess of resin will produce pooling and reduce the integrity of the bond.
8. Incrementally fill and polymerize the cavity with hybrid composite resin until it is level with the occlusal surface.
9. Flow opaque unfilled fissure sealant over the restoration and the entire occlusal fissure pattern and cure for 20 s. There is no need to re-etch the occlusal surface prior to placing the fissure sealant.
10. Remove the rubber dam and check the occlusion.

New techniques for tooth preparation

From the discussion above, it is clear that paediatric dentistry relies heavily on the use of standard high-speed and low-speed handpieces. Standard handpieces allow clinicians to remove carious dentine and shape a cavity. However, in recent years, several hard-tissue removal techniques have been developed that also have a place in modern paediatric dentistry.

Air abrasion

Air abrasion is a technique that uses kinetic energy to remove carious tooth structure. When the aluminium oxide particles hit the tooth surface, without heat or noise of vibration, they remove tooth tissue. This technique requires additional equipment in the dental office for safe particle extraction and requires the use of a rubber dam, but has been shown to be useful in some child patients who may be nervous of the noise or the feeling of conventional handpieces. Care should be taken due to the possibility of particle inhalation when using this method in children with severe dust allergy, open wounds and lung diseases such as asthma.

Laser-assisted dentistry

Laser is an acronym for *l*ight *a*mplification by *s*timulated *e*mission *r*adiation. Dental lasers are devices that use the energy generated by atomic electron shifts producing coherent monochromatic electromagnetic radiation between the ultraviolet and the

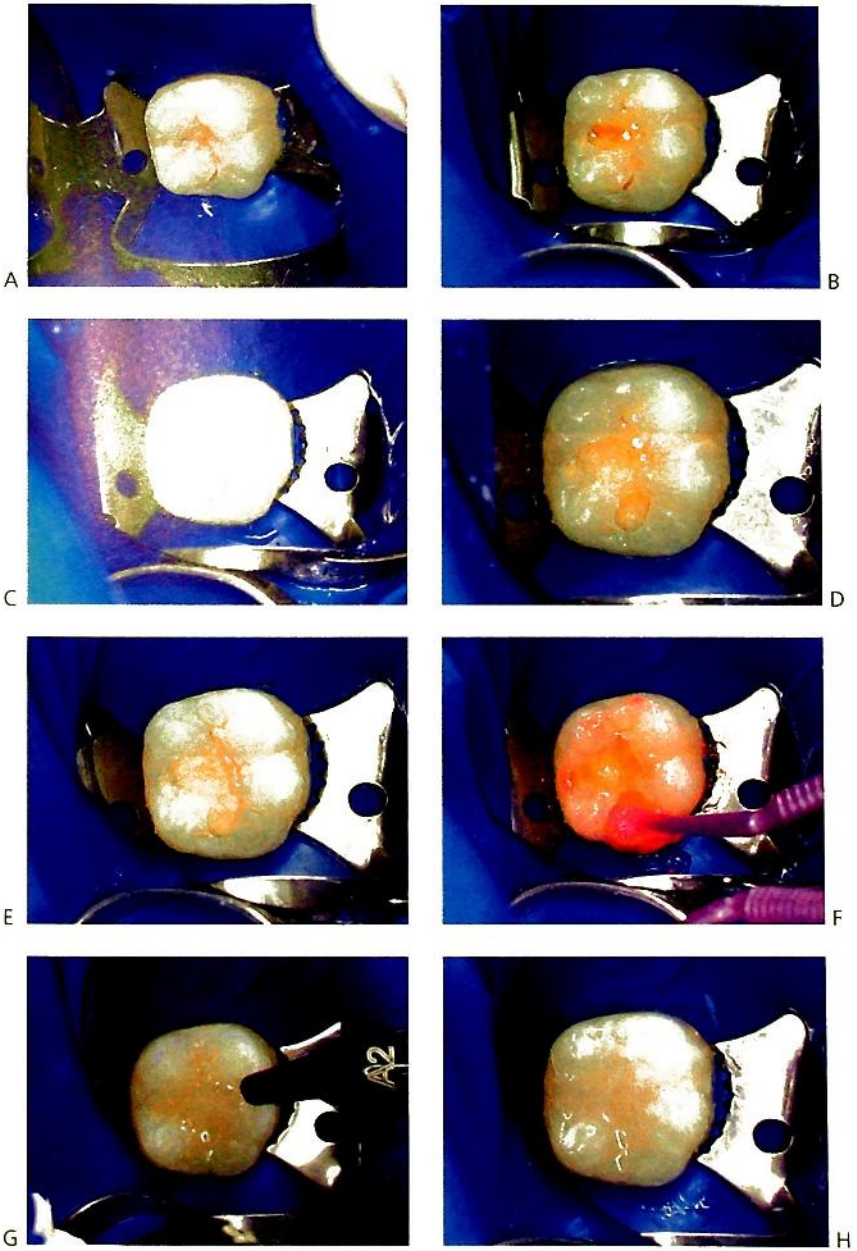


Figure 6.11 Technique for Class I composite restoration in a permanent molar. (A) The true extent of the caries may not be visible from the occlusal surface. (B–D) Progressive investigation of the fissures reveals further extension of dentinal caries. (E) Placement of a glass ionomer base. (F) Etching. (G) Incremental placement with nano-filled composite resin. (H) Final restoration with sealant placed over surface.

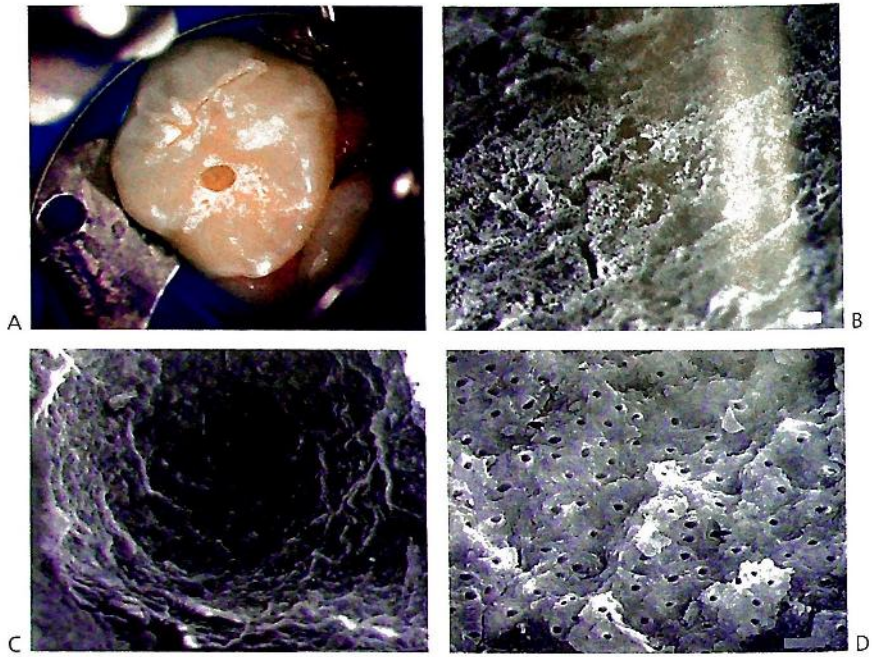


Figure 6.12 Effects of new techniques for tooth preparation on enamel and dentine. (A) Use of an Erbium YAG laser to prepare the enamel prior to placement of a preventive resin restoration and occlusal fissure sealant. (B) Scanning electron micrograph (SEM) enamel surface prepared with air abrasion (×4300 magnification). This may assist in the retention of sealants but it is not superior to normal enamel etching. (C) SEM of dentinal carious lesion prepared with an Erbium YAG hard tissue laser (×180) and at higher power (×700) (D) with complete absence of the smear layer and open dentinal tubules.

far infrared section of the electromagnetic spectrum. The photobiological effects of the lasers most commonly used in dentistry are:

- Laser-induced fluorescence (caries/calculus detection).
- Photoacoustics causing disruption and ablation (soft-and hard-tissue treatments).
- Photothermal effect inducing coagulation and vaporization (soft-tissue treatments).

Bio-stimulation and photochemical effects induced by short-wavelength lasers for treatments including wound healing, analgesia and tissue growth will become more commonplace in time. Laser-assisted fluoride and bleaching treatments also show promising application.

Erbium lasers display bio-resonant properties on neural tissue causing Na^+/K^+ pump blockade and polarization of the A delta fibres and possibly C fibres. For many

applications, local anaesthesia can be reduced and occasionally eliminated due to the analgesic properties of the lasers themselves.

Hard-tissue application

The two lasers most commonly used for dental hard-tissue treatments are in the 2790 nm (ErCr: YSGG (erbium-chromium: yttrium-scandium-gallium-garnet) and 2940 nm (Er: YAG (erbium-doped yttrium aluminium garnet) wavelengths. The tissue is removed by a non-contact beam that ablates based on the photoacoustic affect on water molecules. The water content of the treated tissue and the power density of the laser beam affect the cutting efficiency. Hard-tissue applications include cavity preparation, caries and calculus removal, endodontic treatments, desensitization and bone surgery. The advantages of lasers include:

- Ability to selectively remove only carious dental tissue.
- Limited noise.
- No vibration.
- Ability to cut dental tissue without the need for local anaesthesia (in some cases).

Therefore, lasers can be extremely useful for nervous patients; however, they are expensive and care must be taken during use to ensure that excess heat is not generated, which may be detrimental to the pulp tissue.

It should be noted that these newer modalities result in significant changes to the smear layer compared with those of traditional techniques and clinicians should be aware that these may alter the choice of materials used and their bonding characteristics to both enamel and dentine (Figure 6.12).

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Restoration of primary teeth

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7 Pulp therapy for primary and immature permanent teeth

John Winters, Angus C Cameron, Richard P Widmer

Introduction

Dental caries, trauma and the iatrogenic effects of conservative dental treatment, all provoke a biological response in the pulpo-dentinal complex. This chapter is concerned with the cascade of therapeutic interventions used to promote an adaptive biological response in the pulpo-dentinal complex of the treated tooth, and optimize subsequent growth and development. Therapeutic efforts are directed towards the retention of carious or traumatized teeth, maintaining normal function, with the resolution of, or freedom from, clinical symptoms.

Role of primary teeth

As mentioned in the last chapter, primary teeth play an integral role in the development of the occlusion. Premature loss of a primary tooth through trauma or infection has the potential to destabilize the developing occlusion with space loss, arch collapse and premature, delayed or ectopic eruption of the permanent successor teeth. In general, the effects of early extraction of primary teeth are more profound in the buccal segments than in the anterior dentition.

Effective pulpal therapy in the primary dentition must not only stabilize the affected primary tooth, but also create a favourable environment for normal exfoliation of the primary tooth, without harm to the developing enamel or interference with the normal eruption of its permanent successor. Where these outcomes cannot reasonably be achieved over the clinical life of the primary tooth, it is appropriate to extract the affected tooth and consider alternative strategies for occlusal guidance and maintenance of arch integrity (see Chapter 14).

Immature permanent teeth

All teeth are immature when they erupt. In addition to the important phase of post-eruptive enamel maturation, the roots of newly erupted permanent teeth will take up to 3 years before their growth is completed. During this period, the roots are short, the root apices are wide open, the dentine is relatively thin and the dentine tubules are relatively wide, increasing the permeability of dentine to bacteria. The open apex is associated with excellent pulpal vascularity and the potential for a favourable healing response.

Therapeutic efforts are directed towards preserving the vitality of the pulpo-dentinal complex to facilitate normal root development and maturation (Figure 7.1). If pulp necrosis occurs prior to root maturation, while the affected tooth can still be preserved using non-vital endodontic strategies, it will be compromised with regard to strength,

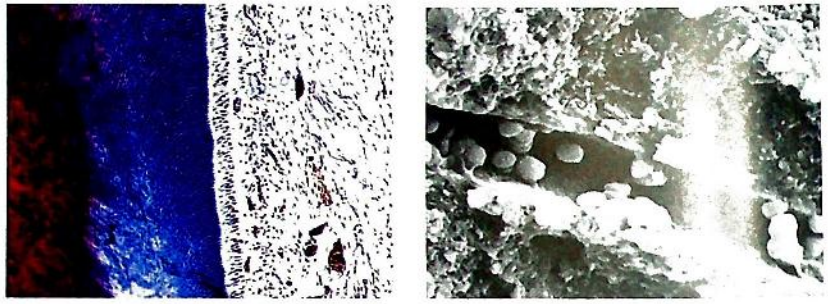


Figure 7.1 (A) Healthy pulp. The aim is preservation of this tissue. (B) Ingress of oral streptococci into dentine tubules. (Courtesy of the Institute of Dental Research, SEM Unit, Westmead.)

root length and apical development. It is important to consider whether the tooth itself is actually restorable in the long term. Retention of a compromised immature permanent tooth with a poor long-term prognosis may still be beneficial for arch integrity and normal alveolar development during the period of dentofacial growth (see Chapter 14).

Evidence for current practice

The current evidence base for pulp therapy in the primary dentition is poor with a demonstrated paucity of prospective randomized controlled trials. The single biggest issue surrounding pulp therapy in the primary dentition is the lack of correlation between clinical symptoms and pulpal status. Hence, at present, there is no single recognized technique for pulp treatment in primary teeth, and a range of different protocols and medicaments are suggested for different combinations of symptoms and clinical findings.

The information in this chapter is based on established clinical practice, retrospective descriptive studies, clinical experience and expert opinion. In general, it is appropriate to use the least invasive intervention that is predictably associated with a healthy, adaptive healing response in the affected primary or permanent tooth. Obviously, effective primary prevention and early intervention will obviate the need for many of the procedures and techniques described later in this chapter.

Clinical assessment and general considerations

Diagnosis of pulpal status

Effective pulpal therapy requires the correct assessment and interpretation of clinical signs and symptoms, leading to an accurate diagnosis of the pulpal condition. Ineffective or inappropriate pulp therapy is associated with both acute and chronic clinical signs and symptoms. Unfortunately, there are no objective or definitive tests to determine the health of the pulpo-dentinal complex in the primary or immature permanent tooth. Clinical signs and symptoms are poorly correlated with actual pulp histology.


A

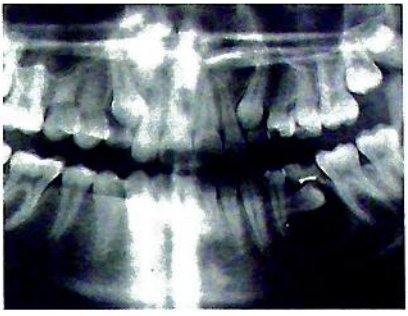

B

Figure 7.2 (A) Large multisurface glass ionomer restorations are inadequate to properly restore primary molars. Persistent coronal microleakage leads to pulp necrosis. (B) Panoramic radiograph showing the results of coronal microleakage and the formation of a large inflammatory follicular cyst associated with the second premolar.

Acute signs and symptoms include:

- Pain.
- Mobility.
- Periapical or intra-radicular abscess.
- Facial cellulitis, including spread of infection into the tissue planes around the airway (Ludwig's angina, see Chapter 10).

Chronic signs and symptoms include:

- Persistent infection.
- Discharging sinus.
- Inflammatory follicular cyst (see Chapter 10).
- Failure of exfoliation of primary teeth.
- Apical fenestration.
- Ectopic permanent teeth (Figure 7.2).

Pulp sensibility tests

Standard techniques of pulp sensibility testing are of limited value in children. These techniques rely on patient feedback in response to thermal and electrical stimulation. In the primary dentition, it is likely that children will not have achieved the cognitive development necessary to respond reliably to a potentially painful stimulus and response challenge. In the immature permanent tooth, raised response thresholds to electrical stimuli are observed. These decrease to normal levels with root maturation and apical closure.

Pain (Figure 7.3)

Young patients frequently have difficulty communicating their experience of pain. It is often not until their pain is severe and prolonged that parents might become aware of and seek treatment for their child. Symptoms of severe, prolonged, spontaneous or nocturnal pain suggest irreversible pulpitis or a dental abscess (Figure 7.3B). A

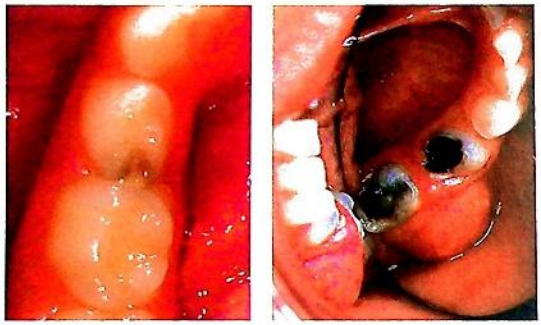


Figure 7.3 (A) Much of the pain that children experience may be caused by food impacting into a cavity. Even without radiographs, it is important to recognize that the pulp will always be involved when the carious lesion is of this size. (B) Buccal swelling not only indicates pulpal necrosis and pus formation but also the loss of bone and perforation of the cortical plate. It may also be difficult to initially determine which tooth is responsible for the swelling; in this case, both teeth should be removed.

history of repeated need for analgesics is also suggestive of pulp necrosis. Dental pain will frequently resolve once a sinus tract establishes drainage, and thus relieves pressure. In these cases, the underlying pathology is still present and must be resolved, despite the lack of obvious discomfort. Chronic infection in the primary dentition can cause disturbances to enamel formation in the permanent dentition (Turner tooth, see Chapter 11) and malocclusion (Fig 7.2B) even in the absence of clinical symptoms or pain.

Other clinical signs

Careful clinical examination of teeth can reveal useful diagnostic information.

- Coronal discoloration is suggestive of pulp necrosis.
- Clinical mobility is associated with loss of bone from infection or imminent exfoliation.
- Marginal ridge fracture in a primary tooth is suggestive of carious pulpal involvement in contact point caries (Figure 7.4A).
- Fracture of the occlusal triangular ridges or carious undermining of the cusps in pit and fissure caries also suggests carious involvement (Figure 7.4B).

Unfortunately, the external appearance of the carious lesion can in some cases, be misleading (Figure 7.5). Persistent symptoms occurring soon after placement of a restoration indicate pulpal pathology. Lack of coronal seal will inevitably lead to pulpal pathology. Radiographic examination is essential to supplement clinical findings and enhance diagnostic accuracy.

Radiographs

Longitudinal radiographs showing normal dentine deposition within the pulp chamber and the roots suggests pulpal health. Irregular pulp calcification or pulpal obliteration suggests pulpal dystrophy, while failure of physiological pulp regression or arrested

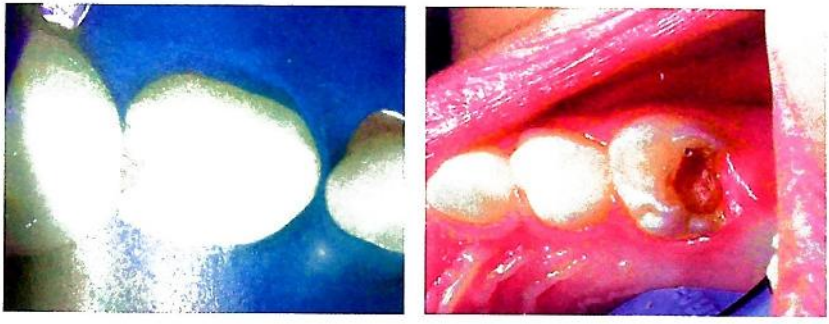


Figure 7.4 (A) Loss of marginal ridge of first primary molar suggests carious pulpal involvement. (B) Undermined triangular ridge or cusp suggests carious pulpal involvement.

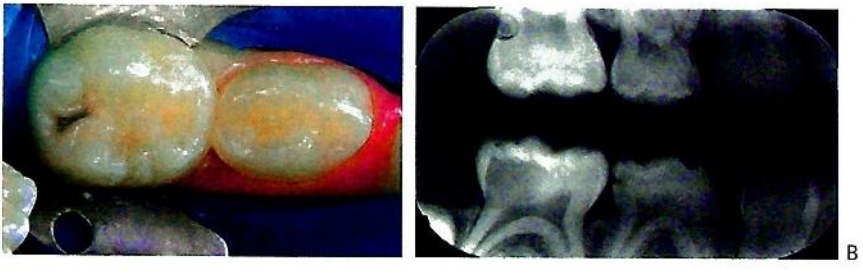


Figure 7.5 (A) Caries may be much more extensive than clinically visible. (B) The full extent of caries is only radiographically evident and shows pulpal involvement.

root development suggests pulpal necrosis. In a single radiographic examination, individual teeth can be compared with their antimere to identify asymmetry.

Clinical signs or symptoms suggesting carious involvement of the pulp require radiographic investigation. Radiographs will show the extent of the carious lesion, the position and proximity of pulp horns, the presence and position of the permanent successor, the status of the roots and of their surrounding bone. Radiographic examination should be considered essential before undertaking endodontic procedures. The presence of caries in the furcation, internal or external root resorption including physiological root resorption, and periapical or furcation bone lesions, are all contraindications to endodontic treatment in the primary dentition.

Primary teeth with these radiographic signs should be extracted.

Swelling

Alveolar swelling, particularly involving the vestibular reflection, facial swelling, coronal discoloration, and the presence of a sinus, are indicators of pulp necrosis and abscess formation (see Figure 7.3B).

Mobility

Inappropriate tooth mobility, tenderness to palpation or a sensation of occlusal interference also suggests abscess formation.

Antibiotic usage to control acute infection (see Odontogenic infection, Chapter 10) may temporarily resolve some or all of these clinical signs, but will not resolve the underlying pathology. A primary tooth that cannot be saved requires extraction despite potential future orthodontic complications.

Factors in treatment planning

Medical history

A thorough medical assessment is essential prior to the commencement of any dental treatment. Medical issues may limit or change treatment options in a number of ways. As pulp therapy necessarily relies on the adaptive healing response after treatment, so patients with a significantly compromised immune system are considered poor candidates for endodontic therapy.

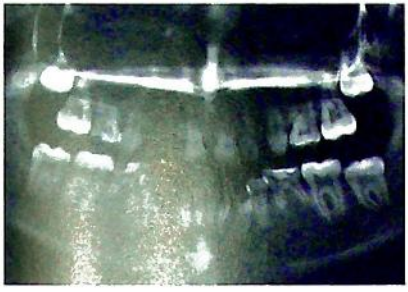
Contraindications

- Congenital cardiac disease (see Appendix E). Patients who are considered to be at risk of bacterial endocarditis should be free of oral infection and any primary tooth with clinical signs of infection should be extracted. There is no evidence to suggest that a primary tooth with a large restoration is more or less likely to become infected if it has undergone endodontic treatment according to established guidelines.
- Immunosuppressed patients and those with poor healing potential (see Immunodeficiency, Chapter 12).

Generally, children with well-managed diabetes present no particular problem in relation to healing potential. The use of long-term corticosteroids for the management of asthma, or asthma, should not affect the decision to retain primary teeth. However, children who are severely immunosuppressed, such as oncology patients, must be treated more aggressively (e.g. extractions).

Indications

- Bleeding disorders and coagulopathies (see Chapter 12). Current management protocols for patients with a bleeding diathesis (such as haemophilia) may use regular, often home-based, factor replacement. Where patients have access to such medical treatment, the decision to extract or retain a pulpally involved primary tooth should not be determined by the bleeding diathesis, but should be based on the same criteria used for any other patient. Consultation with the child's haematologist is essential.
- Hypodontia (i.e. ectodermal dysplasia, Figure 7.6A; see also Chapter 11). In cases of congenital absence of teeth, the decision to extract or retain individual teeth will be influenced by the overall orthodontic strategy. In some cases, there is a requirement to extract primary teeth early to encourage occlusal drift and space closure. In these cases, timing of extractions can be critical, necessitating an interim restoration of the affected primary tooth. In other cases, it is necessary to maintain a



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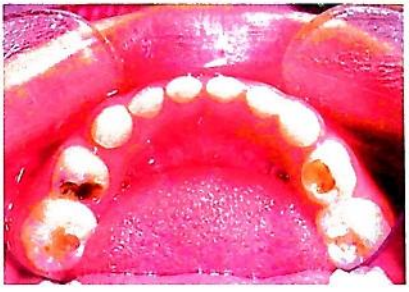

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Figure 7.6 (A) Panoramic radiograph of a child with congenital absence of six premolars. While three of the second primary molars are carious, it is important to retain these teeth. (B) It is important to consider the implications of space management when deciding on the most appropriate options in managing a cariously exposed primary molar. In this case, the tooth '74' is pulpally involved but restoration with a stainless steel crown has been made more difficult due to mesial migration of the second primary molar into the distal carious lesion.

primary tooth without a successor. In the absence of acute symptoms, a formal orthodontic evaluation should be considered.

Behavioural factors

Effective endodontic treatment requires a high level of patient compliance. If a child is unable to cooperate with pre-treatment diagnostic procedures including radiographs, they are unlikely to cope with complex endodontic and associated restorative procedures. Where cooperation cannot be obtained or is fragile, it is reasonable to consider the elective use of general anaesthesia, or even elective extraction of the affected tooth rather than complex endodontic and restorative procedures.

Endodontics requires effective pain control. Even with usually effective doses of local anaesthetic, a child may experience breakthrough pain. This is particularly so on entry to the pulp chamber. The sedative effects of inhalation sedation used in conjunction with local analgesia can facilitate patient comfort and compliance. The use of the rubber dam to isolate the tooth undergoing treatment, and to protect the patient from instruments and medicaments is essential.

Dental factors

Endodontic management should be considered within the overall context of occlusal development, with due consideration to occlusal guidance and space maintenance (Figure 7.6B; see also Space maintenance, Chapter 14). Under normal circumstances, the service life of a primary incisor is 5–8 years, and a primary cuspid or molar is 8–10 years. The early loss of a primary tooth may lead to localized space loss, delayed eruption and ectopic eruption of the permanent successor. Elective extraction may be considered within 3 years of anticipated exfoliation, because accelerated eruption of the permanent successor can be expected.

Long-term success in endodontic therapy requires an effective coronal seal to prevent micro-leakage and the ingress of oral bacteria to the root canals. If the carious

tooth is not restorable, it should be extracted. Pulpotomy and pulpectomy procedures require significant access cavity preparations, which have the potential to weaken the axial walls of the treated tooth. In general, full coverage restoration with a preformed metal crown or a composite resin crown is recommended.

Pulp capping

Indirect pulp capping

Sealing off the advancing carious lesion from the oral environment produces a bacteriostatic response within the body of the lesion, and promotes pulpal healing with the formation of reactionary dentine. This is the basis for indirect pulp capping in both the primary and permanent dentition and is also known as caries control. Indirect pulp capping is also the basis for the atraumatic restorative technique (see Minimal intervention, Chapter 6).

It is uncertain whether the carious lesion in dentine will become sterile and remineralize, or if it merely becomes quiescent with the potential to reactivate if there is leakage around the final restoration, hence there is debate over the necessity of re-entering the tooth to remove the residual caries once there is clinical and radiographic evidence of pulpal healing. Because of the known service life of the primary tooth, there is no indication for re-entering the primary tooth to remove residual caries when the clinical response is favourable.

Ozone and silver fluoride have both been proposed as adjunctive antimicrobial agents in conjunction with indirect pulp capping. At present, there is a lack of evidence to support their superiority over sealing the lesion with standard restorative materials. Ozone may also promote remineralization by oxidization of the lactate-propionate buffering system (pH = 4) within the body of the carious lesion to bicarbonate and water. The depth of residual caries can be no greater than 2 mm when ozone is applied, as ozone will not penetrate more than 2 mm into carious dentine.

Large carious lesions and associated cavity preparations alter the mechanical properties of the treated tooth, reducing the rigidity of the cavity walls in normal function, increasing the potential risk of microleakage. As indirect pulp capping relies on sealing off the residual caries from the oral environment, the residual tooth structure should be carefully evaluated, areas of unsupported enamel removed and weakened cavity walls, which are likely to flex in function (increasing microleakage), should be protected with full coverage. This is of particular importance with approximal lesions where the buccal and lingual walls can be extensively undermined.

Indirect pulp capping in lower first primary molars always requires a preformed metal crown. Severely broken down first permanent molars can be effectively stabilized with preformed metal crowns to allow time for maturation of the pulp and dentine prior to definitive restoration. With growth, there is pulpal regression giving increased dentine thickness for crown preparation, and improved thickness of the radicular dentine giving better root strength. At the completion of dental growth, the restorative options for these teeth can be re-evaluated.

Indications

- Large carious lesion.
- Asymptomatic tooth or mild transient symptoms.
- Preoperative radiograph confirms the absence of radicular pathology.

Technique

- Pain control and isolation.
- Remove superficial caries.
- Remove all peripheral caries, leaving deep caries over pulp.
- Finalize cavity preparation.
- Restore tooth ensuring adequate coronal seal.

Direct pulp capping

Primary teeth

Small pulp exposures can be broadly classified as mechanical (iatrogenic) or carious. Direct pulp capping of carious pulp exposures in primary teeth has a poor prognosis, with failure occurring as a result of internal root resorption. The size of the pulp exposure does not affect prognosis. A pulpotomy should be undertaken in such cases. Uncontaminated mechanical pulp exposures are thought to have a more favourable response to direct pulp capping using hard-setting calcium hydroxide cements. There is inadequate evidence to support the use of other materials currently used, including antibiotic/corticosteroid (Ledermix®), dentine-bonding resins, mineral trioxide aggregate (MTA) as direct pulp capping agents in the primary dentition. Because of the difficulties in determining the pulp status and the vastly superior prognosis of pulpotomy, direct pulp capping cannot be recommended in the primary dentition.

Immature permanent teeth

Direct pulp capping of pinpoint pulp exposures, either mechanical or carious, has a favourable prognosis in the immature permanent tooth. The use of calcium hydroxide and hard-setting calcium hydroxide cements, has been widely reported, however, there is limited evidence to support the use of other materials.

Pulpotomy in primary teeth

Pulpotomy is the most widely used endodontic technique in the primary dentition. The suffix 'otomy' means 'to cut', so pulpotomy is 'to cut the pulp'. The aim of pulpotomy in the primary tooth is to amputate the inflamed coronal pulp and preserve the vitality of the radicular pulp, thereby facilitating the normal exfoliation of the primary tooth.

A pulpotomy cannot be done if the pulp is necrotic

The contemporary pulpotomy traces its origins to nineteenth-century techniques for the mummification of painful, inflamed or putrescent pulpal tissue. Over the twentieth century, the pulpotomy technique changed with fewer stages and reduced duration of application and concentration of medicament. Emphasis is now placed on the preservation of healthy radicular pulp rather than mummification.

Caries removal

- The treated tooth must be rendered completely caries-free before proceeding with the pulpotomy.

The recommendation to remove caries from periphery to pulp not only prevents contamination of the pulpotomy site with carious debris but also reduces the risk of inadvertent pulp exposure. Access to the coronal pulp requires complete removal of the roof of the pulp chamber. Amputation of the coronal pulp requires a clean cut at

the level of the pulpal floor. Residual tissue tags at the amputation site will create problems with haemostasis. High-speed rotary instrumentation with copious water spray irrigation creates the optimal cut.

- If the floor of the pulp chamber is perforated, the tooth must be extracted.

Haemostasis

Haemostasis at the pulpotomy site must be obtained before application of the therapeutic agent. This is achieved with continuous irrigation and gentle dabbing with cotton wool pellets and should occur within 5 min. If bleeding cannot be arrested, the pulpal inflammation is considered to have spread to the roots, and is associated with a poor prognosis. This is referred to as the 'bleeding sign' or a 'hyperaemic pulp'. Pulpectomy or extraction should be considered in these cases.

Pulp medicaments

The therapeutic medicament is applied to the pulpotomy site once haemostasis has been obtained. The pulpotomy site is then covered with a therapeutic base (see below). Traditionally, this has been a zinc oxide-eugenol-based cement. However, eugenol in direct contact with pulp tissue causes chronic pulpitis. It is reasonable to substitute a eugenol-free cement as the therapeutic base. When MTA is used as the therapeutic agent, it will also act as the therapeutic base. Finally, a core material should be used to seal the tooth before the final restoration, ideally with a full coverage restoration.

Earlier texts have suggested that teeth, which are to have a preformed metal crown, should also have a routine elective pulpotomy, regardless of whether they have a carious pulp exposure. This position is no longer tenable given the predictable success of indirect pulp capping. However, the reverse is true, in that all teeth that have had a pulpotomy should be restored with a stainless steel crown. The concept of full coverage also applies to anterior teeth where a full coverage restoration should also be used in those incisors on which a pulpotomy has been performed.

Indications for pulpotomy in primary teeth

- Carious pulp exposure.
- Tooth asymptomatic or mild transient pain.
- Preoperative radiograph confirms the absence of radicular pathology.
- Restorable tooth.

Technique (Figures 7.7, 7.8)

1. Pain control and rubber-dam isolation.
2. Complete removal of caries from peripheral to pulpal.
3. Removal of roof of pulp chamber.
4. Amputation of coronal pulp.
5. Arrest of bleeding at amputation site (see discussion of 'bleeding sign' above).
6. Application of therapeutic agent (see Therapeutic agents used for pulpotomy).
7. Place base directly on to pulp amputation site (IRM® or Cavit®).
8. Place core.
9. Restore tooth with a preformed metal crown (molars) or a composite resin strip crown for anterior teeth.
10. Regular radiographic assessment.

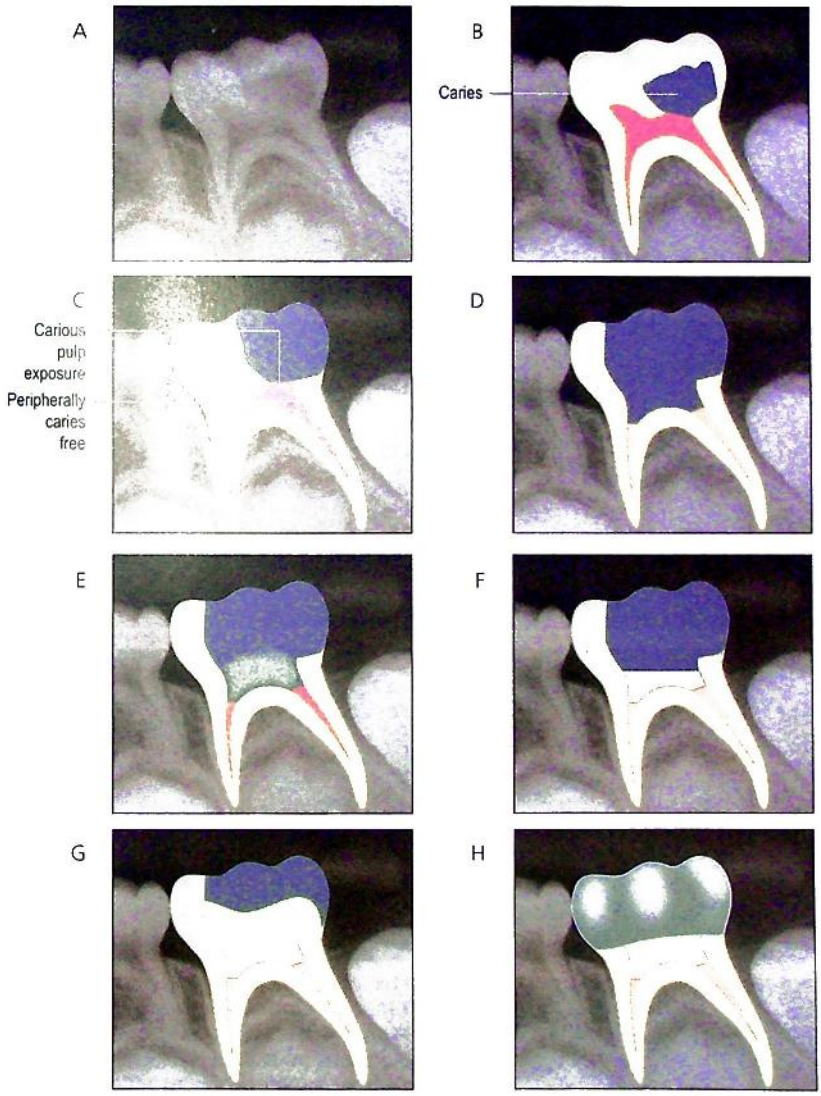

Figure 7.7 Method of performing a pulpotomy. (A) Preoperative radiograph shows deep carious lesion. Clinical history revealed intermittent symptoms on eating with no history of spontaneous pain. (B) Carious lesion identified relative to dental anatomy. (C) Cavity preparation showing complete removal of peripheral caries. (D) After the tooth is rendered free of caries, the roof of the pulp chamber is removed completely, and the pulp is amputated to the level of the pulpal floor. Haemostasis must be achieved at this point before proceeding. (E) The therapeutic agent is applied to the pulpotomy site. (F) Base is applied to completely seal the pulpotomy site. (G) The tooth is built up with a core material. (H) The tooth is restored with a preformed metal crown.

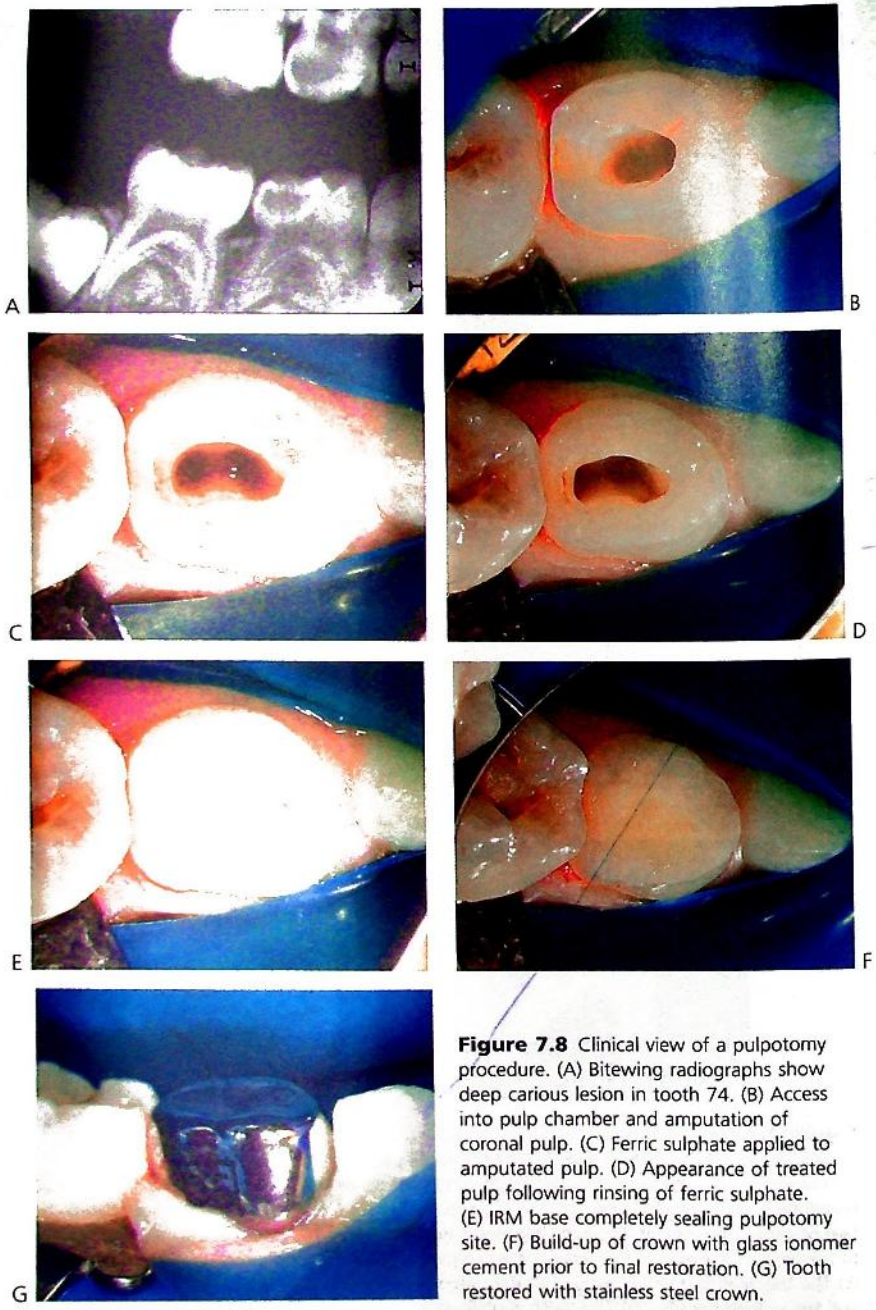

Figure 7.8 Clinical view of a pulpotomy procedure. (A) Bitewing radiographs show deep carious lesion in tooth 74. (B) Access into pulp chamber and amputation of coronal pulp. (C) Ferric sulphate applied to amputated pulp. (D) Appearance of treated pulp following rinsing of ferric sulphate. (E) IRM base completely sealing pulpotomy site. (F) Build-up of crown with glass ionomer cement prior to final restoration. (G) Tooth restored with stainless steel crown.

Therapeutic agents used for pulpotomy in primary teeth

A diverse range of chemicals have been used as pulpotomy agents. As most of these have not been subject to rigorous clinical trials, their use has been based on expert opinion and retrospective studies. In their review for the Cochrane Collaboration, Nadin and colleagues (2003) concluded that based on the available randomized controlled trials (RCTs):

- There is no reliable evidence supporting the superiority of one type of treatment for pulpally involved primary molars.
- No conclusions can be made as to the optimum treatment or techniques for pulpally involved primary molar teeth due to the scarcity of reliable scientific research.
- High quality RCTs, with appropriate unit of randomization and analysis are needed.

The available evidence suggests that formocresol, ferric sulphate, electrocautery and MTA have similar efficacy. Calcium hydroxide appears to have a consistently lower success rate in pulpotomies in primary teeth than these four agents. There are a number of other materials that are of historical significance, or have regional usage, and a number of experimental techniques including bone morphogenic protein and growth factors, which will not be discussed. All current therapeutic agents have toxic effects and must be correctly handled within their therapeutic range. Clinicians should carefully read the Materials Safety Data Sheet for these agents. Cases should be carefully selected within the guidelines recommended.

Mineral trioxide aggregate

MTA is a mixture of tricalcium silicate, bismuth oxide, dicalcium silicate, tricalcium aluminate and calcium sulphate. It is chemically similar to standard cement mix. MTA powder reacts with water to form a paste, which is highly alkaline (pH 13) during the setting phase, then sets to form an inert mass. Clinical success rates for MTA pulpotomy are similar to formocresol and ferric sulphate.

The MTA powder is mixed with water immediately prior to use. The resultant paste is applied to the pulpotomy site using a proprietary carrier or a plastic instrument and is left *in situ* to set. It is covered with a suitable base material prior to restoration of the tooth. The paste should only be applied after haemostasis has been obtained. Persistent bleeding from the pulpotomy site is an indication for pulpectomy or extraction.

Ferric sulphate

Ferric sulphate is widely used in dentistry as a haemostatic agent (Astringident®). It was initially used in pulpotomy as an aid to haemostasis prior to placement of calcium hydroxide. However, as an independent therapeutic agent, ferric sulphate pulpotomy has a success rate of 74–99%. Ferric sulphate is thought to react with the pulp tissue, forming a superficial protective layer of iron–protein complex. The predominant mode of failure is the result of internal resorption.

Ferric sulphate is burnished onto the pulp stumps (pulpotomy site) using a microbrush for 15 s, then rinsed off with water and dried. Persistent bleeding after the application of ferric sulphate is an indication for pulpectomy or extraction.

Electrosurgery

Electrosurgery uses radiofrequency energy to produce a controlled superficial tissue burn. It is both haemostatic and antibacterial. Excessive energy or contact time causes a deep tissue burn with necrosis of the radicular pulp and subsequent internal root resorption. Electrosurgical pulpotomy has a success rate of 70–94%.

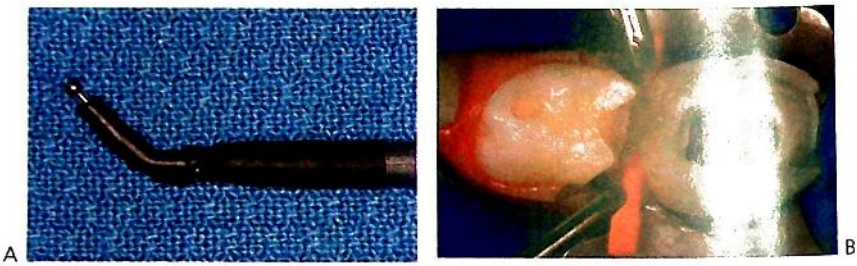

Figure 7.9 (A) Electrosurgical tip used in pulpotomy. (B) Appearance of root stumps following electrocautery.

The electrosurgery unit should be set to coagulate, with a low power setting. A small ball or round-ended tip is applied to the pulpotomy site and briefly activated (Figure 7.9). The site should immediately be flooded with water to remove excess heat. Each pulp stump is treated in turn. If necessary, electrocoagulation can be repeated to control persistent bleeding, until the total cumulative application time is 2 s. Persistent bleeding after this time, is an indication for pulpectomy or extraction. Electrosurgical equipment has the potential to interfere with pacemakers and implanted electronics.

Formocresol

Formocresol has been used in dentistry for over 100 years, and for vital pulpotomy in primary teeth for over 80 years. Its efficacy has been extensively studied, with clinical success rates ranging from 70% to 100%, making it the standard against which newer techniques are compared. The formaldehyde component of formocresol is strongly bactericidal and reversibly inhibits many enzymes in the inflammatory process.

Originally, the aim of using formocresol was to completely mummify (fix) all residual pulpal tissue and necrotic material within the root canal. Current techniques, however, aim to create a very superficial layer of fixation, while preserving the vitality of the deeper radicular pulp.

Formocresol is applied to the pulpotomy site on a cotton wool pledget. Any excess material should be blotted off the pledget prior to application. Traditionally, a 5-min application time has been recommended; however, contact times of only a few seconds are probably equally effective. It is prudent to limit both dose and contact time. Formocresol should only be applied to the pulpotomy site after haemostasis has been obtained. It should never be applied to bleeding tissue.

In 2004, the International Agency for Research on Cancer (IARC) concluded that chronic exposure to high levels of formaldehyde causes nasopharyngeal cancer in humans. In assessing the potential risks of using formocresol clinically, however, it is important to consider the pharmacokinetics of formaldehyde. Formaldehyde is an important intermediate in normal cellular metabolism. It serves as a building block for the synthesis of purines, pyrimidines, many amino acids and lipids, and is a key molecule in one-carbon metabolism. Endogenous formaldehyde is present at low levels in body fluids, with a concentration of 2–3 mg/L in human blood. Application of formocresol results in systemic absorption of formaldehyde, however the absorbed formaldehyde is rapidly metabolized to formate and carbon dioxide with a half-life of

1–2 min. The use of formocresol in dentistry falls within the current permitted exposure limits, and short-term exposure limits for formaldehyde. Formaldehyde does not bioaccumulate.

While much has been written recently concerning the potential toxicity of formocresol, it should be noted also, that exposure to MTA dust and crystalline silica can cause respiratory irritation, ocular damage and skin irritation. The IARC has determined that silica is a known human carcinogen. Similarly, ferric sulphate has been classified as a hazardous, corrosive liquid (Worksafe Australia) and decomposes to form sulphuric acid, that can cause superficial tissue burns if it is not confined to the pulpotomy site. Patients have also suffered earth leakage burns from incorrectly grounded dispersive plates used in electrosurgical equipment.

Clinicians need to be aware of the risks in using any medicament or equipment, use each according to the manufacturers' directions and be familiar with relevant Material Safety Data Sheets.

Pulpotomy in the immature permanent tooth

The aim of pulpotomy in the immature permanent tooth is to amputate the inflamed coronal pulp and preserve the vitality of the remaining pulp to promote apexogenesis (see Chapter 8). Apexogenesis involves the continued normal development of the radicular pulp below the pulpotomy site, resulting in normal root length, thickness of radicular dentine and apical closure. Apexogenesis optimizes root anatomy and strength. The main risk of apexogenesis is the potential for dystrophic pulp calcification in the event that subsequent pulpectomy is required. The biomechanical properties of the root are more favourable after apexogenesis than after apexification, but apexification is the only option once pulp necrosis has occurred in the immature permanent tooth. An alternative to apexification is the use of haematogenous stem cells to induce calcification of the root canal space.

Unlike the primary dentition in which the pulpotomy is always at the level of the pulpal floor, a small carious exposure of the pulp horn of a permanent tooth can be managed by a superficial pulpotomy of only 1–2 mm. This is based on Cvek's pulpotomy. Where there is a large exposure, or multiple exposure sites, a deep pulpotomy is required to the opening of the root canals, or the level of the CEJ in an anterior tooth. The exposure site is continuously irrigated until haemostasis occurs, prior to application of the therapeutic medicament. The therapeutic medicament can be calcium hydroxide powder or paste or MTA. Antibiotic/corticosteroid (Ledermix®) paste has also been used.

Clinical criteria

- Carious pulp exposure.
- Asymptomatic tooth or episodes of mild, transient pain.
- Preoperative radiograph confirms immature roots with open apices.
- Absence of radicular pathology.
- Restorable tooth.

Technique

1. Pain control and rubber-dam isolation.
2. Complete removal of caries.

3. Removal of roof of pulp chamber.
4. Amputation of coronal pulp, either superficially or deep to the opening of the root canal.
5. Arrest of bleeding at amputation site.
6. Application of therapeutic medicament (calcium hydroxide or MTA).
7. Place base directly over the therapeutic medicament (IRM® or Cavit®).
8. Restore tooth with adequate coronal seal.
9. Regular radiographic assessment.

Pulpectomy in primary teeth

Pulpectomy is the complete removal of all pulpal tissue from the tooth. Pulpectomy can only be considered for primary teeth that have intact roots. Any evidence of root resorption is an indication for extraction. Severe infections including acute facial cellulitis associated with primary teeth do not respond well to pulpectomy. Extraction is usually recommended in these cases.

Although the root canal morphology of primary incisors is relatively simple, the root canal morphology of multi-rooted primary teeth is more complex than permanent teeth, with fins, ramifications and inter-canal communications. These anatomical factors inhibit complete chemo-mechanical debridement of the root canal space. The anatomical apex may be up to 3 mm from the radiographic apex, and frequently occurs on the lateral surface of the root, making it difficult to determine the true working length. Over-instrumentation of the primary tooth root canal has the potential to damage the underlying permanent tooth. Electronic measurement of the root canal can assist with the location of the anatomical apex of a primary tooth.

Obturation of the root canal space in a primary tooth must not interfere with normal exfoliation of the permanent successor. This requires a resorbable paste root filling. The exception to this would be where it is planned to retain a primary tooth that does not have a permanent successor. Suitable materials for obturation include unreinforced zinc oxide eugenol cement, calcium hydroxide paste and iodoform paste.

Indications for pulpectomy in primary teeth

- Pulp necrosis in any primary tooth, or carious exposure of vital primary incisor.
- Restorable tooth.
- Preoperative radiograph confirms intact non-resorbed root.
- Retention of tooth is required.

Technique

1. Pain control and rubber-dam isolation.
2. Complete removal of caries.
3. Chemo-mechanical cleaning and preparation of the root canal, taking care to force neither instruments nor debris beyond the anatomical apex. Copious irrigation with sodium hypochlorite.
4. Obturation with a resorbable paste (see above).
5. Restoration to ensure adequate coronal seal.
6. Regular radiographic assessment.

Pulpectomy in immature permanent teeth (Figure 7.10)

Dental immaturity is defined by the lack of apical closure. Necrotic immature permanent molars have a poor long-term prognosis and, except in exceptional circumstances, these teeth should be removed (see Extraction of first permanent molars, Chapter 14). However, retention of such teeth may be important for alveolar development, behavioural reasons or may facilitate subsequent orthodontic treatment by holding space until the optimal time for extraction.

By definition, these teeth have already lost significant amounts of tooth structure due to caries. In addition, endodontic treatment would weaken an already compromised tooth, require apexification over many years (see Chapter 8) and involves significant operative challenges (i.e. isolation, obturation, restoration).

Tables 7.1 and 7.2 summarize the treatment options for primary and immature permanent teeth.

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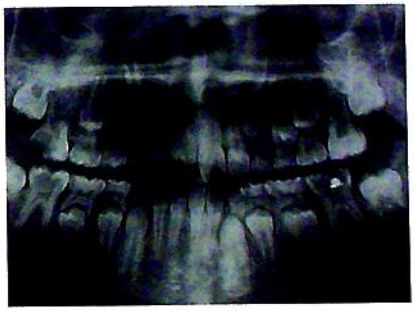

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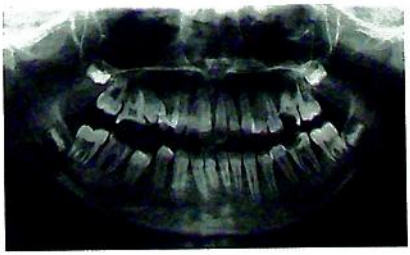

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Figure 7.10 (A,B) The long-term prognosis and the ability to restore a tooth are the overriding factors when assessing whether pulp therapy should be undertaken. In these cases, it is often preferable to extract the first permanent molars and allow the second molars to drift mesially. (C) In this case, the eruption of the second molars does not affect the decision to remove the first molars due to the extensive carious breakdown in addition to the presence of the third molars.

Table 7.1 Treatment options for primary teeth

Clinical event	Signs or symptoms	Pulpal status	Treatment choice
Caries without exposure	No spontaneous symptoms	Healthy or reversible pulpitis	Restore tooth
Caries with possible or near exposure	No spontaneous symptoms	Healthy or reversible pulpitis	Indirect pulp capping
Caries with possible or near exposure	Occasional pain on stimulation	Reversible pulpitis	Pulpotomy
Caries with possible or near exposure	Close to exfoliation		Consider elective extraction
Iatrogenic/ non-carious exposure	No spontaneous symptoms	Healthy	Pulpotomy
Carious exposure	Minimal history of pain No mobility No radiographic evidence of pathology	Reversible pulpitis	Pulpotomy
Carious exposure	Spontaneous pain	Irreversible pulpitis	Pulpectomy Intermediate dressing Extraction
Carious exposure	Draining sinus Swelling Mobility Radiographic pathology (inter-radicular or periapical, root resorption)	Necrotic pulp	Pulpectomy with resorbable dressing or Extraction
Gross caries	Caries through bifurcation Extensive root resorption Tooth not restorable Furcation periapical pathology	Necrotic pulp	Extraction

Table 7.2 Treatment options for immature permanent teeth

Clinical event	Signs or symptoms	Pulpal status	Treatment choice
Caries without exposure	No spontaneous symptoms	Healthy or reversible pulpitis	Restore tooth
Caries with possible or near exposure	No spontaneous symptoms or Occasional pain on stimulation	Healthy or reversible pulpitis	Indirect pulp capping/ caries control
Small pulp exposure	No spontaneous symptoms	Healthy	Direct pulp capping
Carious exposure	Minimal history of pain No mobility No radiographic evidence of pathology	Reversible pulpitis	Pulpotomy and apexogenesis
Carious exposure	Spontaneous pain	Irreversible pulpitis	Pulpectomy and apexification or Extraction
Carious exposure	Draining sinus Swelling Mobility Radiographic pathology	Necrotic pulp	Pulpectomy and apexification or Extraction
Gross caries	Tooth not restorable	Irreversible pulpitis or Necrotic pulp	Extraction

Further reading

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8 Clinical and surgical techniques

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Extraction of teeth in children

The removal of teeth in children can be one of the most stressful procedures for both the operator and patient. While a tooth may be totally anaesthetized, the pressure felt during the extraction can be extremely upsetting and uncomfortable. As one of the most important aspects of clinical practice, dentists need to be skilled, efficient and sensitive in the removal of teeth in children. Teeth should be removed gently with good surgical technique rather than excessive force that may fracture roots or upset the patient.

General principles of tooth extraction in children

Preoperative assessment

- Thorough medical history and informed consent for the procedure.
- Evaluate the tooth to be extracted both clinically and radiographically.
- Identify potentially difficult root anatomy and the proximity of other important structures prior to extraction. Be aware of implications for the permanent successor.
- Clearly identify the tooth to be extracted and confirm again prior to extraction.
- Profound local anaesthesia is vital. Explain the feeling of 'numbness' and the sensation associated with luxation of the tooth prior to commencing the procedure.
- If the child will be unable to cope with the extraction(s) then sedation or general anaesthesia should be considered. Ideally, the decision to sedate a child should be made at the assessment appointment NOT once the child has become upset during the procedure.

Other principles

- Primary teeth do not require a 'primary drive' that is often recommended for permanent teeth.
- Children tolerate the use of luxators or elevators much better than application of forceps. The alveolar bone in children is soft and teeth can be elevated easily to a high degree of mobility prior to a final delivery with forceps.
- If small apical root fragments remain after an extraction, they may be left to resorb, as attempted removal may damage the permanent successor.
- Choose a pair of extraction forceps suited to the required procedure. A wide range of forceps designed for primary teeth is available (Figure 8.1).

Figure 8.1 A selection of paediatric extraction forceps. It is important to use the appropriate size forceps for the tooth to be removed.

Anterior teeth

- Primary incisors, especially if there is a fracture present, should be gently luxated rather than elevated to avoid damage to the permanent incisor.
- Single rooted teeth should be delivered with forceps utilizing a rotational movement (Figure 8.2).

Premolars

- The removal of premolars is usually required for orthodontic reasons and may be the first dental intervention for some children. Extraction of the upper first premolar should be addressed with great care as the root apices may be fine and easily fractured. Surgical removal of a retained root fragment usually involves loss of bone, and will have implications for orthodontic treatment and the ability to move adjacent teeth into this space.
- The use of a 3 mm luxator is invaluable to expand the socket and avoid excessive bone damage prior to application of the forceps.
- Delivery with forceps should be made with gentle movements, avoiding excessive buccal forces that might fracture the buccal bone or fine root tips.

Molars

- Extractions should be clean and atraumatic.
- Avoid gingival injuries by freeing the gingival margin with a flat plastic, luxator or elevator (Figure 8.3).
- Second primary molars are often difficult to remove due to the divergent spread of the roots. Sectioning the tooth vertically can facilitate extraction if the crown is considerably damaged or the roots encircle the crown of the underlying permanent tooth.

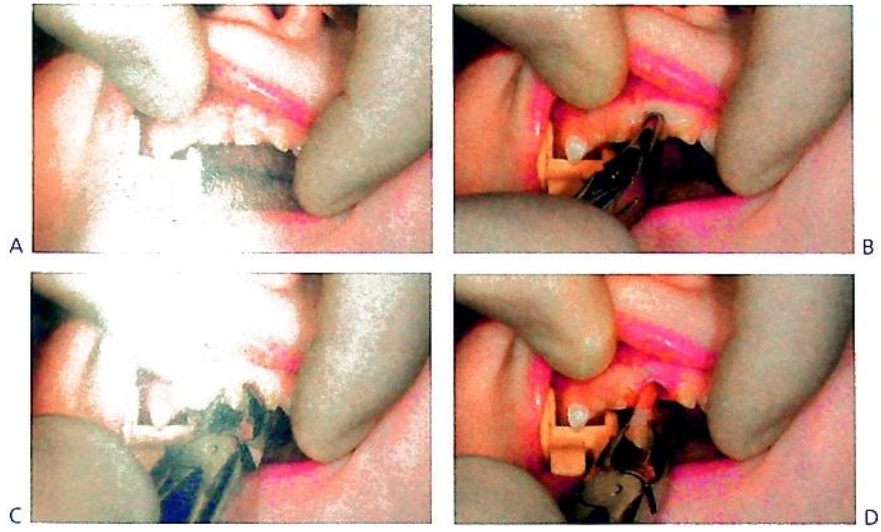


Figure 8.2 Extraction of primary anterior teeth. (A) The alveolus is supported and the upper lip retracted. (B) The beaks of the forceps engage the tooth root, not the crown. Notice the blanching of the attached gingiva. (C,D) The tooth should be delivered with a rotation movement and with minimal apical force that might damage the permanent tooth germ.

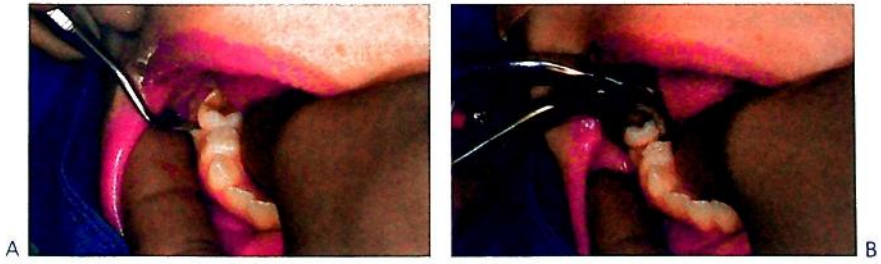
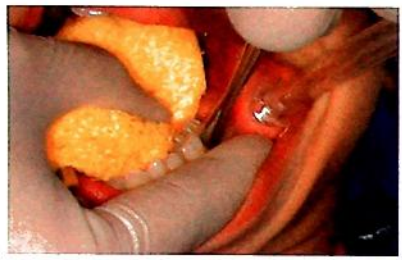


Figure 8.3 (A) When extracting primary posterior teeth it is useful to free the gingiva from the tooth with a flat plastic or a similar blunt instrument to protect it from tearing. (B) Avoid excessive buccal movement that will damage the thin, buccal, cortical plate and the attached gingiva when delivering these teeth.

- Luxation/elevation is essential, however first permanent molars can be difficult to elevate when the adjacent mesial tooth (a second premolar or primary molar) is absent.
- Support the alveolus on either side with fingers.
- Multi-rooted permanent teeth can be extracted by using alternating, slow, buccal and palatal/lingual force or a 'figure of 8' motion in order to expand the alveolar



A

B

C

Figure 8.4 (A) Luxators are delicate and sharp instruments, designed to shear the periodontal attachment and enlarge the tooth socket. The application of the luxator should be vertical along the long axis of the roots. (B) Elevators should be used similarly to a screwdriver, so their application on the tooth root is more horizontal between the embrasure. (C) The index finger should run along the blade and serves to protect the patient if the instrument slips.

bone. While many oral surgery texts recommend the buccal delivery of lower molars, the most dense bone is found on the buccal aspect and excessive movement of a lower permanent molar buccally may result in root fracture, particularly in teeth missing significant amounts of coronal structure.

- 'Cow-horn' pattern forceps are extremely useful in removing either upper or lower permanent molars, especially those with little or no crown remaining on the lingual aspect (Figure 8.5).

Avoiding and managing root fractures

- Avoid root fractures by first luxating or elevating the tooth to a high degree of mobility prior to application of forceps (Figure 8.4).
- If the delivery of the tooth becomes difficult during the extraction – STOP and reassess rather than applying more force that may break the roots.
- Always assess where a permanent tooth germ is positioned prior to elevating roots of primary teeth. If a root is fractured when extracting an ankylosed primary molar, this can usually be left in situ, especially if it is below the interseptal bone.
- Cryer elevators are used to remove interseptal bone between mandibular permanent molar roots to gain access to the roots. Care is required during removal of interseptal bone surrounding primary molar roots so as to avoid damage to the permanent successor.

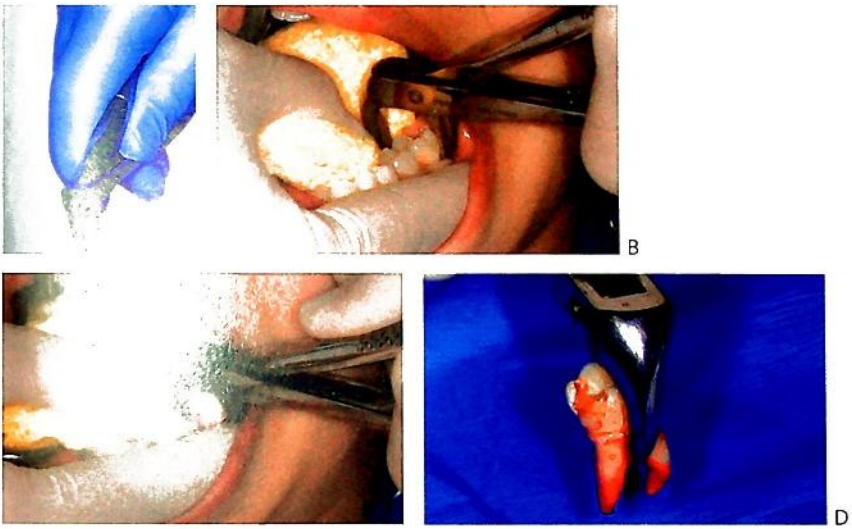


Figure 8.5 (A) 'Cowhorn' pattern forceps engage the bifurcation of a molar tooth (B). (C) As pressure is applied, the beaks are worked further apically and the tooth will rise out of the socket, usually with minimal rotation or buccal movements. These forceps are very useful for badly broken-down molars. While fractures of the crown may occur, the level of the fracture is more coronal and tends to section the tooth, allowing easy delivery of the roots with an elevator. Note the finger support of either side of the alveolus. (D) The beaks of the forceps engage the furcation.

- If it appears impossible to deliver a tooth without a root fracture, then the procedure should be performed as a surgical removal. Ideally this assessment should be made prior to starting the procedure.

Following the extraction

- Examine the extracted tooth carefully.
- If any granulation tissue remains in the socket it should be removed, whilst taking care of the developing tooth germ.
- Obtain haemostasis prior to discharging the child.
- Suture any areas of gingiva or mucosa that may have been torn or damaged.

Postoperative instructions following extractions for children

Always give clear and lucid instructions to the child and caregiver:

- Allow the blood clot to stabilize by avoiding rinsing on the day of extraction.
- The next day, the mouth may be gently rinsed with water. There is little evidence that warm saline or antiseptic mouthwashes are of any real benefit following tooth

extraction in children, but good oral hygiene is essential and gentle toothbrushing can start the day after the extraction. Parents should be advised that halitosis often occurs following extraction or oral surgery.

- Prescribe appropriate analgesics and antibiotics, if required.
- Warn that there should be no sport or excessive play for the remainder of the day.
- Warn of lip biting due to anaesthetized soft tissues.

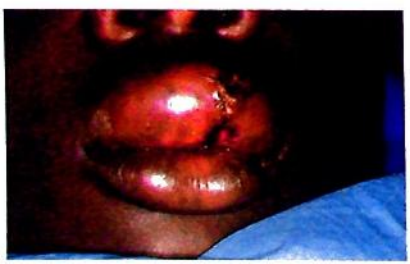
Repair and suturing of soft tissue injuries

Generally, soft tissue wounds should be closed within 24 h. Good closure of wounds allows for more rapid healing by primary intention. Suturing may reduce the sequestration of displaced bony fragments and may prevent bacterial contamination of the gingival sulcus. Furthermore, there is much less pain from the wound if exposed bony defects are well covered with periosteum and gingival tissues. Deeper lacerations of the lip will involve the muscle layer and it is important to close this as a separate layer to prevent formation of a 'dead space' will easily become infected (Figure 8.6). It is essential that the wound is properly debrided and free of contamination from foreign bodies or bony spicules prior to apposition of tissues. Any wound involving skin, including those crossing the vermillion border of the lip, require precise and expert skill to facilitate the best possible result. Often, this requires timely referral to an appropriate surgeon.

Cyanoacrylate (tissue glue) is now commonly used for closure of smaller soft tissue wounds on the face and scalp in children without having to give local anaesthetic. Currently, the literature is equivocal as to whether suturing or gluing produces better outcomes, although it is clear that gluing is far less traumatic for the child and much faster.

Choice of material (Table 8.1)

The choice of suture material and needle will depend on:



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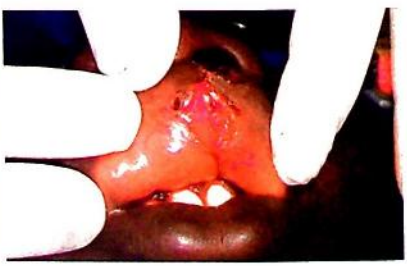

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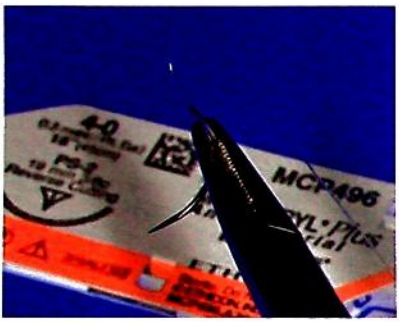
Figure 8.6 (A) When closing any wound, it is essential not to leave a dead space. This laceration to the upper lip was closed only superficially, leaving the muscle layers open. A large abscess developed within 12 h, requiring reopening of the wound, drainage and debridement and reclosure including the muscle and the mucosa (B).

Table 8.1 Some indications for the selection of suture materials in paediatric dentistry

Suture	Indications	Size	Needle	Absorption	Tissue reaction	Notes
Surgical gut	Extraction suture	3-0	Cutting	Completely digested by 70 days. Effective strength for 2–3 days in the oral cavity	Moderate	Used for tissue closure where strength is required for 1–2 days
Chromic catgut	General closure	4-0	Taper	Completely digested by 110 days, but in the oral cavity it has effective strength for up to 5 days	Moderate but less than plain gut	Excellent for oral tissue closure when longer life is required compared with plain gut
Polyglycolic acid Polyglactin	Alveolar mucosa Attached gingiva Large flaps where strength is required but a resorbable suture is desirable	4-0 5-0 3-0 4-0 3-0	Taper Cutting Cutting	Completely absorbed by hydrolysis after 90 days. Faster absorption when exposed to the oral environment. Good strength for least 2 weeks.	Mild	Polyglycolic acid has great advantages for use in the oral cavity in children. It has good strength over 7 days and is resorbable. It is often retained for longer periods however, and has a tendency to accumulate plaque due to its braided nature. Tapering? needles are useful where tissues are friable
Monofilament Nylon	Large flaps where strength is required (i.e. palate)	3-0 4-0	Cutting	Essentially a non-resorbable material, but degrades at 15–20% per year	Extremely low	Excellent tissue reaction and strength. Monofilament material is extremely clean and allows good wound healing but needs to be removed
	Skin	6-0	Cutting			Skin closure must be performed with 6-0. Sutures should be removed before 7 days
Surgical silk	General closure of most oral tissues where a non-resorbable suture is required	3-0 4-0	Cutting	Completely degraded by 2 years	Moderate	Traditional suture material, used where strength was required. Its use has diminished with the availability of materials such as polyglycolic acid. A braided material and therefore not as clean as monofilament



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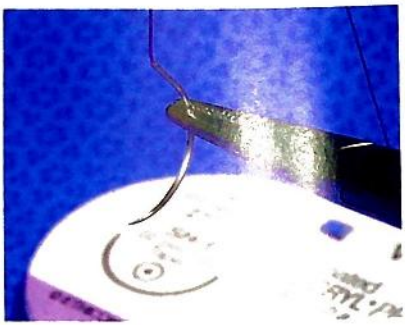

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Figure 8.7 (A) Surgical nylon 4-0 on a reverse cutting needle. This monofilament suture material has excellent tissue reaction and strength. The reverse cutting needle has its cutting edge on the convex surface, which avoids tearing. Cutting needles are used for thick, keratinized tissue such as attached gingiva or palatal mucosa. (B) Polyglactin is a resorbable, braided material. They also have good tissue reaction but tend to accumulate plaque and can become quite dirty in the mouth prior to their loss after 2 weeks. The taper needle is excellent for friable or thin alveolar mucosa.

The type and location of the wound to be closed

- Reverse cutting edge needles should be used in sites involving keratinized gingiva.
- Tapering needles are suitable for non-keratinized gingiva.
- Monofilament materials such as nylon must be used for skin to minimize tissue reaction (Figure 8.7A).
- Internal (muscle) closure must be resorbable.
- Suturing of torn or lacerated gingival tissues should be conducted using a fine suture, such as a 4-0 or 5-0 resorbable suture (Dexon/Vicryl). Polyglactin or polyglycolic acid sutures have good traction strength for at least 3 weeks and have far less tissue reaction than catgut. They are resorbable but because they comprise braided material, they are not nearly as clean as monofilament sutures. Where strength is required, and removal of the sutures is not an issue, monofilament nylon is preferable.

The required strength and length of time required

- Keratinized and thick tissue such as the palate require thicker suture material (3-0).
- Large but thin, friable flaps may still need the strength of a thicker material without the risk of the suture tearing the mucosa.

Whether the material needs to be removed – resorbable or non-resorbable

- Resorbable sutures such as polyglactin/polyglycolic acid (4-0 or 3-0) are preferable for use in young children or where behavioural issues are a concern (Figure 8.7B).

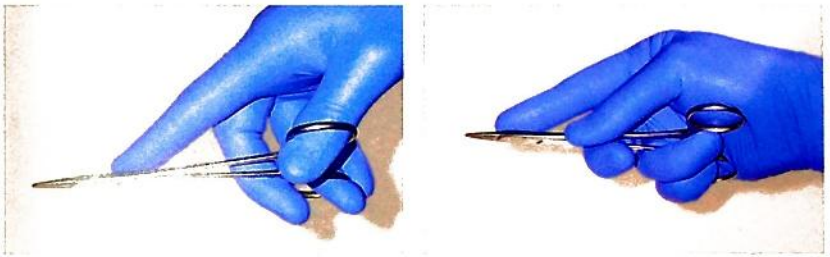


Figure 8.8 (A) Suture needle holders may be held in a scissors or palm grip (B) with the index finger supporting the instrument.

Instruments

While each surgeon will have their own individual preference of surgical instrumentation, the following instruments are those commonly used in many oral surgical suturing situations.

Needle holders

Many different patterns of needle holders are available. The most convenient holders are around 15 cm in length with tungsten carbide beaks and a locking, ratchet handle. For very fine suturing, iridectomy-type (microsurgical) needle holders may be useful. Needle holders can be held in a scissors or a palm grip, but in either case, the index finger should support the blades (Figure 8.8).

Toothed tissue forceps

Always use toothed forceps to hold tissue that would otherwise be crushed with a non-tooth pattern. A straight pattern such as Gillies or McIndoe is sufficient for most procedures, but for fine procedures, a smaller Adson-type is used. Tissue forceps are held in a pen grip.

Fine suture scissors

Almost all scissors are made for use in the right hand and any surgical assistant will be aware of how difficult it is to cut sutures using the left hand. Good suture scissors must be of adequate length to reach into the mouth and while the blades can be short, they must be sharp and maintained.

Skin hooks

The use of skin hooks is usually confined to extra-oral work but are invaluable for mobilizing and everting flap and tissue margins.

Suturing techniques

- **Simple interrupted** – This is the most common suture used in the oral cavity. It is used for interdental suturing of flaps and relieving incisions (Figure 8.9).
- **Horizontal mattress** – The horizontal mattress suture applies force across the wound margin and can be placed across an extraction site. It can also be used to evert wound margins (Figure 8.10).

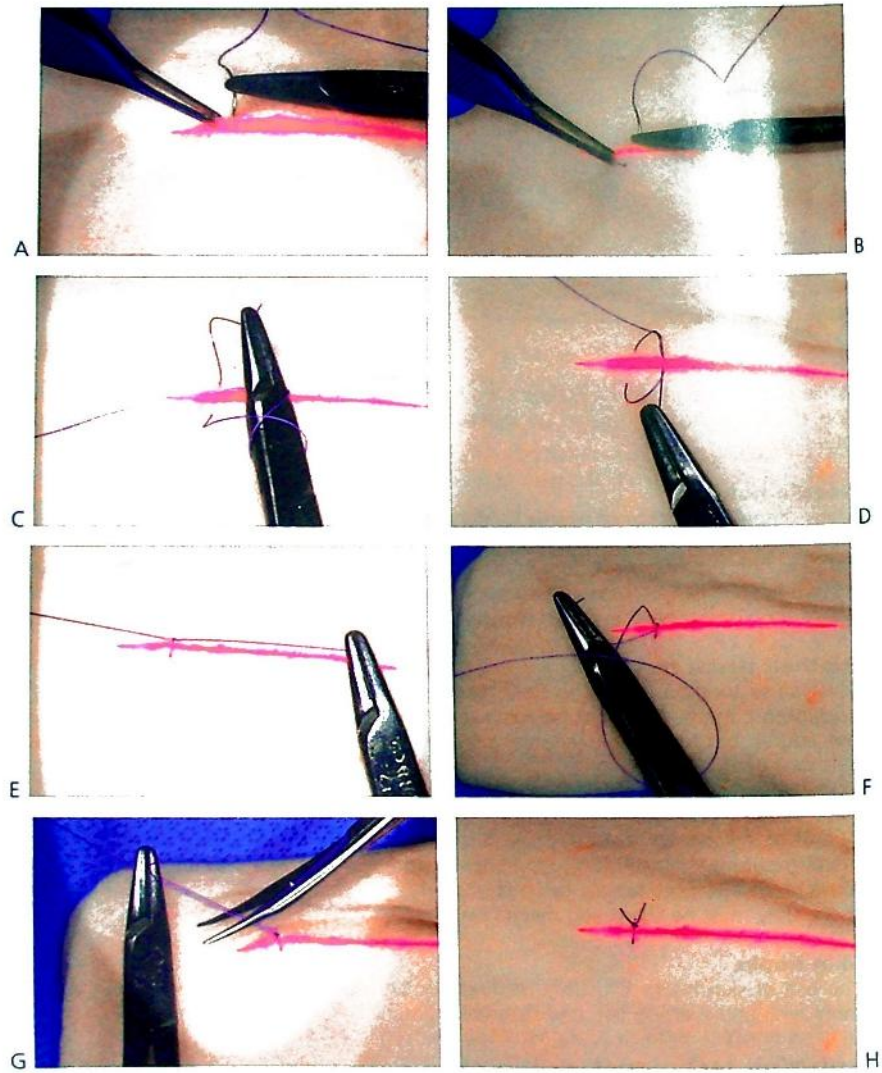


Figure 8.9 Technique for simple interrupted suture. (A,B) Each side of the flap is held and everted with toothed tissue forceps as the needle is passed in an arc from one side of the wound to the other. (C,D) A double throw knot is made and is tightened and locked by pulling on the long (or needle) end of the suture (E). Avoid pulling on the short end (held by the needle holder), as this will create a long tag end. (F) A reverse throw knot is made and the suture is cut short with fine scissors (G). (H) Note that the knot is sitting on tissue and not overlying the wound.

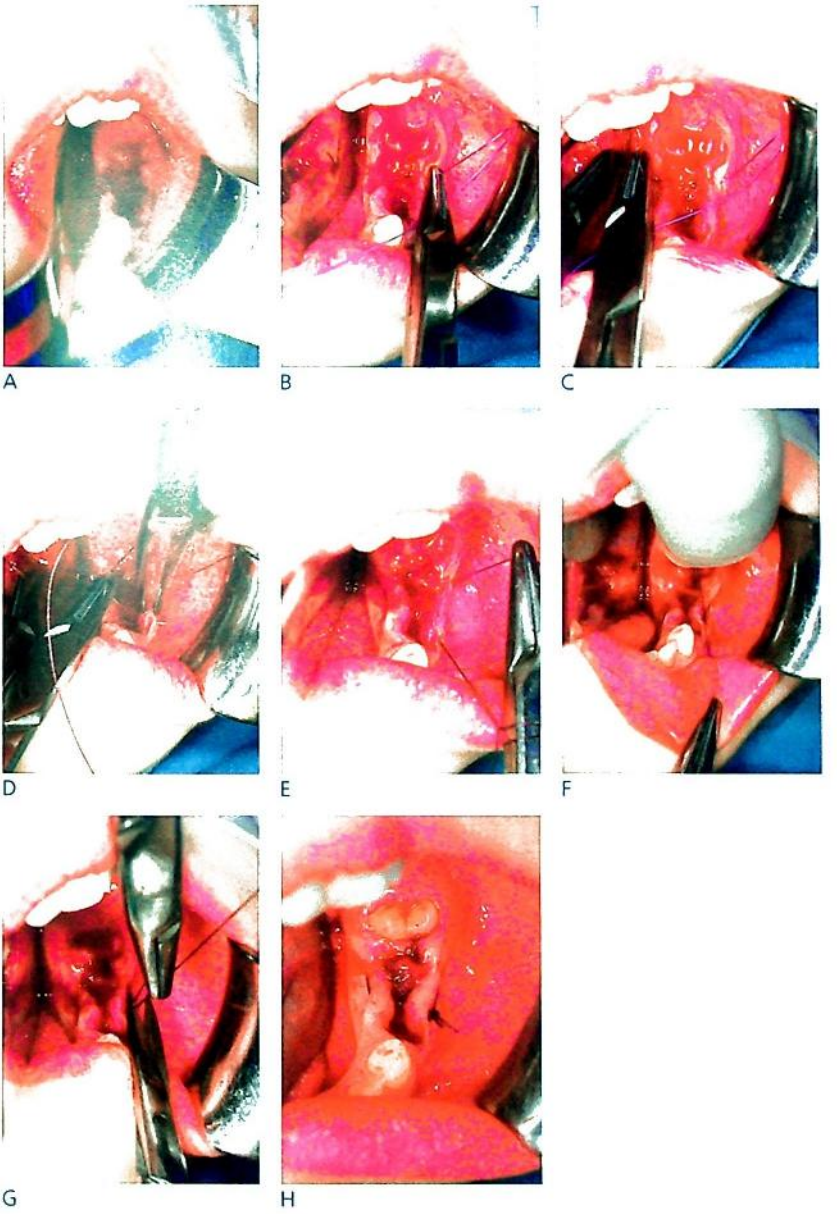


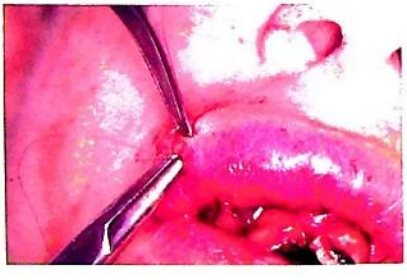
Figure 8.10 Horizontal mattress suture. (A) A large wound is left following the extraction of teeth 74 and 75. (B) The suture needle is passed through the interdental papilla on the buccal through to the lingual side (C). Approaching from the lingual, the needle is then passed back to the buccal through the more mesial papilla (D). (E,F) The suture is tied on the buccal side and cut leaving short tag ends (G). The final appearance showing good repositioning of the flaps and compression of the socket (H).

- **Vertical mattress** – This is frequently used to evert skin margins or in deep muscle closure in the lip.
- **Haemostatic** – This suture crosses over the tooth socket and can help retain packs for haemorrhage control.
- **Continuous** – An interlocking continuous suture is used for long wounds, particularly in the buccal vestibule. It has the disadvantage that if there is a break at any point, then the whole wound may open.
- **Subcuticular** – This is a form of running suture where a skin suture is hidden below the epidermis.

Clinical Hints

- Prior to suturing a site, examine it thoroughly for tooth fragments. If in any doubt, radiograph the site.
- Remove any jagged, damaged and necrotic soft tissues tags and freshen up old wound margins with a scalpel.
- Suture with adequate stress and tension achieving complete haemostasis.
- Leave suture ends short if using resorbable materials, but longer if the material is to be removed. Very short tag ends often become buried and are difficult to remove.
- Remove skin sutures on the face within 5 days to prevent scarring of puncture sites (Figure 8.11).

A



B

C

Figure 8.11 (A) When closing skin lacerations, it is essential that the key suture (the positioning suture) is at the vermilion border (B). A fine monofilament non-resorbable suture such as 6-0 nylon is used to minimize scarring (C).

Surgical removal of supernumerary teeth or impacted canines

Identification of the tooth

- Identify the tooth indicated for removal prior to surgery. It is good practice to confirm with the surgical assistant which teeth are to be removed by noting them on the radiographs and then on the patient.
- Most maxillary supernumeraries lie in the palate or in the midline between the central incisors. Midline inverted, conical supernumeraries often lie adjacent to the anterior nasal spine and are best removed via a labial approach. Tuberculate teeth are commonly palatal and inferior to the central incisor.
- Supernumeraries in the premolar region lie lingual and inferior to the premolars.
- Canines may be impacted buccally or palatally, although invariably, the root apex lies in close approximation to the floor of the sinus.

Radiology

- Panoramic radiographs.
- Palatally impacted canine crowns will appear to be larger on panoramic films.
- Tube-shift techniques.
- CT and cone-beam tomography (CBCT).

Surgical technique

Anaesthesia

- Good anaesthesia is essential (Figure 8.12A). Many younger children will be managed under general anaesthesia; however, perioperative local anaesthesia is still required. A local anaesthetic with a vasoconstrictor will aid haemorrhage control at the surgical site.

Mucoperiosteal flaps

- A large flap will heal in the same amount of time as a small flap. Therefore an appropriate sized flap should be raised to ensure adequate access to remove the tooth (Figure 8.12B).
- When removing a palatal supernumerary tooth, a full thickness palatal flap should be raised at least from the distal of the canine and beyond the midline (Figure 8.12C). Preserve the architecture of the palatal interdental papillae. The neurovascular bundle should be retained if possible, but if division is necessary to extend the flap, then a haemostat can be applied and the nerve bundle sectioned proximal to the forceps.
- When the tooth is labially impacted, a labial or buccal approach is indicated. A crevicular incision preserving the interdental papilla will be aided by the use of a relieving incision to reflect the flap.

Bone removal

- Access to the tooth can be made with hand instruments such as a 3 mm bone chisel or surgical drills (Figure 8.12D). The overlying bone is normally removed with

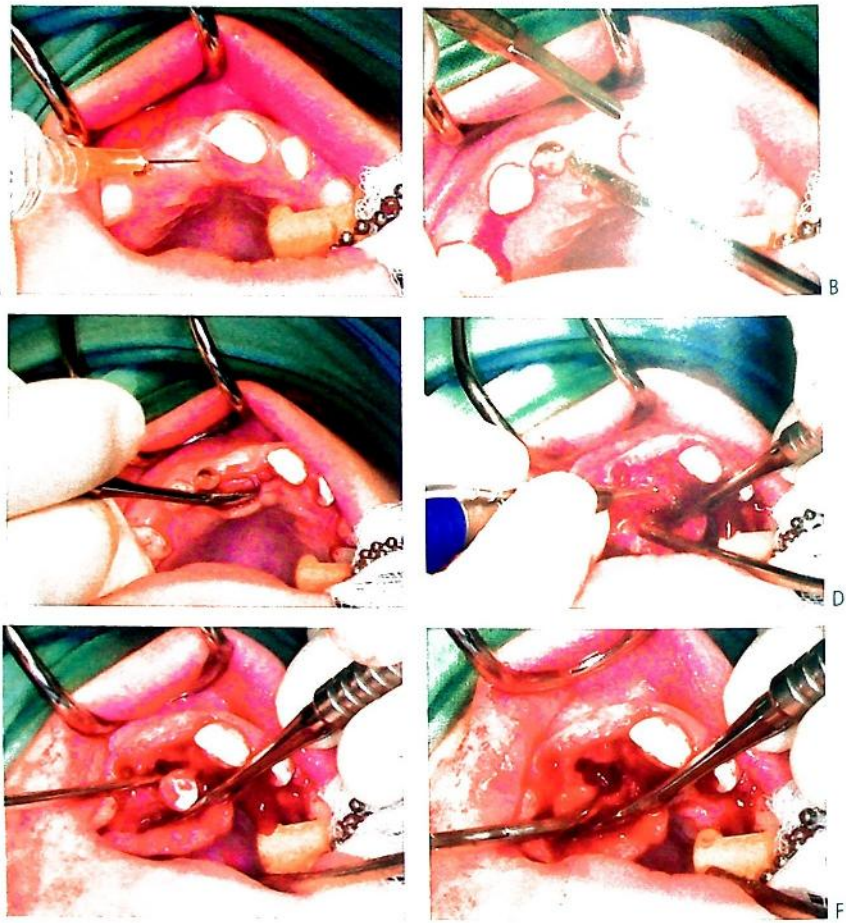


Figure 8.12 Surgical removal of a maxillary supernumerary tooth (A) Labial and palatal anaesthesia. (B) Crestal incision from at least the distal of the canine to the contralateral side. (C) Elevation of a full-thickness mucoperiosteal flap. (D) Removal of bone with bur. Note how the flap is protected by the periosteal elevator. (E) Exposure of the supernumerary and elevation with a Warwick James elevator. (F) Removal of the supernumerary tooth follicle with a pair of haemostats.


G

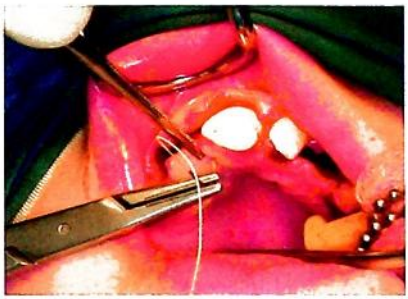

H

Figure 8.12 *Continued* (G) The socket is thoroughly irrigated and in this case, bone overlying the impacted upper right central incisor is removed to encourage eruption. (H) Closure of the flap with a resorbable suture.

a round bur and then the crown is more fully exposed by guttering around the crown to enable the application of an elevator.

Tooth removal

- Apply an appropriate elevator to the root to deliver the tooth (Figure 8.12E). The tooth should be luxated without placing any additional force on the adjacent teeth. Surgeon's fingers should support the adjacent teeth.
- Occasionally, sectioning of the tooth may be required to facilitate removal. Never attempt to section a tooth without first elevating and establishing some movement within the socket.
- Remove the dental follicle carefully with a pair of haemostats or gentle curette with Mitchell's trimmer (Figure 8.12F).

Closure

- Irrigate the socket and operating site copiously, checking for any bony spicules or jagged margins (Figure 8.12G).
- Smooth off the bony margins with a bone file or bone rongeurs.
- Close the flap with an appropriate suture material (see above). Ensure complete haemostasis is achieved (Figure 8.12H).

Postoperative care

- Provide adequate analgesia perioperatively and postoperatively. Depending on the amount of bone removal and the size of the bony defect, postoperative antibiotics are often not required. If antibiotics are required, a single perioperative administration is preferable.
- Give good clear oral hygiene instructions.
- Arrange an appropriate postoperative recall appointment.

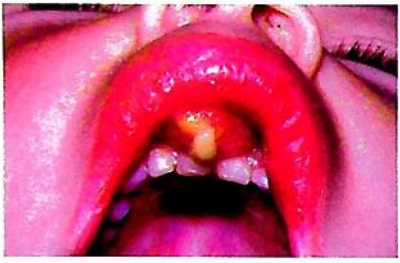


Figure 8.13 Any collection of pus requires drainage, whether that occurs spontaneously or surgically.

Incision and drainage of abscess

Any collection of pus requires drainage (Figure 8.13). Fortunately, children usually attend the dentist (or doctor) early with odontogenic infections that have spread to involve the fascial planes of the face and typically, these present as a cellulitis. When treated inappropriately with repeated antibiotics and without removal of the cause (i.e. extraction of the offending tooth), or with particularly virulent organisms, then an abscess may develop. An abscess is a collection of pus within a cavity. An abscess will not resolve by itself and pus will track to the most dependent point and in the case of head and neck infections; extraorally or between tissue planes.

This may be life-threatening and any posterior spread of pus from a tooth in the upper arch may spread from the canine fossa to the antrum, the pterygopalatine fossa, the orbit, the cavernous sinus and the brain. A submandibular abscess may spread to the floor of mouth, the buccal spaces, the pterygomandibular space, the parapharyngeal spaces and neck and ultimately the mediastinum.

The following cases represent surgical emergencies and require urgent and immediate care and/or referral:

- A floor of mouth swelling, particularly those that have crossed the midline.
- Dysphagia or respiratory obstruction.
- Trismus.
- A fluctuant enlarging swelling in the head and neck.
- A enlarging swelling associated with acute fever, particularly a spiking temperature.

Clinical presentation

Cellulitis

- A hard, brawny swelling.
- Diffuse and tender.
- Warm to touch.

Abscess

- A soft, warm and painful swelling, usually fluctuant.
- Usually circumscribed, may be very well localized in the mouth or more diffuse if extraoral.

Surgical technique

Remember that such infections are serious and potentially life-threatening and prompt referral to an experienced surgeon is warranted.

Anaesthesia

Obtaining adequate anaesthesia may be difficult, so block injections are preferable. Where there is a significant swelling either intra-orally or extra-orally, general anaesthesia will be required to adequately manage and protect the airway and to undertake the procedure.

Intra-oral (Figure 8.14)

- Protect the airway with gauze from pus or irrigating solutions that may go down the throat.
- Extract the tooth.
- Raise a flap and allow drainage of subperiosteal pus.
- An incision into the buccal or labial sulcus may also be required to establish drainage of any pus above periosteum.

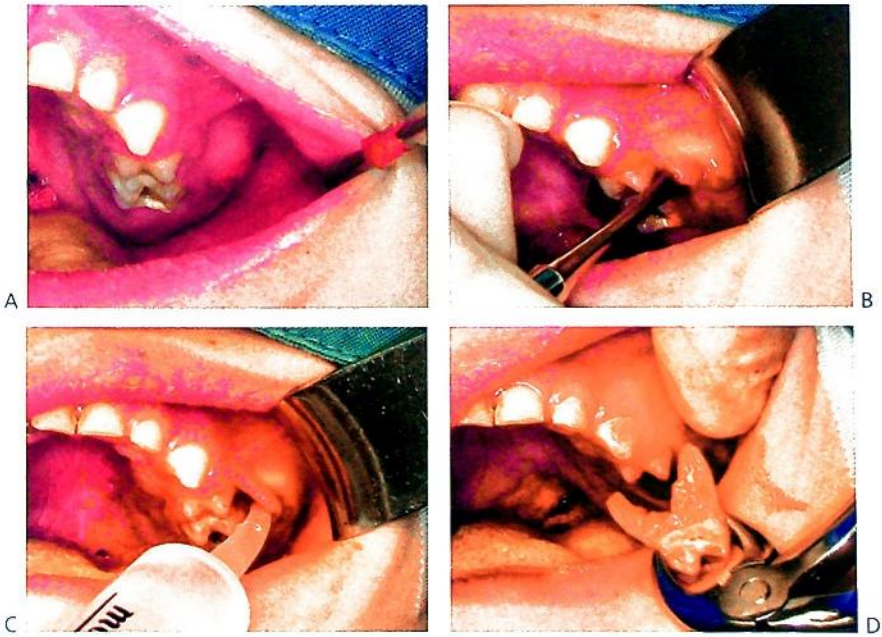


Figure 8.14 Management of an intraoral abscess. (A) A large fluctuant swelling associated with tooth 65. (B) Elevation of a flap to drain subperiosteal pus. (C) Irrigation of the abscess cavity. (D) Extraction of the offending tooth.

- Irrigate with copious sterile saline or a 50:50 mixture of povidine iodine and water?
- Suturing a drain through the incision may be required to maintain drainage.

Extra-oral (Figure 8.15)
Extra-oral drainage is typically only required for mandibular swellings.

- Prepare and drape the skin.
- Incise over the most dependent point of the abscess with the No.15 blade. Hilton's method is used to incise into the abscess along any skin folds and along a line inferior to (if possible) the lower border of mandible. The incision must avoid the

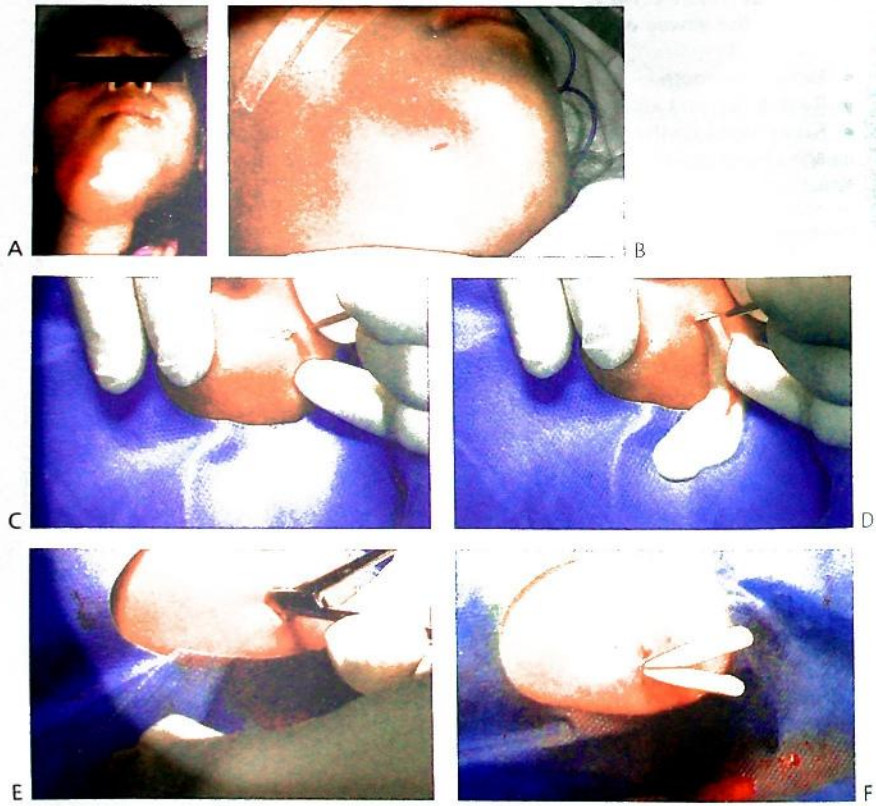


Figure 8.15 Drainage of large extra-oral abscess. (A) This child is acutely unwell and has a large collection of pus in the submandibular space with dysphagia. (B,C) The swelling is fluctuant and an incision is made into the abscess. (D,E) Drainage is established with a pair of haemostats opening into the cavity and exploring the tissue space. (F) The cavity is kept patent with a flexible drain.

marginal branch of the facial nerve, the facial artery and the lower lobe of the parotid gland.

- Enter the abscess cavity with a pair of haemostats, opening the beaks of the forceps to bluntly dissect and establish a flow of pus. It may be necessary to investigate other tissue spaces such as the sublingual, submasseteric or pterygomandibular spaces.
- Take a sample of pus for culture and antibiotic sensitivity testing.
- Copiously irrigate the cavity.
- Suture a drain into the depth of the cavity and place a dressing over the skin.

Lingual frenotomy (Figure 8.16)

A lingual frenotomy (simple cutting of the frenulum) is a procedure indicated in those infants where a significant tongue-tie is affecting breast-feeding. A lactation consultant or speech pathologist must assess attachment and feeding practices in order to determine the need for a frenotomy. It is normally performed on babies from birth to 4 months of age. Local anaesthesia is usually not required.

Breast-feeding problems associated with ankyloglossia include:

- Difficult attachment onto the breast.
- Prolonged feeding times.

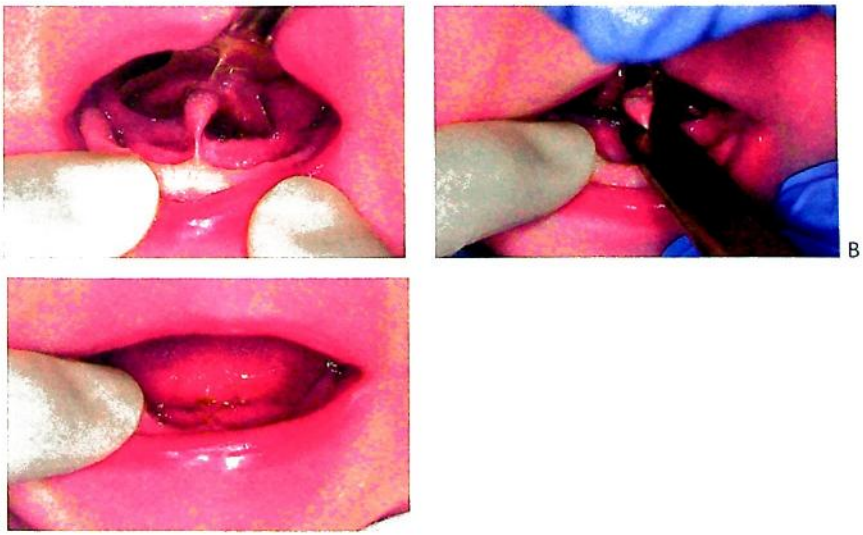


Figure 8.16 Lingual frenotomy. (A) The tongue is retracted. (B) An incision is made in the frenum with blunt-ended dissecting scissors. (C) There is minimal bleeding and the infant can commence breast-feeding immediately.

- Frequent feeding.
- Nipple pain or damage.
- Recurrent mastitis.
- Low weight gain or failure to thrive.

Bottle-feeding problems associated with ankyloglossia include:

- Clicking sounds made by the tongue during feeding.
- Poor saliva control and drooling.
- Swallowing of air while feeding.
- An inconsolable 'colicky' child.

A lingual frenotomy is simple and quick with few complications. The frenum is usually a very fine translucent tissue in babies, although clinicians should be aware of the risk of a small amount of bleeding and possible postoperative infection. To minimize the risk of infection, parents are advised to sterilize/disinfect any nipple shields, pacifiers and bottles adequately.

Clinical procedure

- The infant is wrapped (swaddled) to minimize movement.
- A Lorenz retractor/grooved director is used to retract the ventral surface of the tongue and to stretch the lingual frenulum.
- Blunt-ended scissors are used to release the lingual frenulum taking care not to injure the submandibular ducts or the ventral surface of the tongue. The cut is made superior to Wharton's (submandibular) duct extending posteriorly but NOT involving muscle. The frenum may also be released using a soft tissue laser.
- Once the frenum is released, the baby is immediately placed on the breast/bottle to begin feeding. Postoperative feeding helps to comfort the baby and assists in haemostasis.
- Once haemostasis is achieved, the baby can be discharged.

Reports indicate that this simple procedure leads to successful breast-feeding in most cases.

Lingual frenectomy (Figure 8.17)

A frenectomy is normally carried out under local anaesthesia in older children and under general anaesthesia in younger children. Frenectomy involves the surgical incision of the frenum, establishing haemostasis and suturing of the wound. In cases where there is a very short frenum and the floor of the mouth is shallow, a *Z*-plasty is performed sometimes.

Clinical procedure

- Local anaesthesia is administered into the tip of the tongue and the floor of the mouth on either side of the frenum.
- The tongue may be secured with a large stay suture through the dorsal surface to mobilize, retract and control the tongue.

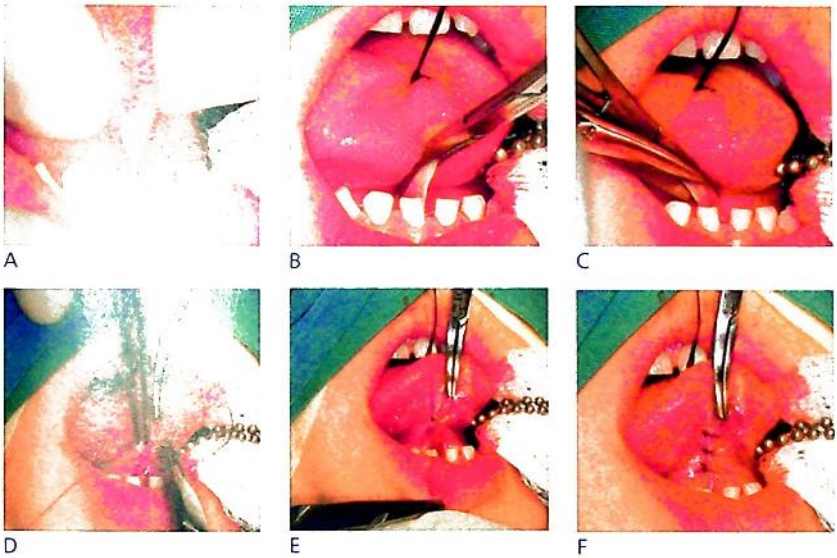


Figure 8.17 Lingual frenectomy. (A) A severe tongue tie with the insertion of a short frenum into the tongue tip. (B) A stay suture stabilizes the tongue and a haemostat is placed from the insertion of the frenum to a point in the floor of the mouth superior to the submandibular duct orifice. (C) The frenum is cut using the haemostat as a guide. (D,E) Closure of the wound with a 4-0 resorbable suture. (F) Haemostasis and final closure.

- A small curved haemostat is clamped parallel to the tongue from the insertion of the lingual frenulum at the tongue tip to a point at the greatest depth and most inferior aspect of the tongue. Iris scissors are used to cut along the jaws of the haemostat, releasing the frenum. A small transverse (horizontal) incision at the base of the haemostat allows for an extension of the incision and lengthening of the base of the tongue.
- The haemostat is then removed and the surgical site is closed with 4-0 resorbable sutures on the ventral surface of the tongue. Do not place sutures in the floor of the mouth as it leads to scarring.
- Postoperative instructions should include analgesia if required. Tongue-tie exercises as prescribed by the speech pathologist are commenced 2–3 days postoperatively.

Biopsy of soft tissue lesions (Figures 8.18, 8.19)

A definitive diagnosis of soft tissue lesions can only be made following histopathological examination, however biopsy procedures in children are not without potential

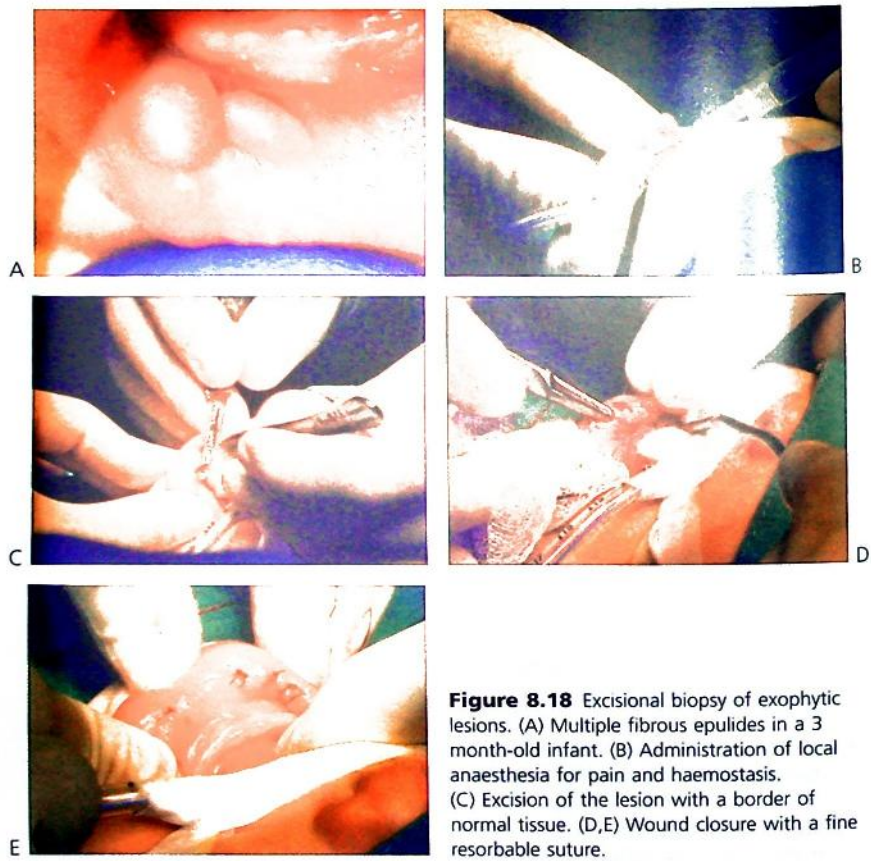
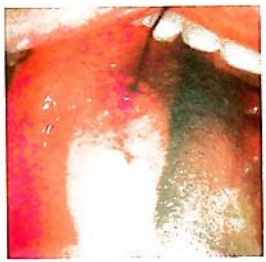


Figure 8.18 Excisional biopsy of exophytic lesions. (A) Multiple fibrous epulides in a 3 month-old infant. (B) Administration of local anaesthesia for pain and haemostasis. (C) Excision of the lesion with a border of normal tissue. (D,E) Wound closure with a fine resorbable suture.

complications. Consideration must therefore be given as to how the procedure is to be performed, with younger children usually requiring general anaesthesia. There is a risk of damage to adjacent structures, possible scarring and the excessive removal of tissue. Fortunately, life-threatening pathology in the oral cavity of children is rare and there may be little benefit to the patient in removing tissue, simply to confirm the diagnosis of a benign condition. Therefore if a malignancy or other serious condition is suspected, then the child must be referred to a clinician who is able to manage or treat the patient appropriately.

- An excisional biopsy is recommended for small lesions to completely excise the lesion and to confirm the diagnosis. The biopsy must include a border of normal tissue.



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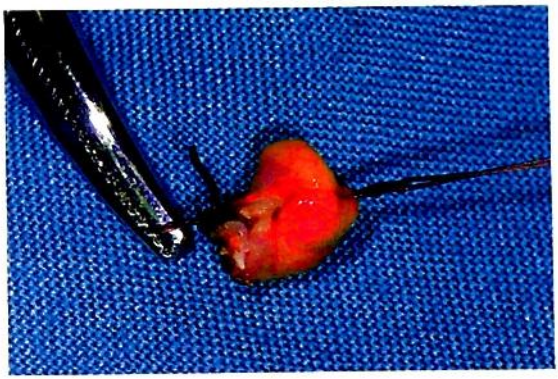

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Figure 8.19 (A) In this case, a suture has been placed through the lesion to mobilize it and another (B) around the base in case of haemorrhage. The excised lesion is then sent for histopathological examination. (C) The operculum overlying the erupting 46 tooth has been removed and haemorrhage has been controlled with diathermy.

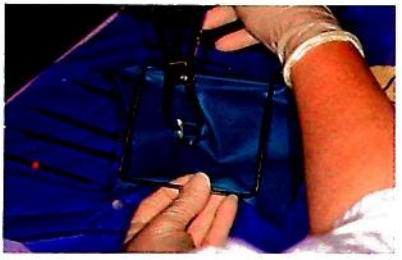
- An incisional biopsy is performed on larger lesions prior to complete resection. Incisional biopsies must include the most representative areas of the lesion together with a border of normal tissue to allow study of the margins.

Surgical procedure

- Adequate anaesthesia is essential. While block anaesthesia may be beneficial, infiltration around the lesion aids haemostasis and provides a dry operating field.
- Elliptical incisions surrounding the lesion and tapering to meet at a point under the lesion are placed using a scalpel and No.15 blade.
- Tissue is carefully dissected out and placed in an appropriate reagent, usually buffered formalin, for transfer to the pathology lab.
- Primary closure is preferred and the biopsy site is sutured with a 4/0 resorbable suture.
- It is essential that the histopathological report is reviewed and the patient followed-up with appropriate treatment.

Placement of a rubber dam (Figures 8.20, 8.21)

The use of a rubber dam in restorative procedures is invaluable in restorative dentistry. While there is reluctance by many clinicians to use a rubber dam, once the technique has been mastered, it becomes a simple and time-saving procedure.



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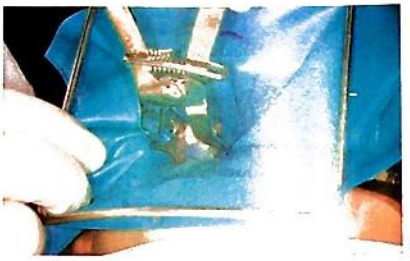

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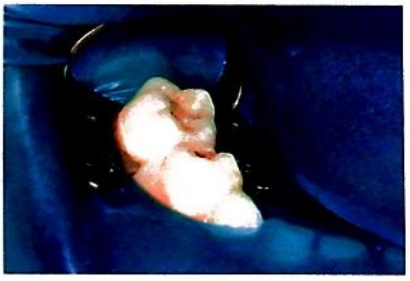
C

Figure 8.20 Placement of a rubber dam over a single tooth. (A) The rubber dam, frame, a winged clamp and clamp forceps are arranged and placed over the tooth in one step (B). (C) The rubber is then lifted from the wings with a flat plastic instrument to seal the tooth.



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B

Figure 8.21 (A) The split dam technique where a large hole (or two holes joined together) is stretched from the most distal clamped tooth to the mesial of the canine. (B) In the operating room, often two arches can be isolated in this manner.

Advantages

- Protection of the airway from aspiration of instruments, materials or medicaments.
- Provides retraction and protection of soft tissues.
- Improved infection control.

Improves efficiency due to maintenance of a dry field

- Superior access and visibility.
- Patient compliance.
- Enhanced gas control with inhalation sedation (N_2O).

Modern adhesive restorative materials do not tolerate moisture contamination and stainless steel crowns can be easily aspirated or ingested by accident in a reclining child patient.

Procedure

When applying a rubber dam, it can be referred to as a 'raincoat for the tooth'. Clamps can be called a 'tooth ring' and introduce the idea that 'it hugs the tooth tightly'. It is important not to place the clamp on the gingiva, as this often causes bleeding and unnecessary discomfort.

Clamps

- Remember to always secure the clamps with dental floss in order to minimize the risk of ingestion/aspiration.
- The No.13A clamp is ideal for quadrant 1 and 3 (odd numbered quadrants) and the No.12A for quadrants 2 and 4. A No. 9 anterior clamp is perfect for anterior strip crowns.
- The larger circumference of the clamp should be directed towards the buccal aspect. Place the clamp on the tooth surface and guide it down the tooth until it is well seated below the bulbosity of the crown. Winged clamps are useful if the rubber dam, clamp and frame are to be placed in one step.

Placement (Figure 8.20)

- Local anaesthesia is generally required when the clamp is placed below the gingival margin sits below the gingival margin or the beaks of the clamp impinge into the interdental papilla. A standard buccal infiltration, supplemented with an intra-papillary injection is usually sufficient.
- A split-dam technique is frequently used in children using one hole that extends from the second molar (or first permanent molar) to the mesial of the canine. This can be achieved by placing three adjoining holes in the rubber dam sheet and allows quadrant dentistry to be completed with ease (Figure 8.21A).
- Alternatively, cut one hole for each tooth to be isolated and then stretch the rubber dam sheet over the clamp and individual teeth. Dental floss can be used to guide the rubber through the contact point. Rubber strips or tubes can aid with retention when dealing with open contacts. Finally, stretch the rubber dam onto a frame.
- The whole rubber dam apparatus can be removed by removing the clamp.
- For procedures under general anaesthesia, the double-dam technique is ideal as it reduces the treatment time (Figure 8.21B).
- Use latex-free rubber dam in case of latex allergy.

9 Trauma management

Angus C Cameron, Richard P Widmer, Paul Abbott, Andrew A C Heggie, Sarah Raphael

Introduction

The management of dentoalveolar trauma in children is distressing for both the child and the parent (Figure 9.1), and often difficult for the dentist. However, trauma is one of the most common presentations of young children to a paediatric dentist. The patient's emergency must be the dentist's routine. The child should be carefully assessed regarding treatment needs before commenting to parents because many cases are not as bad as they first appear. Initial reassurance to both parent and child is of great value. Trauma not only compromises a previously healthy dentition but may also leave a deficit that affects the self-esteem and quality of life, and it commits the patient to lifelong dental maintenance.

Guidelines for management of dental injuries

The International Association of Dental Traumatology published guidelines in 2007 with recommendations for the management of dental injuries based on a review of the literature and consensus opinions. These guidelines provide views on care based on the published evidence and the opinions of professionals who practise in this field. As is stated in the guidelines, there is no guarantee of success and as further research is published, clearly the recommendations in these guidelines will be updated. The practitioner should be aware that clinical judgement is still required, depending on the presentation of each case. An internet-based set of guidelines has also been developed and sponsored by the International Association for Dental Traumatology (www.dentaltraumaguide.org). This is a free website that practitioners can use to quickly and easily access information about how to manage dental injuries, the prognosis of the teeth and many other details.

Aetiology

Most injuries are caused by falls and play accidents. Luxation injuries to upper anterior teeth predominate in toddlers because of their frequent falls during play and attempts at walking. Injuries are generally more common in boys. Blunt trauma tends to cause greater damage to the soft tissues and supporting structures, whereas high-velocity or sharp injuries cause luxations and fractures of the teeth.

Figure 9.1 The presentation of a child with trauma is distressing for parent and child. The child in other instances may be oblivious to what has happened and is happily playing in the surgery.

Table 9.1 Causes of maxillofacial injuries in children

	Injuries occurring at each age group (%)			Total injuries (%)
	0–5 years	**5–10 years**	**10–15 years**	
Falls	50.1	32.8	17.1	43.2
Play accidents	39.5	43.5	17	17.7
Motor vehicle accidents	31.9	44.1	24	17.4
Sporting accidents	9	29.5	61.5	8.3
Dog bites	63.3	29.6	7.1	6.4
Fights and assaults	–	21.9	78.1	1.4
Child abuse	80	20	–	1
Others				4.6

Source: Hall (1994).

Predisposing factors

- Class II division 1 malocclusion.
- Overjet 3–6 mm – double the frequency of trauma to incisor teeth compared with 0–3 mm overjet.
- Overjet >6 mm – three-fold increase in the risk.
- The study summarized in Table 9.1 by Hall (1994), from the Royal Children's Hospital in Melbourne, shows that falls and play accidents account for the majority of injuries. Lam and others (2008) also reported that dental trauma in an Australian rural population was most frequently the result of falls, accidents while playing and while participating in sports. Importantly, although accounting for only 1% of all injuries, over 80% of child abuse occurs in the very young child.

Frequency

- 11–30% of children suffer trauma to the primary dentition. This figure may represent up to 20% of all injuries in preschool children.
- 22% of children suffer trauma to the permanent dentition by the age of 14 years.
- Male : female ratio is 2 : 1.
- Peak incidence is at 2–4 years and rises again at 8–10 years.
- Upper anterior teeth are the most commonly involved teeth, especially the central incisors (in both the primary and permanent dentitions).
- Usually only a single tooth is involved, except in cases of motor vehicle accidents and sporting injuries.

Dog bites account for a significant number of injuries and every year several children are killed by dogs. It is common that the dog is known to the child and it cannot be stressed too highly that children must be supervised when around even the most timid of animals.

Child abuse

Child abuse is defined as those acts or omissions of care that deprive a child of the opportunity to fully develop his or her unique potential as a person either physically, socially or emotionally. There are four types of child abuse:

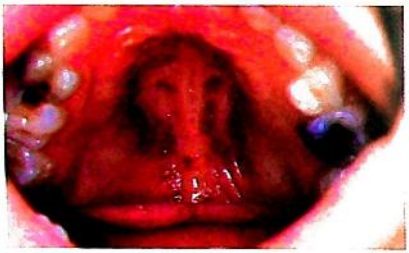
- Physical abuse.
- Sexual abuse.
- Emotional abuse.
- Neglect.

Dental neglect is the knowing failure of a parent or guardian to access treatment of orofacial conditions for a child. When left untreated, such conditions may adversely affect a child's normal growth and development.

The true incidence of child abuse and neglect is unknown, and although there is increasing awareness and reporting, professionals are still reluctant to deal with it. The first step in preventing abuse is recognition and reporting. Dentists are in a strategic position to recognize and report mistreated children because they often see the child and parent/caretaker interacting during multiple visits and over a long period of time.

The orofacial region is commonly traumatized during episodes of child abuse (Figure 9.2). Injuries that do not match the given history, bruising of soft tissue not overlying bony prominences or injury that takes the shape of a recognizable object, and multiple injuries of different ages, may be the result of non-accidental trauma. Bite marks in children represent child abuse until proven otherwise. The characteristics and diagnostic findings of child abuse, and the protocol of reporting such cases, should be familiar to the dentist so that appropriate notification, treatment and prevention of further injury can be instituted.

Whenever injuries are inconsistent with the history, the patient must be investigated for abuse. There is a legal obligation in some countries or states to report the suspicion of child abuse or sexual assault. In Australia, child abuse teams are available at all paediatric hospitals or through the departments of family and community services.



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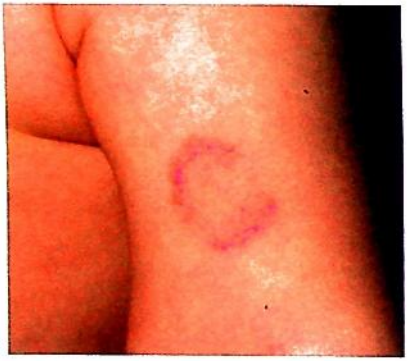

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Figure 9.2 (A) Child abuse caused by sexual assault by a family member. Commonly the perpetrator is known to the child. (B) Bruising on the arm of an infant discovered during routine dental treatment under general anaesthesia. (C) An 18-month-old infant who was bitten by an older child. Good photographic records are required and the wounds should not be washed until specimens for DNA testing of saliva are taken. The child assault team will organize appropriate input from social workers, paediatricians and the police, if necessary. The dentist should also be aware of the legal requirements for recording of evidence (i.e. standardized photography with measuring scale).

History

As dental injuries may become the subject of litigation or insurance claims, a thorough history and examination is mandatory. Where possible, injuries should be photographed. An accurate history gives important information regarding:

- Status of the dentition at presentation.
- Prognosis of injuries.
- Other injuries sustained.
- Medical complications.
- Possible litigation.

Questions to ask

- When, where and how did the trauma occur?
- Were there any other injuries?
- What initial treatment was given?
- Have there been any other dental injuries in the past?
- Are current immunizations up-to-date?

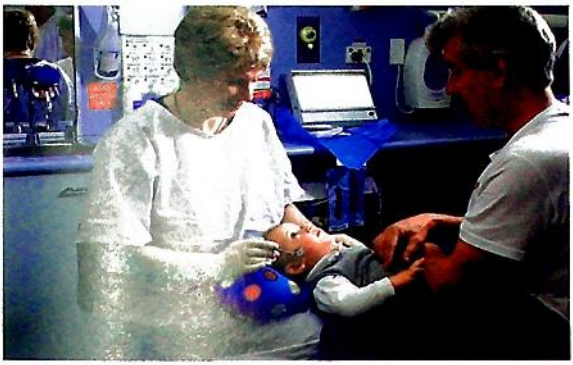


Figure 9.3 One of the most convenient ways to examine young children is with the child's head in the dentist's lap. The child can see the parent, who gently restrains the arms. This gives an excellent view of the upper teeth and jaws, where most trauma occurs.

Examination

Examination should be undertaken in a logical order. It is important to examine the whole body, as the patient may present first to the dentist and other injuries may also have occurred (Figure 9.3 and see Chapter 1).

Trauma examination and records

- Extra-oral wounds and palpation of the facial skeleton (Figure 9.4).
- Injuries to oral mucosa or gingivae.
- Palpation of the alveolus.
- Displacement of teeth.
- Abnormalities in occlusion.
- Extent of tooth fractures, pulp exposure, colour changes.
- Mobility of teeth.
- Reaction to pulp sensibility tests and percussion.

Assessment of cranial nerves involved in facial trauma

I	Olfactory	Olfaction
II	Optic	Vision
III	Oculomotor	Movements of the globe
IV	Trochlear	Superior rectus
V	Trigeminal	Muscles of mastication
VI	Abducent	Lateral rectus
VII	Facial	Muscles of facial expression
VIII	Vestibulocochlear	Hearing and balance
IX–XII	Hypoglossal	Tongue, pharyngeal and shoulder function

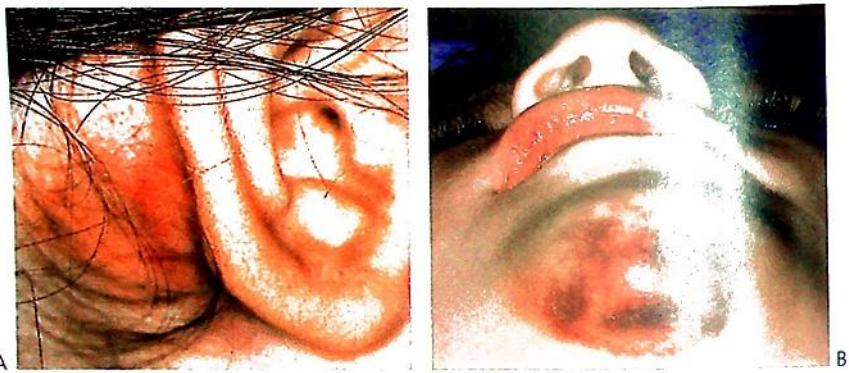


Figure 9.4 (A) The 'battle sign', or bruising of the mastoid region, is associated with a base-of-skull fracture. Examination must include all areas of the head and neck, which often requires parting the hair to detect lacerations and bruising. (B) Bruising is a collection of blood which will fall to the most dependent point. The chin-point ecchymosis shown here is often associated with gingival degloving, laceration and/or a mandibular fracture.

Head injury

Closed head injury is the most common cause of childhood mortality in accidents. Between 25% and 50% of all accidents in children aged up to 14 years involve the head. If there is any suggestion that a head injury has been sustained, the child should be immediately medically assessed, preferably in a paediatric casualty department.

Signs of closed head injury

- Altered or loss of consciousness.
- Bleeding from the head or ears.
- Disorientation.
- Prolonged headache.
- Nausea, vomiting, amnesia.
- Altered vision or unilateral dilated pupil.
- Seizures or convulsions.
- Speech difficulties.

Dentoalveolar injuries may take second place if there is central nervous system involvement. As a head injury may be long-lasting, initial management and replantation may be possible in consultation with other medical practitioners. If there is any loss of consciousness, hourly neurological observations should be commenced. The Glasgow Coma Scale is commonly used in Accident and Emergency departments to assess the severity of head injury and prognosis (see Appendix H).

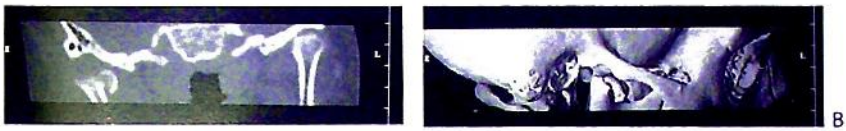


Figure 9.5 Use of computed tomographic reformatting to visualize fractures to the mandibular condyle. (A) Coronal section showing an intracapsular fracture with medial displacement of the condylar head due to the pull of medial pterygoid muscle. (B) 3-dimensional reconstruction showing the degree of displacement of the condylar head following chin-point trauma.

Investigations

Radiographs

The request for radiographs should only be made after a thorough history and clinical examination. There is great value in using extra-oral films in young children, e.g. panoramic radiographs. In the very upset or difficult child, it may be the only way that some clinical information can be gained in the acute phase of management.

When taking intra-oral radiographs, several periapical images from different angulations should be taken for each traumatized tooth, plus an occlusal radiograph. These are especially important to determine the presence of root fractures and tooth luxations. As a baseline, all traumatized teeth should be radiographed to assess:

- Stage of root development.
- Injuries to the roots and supporting structures.
- Degree and direction of luxation or displacement.

Guide to prescription of radiographs

Dentoalveolar injuries

- Anterior maxillary occlusal or anterior mandibular occlusal.
- Panoramic radiograph.
- True lateral maxilla for intrusive luxations of primary anterior teeth.

Condylar fracture (Figure 9.5)

- Panoramic radiograph, closed and open mouth.
- Cone-beam tomography (CBCT) or computed tomography (CT) scan.
- Reverse Townes view.

Mandibular fracture

- Panoramic radiograph.
- True mandibular and anterior mandibular occlusal (for parasymphysial fractures).
- Cone-beam tomography (CBCT) or computed tomography (CT) scan.
- Lateral oblique (this is rarely used today except in cases where a CT is unavailable).

Maxillary fractures

- CT scan.

New imaging technologies have superseded older-style views such as the lateral oblique, the reverse Townes' and Waters' (occipitomental 30°) projections. While such radiographs may be valuable in particular cases, contemporary practice indicates the use of fine-slice CT or cone-beam tomography for an accurate assessment of middle third fractures in children.

Pulp sensibility tests

Pulp sensibility tests provide an essential baseline measure of the pulp status. It is common that the initial responses at presentation may be inaccurate; however, it is important that results are recorded for later comparison. The results of pulp sensibility tests performed immediately following trauma are also very useful predictors of the prognosis of traumatized teeth. Teeth that do respond to these tests are more likely to recover than teeth that do not respond. Young children often find it difficult to discriminate between the touch of the tester and the actual stimulus itself, so the clinician must be aware of the possibility of false results. In cases that are difficult to diagnose, isolation of individual teeth under rubber dam may be required.

Pulp sensibility tests are used to help assess the status of the pulp. Previously and erroneously termed 'vitality tests', the contemporary terminology (i.e. sensibility tests) stresses the fact that the neural and vascular components of the pulp tissue need individual consideration. Sensibility is defined as the 'ability to respond to a stimulus' – which is what is tested with thermal and electric pulp tests. It is important to understand that a tooth may not respond to a thermal or electric pulp test but it may still have an intact blood supply. Such discrimination of the health of the pulp is important in planning treatment.

Thermal pulp tests

Responses to cold stimuli give the most reliable and accurate results in children (even with immature teeth). The carbon dioxide (dry ice) pencil is regarded as the most convenient. Cold sprays may also be used but they are not as accurate or as reliable. Cold tests have the advantage that assessment of the pulp is possible while temporary crowns and splints are in place.

Electric pulp tests

Electric pulp tests may give a graded response to stimuli. When using these instruments, the current should be slowly increased so that sudden painful stimulation of the tooth is avoided.

Percussion

There are two reasons to percuss teeth:

- Tenderness to percussion gives information about the extent of damage to the periapical tissues and the periodontal ligament. Tenderness to percussion can also indicate that a tooth has been concussed or subluxated. The percussion of luxated teeth will usually be painful but there is no need to percuss teeth that are obviously luxated on visual examination.
- The sound in response to percussion, especially during follow-up examinations, is also an important indicator of the presence of ankylosis.

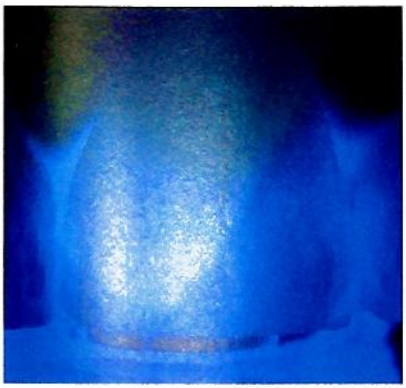


Figure 9.6 Transillumination to detect enamel infarctions.

Transillumination (Figure 9.6)
This is an extremely useful, non-invasive technique to assess the presence of cracks and/or fractures, and subtle alterations in crown colour which may indicate a change in the pulp status.

Other considerations in trauma management

Having carefully assessed the patient, the only treatment necessary may be to reassure the child and parent, and to discuss the various possible sequelae such as pulp necrosis, resorption, infection and facial swelling.

Fasting requirements

If the patient requires extensive work under general anaesthesia, it is important to check fasting details. A child over 6 years of age must be fasted for at least 6 hours without solids or liquids. Children under the age of 6 years must be fasted for 6 hours without solids and 2 hours without liquids.

Immunizations

If a child has sustained an injury that involves contamination of the wound with soil, especially from a farm area, their tetanus immunization status must be determined. If the child has completed their normal immunization schedule, under normal circumstances boosters are not required.

Maxillofacial injuries

Fractures of the facial bones are uncommon in children and account for less than 5% of all maxillofacial fractures. Consequently, few surgeons have extensive experience in this area and the management of these cases must embody an understanding of the implications of such injuries for the growing child (Figure 9.7).

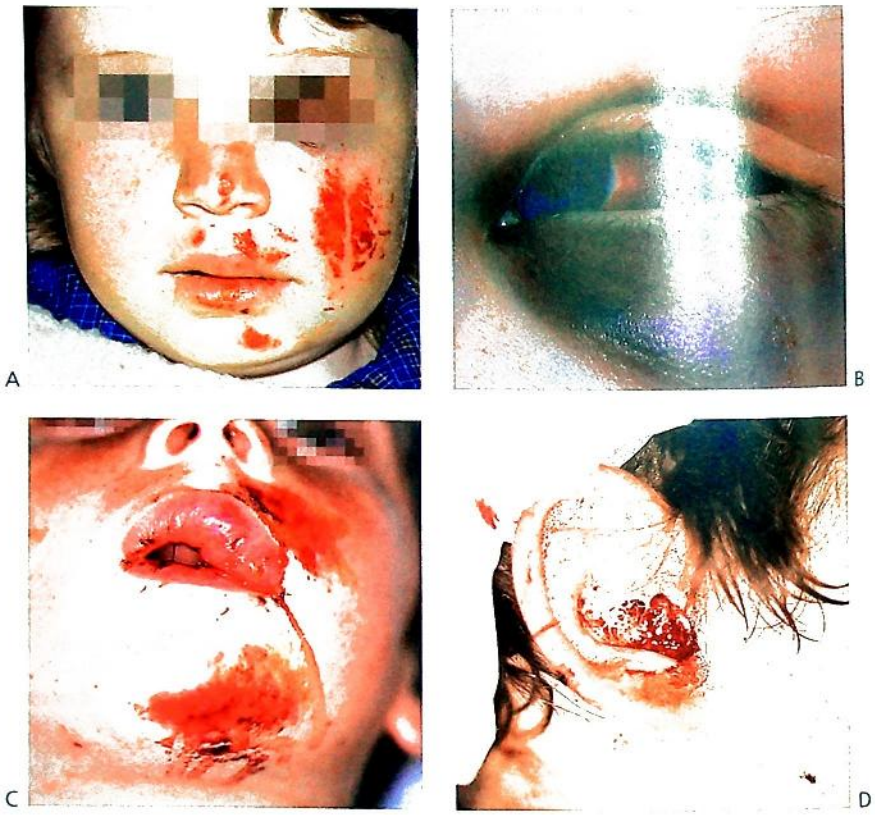


Figure 9.7 (A,B) This girl fell from a Tarzan rope on to her face. There is extensive ecchymosis and subconjunctival haemorrhage. While many of the signs of a zygomatic fracture are present, the immaturity of the frontozygomatic suture allowed for some displacement and there was no fracture evident. (C,D) Many children suffer chin-point trauma and it is important to check the mandibular condyles. This boy sustained a right subcondylar fracture. There was bleeding from the external meatus as the condyle had perforated the anterior wall of the meatus. Under no circumstances should the ear be suctioned because the ossicles may be removed if the tympanic membrane is ruptured.

Principles of management

Management of maxillofacial trauma is complicated in a child by the unerupted dentition, anxiety, growth considerations and the common association of closed head injuries that may delay definitive treatment. The use of internal fixation such as miniplates and screws must be undertaken with care due to the potential for damaging

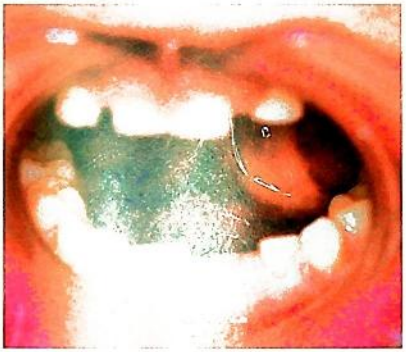


Figure 9.8 The sublingual haematoma is pathognomonic for a fractured mandible in the symphysis or in the canine region of the body.

developing tooth buds. Intermaxillary fixation, occasionally in conjunction with transosseous wires is well tolerated in children. While arch bars may be used as dental fixation, silver cap splints may be still used effectively. With accurate reduction, fixation and immobilization, fractures unite within 3 weeks. Prophylactic antibiotic treatment and strict oral care must be maintained. Non-union or fibrous union is rare.

Fractured mandible

Most mandibular fractures involve the parasymphysial region (due to the position of the unerupted canine) and the condylar neck either in isolation or in combination.

Clinical signs

- Pain, swelling.
- Trismus.
- Occlusal discrepancies.
- Stepping at the lower border.
- Sublingual/buccal ecchymosis (Figure 9.8).
- Chin asymmetry.
- Paraesthesia of the mental nerve distribution.

Management

- Reduction and fixation with arch bars and wire or elastic intermaxillary fixation.
- Splints may be attached with glass ionomer or black copper cement or retained with circum-mandibular wiring.

Condylar fractures

Fractures of the mandibular condyle are likely to be under-diagnosed in children and comprise up to two-thirds of all mandibular injuries. This injury usually results from trauma to the lower border of the chin. If a subcondylar fracture occurs, the condylar head is usually displaced antero-medially by the action of the lateral pterygoid muscle. Depending on the displacement of the fragments and the compensatory posturing of the mandible, there may be deviation of the chin to the affected side or there may be

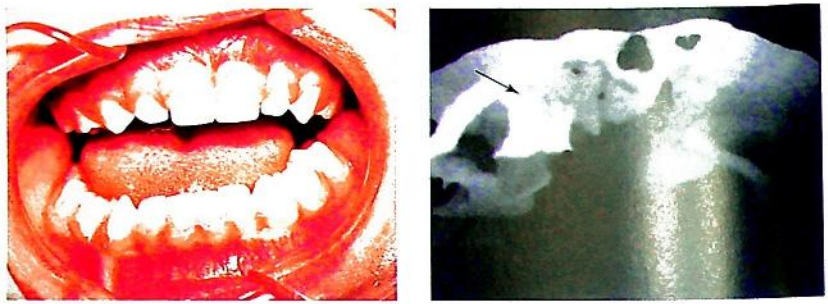


Figure 9.9 (A,B) Mandibular asymmetry caused by a dislocation of the left condyle after play equipment fell on this young girl. Imaging of these injuries can be difficult and in this case a CT scan was performed with 3-dimensional reconstruction to detail the injury. The CT demonstrates the dislocation, with the condylar head (arrow) anterior to the articular eminence and lying under the zygomatic arch. As is common with these injuries in children, an intracapsular fracture-dislocation is present, which remodelled itself without treatment. Normal function was achieved within 6 months.

no occlusal disharmony. Bleeding from the external meatus may occur due to perforation of the anterior wall of the auditory canal by the condylar head (see Figure 9.7C,D). Bleeding or discharge from the ear should be investigated by an otolaryngologist but suctioning of the external meatus is contraindicated due to the potential for disturbance of the ossicular chain should there be a perforation of the tympanic membrane. Displacement of the condylar head into the middle cranial fossa has been reported but is a rare event.

Management

Treatment is almost always conservative with a short period of rest followed by active movement to prevent temporomandibular joint ankylosis. Fractures involving telescoping of the condyle and distal fragment may be successfully treated with functional appliances for 2–3 weeks or longer, allowing better remodelling. Bilateral subcondylar fractures may result in significant displacement and an anterior open bite. A short period of intermaxillary fixation with posterior bite blocks to distract the fragments may be indicated where there has been gross displacement of the condylar head, or in severe cases of bilateral condylar fracture.

As the condylar neck is relatively broader in the child with a greater volume of cancellous bone, fractures of the articular surface are more common than in the adult. In cases of intracapsular fracture (Figures 9.5, 9.9), follow-up over many years will enable detection of any growth disturbance. Should there be a limitation of opening or frank ankylosis, early intervention to mobilize and reconstruct the mandible is recommended.

Maxillary fractures

Middle-third fractures are rare in children and usually present with other severe craniomaxillofacial and head injuries. Mid-facial fractures tend not to follow the typical

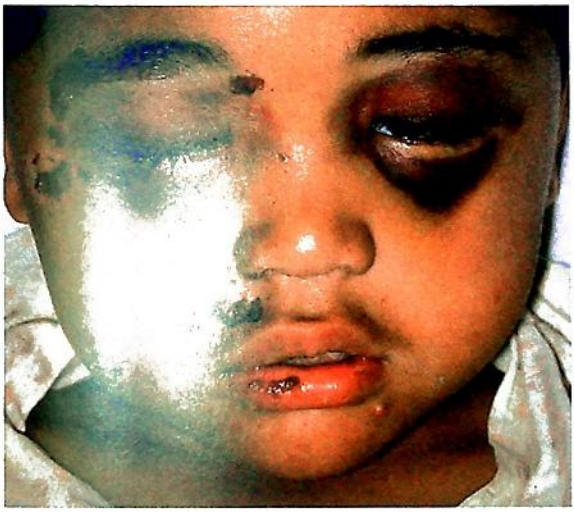


Figure 9.10 Middle-third fracture of the face in a child involved in a motor vehicle accident. Note the bilateral periorbital ecchymosis and swelling resulting in closure of the eyes. Despite the appearance, there was only minimal displacement of the maxilla, although external fixation was required to reduce the depressed nasal fracture.

'Le Fort lines', as the immature skeleton results in more greenstick and incomplete fractures.

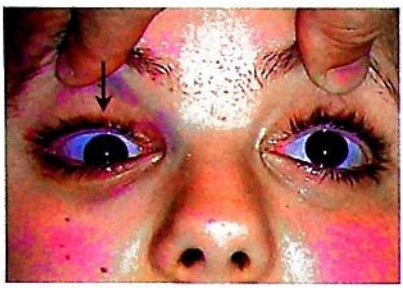
Orbital floor 'blow-out' and orbital roof 'blow-in' are seen and may require urgent reduction, particularly where orbital contents, such as fascia and muscle are trapped, thus preventing normal ocular movements.

Clinical signs

- Facial swelling and periorbital ecchymosis (Figure 9.10).
- Periorbital surgical emphysema.
- Subconjunctival haemorrhage, with no posterior limit.
- Diplopia.
- Nausea, vomiting and photophobia often occurs with inferior rectus muscle entrapment.
- Orbital rim contour deformaties.
- Mid-facial mobility.
- Infraorbital paraesthesia (Figure 9.11).
- Cerebrospinal fluid rhinorrhoea and epistaxis.
- Occlusal discrepancies.

Management

- Conservative management is usual unless there is significant displacement of the mid-facial complex. In this situation, open reduction with or without semi-rigid internal fixation is required.



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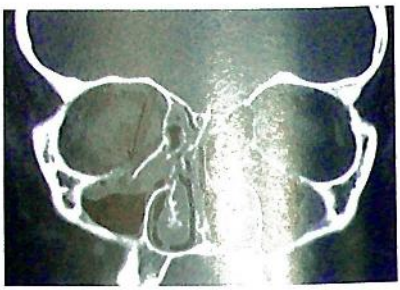

B

Figure 9.11 (A) Limitation of upward gaze associated with right orbital floor 'blow-out' fracture. (B) Coronal CT scan demonstrating 'trapdoor' orbital floor fracture with tissue entrapment.

- Simple maxillary fractures are managed with cap splints or arch bars with intermaxillary fixation.
- 'Trap-door' orbital floor fractures with soft tissue entrapment is a surgical emergency and must be explored as soon as possible to reduce the orbital contents.

Sequelae of fractures of the jaws in children

Closed head injury

Children who sustain middle-third facial injuries usually have concomitant head injuries. Head injuries occur in 25% of cases of facial trauma. These children spend extended periods in intensive care units, may undergo personality changes, suffer post-traumatic amnesia and may have episodes of neuropathological chewing.

Tooth loss

Approximately 10% of children who sustain fractures of the jaws will also have loss of permanent teeth.

Developmental defects of enamel

In addition to the damage caused by displacement of primary teeth into the crypts of permanent successors (see 'Sequelae of trauma to primary teeth' later in the chapter), unerupted teeth in the line of jaw fractures may also be damaged. Defects may include:

- Hypoplasia or hypomineralization of enamel.
- Dilaceration of crown and roots.
- Displacement of the developing tooth within the bone.
- Arrest of tooth development with pulp canal calcification.

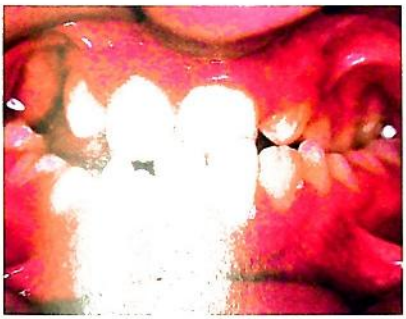

Figure 9.12 Maxillary hypoplasia and growth retardation, following in a child, 8 years after sustaining a middle third fracture.

Intra-articular damage to the temporomandibular joint

There is always a risk of ankylosis of the temporomandibular joint after significant displacement of the condylar head, intracapsular fracture or a failure to achieve early mobilization of the joint. Treatment of the ankylosis involves condylectomy and joint reconstruction with a costochondral graft in later childhood.

Growth retardation

Maxillary (Figure 9.12) and mandibular growth retardation may occur following major trauma. Significant scarring of soft tissues and/or tissue loss may inhibit jaw growth. Mandibular asymmetry with antegonial notching may occur on the affected side after subcondylar fracture. The key to management is to correct asymmetries early to avoid secondary maxillary deformity.

Luxations in the primary dentition

General management considerations

There is general agreement that most injuries to the primary dentition can be managed conservatively and heal without sequelae. As a general rule, either leave and observe or extract the tooth.

Immunization

If the child is not fully immunized then a tetanus booster is required: tetanus toxoid 0.5 mL by intramuscular injection.

Antibiotics

Unless there are significant soft-tissue or dentoalveolar injuries, antibiotics are not usually required. Antibiotics are prescribed empirically as a prophylaxis against infection, but they are not a substitute for proper debridement of wounds. All drugs should be prescribed according to the child's weight (see Appendix E).

Luxations

Up to 2 years of age, the most common injuries to the primary teeth are luxations involving displacement of the teeth in the alveolar bone.

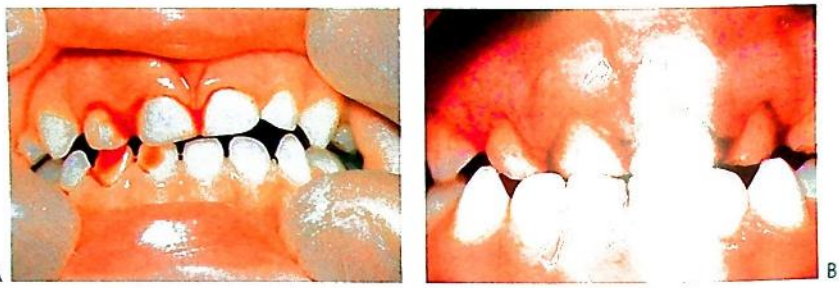

Figure 9.13 (A) Subluxation of the upper right incisors with minimal displacement. (B) Palatal luxation of the upper incisors resulting in an occlusal interference. These teeth can be repositioned by digital pressure, only to relieve the interference. Further anterior movement may damage the permanent teeth.

Concussion and subluxation (Figure 9.13)

Concussion is an injury to the tooth and ligament without displacement or mobility of the tooth. Subluxation occurs when the tooth is mobile but is not displaced. Both involve minor damage to the periodontal ligament. Teeth with these injuries will be tender to percussion. There will be haemorrhage and oedema within the ligament, but gingival bleeding and mobility only occurs if the teeth have been subluxated.

Management

- Periapical radiographs as baseline.
- Soft diet for 1 week.
- Advice to the parents of possible sequelae, such as pulp necrosis and infection.
- Individualized follow-up.

Intrusive luxation

Intrusive injuries (Figure 9.14) are the most common injuries to upper primary incisors. Newly erupted incisors often take the full force of any fall in a toddler. There is usually a palatal and superior displacement of the crown, which means that the apex of the tooth is forced away from the permanent follicle.

Management

- If the crown is visible and there is only minor alveolar damage – leave tooth to re-erupt.
- If the whole tooth is intruded – extract.

Clinical Hint

Where the apex of the primary tooth has perforated the labial alveolar cortical plate, the tooth should be extracted. The decision on whether to extract or to allow re-eruption is very much a clinical one, and is based on the presentation of the injuries and the assessment of the child. More severe injuries, involving alveolar bone and gingivae, often necessitate extraction.

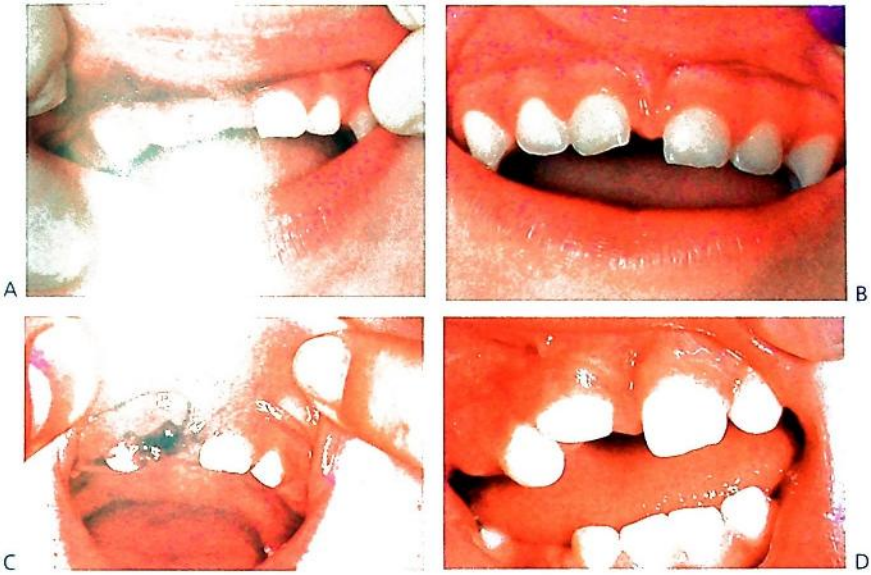

Figure 9.14 (A) Many intruded primary teeth will re-erupt (B). (C) The decision as to whether or not to extract is dependent on the degree of displacement and direction of displacement of the crown and the amount of gingival and alveolar damage. An intrusive luxation of the upper right central incisor in a 12-month-old child. Note the displacement of the gingiva, indicating that the tooth has not been avulsed. (D) The tooth partially re-erupted within a month.

Extrusive and lateral luxation (Figure 9.15)

Treatment is dependent on the mobility and extent of displacement. If there is excessive mobility the tooth should be extracted.

Avulsion (Figure 9.16)

- Avulsed primary teeth should **not** be replanted.
- Replanting an avulsed primary tooth may force the blood clot in the socket, or the root apex itself, into the developing permanent tooth. The other main reason is lack of patient cooperation. There are cases in which the parent or caregiver has replanted the tooth and it appears to be stable; in these cases the tooth could be left *in situ* but it should be splinted to prevent it being inhaled or swallowed.
- Unless significant soft-tissue damage is present, antibiotics are not required.
- Splinting of primary teeth may be difficult in young, traumatized children and if successfully placed, the splint must then also be removed later when the child may be less compliant.

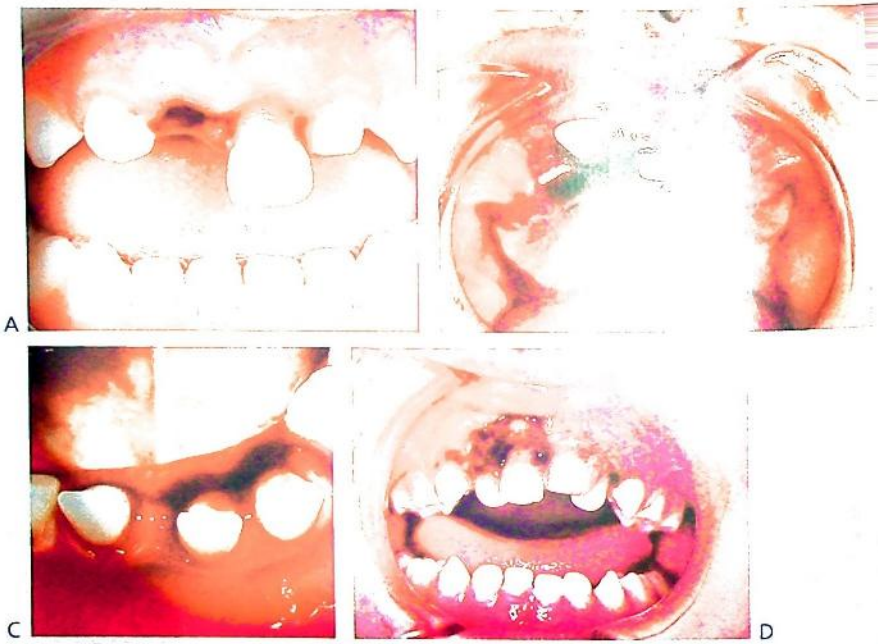

Figure 9.15 (A) Extrusive luxations result in increased mobility necessitating removal of the tooth. There is no indication for repositioning such teeth. (B) Similarly, lateral luxations of this magnitude require removal of the tooth. xation. (C) This child presented 1 week following lateral luxation of the lower primary incisors with continued gingival oozing. He was subsequently diagnosed with Christmas disease (factor IX deficiency). (D) Gross displacement of all upper anterior teeth with gingival degloving and loss of the labial plate. This child had the displaced teeth extracted, and debridement and suturing of the gingiva under general anaesthesia.

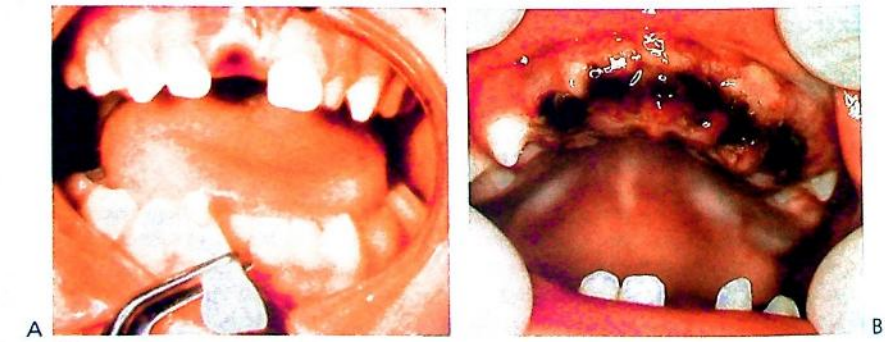

Figure 9.16 (A) There is almost no indication for the replantation of an avulsed primary tooth. There is more risk of damage to the permanent tooth than there is benefit gained by replacing the tooth. (B) A child involved in a motor vehicle accident resulting in six avulsed primary teeth, but with surprisingly very little dentoalveolar damage. A chest radiograph was required to ensure that no teeth were swallowed or aspirated.

Fractures of primary incisors

Crown fractures not involving the pulp (Figure 9.17A)
Unlike the permanent dentition, primary teeth are more commonly displaced rather than fractured. Enamel and dentine fractures may be smoothed with a disc and, if possible, cover the dentine with glass ionomer cement or composite resin. Paediatric strip crowns are often useful. A possible sequel is pulp necrosis and/or grey discolouration. If the pulp does become necrotic, then it may subsequently become infected, leading to an apical abscess.

Complicated crown/root fractures (Figure 9.17C–E)
More commonly, fractures of primary teeth involve the pulp and extend below the gingival margin. Commonly, there are multiple fractures in individual teeth. In these cases, it is not feasible to adequately restore the tooth and therefore it should be extracted. Often the fracture is not immediately evident, but the child may present several days after the trauma with a pulp polyp which is causing separation of the fragments. Such a proliferative response is a protective mechanism and is not painful. Management of such a case should be by extraction of the tooth.

Management

- Most of the discomfort results from the movement of fractured pieces of tooth that are still held by the gingiva or periodontal ligament. In the emergency management of such teeth, these loose tooth fragments should be removed.
- The remaining tooth can be extracted when convenient. This may necessitate the use of sedation or a short general anaesthetic.
- If a small piece of root remains in the socket after a fracture, it may be safely left *in situ* where it will be resorbed as the permanent tooth erupts. It is important to keep parents adequately informed in these situations.

Root fractures (Figure 9.16B)
As mentioned above, when children fracture primary incisors, there is usually a complex crown/root fracture that extends below the gingival margin and extraction is indicated. Isolated root fractures are uncommon. Normally, no treatment is necessary for primary incisors with horizontal or transverse root fractures. If, at regular review, the pulp shows signs of necrosis and infection, with excessive mobility or sinus formation, the coronal portion should be extracted. The apical root fragments are usually removed by resorption as the permanent tooth erupts.

Dentoalveolar fracture (Figure 9.17F)
This is more common in the mandible with the anterior teeth being displaced anteriorly with the labial alveolar cortical plate. It is often desirable to reposition the teeth with the bone to maintain the alveolar contour. This can be achieved with a thick nylon suture (2–0) passed through the labial and lingual plates of the bone. Teeth that are excessively mobile should be carefully dissected out of the sockets preserving the labial plate, which is then repositioned and sutured.

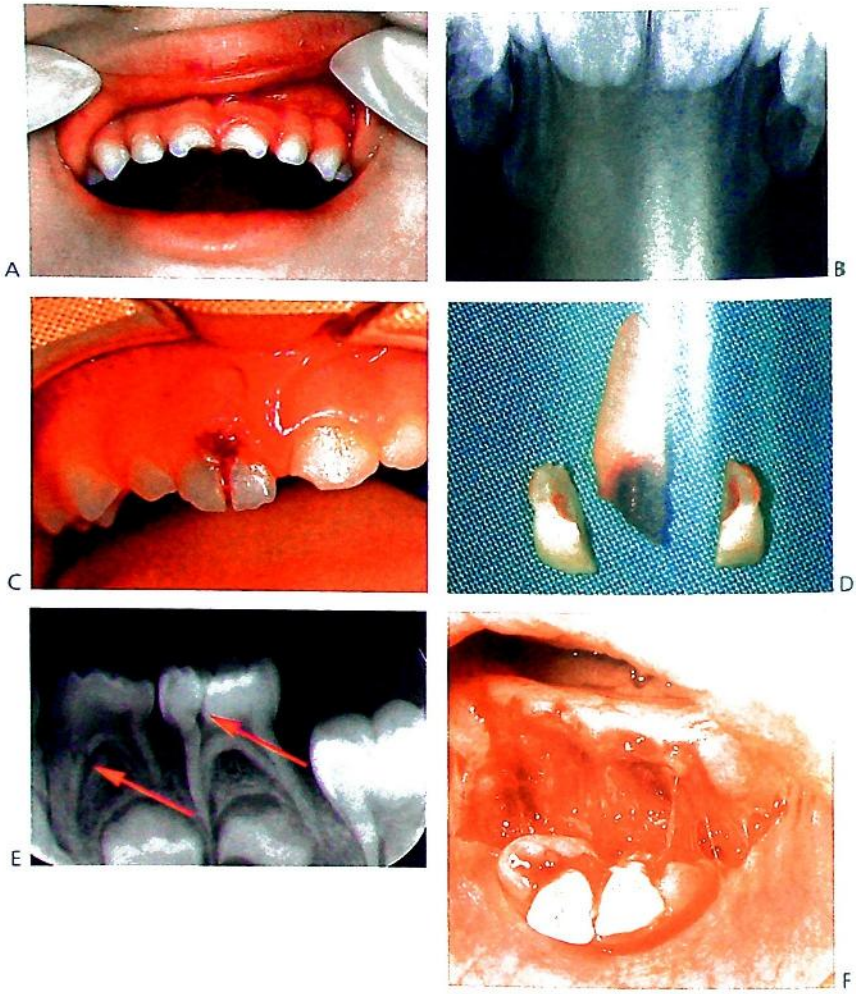

Figure 9.17 (A) Minor enamel/dentine fractures can be smoothed with a disc or left untreated. (B) Root fractures require no treatment unless the coronal fragments are excessively mobile. The pulp in the apical portions remain normal and the roots will resorb normally. (C) A complex crown/root fracture involving the upper left primary central incisor. These teeth are not suitable for restoration and need to be extracted. The extent of the subgingival fracture can be seen in (D). These teeth are often difficult to remove and care must be taken to avoid damage to the permanent teeth if using elevators or luxators to remove large fragments of root. (E) Complicated crown and root fractures of the first and second primary molars. (F) A dentoalveolar fracture in a 6-month-old infant. In these cases, it is important to reposition the bone, with or without the teeth. A thick (2–0) nylon suture passed through both labial and lingual plates can be used to provide fixation for the fragment. Teeth usually survive this trauma and there are few untoward sequelae for the permanent teeth.

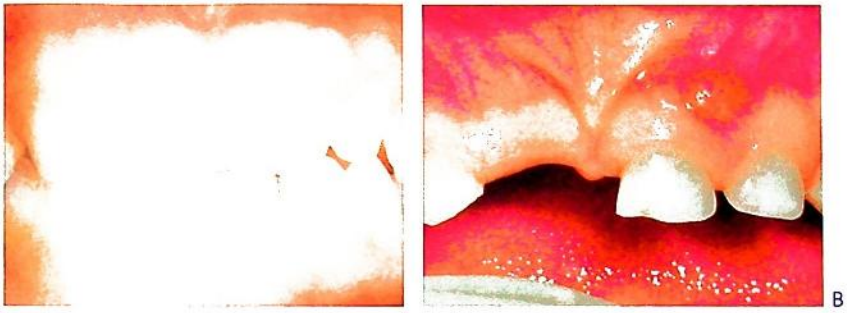

Figure 9.18 (A) Discolouration of the crown following trauma to the primary incisors. Unless an abscess is present (B), these teeth do not require treatment other than monitoring and reassurance of the parents.

Sequelae of trauma to primary teeth (Figures 9.18, 9.20)

It is important to discuss with parents the sequelae of luxated or avulsed primary incisors. Although it may be difficult to accurately predict the prognosis for the unerupted permanent teeth, parents appreciate having an idea of the possible outcomes. In cases that have been followed up in studies, up to 25% of children are left with some developmental disturbance of the permanent tooth.

Damage to the unerupted permanent dentition occurs more often with intrusive luxation and avulsion in very young children. It is important to warn parents of possible problems with permanent teeth and also to reassure them that, with modern restorative materials, minor defects are easily repaired. Sequelae in the permanent dentition depend on:

- Direction and displacement of the primary root apex (Figure 9.19).
- Degree of alveolar damage.
- Stage of formation of the permanent tooth.
- Treatment provided to the primary tooth.

Possible damage to primary and permanent teeth

- Necrosis of the pulp of the primary tooth with grey discolouration and possible infection of the root canal system followed by abscess formation (Figure 9.19).
- Internal resorption of the primary tooth.
- Ankylosis of the primary tooth. Commonly, intruded primary teeth will fail to fully erupt but will exfoliate normally. In rare cases, extraction may be required just prior to eruption of the permanent incisor.
- Hypoplasia (Figure 9.20E) or hypomineralization (Figure 9.20B) of succedaneous teeth (see also Chapter 11).

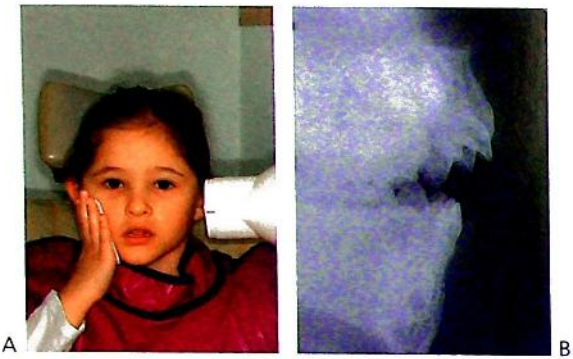

Figure 9.19 (A) Technique of taking a true occlusal maxillary radiograph. This film gives a good localization of the position of the primary root apex in relation to the central incisors. (B) The root apex is clearly visible, just underneath the anterior nasal spine, having perforated the labial plate. In this situation, damage to the unerupted permanent tooth is less likely.

- Dilaceration of the crown, or root of the permanent tooth; varies by developmental stage of the permanent tooth at the time of trauma (Figure 9.20C,D).
- Resorption of the permanent tooth germ.

Treatment options

- If the primary tooth is discoloured, but asymptomatic, no treatment is usually indicated. Masking a discoloured tooth with composite resin may be an option if aesthetics are a concern. If an abscess is present, pulpectomy (and subsequent treatment of the root canal system) or extraction is indicated.
- Hypoplasia and hypomineralization of the permanent teeth can be restored with composite resin.
- Dilaceration of the crown or root of the permanent tooth often necessitates surgical exposure and bonding of chains or brackets for orthodontic extrusion (see Chapter 11 for details of surgical procedure). Severe cases may be untreatable and such teeth may need to be removed.

Crown and root fractures of permanent incisors

Crown infractions

When there is an infraction (or crack) of the enamel, there is no loss of tooth structure. Infractions do not usually cross the dentino-enamel junction and usually require transillumination or indirect light to be identified (see Figure 9.6). However, it is impossible to determine the depth or extent of an infraction and whether it involves dentine or not.

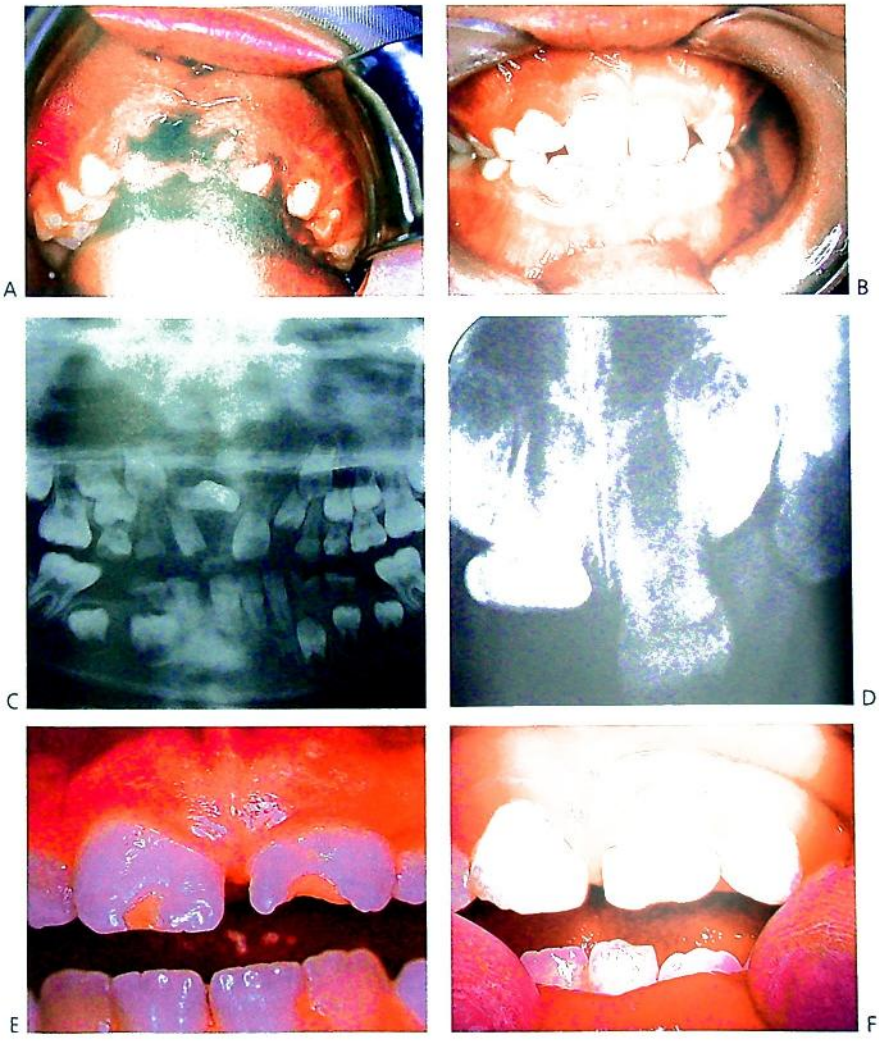

Figure 9.20 (A) It is often difficult to predict sequelae. For example, this case of severe intrusion, and alveolar disruption, has caused little damage other than mild hypocalcification of the permanent incisors (B). (C) Displacement and dilaceration of the upper-right permanent central incisor, following avulsion of the primary precursor tooth, at 18 months of age. (D) Severe dilaceration of the crown of the upper left central incisor. (E) Hypoplasia of the permanent central incisors resulting from trauma in the primary dentition. (F) Restoration of dilacerated teeth is extremely difficult, especially when the defect involves the gingival margin.

Management
- Pulp sensibility tests.
- Periapical radiographs taken from several angulations to exclude other injuries.
- Occlusal radiograph to exclude other injuries.
- Cover the infractions with two coats of light-cured resin bonding liquid as a temporary means of protecting the pulp by preventing bacterial penetration during the early healing phase.

Review
- Pulp sensibility testing after 3, 6 and 12 months.
- Periapical radiographs at each review.

Uncomplicated crown fractures

Uncomplicated crown fractures are confined to the enamel only or they may involve the enamel and dentine, but they do not involve the pulp. The most common presentation is an oblique fracture of the mesial or distal corner of an incisor.

Management
- Baseline pulp sensibility tests.
- Baseline periapical radiographs taken from several angulations to exclude other injuries.
- Occlusal radiograph to exclude other injuries.
- Enamel-only fractures – smooth over the sharp edges with a disc or restore with composite resin if required.
- Enamel and dentine fractures – cover the dentine with glass ionomer cement and then restore the crown with composite resin either immediately or at review (Figures 9.21, 9.22).

Review
- Pulp sensibility testing after 3, 6 and 12 months.
- Periapical radiographs at each review.

Clinical Hint

It is extremely important to cover the exposed dentine of permanent incisors as soon as possible. This is to prevent direct irritation of the pulp due to entry of bacteria via the dentinal tubules. Parents often save the fractured piece of a permanent incisor that can sometimes be used to restore the tooth by being bonded back onto the tooth with composite resin (Figure 9.22).

In the very immature tooth, where there is a questionable pulp exposure, an elective Cvek pulpotomy (see below) may be indicated. This will ensure normal development of the apex and prevent the need for any possible open apex endodontic procedure (apexification).

Figure 9.21 (A,B) Composite resin restoration on a proximal fracture. Retention is aided by using a long bevel over the labial surface. The dentine is protected with a glass ionomer base.

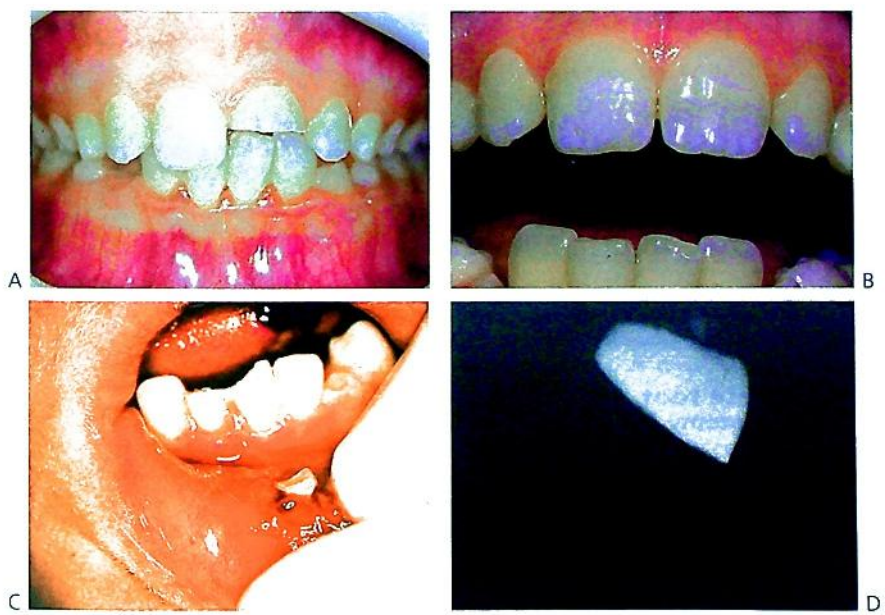

Figure 9.22 Restoration of a fractured enamel fragment by bonding the fragment back on to the tooth. (A) A chamfer or bevel is placed around the fragment and remaining crown and the dentine covered with glass ionomer cement. (B) Composite resin is then used to bond the fragment to the crown. It is often impossible to re-create the subtle hypocalcific flecks in a crown with composite resin alone; the replacement of the fractured piece is a good alternative technique if the fragment can be found. (C) Always look for fragments of tooth in the soft tissues. It is essential that they are removed at the time of the trauma, as they are extremely difficult to find once the tissues have healed. (D) Radiographs are useful in localizing tooth fragments within the lip.

Prognosis
Pulp necrosis after extensive proximal fracture:
- No protective coverage of dentine: 54%.
- With dentine coverage: 8%.

Complicated crown fractures (Figure 9.23)
- Fractures involving enamel, dentine and exposure of the pulp.
- Involves laceration of the pulp and its exposure to the oral environment and bacteria within the mouth.
- Healing does not occur spontaneously and untreated exposures will result in pulp necrosis and subsequent infection of the root canal system leading to apical periodontitis and possible apical abscess.

The time elapsed since the injury and the stage of root development will influence treatment. If the tooth is treated within several hours of the exposure, conservative management is appropriate. After several days, microabscesses may occur within the pulp, and more radical pulp amputation will be required.

Management
- Baseline pulp sensibility tests – to assess adjacent teeth for possible injury.
- Baseline periapical and occlusal radiographs taken from several angulations to exclude other injuries.
- The aim of managing the exposed pulp is to preserve the non-inflamed pulp tissue and for it to be biologically walled off by a hard-tissue barrier (Cvek 1978).

In almost all situations, if the pulp tissue can be covered with a calcium hydroxide dressing, it is possible for a dentine bridge to form over the exposed pulp. It is undoubtedly preferable to preserve the pulp rather than to do root canal treatment.

Incomplete root apex with a clinically normal pulp
Cvek pulpotomy (apexogenesis) (Figure 9.24)
The Cvek pulpotomy procedure involves the removal of contaminated pulp tissue with a clean round high-speed diamond bur, using saline or water irrigation. A non-setting

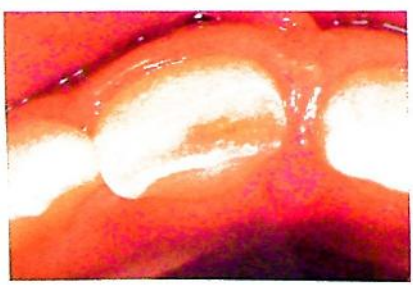

Figure 9.23 Assessment of any pulp exposure is essential, especially when the tooth is immature. The exposure of the mesial pulp horn is quite small and there is no haemorrhage, so it may be easily missed. Immediate coverage and dressing will help to prevent pulp necrosis and infection, and the subsequent need for an open apex endodontic procedure.




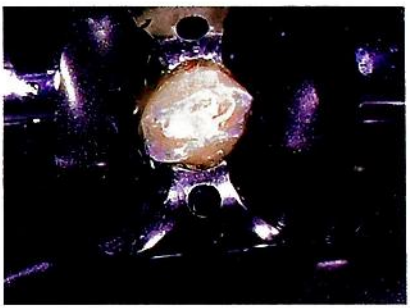

Figure 9.24 Cvek pulpotomy. (A) Traumatic pulp exposure of an upper central incisor. (B) Obtaining access to the pulp chamber with a high-speed diamond bur with copious saline irrigation. (C) Removal of 2 mm of pulp tissue to a level with no contaminated pulp tissue. (D) Placement of non-setting calcium hydroxide dressing over the pulp tissue.

calcium hydroxide paste is then placed directly onto the uncontaminated pulp tissue (see Step 5. below). The steps are as follows:

1. Administer local anaesthesia.
2. Place rubber dam to isolate the operating field – this is mandatory.
3. Remove 1–2 mm of pulp using a high-speed diamond bur, as described above.
4. Wash the pulp with saline until the haemorrhage stops. Any blood clot should then be gently rinsed away.
5. Place a non-setting calcium hydroxide paste over the remaining pulp and then cover this paste with a hard-setting calcium hydroxide cement or liner. It is essential that the calcium hydroxide is placed over pulp tissue, and not over a blood clot.
6. Place a glass ionomer cement base over the calcium hydroxide and restore the tooth with composite resin.

This technique does not need to be limited to the coronal pulp. A 'partial pulpotomy' may be performed at any level of the pulp space, as there are great benefits in preserving the apical part of the pulp in traumatized incisors.

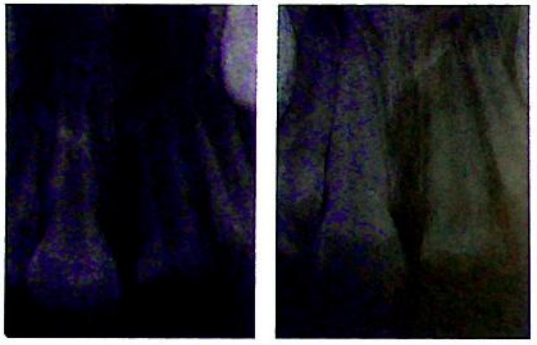

Figure 9.25 (A) Pulp exposure in an immature central incisor. (B) A Cvek pulpotomy (apexogenesis) has allowed normal root development with a dentine barrier in the crown. This significantly strengthens the root, especially at the cemento-enamel junction.

Review

- 6–8 weeks and then at 6 and 12 months with pulp sensibility tests.
- Periapical radiographs at each review to check for continued root development and narrowing of the root canal space as the root develops (Figure 9.25).

Prognosis

- Favourable pulp healing 80–96%.

Incomplete root apex with a necrotic pulp (Figure 9.26)

Pulp necrosis is unlikely to occur immediately after trauma that results in a complicated crown fracture. It is more likely to be diagnosed at follow-up examinations. If the pulp of a tooth with a complicated crown fracture becomes necrotic and infected, then removal of the pulp and subsequent root canal treatment is required. Although there is no difference in the prognosis of root canal treatment in immature teeth compared with mature teeth, the long-term survival of a tooth with an open apex may be compromised. This is caused by the thin dentine walls of the root, especially in the cervical third, and a shortened root which make the tooth susceptible to fracture during function or if there is further trauma to the tooth. Endodontic treatment of immature anterior teeth is complicated because of the inability to create an apical seat, the thin dentinal walls, and the difficulty in filling the root canal by traditional methods such as lateral compaction of gutta percha.

Management

The aim of management is to create an apical hard-tissue barrier against which the root canal filling can be placed. The formation of this apical hard tissue barrier is stimulated by using long-term intra-canal calcium hydroxide dressings (apexification).

Technique (apexification)

1. Administer local anaesthesia.
2. Place rubber dam – this is mandatory for all root canal treatment.

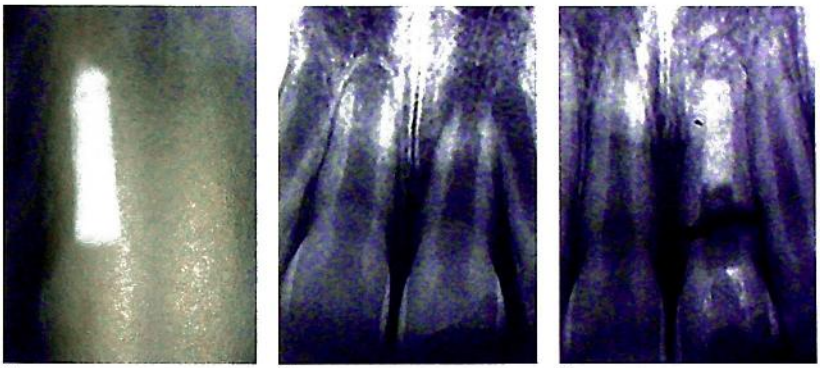

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Figure 9.26 (A) Open apex root canal treatment requiring an apexification procedure. (B,C) The long-term prognosis of these teeth is not ideal with some sustaining subsequent root fractures because of inherent weakness in the cervical region.

3. Prepare an access cavity through the palatal or lingual surface of the crown.
4. Remove any necrotic pulp tissue from the canal with a barbed broach.
5. Biomechanically prepare the canal to a level 1 mm short of the radiographic apex.
6. The canal should be carefully instrumented to completely remove necrotic tissue and debris, while also preserving as much tooth structure as possible. The apical root, being very thin, is weak and may fracture if undue pressure is exerted. Very little instrumentation of the canal walls is required.
7. Irrigate thoroughly with 1% sodium hypochlorite to dissolve pulp tissue remnants and to disinfect the root canal system.
8. Ledermix® paste should be placed as the initial dressing followed by calcium hydroxide to create a 50:50 mixture of these two medicaments. The mixture is very effective at reducing periapical inflammation, reducing pain and controlling infection within the root canal. The pastes can be inserted into the root canal using a spiral root filler in a low-speed handpiece, run at a very low speed.
9. Place a small pledget of cotton wool in the coronal pulp chamber and then place a temporary restoration in the access cavity using a temporary filling material such as Cavit, or a double-layer temporary restoration using Cavit® and IRM®.
10. After 4–6 weeks, the patient should be reviewed. If there are no symptoms or other problems, then under rubber dam isolation, the temporary filling material should be removed and the canal should be thoroughly irrigated to remove the previous dressing. After drying the canal, it should be re-dressed with a non-setting calcium hydroxide paste.
11. Compress the calcium hydroxide with a cotton wool pellet to ensure good condensation in the canal and to allow contact with the apical tissues. Another temporary restoration should then be placed in the access cavity.

12. Review the child every 3 months and change the calcium hydroxide dressing each time in the manner described above. The formation of an apical hard tissue barrier typically takes about 12 months but it may take up to 18 months. Once the barrier has formed, the canal should be filled with gutta-percha and cement. Root canal filling with gutta-percha is performed using either a warm vertical compaction technique, or lateral compaction. An impression of the apical seat may be made with heat-softened gutta-percha which is then cemented into the canal with a root canal cement. Whichever technique is used, it should be stressed that gentle pressure must be applied to avoid splitting the root or breaking the hard tissue barrier off the root and pushing it into the periapical tissues. Thermoplasticized gutta-percha delivery systems are often invaluable in these cases.
13. Remove the gutta-percha and cement from within the crown part of the tooth. Gutta-percha can be easily removed with a hot instrument and then the remainder should be vertically compacted into the coronal third of the canal while it is still warm. The access cavity should be thoroughly cleaned by wiping it out with cotton pellets soaked in alcohol to remove the root canal cement. This should be repeated 2–3 times to ensure complete removal of the cement in order to avoid discolouration of the tooth.
14. Restore the access cavity with a base of Cavit, followed by a glass ionomer cement to replace dentine and finally composite resin. The Cavit will facilitate any further access to the root canal system should it become necessary in the future.

In immature teeth, occasionally a small root apex may develop, although the pulp otherwise appears necrotic. This is caused by surviving remnants of Hertwig's epithelial root sheath. Such a situation requires no management or change to the treatment being provided for the tooth.

Review

- Review 6 months after the root filling has been completed and then annually for at least 5 years to monitor the tooth and the periapical tissues.
- Periapical radiographs at each review.
- Adjacent teeth should also be monitored in the usual manner following trauma.

Filling an open apex tooth without apexification

An alternative approach that has been advocated in recent years is the use of a material known as mineral trioxide aggregate (MTA) to fill the apical few millimetres of an open apex tooth without first having to use long-term dressings of calcium hydroxide. Although sometimes called 'MTA apexification', it is not an apexification procedure, since an apical hard tissue barrier is not formed prior to root filling the tooth. It is more accurate to consider this procedure as filling an open-ended root canal. MTA is a mixture of tricalcium silicate, dicalcium silicate, tricalcium aluminate, tetracalcium aluminoferrite, calcium sulfate and bismuth oxide. It is chemically very similar to Portland cement and has similar handling and physical properties.

Initially, the root canal system must be cleaned and disinfected – this can be achieved through the use of irrigating solutions such as sodium hypochlorite and EDTAC, plus the use of an appropriate intra-canal medicament. The medicament

chosen will depend on the presenting condition of the pulp or root canal. Ledermix paste may be used if there has been irreversible pulpitis, while either a 50:50 mixture of Ledermix paste and calcium hydroxides or just calcium hydroxide alone should be used if the root canal system had been infected.

Once the canal has been disinfected and dried, the MTA can be placed in the apical few millimetres of the canal. Special instruments and magnification are required to achieve an adequate filling, as it is very technique sensitive and difficult to do. The MTA needs to be left to set for at least several days before the remainder of the canal can then be filled with conventional materials (such as gutta-percha and cement) and techniques (such as lateral compaction).

It is claimed that this technique reduces the chances of root fractures occurring later since the dentine is not exposed to long-term calcium hydroxide. However, MTA releases calcium hydroxide and therefore the effects of this need further investigation. The other disadvantages of this procedure are the high costs of the material, the need for two appointments to do the root canal filling, the slow setting time and the technical difficulties of placing the material without any of it being pushed into the periapical tissues.

New methods to manage open apex teeth with pulp necrosis and infection

An emerging prospect for the management of teeth with incomplete root development where the pulp has necrosed and become infected is the concept of 'pulp regeneration' (Figure 9.27). The aim is to achieve revascularization of the root canal system and regeneration of tissue that is capable of producing what radiographically appears to be dentine. To date, several cases have been reported in the literature showing that this is feasible, especially in premolar teeth that had developmental defects such as dens evaginatus. The prospect of using this approach for traumatized teeth with an open apex is being researched and shows promise. Although there are no established guidelines published yet, the initial approach is to:

1. Disinfect the root canal system by using sodium hypochlorite irrigating solution, followed by.
2. Antibiotics as an intracanal dressing. A triple antibiotic paste (ciprofloxacin, metronidazole and minocycline) has been advocated but some authors have reported only using one or two antibiotics.
3. At the next appointment, the antibiotic paste is removed and bleeding is induced in the periapical tissues by instrumenting through the apical foramen with a root canal file. The aim of this is to get blood within the canal which then clots to form a matrix for cell regeneration (Figure 9.27A).
4. Once the clot has formed in the canal, a cement such as MTA is placed in the coronal part of the root canal followed by restoration of the crown of the tooth (Figure 9.27B).

The tooth should then be reviewed after 6 and 12 months to determine whether there is further root development and hard tissue formation along the canal walls (Figure 9.27C). Research is currently being carried out to determine whether the procedure can be more predictable if stem cells, growth factors, tissue scaffolds or other tissue engineering techniques are used.

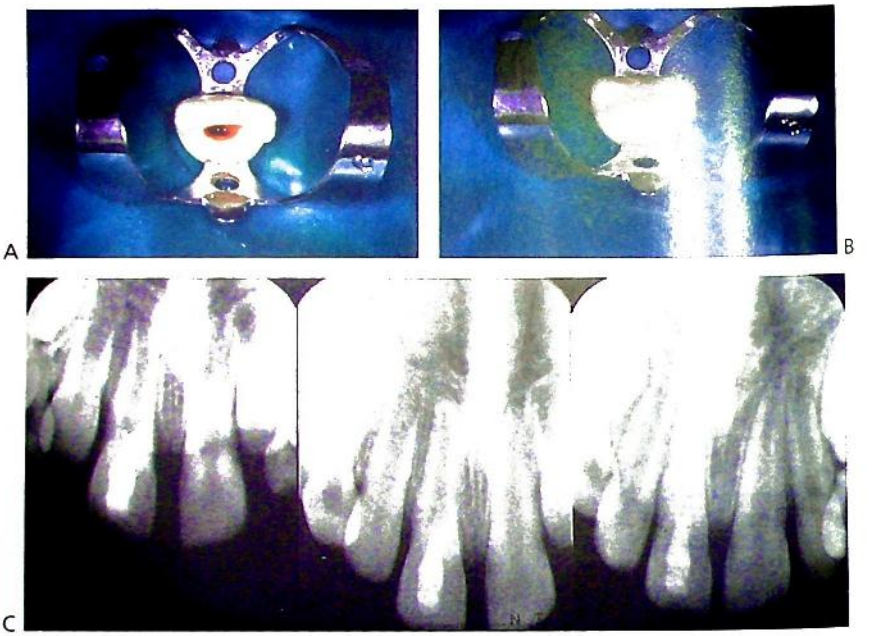

Figure 9.27 Pulp revascularization. (A) Haemorrhage is induced into the canal by passing a file through the apex of this immature tooth. (B) Mineral trioxide aggregate (MTA) is placed over the clot at the level of the cemento-enamel junction (CEJ). (C) Periapical radiographs of an open-apex tooth treated with this new technique. Over 3 and 12 months, there has been further development and closure of the root apex.

It is important to understand that the above procedures for regeneration are largely based on case reports at present, and guidelines need to be established. Current recommendations are that regenerative procedures in traumatized infected incompletely developed permanent teeth should only be performed if the tooth is not suitable for apexogenesis, or root canal treatment and apexification.

Mature root apex

If the pulp of a permanent anterior tooth is exposed by trauma, and the period of exposure is short, it need not be removed, regardless of the apical development. The Cvek (partial) pulpotomy can be used to attempt to preserve the pulp. If there are restorative considerations (i.e. the need for a post), it may be better to remove the pulp and perform root canal treatment immediately.

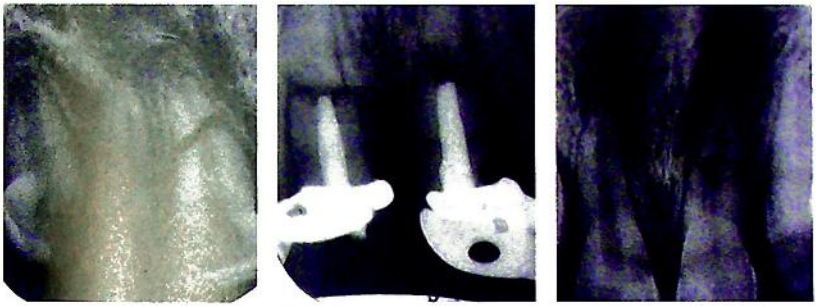

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Figure 9.28 (A) Root fractures near the apex of the root often require no treatment. In most cases, the pulp in the apical fragment remains normal. (B) When the coronal fragments become necrotic and infected, root canal treatment should only be performed up to the fracture line. Long-term calcium hydroxide treatment is required because the root canal at the fracture site will be wide open. In this case, bone is interposed between the two fragments. (C) Not all pulps in the coronal fragments become necrotic. Healing of an apical third root fracture. The pulp in the apical fragment is normal and there has been pulp canal calcification in the coronal two-thirds. In this case, there is probably bone interposed between the two fragments.

Root fractures (Figure 9.28)

- A fracture involving enamel, dentine and the cementum may or may not involve the pulp. Pulp necrosis occurs in 25% of teeth with root fractures and is related to the degree of displacement of the fragments. External inflammatory and replacement root resorption are rare.
- To check for horizontal root fractures, alter the vertical angulation of periapical radiographs. When looking for vertical root fractures, change the horizontal angulation. An occlusal radiograph is particularly useful and therefore indicated in all cases.
- Sometimes, a horizontal root fracture is not initially evident. This is because the fracture site opens up under the influence of the inflammatory reaction several days after the injury. Thus, for all traumatized teeth, it is important to take a subsequent radiograph within 2 weeks.

Frequency

- Permanent dentition: 2–4%.

Tissue responses following root fractures

- Healing by hard-tissue union with calcified tissue (osseodentin).
- Healing by interposition of bone.
- Healing by interposition of fibrous connective tissue.
- Granulation tissue in the fracture line, indicating coronal pulp necrosis and infection.

Management

- Radiographs – several vertical and/or horizontal angulations of periapical radiographs plus an occlusal radiograph are usually required to adequately determine the extent of the fracture.
- Reposition the coronal fragment.
- Place a rigid splint with composite resin and wire or an orthodontic appliance for 3–4 months if the coronal fragment is mobile.
- Root fractures in the apical few millimetres often require no treatment.

Review

- Review at 4, 8 and 12 weeks with pulp sensibility testing.
- Remove splint after 3–4 months.
- Review at 6 months, 12 months and then annually for 5 years.
- Take periapical radiographs at all review appointments.

Pulp necrosis and infection of the coronal fragment (Figure 9.28B)

It is uncommon for the apical fragment to develop pulp necrosis and it will usually undergo pulp canal calcification which requires no treatment. If pulp necrosis and infection of the coronal fragment occurs, there will be radiographic signs of bone loss at the level of the fracture. Symptoms, such as pain, excessive mobility, gingival swelling or a draining sinus, may also indicate that the coronal pulp has necrosed and become infected. These problems will not be evident at the time of the trauma so endodontic treatment should not be commenced then. These problems will only become evident during the review of root-fractured teeth and they may take several months or even longer to occur. If they occur, then the tooth should be managed as follows:

- Remove the pulp from the coronal fragment. Never advance an endodontic instrument through the fracture line.
- Take a periapical radiograph to determine the 'working length' at approximately 1.0 mm coronal to the fracture line.
- Biomechanically prepare the root canal to the working length.
- Place a 50:50 mixture of Ledermix paste and a calcium hydroxide paste as an initial dressing to control the infection and reduce the inflammation in the fracture line.
- Place a temporary restoration in the access cavity and arrange to review the patient in about 4 weeks.
- At the 4-week review appointment, open the access cavity and irrigate the root canal to remove the initial dressing. Then, place a non-setting calcium hydroxide paste to induce the formation of a hard-tissue barrier at the end of the coronal fragment (Figure 9.24B). The calcium hydroxide dressing should be replaced every 3 months until the hard tissue barrier has formed. This may take up to 18 months.
- Place a root canal filling using gutta-percha and cement once the barrier has formed, using a similar technique to that described above following apexification. The access cavity can then be restored, also as described above for apexification cases.

Pulp necrosis and infection of both the apical and coronal fragments

When the apical fragment shows signs of pulp necrosis and infection, the prognosis is poor. Root canal treatment of the coronal fragment should be performed followed by surgery to remove the apical fragment. There have been case reports of intra-radicular splinting and endodontic implants, but both have a very poor long-term prognosis.

Crown/root fractures

The coronal fragments should always be removed to fully assess the extent of the fracture (Figure 9.29).

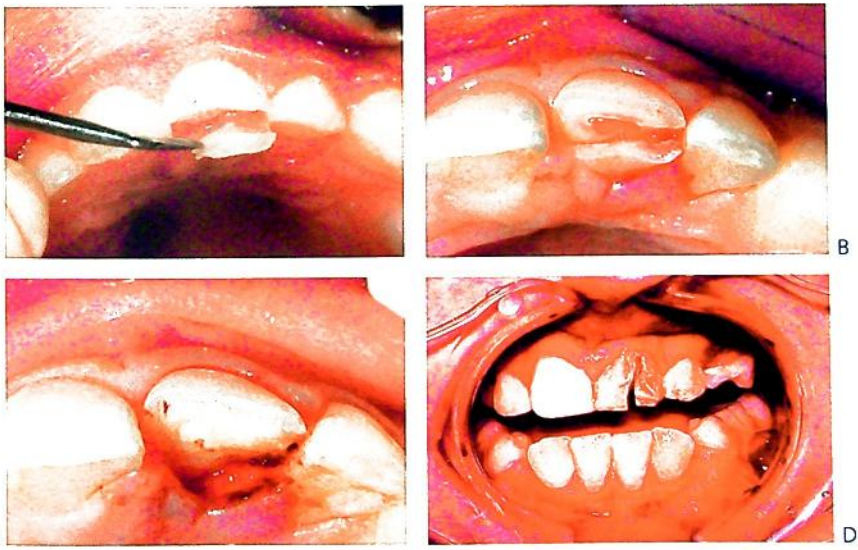


Figure 9.29 Crown/root fractures. (A) The coronal fragment of a crown/root fracture should always be removed to investigate the full extent of the fracture. (B) This case shows a complicated crown/root fracture (i.e. pulpally exposed) with the fracture extending just above the alveolar crest on the palate. (C) The fractured portion has been removed and it is clear that the fracture extends approximately 3 mm below the gingival margin. The pulp was capped, and the access cavity filled. Treatment will involve periodontal-flap surgery and placement of a crown with an extended shoulder. Alternatively, orthodontic extrusion may be required. (D) Unfortunately, vertical crown/root fractures are untreatable and such teeth should be extracted. Retention in the short term may be valuable to preserve bone while planning for possible orthodontics, or implants, when growth has finished.

Uncomplicated crown/root fracture

Where the fracture extends just below the gingival margin (Figure 7.29A), cover the dentine with a glass ionomer cement initially and then restore the tooth with composite resin or a crown. Only the crown part of the tooth should be restored to allow re-attachment and new cementum formation on the fractured root surface, i.e. do not restore the root portion.

Complicated crown/root fracture (i.e. with pulp exposure)

If the fracture extends below the crestal bone and the root development is complete, remove the coronal fragments to assess the extent of the fracture (Figure 7.29B). Root canal treatment is required. Ledermix paste or calcium hydroxide may be placed as the initial endodontic dressing.

If the crown/root fracture does not extend below the crestal bone, and the root development is complete, a Cvek pulpotomy may be performed (Figure 7.29C). This type of fracture may be restored with composite resin whereas deeper fractures may need a cast restoration or may require surgical treatment to expose the margins for restoration.

The prognosis for a tooth with a complicated crown/root fracture is poor.

Options for management

- Gingivectomy to expose the fracture margin. If the fracture is minimal, and just below the gingival margin, then restoration of the root surface may be performed with glass ionomer cement and a crown build-up in composite resin.
- Cast crown with extended shoulder with or without periodontal flap procedure.
- Orthodontic extrusion of the root to expose the fracture margin.
- Extraction.
- Root burial (or decoronation).

Orthodontic extrusion

This may be a viable option, provided there is adequate root length to support a crown. However, because of the narrower emergence profile of the root compared with the crown of a normal tooth, a satisfactory aesthetic result may be difficult to achieve. A gingivoplasty will almost always be required to reposition the gingival margin after the tooth has been extruded and then retained for an adequate period of time. Fixed appliances are placed to extrude the root so that the margin is exposed. A pericision is often advisable.

Root burial or decoronation (Figure 9.30)

In cases of sub-alveolar root fracture, root burial (decoronation) may be an alternative to extraction to preserve the alveolar bone. The root is 'buried' below the alveolar crest (i.e. the root is reduced in length from a coronal direction until it is entirely within bone) and a coronally repositioned flap is raised to cover the defect with periosteum. In this way, it is possible for bone to grow over the root surface (Figure 9.30.E,F). The pulp may be normal or root canal treatment may be necessary. This technique is valuable in the preservation of the labio-palatal width of the alveolus, which may be essential if an osseointegrated implant is required later, as it may negate the need for ridge augmentation.

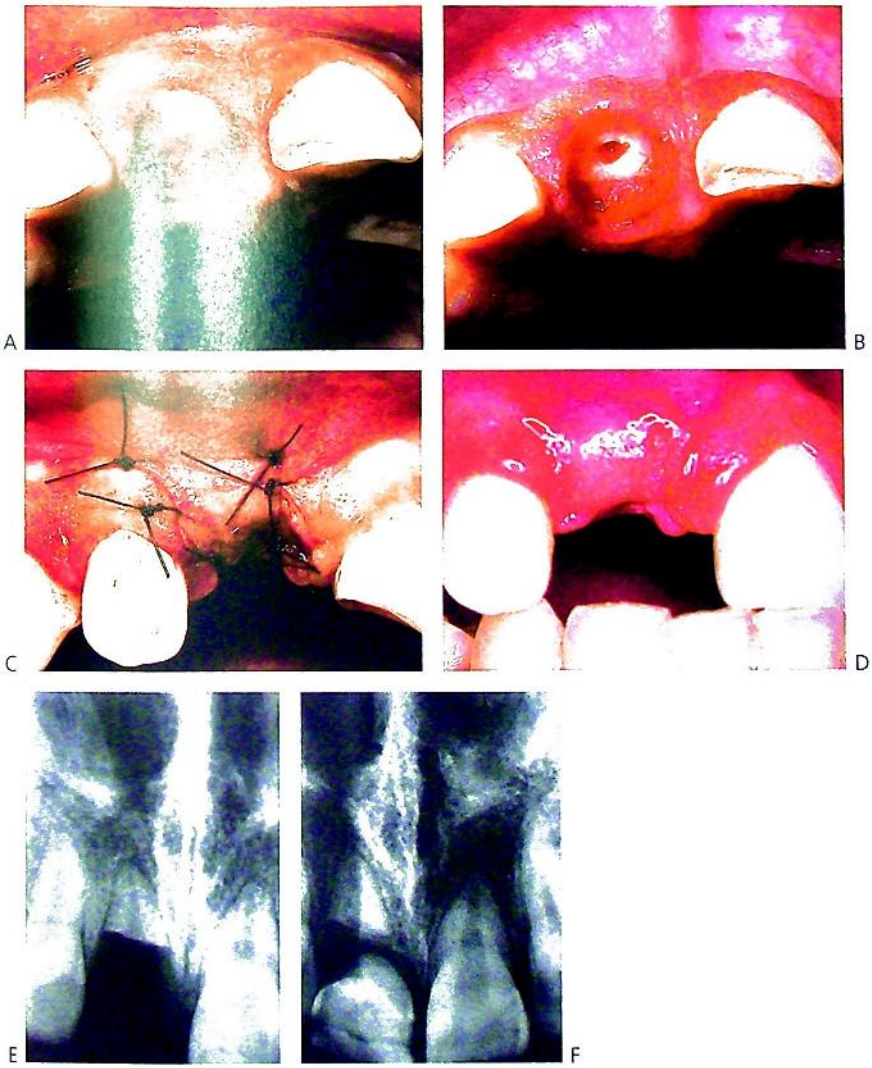


Figure 9.30 Root burial of a tooth fractured below the alveolar crest. Root burial may be an alternative to extraction in these cases. (A) This root has been traumatized with the fracture extending from the gingival margin on the labial to a level below the alveolar crest on the palatal. (B) The root was sectioned 1–2 mm below the crestal bone, and (C) covered with a coronally repositioned mucoperiosteal flap. (D) Healing after 2 weeks. (E,F) Bone growth has been stimulated over the root. This preserves the alveolar height for later prosthodontic work. The original crown has been contoured and attached to adjacent teeth with composite resin.

Crown/root fractures in immature teeth

When complex crown/root fractures occur in teeth with incomplete root formation, consideration should be given to maintaining the pulp, where possible, to allow continuation of root development. However, if the pulp has undergone necrosis and infection, then endodontic treatment including apexification will be necessary. It is worth noting, however, that complicated crown/root fractures tend to occur in mature teeth, where consideration of apical development is unnecessary. As a general comment, if the complex fracture extends below the crestal bone, then the prognosis is poor.

Luxations in the permanent dentition

Concussion and subluxation

These teeth are treated symptomatically. Concussed teeth will have a marked response to percussion, but the tooth will be firm in the socket. A subluxated tooth (Figure 9.31A) will exhibit increased mobility but will not have been displaced and there are no radiographic abnormalities. They are tender to percussion and mobility is usually increased.

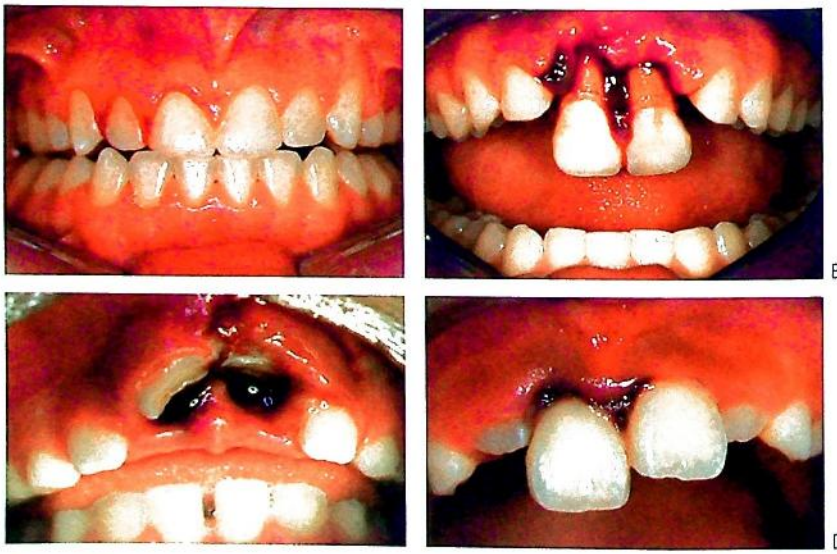


Figure 9.31 Luxations in the permanent dentition. (A) Subluxation. (B) Extrusion. (C) Intrusion. (D) Lateraluxation. Often, there is a combination of injuries, e.g. a lateral and intrusive luxation.

Management
- Pulp sensibility tests and radiographs (several periapical views plus an occlusal view).
- Relieve from occlusion; splinting is not usually required.
- Soft diet for 2 weeks.

Review
- Pulp sensibility testing at 1, 3, 6 and 12 months.
- Radiographs at each review.
- It is important to follow-up these teeth for at least 12 months (to check the pulp status, colour, mobility) and radiographically, to assess changes in the size of the pulp chamber and root development, as both of these indicate that the pulp has recovered and returned to a clinically normal state.

Prognosis
- Pulp necrosis in 3–6% of cases – will depend on any concurrent injuries (e.g. infractions, crown fractures, etc.).

Lateral and extrusive luxation (Figure 9.31)

Teeth may be luxated in any direction and will usually need repositioning and splinting. Repositioning can be achieved with digital pressure. Ideally, forceps should not be used since they can damage the root surface and this predisposes the tooth to root resorption. Luxated teeth are easily identified by visual examination as the tooth is obviously displaced, potentially mobile and with radiographic changes to the periodontal ligament. Pulp sensibility tests may give negative results initially.

Management
1. Reposition under local anaesthesia. Early repositioning is important (Figure 9.32A,B), as it is often extremely difficult to reposition the tooth if the patient presents later (especially after 24 hours) due to the presence of blood clots in the original socket space.
2. Suture gingival lacerations – particularly check for 'degloving' of the palatal gingivae with all luxated teeth by using an instrument (e.g. flat plastic instrument) to check whether the tissue is still attached or not. They may visually appear to be attached but may not actually be attached. If in doubt, it is better to suture the tissues to ensure the ideal conditions for healing are created.
3. Place a flexible splint using composite resin and fishing line or orthodontic appliances for 10–14 days weeks for extrusive luxation. Lateral luxation cases should be splinted with a more rigid splint for 4–6 weeks (because of concomitant alveolar bone fracture) by using wire and composite resin or orthodontic appliances.
4. Prescribe antibiotics, tetanus prophylaxis and 0.2% chlorhexidine mouthrinse if required.

Lateral luxations always have a fracture of the alveolar socket wall and hence it is important to mould the bone back into the correct position. Fragments of bone attached to the periosteum should be retained.

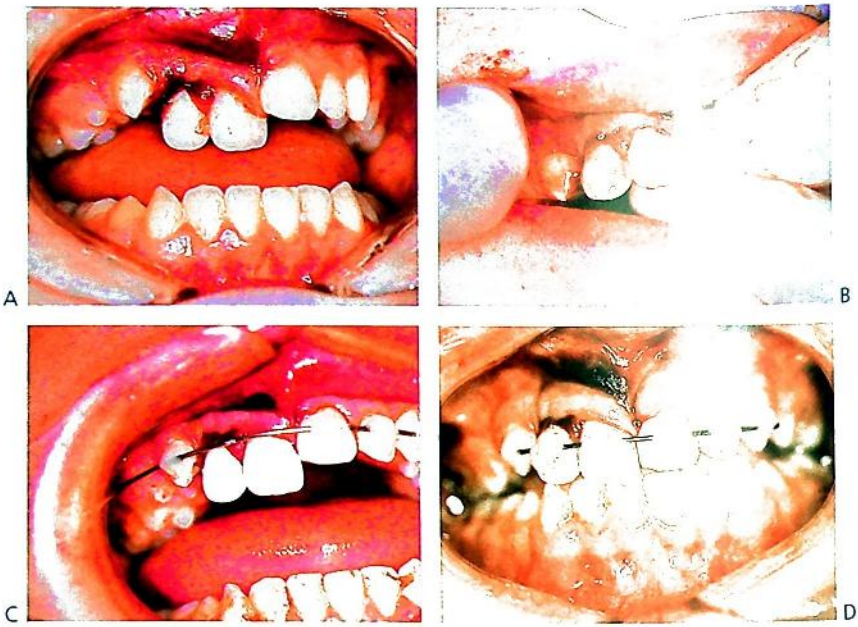


Figure 9.32 (A) Lateral luxation (palatal) with a dentoalveolar component involving the upper right central and lateral incisors. (B) The block of teeth and bone is manually replaced with finger pressure. (C,D) A rigid composite resin and wire splint is placed. When placing a splint, attach and stabilize uninvolved teeth before splinting the displaced segment.

Review

- Review every 2 weeks while the splint is in place, and then after 1, 3, 6 and 12 months. Subsequently annual reviews for up to 5 years.
- Take pulp sensibility tests at each review.
- Take radiographs at each review.

Prognosis

- Depends on the degree of displacement and apical development, with excellent healing in immature teeth. Also depends on whether any concurrent injuries such as a crown fracture.
- Pulp necrosis and infection occurs in 15–85% of cases and is more prevalent in teeth with closed apices. Also depends on whether any concurrent injuries such as a crown fracture.
- Pulp canal calcification often occurs in teeth with immature apices.
- Resorption is rare.
- Transient apical breakdown (2–12%) is a repair process where the apical foramen appears to 'open up' via a resorptive process to allow revascularization of the pulp

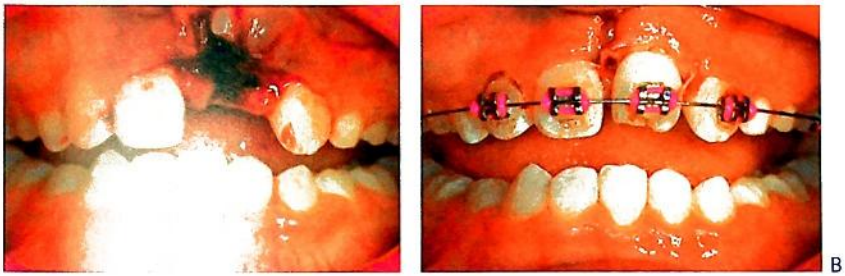


Figure 9.33 (A) Intrusion of the upper left central incisor. Early mobilization is essential to prevent ankylosis and to allow access to the palatal surface to perform root canal treatment. (B) The tooth was surgically repositioned and splinted with orthodontic appliances.

to occur. There is also an expansion of the apical periodontal ligament space. Essentially, there is some resorption followed by repair so the process is really a remodelling process. There is no indication for root canal treatment, unless there are other indicators of infection of the root canal system.

Intrusion

Intrusion (caused by pushing or forcing the tooth into the alveolar bone) is one of the worst injuries that can occur (Figure 9.31C, 9.33). There is extensive damage to the supporting structures (i.e. crushing of the periodontal ligament and bone) and the neurovascular bundle that supplies the pulp. There is much discussion about whether intrusively luxated teeth should be repositioned or allowed to re-erupt on their own. Treatment may well depend on the state of apical development but, as a general rule, repositioning and splinting of intruded teeth is preferred. Incompletely developed teeth may have the potential to re-erupt and therefore these teeth can be left to see if spontaneous re-eruption occurs.

Management

Current opinion suggests that early repositioning of intruded fully developed permanent teeth is essential. The aims of repositioning are to disimpact the tooth in order to avoid ankylosis, minimize pressure necrosis of the periodontal ligament and allow access to the palatal surface of the tooth to remove the pulp immediately. It is highly unlikely that the pulp will survive and therefore, its removal is important, as this will help to reduce the possibility of external inflammatory resorption occurring.

Repositioning

Teeth with incomplete root formation

- If the crown remains visible and there is a very wide immature apex (>2 mm), the tooth may be allowed to re-erupt spontaneously.
- If there is no improvement in position over 3–4 weeks, then rapid orthodontic repositioning is required.

Teeth with complete root formation

- Immediate repositioning is preferred for mature teeth (see above).
- Gently reposition the tooth with fingers or with forceps applied only to the crown. Avoid rotating the tooth in the socket.

Or

- Fixed orthodontic appliances can be used to apply traction to the intruded tooth over a 2-week period (Figure 9.33B).
- Extrusion should be rapid so that the palatal surface is exposed and an access cavity can be made as soon as possible.

Endodontic treatment

- Removal of the pulp is essential in almost all cases. The only exceptions are partially intruded, extremely immature teeth that are being left to re-erupt (with regular monitoring).
- Ledermix paste should be placed as the initial dressing for 3 months (change the dressing after 6 weeks to ensure adequate concentrations within the canal) to reduce the chances of external inflammatory resorption. This can then be followed by a 50:50 mixture of Ledermix paste and calcium hydroxide for 2–3 months before placing a root canal filling.
- If the apex is immature, then a further period of calcium-hydroxide therapy will be required for apexification before root-canal filling.

Review

- It is essential that these teeth are regularly reviewed. External inflammatory resorption can occur very rapidly if preventive measures have not been used (such as immediate root canal treatment with Ledermix paste dressings). External replacement resorption may also occur very rapidly due to the damage to (especially crushing of) the root surface and periodontal ligament during the injury, and an immature tooth may be lost within a number of weeks.
- Review every 2 weeks during the splinting phase, then at 6–8 weeks, 6 months, 12 months and yearly, for 5 years.

Prognosis

- Mature teeth undergo pulp necrosis in almost all cases (>96%), especially if there are also concurrent injuries such as a crown fracture, and there is a high prevalence of replacement resorption and ankylosis if not treated as above because of the damage to the root surface.
- Immature teeth that re-erupt show pulp necrosis in 60% of cases and ankylosis in up to 50% of cases.
- Teeth treated early have a much better prognosis.

Dentoalveolar fractures

With luxation of teeth, the alveolar plate can be fractured or deformed. Use firm finger pressure on the buccal and lingual plates to reposition. It should be remembered that

alveolar fractures can occur without significant dental involvement. These alveolar fractures should be splinted for 4 weeks in children (or 6–8 weeks in adults). Laterally luxated, intruded and avulsed teeth always have an alveolar bone fracture and/or displacement. Firm pressure is needed to realign the bony fragments once the tooth has been repositioned. Splinting may be rigid or semi-rigid and is dependent on the degree of injury and the number of teeth involved (Figure 9.32).

Pulp status

Pulp sensibility tests only test the ability of the pulp's nerves to respond to the stimulus that is applied; they do not provide any information about the presence or absence of blood supply or the histological status of the pulp. When determining the status of the pulp in luxated permanent teeth, beware of false test results. The pulp may not respond to a stimulus because of damage to the sensory nerves of the pulp, even though the tooth's vascularity is maintained. It may take up to 1 year (or never) to get a response from such a pulp. Thus, one must be careful to judge the patient's signs and symptoms before commencing root canal treatment. Regular radiographs are required to assess root development and growth, evidence of external or internal root resorption, and changes in the size and shape of the pulp chamber. Clinically, changes in colour, excess mobility, tenderness to percussion and a draining sinus are important diagnostic signs of an infected root canal system. A necrotic pulp does not cause apical periodontitis – it is only when the necrotic pulp becomes infected that a periapical response occurs. Hence, the most important thing to assess is whether the root canal system is infected or not.

Radiographs

It is important to remember that when teeth have been luxated they may also have had a crown or root fracture. Crown fractures are usually obvious but root fractures may be hidden or not yet apparent. Therefore, radiographs are always essential.

Avulsion of permanent teeth (Figure 9.34)

If a permanent tooth is avulsed, the chance of successful retention is enhanced by minimizing the extra-oral time. Even if the tooth has been out of the mouth for an extended period, it is usually still better to replant the tooth, with the knowledge that the tooth may ultimately be lost. In the mixed dentition, this is important, as replantation of even questionable teeth will allow normal establishment of the arch, occlusion and aesthetics. Furthermore, orthodontic treatment planning is simpler if the tooth remains in the socket. These teeth are usually lost by replacement resorption, which has the benefit of preserving the alveolar bone height, making prosthodontic replacement much simpler.

First aid advice

It is important that parents, caregivers and teachers have access to appropriate advice on the management of avulsed teeth. Timing is essential and this information can be given over the telephone:

- Keep the child calm.
- Do not allow the child to eat or drink. If sedation or anaesthesia is required for extensive injuries, then the child may need to be fasted.

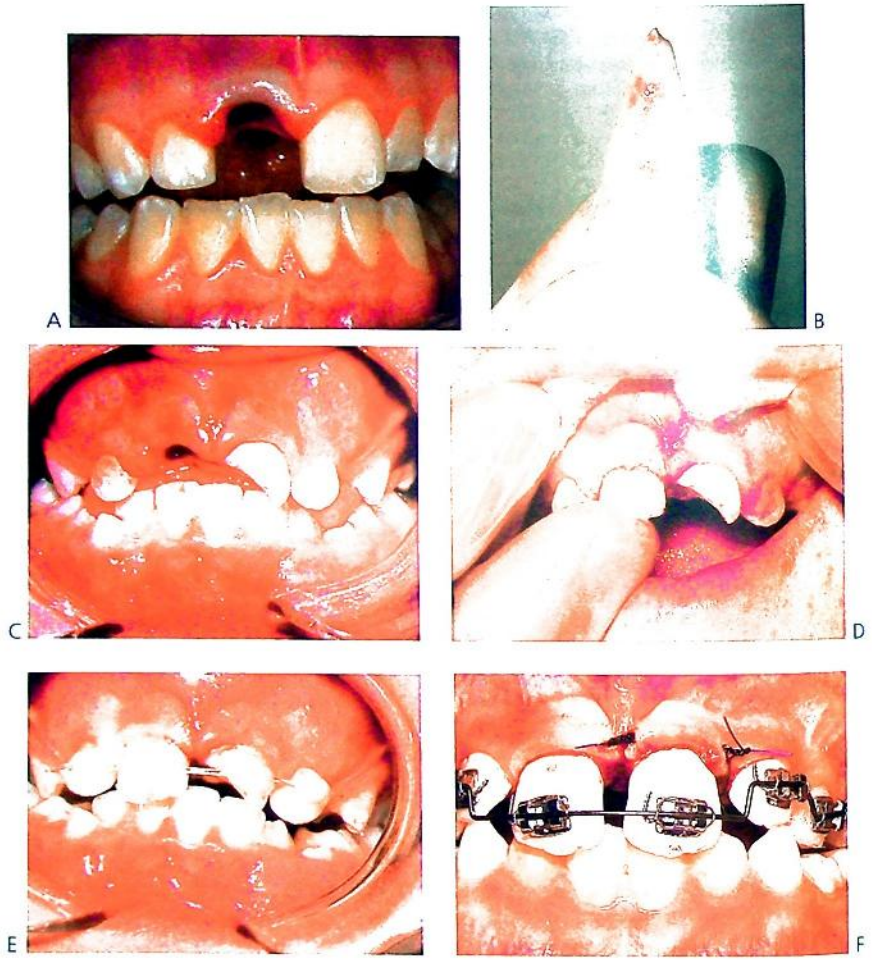


Figure 9.34 Management of avulsion. (A) With avulsions, there may be few other injuries. In other cases, there may be extensive damage to the supporting tissues. (B) Always hold the tooth by the crown and gently debride the root surface with saline. (C) The socket should be irrigated and clear of debris. (D) Replant with firm pressure. The tooth will usually click back into position. (E) Splint with a flexible splint, such as composite and nylon fishing line, to allow some physiological movement. (F) Orthodontic appliances are extremely useful when splinting traumatized teeth. The wire should be passive and allow physiological movement. Placement of a wire through orthodontic brackets allows the splint to be removed and the mobility of the tooth assessed.

- Locate the tooth and hold by the crown only. Always check the patient's clothing for avulsed teeth that are thought to be lost.
- Replant the tooth immediately if clean. If the tooth is dirty, it should be washed – preferably with milk if available, otherwise saline or the patient's saliva. As a last resort, very briefly rinse under cold water (10 s only).
- Hold the tooth in place by biting gently on a handkerchief or clean cloth, or use aluminium foil or similar and seek urgent dental treatment.
- If unable to replant the tooth, store it in isotonic media to prevent dehydration and death of the periodontal ligament cells. Use:
  - Milk (the preferred solution).
  - Saline.
  - Saliva.
  - Wrap in plastic cling wrap (with some saliva to keep it moist).
  - **Do not use water** as this will result in hypotonic lysis of the periodontal ligament cells.
- Seek urgent dental treatment.

Time is essential! The long-term prognosis of the tooth is severely reduced after 10 min of being dry and out of the mouth. Do not waste time searching for an ideal storage medium, replant the tooth!

Management in the dental surgery

The following are guidelines for replanting avulsed permanent teeth.

Tooth replanted prior to arrival

Debride the mouth but do not extract the tooth.

Tooth maintained in storage solution with extra-oral time <60 min

1. Gently debride the root surface under copious saline, milk or tissue-culture media (Hanks balanced salt solution) irrigation. When holding teeth, always do so by only holding the crown with a wet gauze square (teeth can be very slippery, see Figure 9.34B).
2. Give local anaesthesia and gently debride the tooth socket with saline to remove any blood clot, but do not curette the bone or remaining periodontal ligament (Figure 9.29C).
3. Replant the tooth gently with finger pressure (Figure 9.34D). The tooth usually 'clicks' back into the correct position if there has not been too much bone damage and there is no blood clot left in the socket.

Tooth is dry or extra-oral time is >30 min

1. Remove any necrotic periodontal ligament by soaking the tooth in saline and gently debriding the root surface with saline-soaked gauze. There is conflicting evidence as to the most appropriate way in which to remove necrotic debris and remnants of the periodontal ligament – however, damage to the cementum must be avoided and mechanical instrumentation should be avoided. The tooth should also be soaked in sodium fluoride for 20 min. It is essential that the tooth be rehydrated prior to replantation.

2. Give local anaesthesia and gently debride the tooth socket with saline to remove the blood clot; do not curette the bone or remaining ligament.
3. Replant the tooth gently with finger pressure.

Management following replantation

1. Splint for 14 days (Figure 9.34E,F).
2. Reposition and suture any degloved gingival tissues and suture all lacerations.
3. Under rubber dam isolation, cut an endodontic access cavity and remove the pulp. Irrigate the canal with sodium hypochlorite solution and then dry it. Place a Ledermix paste dressing in the canal in order to reduce the chance of external inflammatory root resorption. Continue root canal treatment as outlined above for intruded teeth.
4. Prescribe a high-dose, broad-spectrum antibiotic and check current immunization status.
5. Account for any lost teeth. A chest radiograph may be required.
6. Normal diet and strict oral hygiene including chlorhexidine gluconate 0.2% mouthwash.

Splinting of avulsed teeth

- Orthodontic brackets with a light archwire (0.014"). Orthodontic appliances are particularly useful as the time taken to apply the brackets is half that to set composite resin (Figure 9.34F).

Or

- Composite resin and nylon fibre (0.6 mm diameter) such as fishing line (20 kg breaking strain).

Splints should be flexible to allow normal physiological movement of the tooth. This helps to reduce the development of ankylosis and replacement resorption; however, if there is a bone or root fracture present, then a rigid splint must be used so that there is no movement of the teeth or bone segments.

Splints should generally stay in place for 10–14 days if there are no complicating factors such as alveolar or root fractures. When bone fractures are present, the splint should be retained for 4–6 weeks. If there is a root fracture, then the splint is usually required for 3 months. Avulsed teeth with immature apices that were kept dry prior to replantation may require splinting for up to 4 weeks. The occlusion may need to be relieved when the degree of overbite or luxation is such that the tooth will receive unwanted masticatory force. This can be achieved by minimal removal of enamel, or construction of an upper removable appliance, or placement of composite resin on the molars to open the bite. However, some physiological movement is necessary.

As a general rule, all teeth should be replanted whether wet or dry. Although the prognosis of a dry tooth may be poor, it is usually preferable to have the tooth present during growth than not at all. Always keep options open for future treatment.

Orthodontic splinting is always preferable but obviously requires suitable training and access to equipment. It does not matter which orthodontic bracket system is used. The most important point is that any arch wire placed for splinting is passive and will not move adjacent teeth. There are certain advantages over the use of a composite resin splint, in particular:

- Easier and quicker to place.
- Allows the splint to be readily removed and replaced so that the mobility of the teeth can be monitored.
- Easier to maintain oral hygiene.
- Less time to remove and less chance of damage to the teeth following removal of composite resin (often used to excess).

If composite resin splints are used, then choose a distinct shade of resin, avoid filling embrasures and try to minimize the amount of resin used. All these points assist in the later removal of the splint. It is more comfortable to delay removal of the splint until after any endodontic procedures, such as pulp extirpation, have been commenced.

Root canal treatment

Immature root apex

If a tooth has been avulsed, replanted within a short period, the apex is *extremely* immature (>2 mm) and the child is <8 years old, then root canal treatment is only needed if symptoms and clinical signs indicate that the pulp space has become infected. Hence, such teeth should not have root canal treatment commenced immediately after the replantation; instead they should be monitored to see whether the pulp revascularizes.

If the canal becomes infected, the root canal should be dressed with Ledermix paste, placed initially for 6 weeks followed by another Ledermix paste dressing for a further 6 weeks (i.e. total period 3 months). Calcium hydroxide treatment can then be used to induce apexification (see above). The calcium hydroxide should be a non-setting paste. This is changed 3-monthly until an apical hard tissue barrier has formed and root canal filling is possible. The rate of long-term survival of immature teeth is only 30% even if replanted early.

Mature root apex

In all other situations, in which the apex of the avulsed tooth is <2 mm open or closed, root canal treatment should be commenced immediately after replantation in order to prevent external inflammatory root resorption. The initial dressing should be Ledermix paste for two periods of 6 weeks each (i.e. total 3 months) followed by either a 50:50 mixture of Ledermix paste and calcium hydroxide or just calcium hydroxide alone. The root canal filling can usually be completed after 5–6 months.

Generally, it is best to always replant avulsed teeth even if they have a poor prognosis. Even with appropriate treatment, these teeth will often be lost by progressive replacement resorption, but the positive benefit being that the alveolar bone height is maintained. The only exceptions are those cases with very immature roots where ankylosis will prevent alveolar bone growth and may complicate future orthodontic and prosthodontic management.

Complications in endodontic management of avulsed teeth

External inflammatory root resorption (Figure 9.35A)

This is the progressive loss of tooth structure by an inflammatory process caused by the presence of bacteria in the root canal system and damage to the root surface. This

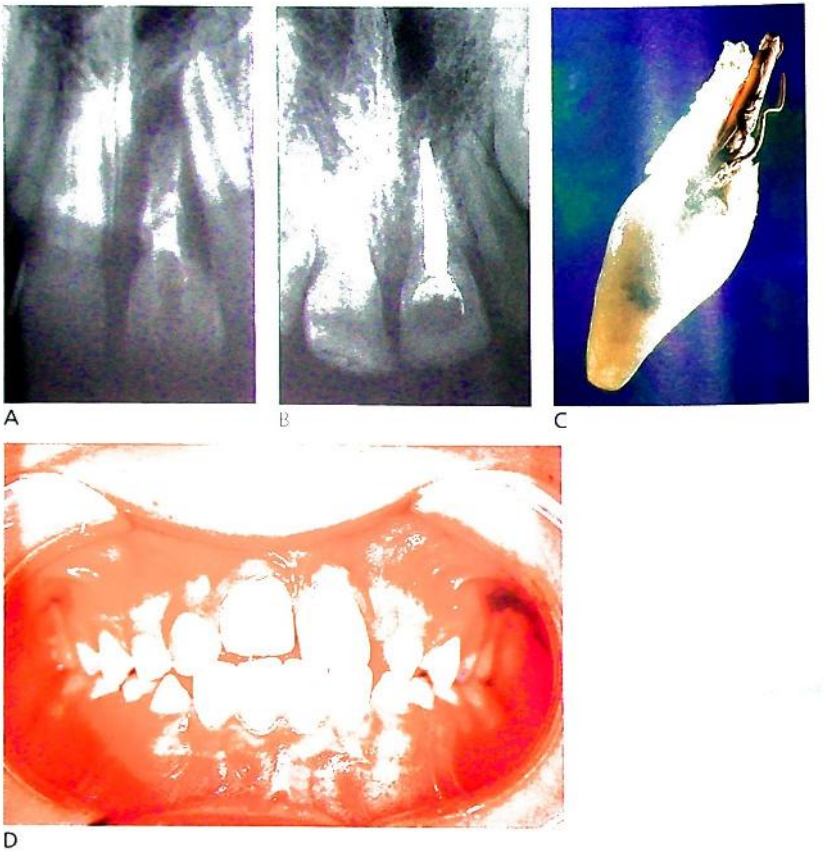


Figure 9.35 (A) Inflammatory root resorption resulting from a failure to adequately disinfect and medicate the root canal. (B) The root is being replaced by bone around the gutta-percha root filling. Note the slight infraocclusion of this tooth. (C) Replacement resorption of an avulsed central incisor. Note the ankylosis on the labial aspect of the root. (D) Ankylosis and subsequent infraocclusion is a significant problem when permanent teeth are traumatized before the cessation of growth. There is retardation of alveolar growth and the tooth is ultimately lost.

resorption can be prevented or managed with appropriate treatment. Factors in prevention and management include:

- **Prophylactic antibiotics**: a broad-spectrum antibiotic (e.g. tetracycline, amoxicillin or Penicillin V) should be given as soon as possible after avulsion and continued for 1 week. Although tetracyclines are preferred, they should be avoided in children where staining of other teeth may occur.

- **Pulp removal**: This should be done as soon as possible after the replantation – that is, on the day of the injury once the tooth has been replanted and stabilized with a splint. It must not be done outside the mouth. Any delays in commencing root canal treatment are likely to lead to inflammatory resorption.

Avoid medicaments that may cause inflammation, such as calcium hydroxide, in the first 3 months after trauma. Ledermix paste is an ideal first-dressing medicament as it has been shown to prevent inflammatory root resorption and inhibit the action of clastic cells.

Management
If inflammatory resorption is detected, the canal must be thoroughly re-instrumented, irrigated and then dressed with Ledermix paste for 3 months, but changing the dressing every 6 weeks. Calcium hydroxide can then be placed for a further 3 months after which time, if there is no progression of the resorption, the root canal can be filled.

External replacement root resorption (Figure 9.35B–D)

This is the progressive resorption of tooth structure and replacement with bone, as part of continual bone remodelling. It results from damage to the cementum and/or periodontal ligament or from replantation of dry teeth. It cannot be treated, so the aim must be to prevent replacement resorption and subsequent ankylosis. Factors in prevention and management include:

- **Extra-oral time**: prognosis decreases dramatically after 15 min if the tooth is dry. Approximately 50% of the periodontal ligament cells are usually dead after 30 min and all are dead after 60 min.
- **Storage media**
  - Milk is the best medium and may keep cells viable for up to 6 h. It has the advantage that it is pasteurized with few bacteria, is readily available and is cold. There appears to be no difference between low-fat and skimmed milk, but yoghurt and sour milk should be avoided due to their low pH.
  - Saliva is suitable for up to 2 h.
  - Saline and plastic cling wrap will maintain cells for 1 h.
  - Water is hypotonic and causes cell lysis, so it should be avoided.
  - Tissue culture media such as Hank's balanced-salt solution or RPMI 1640 (Roswell Park Memorial Institute tissue culture medium) is also appropriate, if available, and may give up to 24 hours' cell survival.
- **Mechanical damage**: ankylosis will result if the cementum has been removed or damaged.
- Risk increases with increased handling during transport and replantation.
- **Splinting**: flexible splinting allows physiological movement and results in less ankylosis and less replacement resorption.
- Extra-oral root canal treatment should be avoided since it will increase the likelihood of replacement resorption and ankylosis due to the prolonged extra-oral time, damage during treatment and the effects of toxic substances such as irrigating solutions and root canal cements.

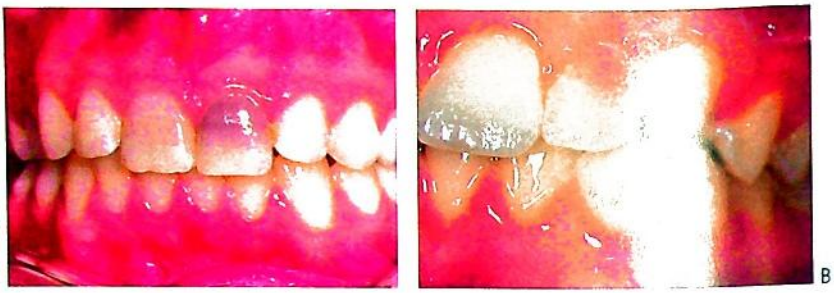


Figure 9.36 Complications post-trauma. (A) Discolouration of the crown of a previously luxated upper left central incisor due to breakdown of the temporary restoration in the access cavity. (B) Internal resorption following trauma to the upper left lateral incisor.

Management

- No treatment is possible.

Questions concerning the management of avulsed teeth

Despite a plethora of literature supporting the different procedures for managing avulsed teeth, the clinical reality remains that teeth that have been out of the mouth for more than 30 min have a poor prognosis (Figure 9.36).

- There is good evidence to support the use of specialized storage media, however, they are rarely available at the scene of an accident. Many dental injuries occur on weekends during sport. It would be interesting to research the average time taken to get a traumatized child to a dentist on a Saturday afternoon.
- There is a social cost following trauma, including absence from school (for the child) and work (for the parent) in attending multiple appointments, and loss of self-esteem. There are also financial considerations of complex restorative and endodontic treatment for teeth that often have a very poor outcome.

Parents and children should be given a clear indication about the probable outcomes of treatment. Heroic work is often performed with all good intentions when the prognosis is questionable (Barrett & Kenny 1997). In some cases, it may be preferable to retain hopeless teeth where replacement resorption will preserve bone. In other cases, the retardation of alveolar bone growth accompanying ankylosis in a growing child may necessitate early removal. Always keep the options open and consider the following questions:

- What is the long-term prognosis of the tooth?
- Are there orthodontic considerations such as the implications of ankylosis or space loss?
- Is the tooth important in the development of the occlusion?

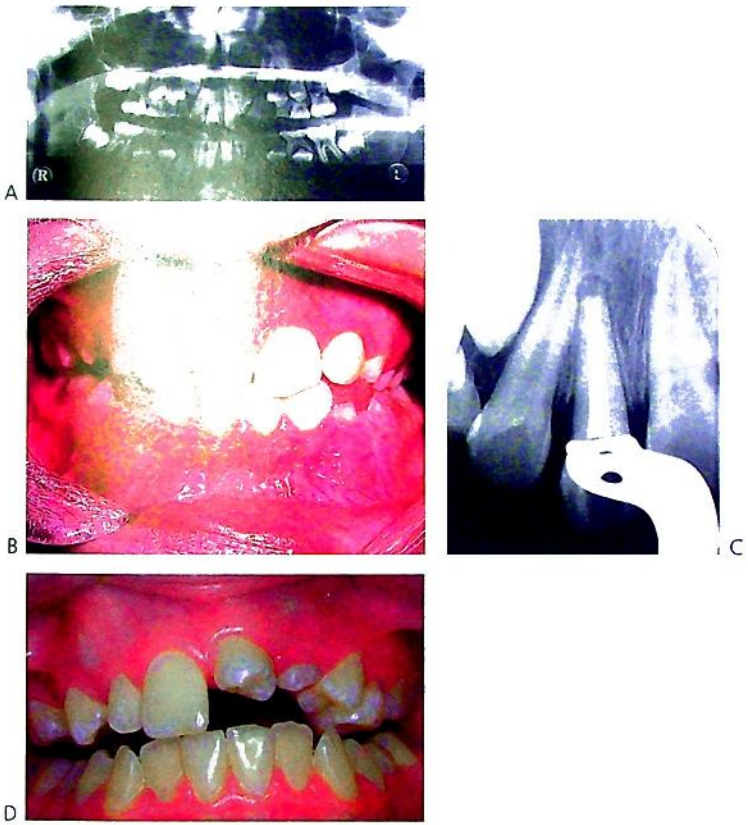


Figure 9.37 Autotransplantation. (A) The upper right central incisor in this boy was avulsed and undergoing resorption. A supernumerary lateral incisor was present lying palatal to the upper left central incisor. This tooth was autotransplanted into the socket of the traumatized central incisor. (B) Healing after 2 months. (C) Completion of root canal treatment at 6 months following transplantation showing good bone and periodontal healing. (D) Autotransplantation of a premolar. Notice the rotation of the crown by 45° to improve the emergence profile of the tooth prior to restoration with a composite resin strip crown.

Autotransplantation (Figure 9.37)

Autotransplantation has been successfully used in the management of tooth loss following trauma. It may be used in management of complicated crown/root fractures, replacement of the missing anterior teeth, and after avulsion injuries.

Good case selection is essential.

Table 9.2 Selection of donor tooth for autotransplantation

Donor tooth	Recipient site
Third molars	First molars
Lower first premolar	Upper central incisor
Lower second premolar	Upper lateral incisor
Supernumerary/supplemental teeth	Upper incisors
Lower incisors	Upper lateral incisor
Upper premolars	Depends on root shape

Indications

- Traumatized anterior tooth with poor long-term prognosis.
- Donor tooth is favourable with respect to the stage of root development, size and shape of crown, etc.
- Cases with Class I or Class II malocclusion with moderate to severe crowding involving extraction of premolars.

Autotransplantation must be considered as part of an overall treatment plan for the patient and other alternatives such as orthodontic space closure, fixed and removable prosthodontics and osseointegrated implant placement must be considered.

Success rates

- There is a 94% success rate for open apex.
- There is an 84% success rate for closed apex.

Procedure for autotransplantation

1. Selection of donor tooth (Table 9.2) – usually a premolar – consider:
   - The stage of root development.
   - The optimal time for transplantation is when the root is ½ to ¾ formed.
2. Analysis of recipient site:
   - Size and shape of recipient area.
   - Need for socket expansion or instrumentation.
   - Need to partially rotate the donor tooth.
3. Surgical procedure:
   - Remove the traumatized incisor as carefully as possible to minimize alveolar damage.
   - Prepare the socket – the socket can be enlarged if required and then irrigated with saline. Any necrotic or foreign debris such as gutta-percha, intracanal medicaments or granulation tissue must be removed.
   - Make an incision into the periodontal ligament of the donor tooth through the gingival margin; a collar of attached gingiva may be included with the graft.
   - Gently extract the tooth, avoiding damage to the root surface.
   - Position donor tooth into recipient site. This usually involves rotation of a premolar tooth about 45–90°.

Table 9.3 Healing and prognosis after autotransplantation of premolars

Root development	Root resorption		Pulp revascularization	Root formation
	Inflammatory resorption	Replacement resorption		
Stage 1 Initial root formation				77% normal root length
Stage 2 ¼ root formation	3%	6%	100% pulp revascularization	66% arrested root formation
Stage 3 ½ root formation				88% normal root length
Stage 4 ¾ root formation			87% pulp revascularization	Up to 98% normal root length
Stage 5 Root formation complete with apical foramen wide open				
Stage 6 Root formation complete with apical foramen half closed	9%	18%	No revascularization	Up to 98% normal root length
Stage 7 Root formation complete with apical foramen closed	25%	38%	0% pulp revascularization	Complete root development

After: Andreasen et al. (1990).

4. Splint with a flexible splint.
5. Follow-up as per protocols for avulsed teeth.

The need for root canal treatment will depend on the degree of root development (Table 9.3) and recovery of the pulp after the transplantation.

Reasons for an unfavourable outcome

- Inflammatory resorption or ankylosis, replacement resorption:
  - Closed apex – 20% root resorption.
  - Open apex – 3% root resorption.
- Pulp necrosis and infection.
- Infraocclusion.
- Incomplete root formation.
- No primary healing.

Details of healing and prognosis are shown in Table 9.3.

Internal bleaching of root-filled incisors

One consequence of trauma is tooth discolouration. Internal bleaching is a common procedure following root canal treatment. The integrity of the root canal filling is paramount and, above all, bleaching should not be carried out below the cemento-enamel junction because of the risk of initiating external invasive resorption.

Method

1. Bleaching must be carried out under rubber dam isolation.
2. Ensure adequate root canal filling and remove the gutta-percha to a level 3 mm below the cemento-enamel junction.
3. Place a Cavit base to just above the cemento-enamel junction.
4. Ensure that the access cavity is clean and free of all debris.
5. Acid etch the access cavity to open the dentine tubules and then rinse with water.
6. Place a thick, dry mixture of sodium perborate and hydrogen peroxide into the cavity and then place a temporary filling using Cavit. The bleaching mixture should remain in the tooth for 1 week, after which the tooth colour is evaluated. The procedure may be repeated several times if required.
7. Once good colour modification has been achieved, the access cavity can be restored. Cavit can be left as a base over the gutta percha and also on the labial wall of the access cavity. This will facilitate further access to the root canal system if endodontic re-treatment becomes necessary. Leaving Cavit on the labial wall of the cavity will allow further internal bleaching in the future if the tooth discolours again and it avoids the removal of dentine at that time. The remainder of the pulp chamber should be filled with a glass ionomer cement followed by a layer of composite resin.

Soft-tissue injuries

Alveolar mucosa and skin

Bruising (Figure 9.38)

The simplest and most common type of soft-tissue injury is bruising (contusion). This will often be present without any dental involvement. Treatment is symptomatic. However, be careful to check in the depths of the labial and buccal sulci for any other deep soft-tissue wounds (e.g. lacerations) or degloving-type injuries.

Lacerations (Figures 9.39, 9.40)

- Often a full-thickness laceration of the lower lip can be undetected because of the natural contours of the soft tissues or the tentative examination of an upset child. If there has been a dental injury, always look for tooth remnants in the lips.
- Careful suturing of skin wounds will be needed to avoid scarring and should be performed only by those who are competent to do so. Skin wounds must be closed within the first 24 h and preferably within 6 h.
- Any debris, such as gravel and dirt, must be removed by scrubbing with a brush and an antiseptic surgical solution such as 2.5% povidone iodine or 0.5% chlorhexidine acetate.

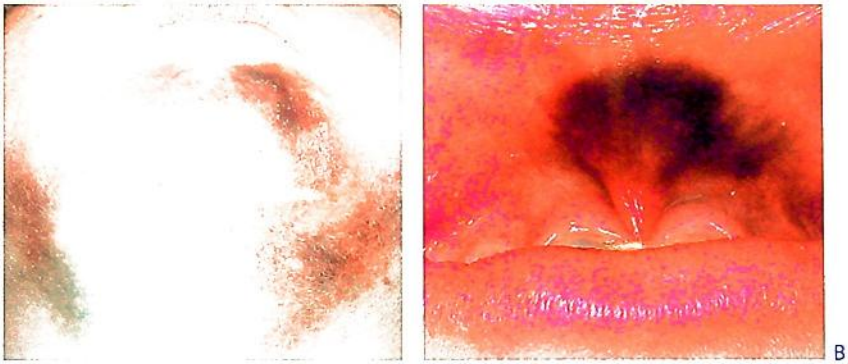
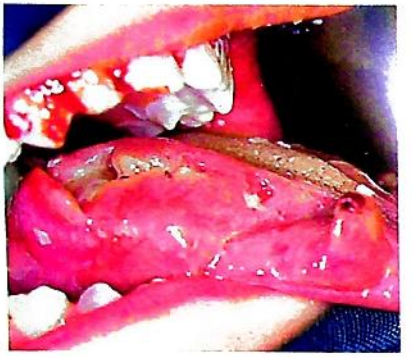


Figure 9.38 (A) Bruising of the chin is usually associated with severe degloving (see Figure 9.41B). (B) Bruising of the labial frenum may occur from a blow across the face; child abuse should always be suspected.

Figure 9.39 (A) When upper teeth are intruded, the lip is often bitten and it should be assessed for a through-and-through laceration. (B) These lacerations must be closed in three layers: the muscle, mucosa and skin. Always check lip lacerations for the presence of any tooth fragments if there are fractured teeth.

- Ideally, skin edges should be excised with a scalpel to remove necrotic tags and irregular margins.
- Muscle closure and deep suturing is achieved with a fine resorbable material such as polyglactin or polyglycolic acid.
- Final skin closure is with 6–0 monofilament nylon on a cutting needle.



A

B

Figure 9.40 (A) Lacerations may be caused by self-mutilation. This child has a peripheral sensory neuropathy (congenital indifference to pain). Attempts to make splints that would stop her behaviour failed and, after much agonizing, a full clearance was performed. (B) A severe laceration of the palate caused by this child falling with a straw in her mouth. In many cases, small lacerations will granulate and heal without intervention. However, large lacerations require suturing.

Attached gingival tissues

Degloving (Figure 9.41)

One of the most common injuries is degloving, which is when a full-thickness muco-periosteal flap is stripped off the bone, with the separation line usually being the mucogingival junction (Figure 9.41A). These injuries tend to occur after blunt trauma and a common presentation is a large collection of blood in the submental region (Figure 9.38A). The flap should be tightly sutured and a pressure dressing placed if the lower arch is involved. This prevents the pooling of blood and prevents swelling in the submental region, which may compromise the airway.

Interdental suturing of displaced gingival tissue is very important, especially where palatal tissue is involved (e.g. with lateral luxation). The close re-adaptation of tissues to the tooth surface will help preserve alveolar bone especially interdentally. Suturing will also help keep the tooth in position.

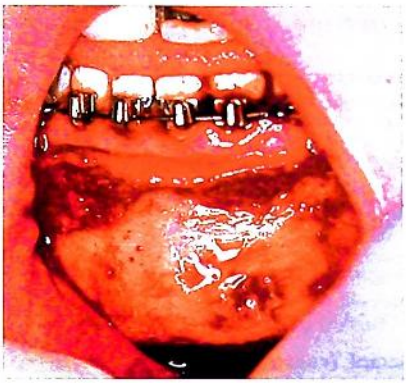
Suturing (see Chapter 8)

Prevention

Education of parents and caregivers

- Seat belts and child restraints.
- Helmets for bike riding.
- Mouth guards.

A



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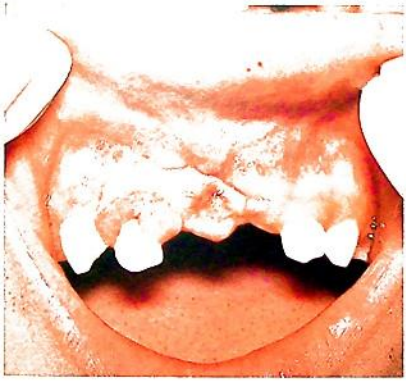

C

Figure 9.41 (A) Gingival degloving in a young child. Small tears such as this can be repositioned without suturing and will granulate well. (B) Severe degloving of the mandible separating at the mucogingival junction from molar to molar and to the level of the hyoid. The mental nerve on the left was severed. (C) Inadequate repositioning of a degloving injury has resulted in delayed healing and soft tissue loss. It is essential that these injuries should be treated early.

- Face guards.
- Supervision of pets, especially dogs.

While seatbelts and child restraints are covered by legislation, and helmets for bike riders are mandatory in many countries, the failure of parents to observe these regulations often results in unnecessary childhood craniofacial trauma. It has been the authors' experience that there is often little trauma seen from sports, as most children are wearing mouth guards; nevertheless, there is a disproportionate amount of trauma seen from leisure activities such as skateboarding, swimming and other 'non-contact' sports.

Educating parents, caregivers and teachers about primary care for dental trauma is essential. The correct protocols for dealing with avulsed teeth should be available to all schools and sporting clubs.

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10 Paediatric oral medicine, oral pathology and radiology

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Introduction

Although some disorders are confined to the mouth, oral lesions may be a sign of a systemic medical disorder. The majority of oral pathology seen in children is benign; however, it is essential to identify or eliminate more serious conditions. The presentation of pathology in children is often different from adult pathology and the subtleties of these differences are often important in diagnosis. In addition, many lesions change in form or extent with growth of the body. In this chapter, conditions will be grouped according to presentation, followed by a review of the most common causes and management of orofacial infections. It is important to remember that one disease entity may have different presentations while one presentation, e.g. an ulcer, may be representative of many different diseases.

Orofacial infections

Differential diagnosis

- Bacterial infections:
  - Odontogenic.
  - Scarlet fever.
  - Tuberculosis.
  - Atypical mycobacterial infection.
  - Actinomycosis.
  - Syphilis.
  - Impetigo.
  - Osteomyelitis.
- Viral infections:
  - Primary herpetic gingivostomatitis.
  - Herpes labialis.
  - Herpangina.
  - Hand, foot and mouth disease.
  - Infectious mononucleosis.
  - Varicella.
- Fungal infections:
  - Candidosis.

Odontogenic infections

The basic signs and symptoms of oral infection should be familiar to all clinicians.

Acute infection usually presents as an emergency:

- A sick, upset child.
- Raised temperature.
- Red, swollen face.
- Anxious and distressed parents.

Chronic infection typically presents as an asymptomatic or indolent process:

- A sinus may be present (usually labial or buccal).
- Mobile tooth.
- Halitosis.
- Discoloured tooth.

Presentation

- Children tend to present with facial cellulitis rather than an abscess with a large collection of pus. The child is usually febrile. Pain is common, although if the infection has perforated the cortical plate the child may not be in pain. The mainstay of treatment is removal of the cause of the infection. Too often, antibiotics are prescribed without consideration of extraction of the tooth or extirpation of the pulp.
- Maxillary canine fossa infections are predominantly Gram-positive or facultative anaerobic infections (Figure 10.1A). They may be misdiagnosed as a periorbital cellulitis (which is typically caused by *Haemophilus influenzae* or *Staphylococcus aureus* from haematogenous spread). Posterior spread may lead to cavernous sinus thrombosis and a brain abscess.
- Mandibular infections which spread inferiorly may compromise the airway and there is the possibility of mediastinal involvement.
- Young patients may be dehydrated at presentation. It is important to ask about their fluid intake and ascertain whether the child has urinated during the previous 12 h (see Appendix B).

Management

The treatment of infection follows two basic tenets:

- Removal of the cause.
- Local drainage and debridement.

Criteria for hospital admission

- Significant infection present or spiking temperatures >39°C.
- Floor of mouth swelling or swelling that crosses the midline.
- Dehydration.

Use of antibiotics

- Antibiotics should not be considered automatically as a firstline of treatment unless there is systemic involvement. In a child, a temperature of 39°C or higher can be considered a significant rise (normal ~37°C).

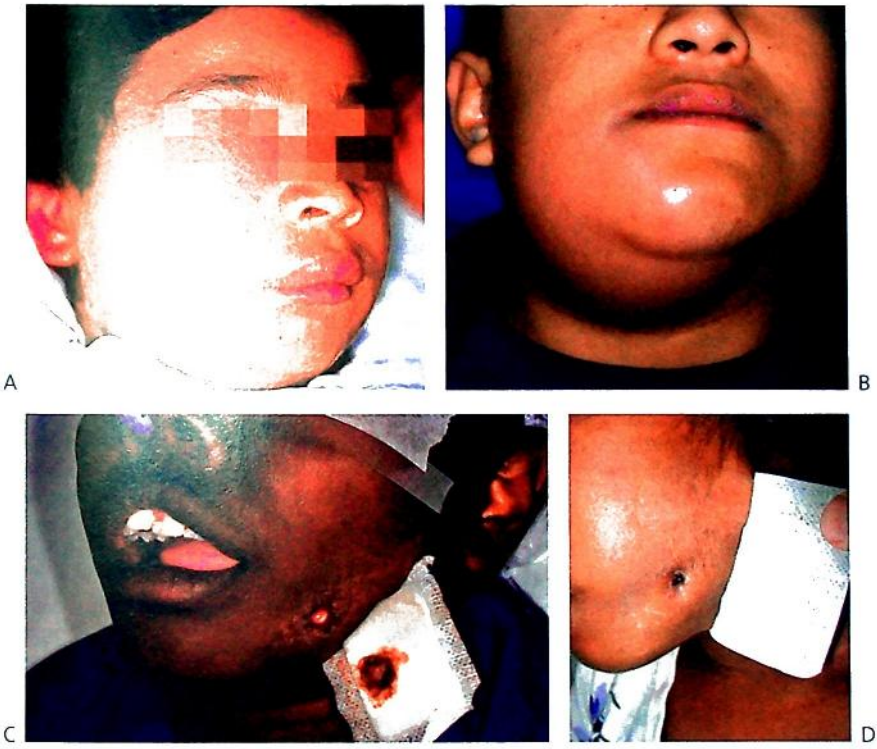


Figure 10.1 Severe facial swellings associated with odontogenic infections. (A) The right eye was almost closed from the spreading infection. (B) This child required extra-oral drainage of the facial swelling, which was caused by involvement of the floor of mouth as well as the submandibular and sublingual spaces. He required hospitalization and was placed on high-dose intravenous penicillin supplemented with metronidazole. (C) Extra-oral drainage of a long-standing abscess. Although this child was placed on repeated courses of antibiotics, no attempt was made to remove the cause of the infection, namely a carious tooth. (D) Healing 24 h after removal of the mandibular left first permanent molar tooth and debridement of the sinus tract draining sinus.

- If a child has a systemic infection resulting from a local focus of dental infection i.e. a sick child with a high temperature, an obvious spreading infection of the face and regional lymphadenopathy, antibiotics should be administered.
- Immunosuppressed patients or those with cardiac disease should receive antibiotics if infection is suspected.

General considerations

- Extraction of involved teeth.

Or

- Root canal treatment for permanent teeth if it is considered important to save particular teeth (see Chapter 7).
- Oral antibiotics if systemic involvement.

Amoxicillin is usually the drug of first choice. This has the advantage that it is given only three times a day, achieves higher blood levels and is a more effective antibiotic than, e.g. phenoxymethylpenicillin (Penicillin VK). Often the extraction of the abscessed tooth alone will bring about resolution without antibiotic treatment

Severe infections

- Hospital admission.
- Extraction of involved teeth. It is impossible to drain a significant infection solely through the root canals of a primary tooth.
- Drainage of any pus present. If the diagnosis or the correct management of an infection in the mandible has been delayed and the swelling has crossed the midline, or if there is swelling of the floor of the mouth, then extra-oral drainage with a through-and-through drain should be considered (Figure 10.1B). If a flap is raised, any granulation tissue should be removed and the area well irrigated. Flaps should be apposed but not tightly sutured. Soft flexible drains such as Penrose drains are better tolerated in children than are corrugated drains.
- Swabs for culture and sensitivity. It is important to take specimens for culture, even though empirical antibiotic treatment needs to be commenced immediately. Should the infection not respond to the initial antibiotic treatment, the results of the culture and sensitivity tests can be used to determine subsequent management.
- Intravenous antibiotics. Benzylpenicillin is the drug of first choice (up to 200 mg/kg per day).
- First-generation cephalosporins may be used as an alternative. However, if the child is allergic to penicillin, there may be cross-allergenicity and in these cases, it would be prudent to avoid cephalosporins. In these cases, clindamycin would be a better choice.
- In severe infections, particularly deep-seated infections and those involving bone, metronidazole can be added. The flora of most odontogenic infections is of a mixed type and anaerobic organisms are thought to have a significant role in their pathogenesis.
- For any antibiotics administered, adequate doses must be used – **treat serious infections in the head and neck seriously**.
- Maintenance fluids, adding 10–12% for every degree over 37.5°C, until the child is drinking of their own accord.
- Give 0.2% chlorhexidine gluconate rinses.
- Give adequate pain control with paracetamol suspension (orally), 15 mg/kg, 4-hourly, or by suppository (rectally).
- If the eye is closed because of collateral oedema, it may be appropriate to apply 0.5% chloramphenicol eye drops or 1% ointment to prevent conjunctivitis.

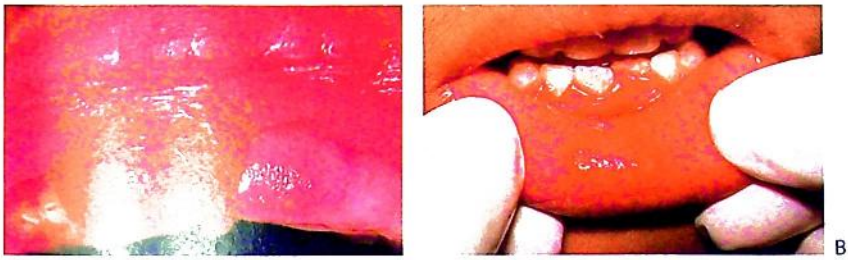


Figure 10.2 Different presentations of primary herpetic gingivostomatitis. (A) Infection with primary herpes often occurs at the time of eruption of primary teeth. (B) Common presentation with multiple small ulcers on the lower lip, and gingival swelling and inflammation.

Osteomyelitis

Rarely, odontogenic infection may lead to osteomyelitis, most commonly involving the mandible. Radiographically, the bone has a 'moth-eaten' appearance. Curettage of the area is required to remove bony sequestra and antibiotics are given for at least 6 weeks, depending on the results of microbiological culture and sensitivity test results. A variant of osteomyelitis has been reported in children and adolescents, termed juvenile mandibular chronic osteomyelitis. In this condition, there is often no obvious odontogenic source of infection; there is limited response to a number of different treatment modalities.

Primary herpetic gingivostomatitis (Figure 10.2)

This is the most common cause of severe oral ulceration in children. It is caused by herpes simplex type 1 virus. Occasional cases of type 2 (the usual cause of genital herpes) infection have been reported, mainly in cases of sexual abuse. The clinical appearance of the two different strains of herpes simplex virus are, however, clinically identical in the orofacial region. Although the majority of the population has been infected with the virus by adulthood, less than 1% manifest an acute primary infection. This usually occurs after 6 months of age, often coincident with the eruption of the primary incisors. The peak incidence is between 12 and 18 months of age. Incubation time is 3–5 days with a prodromal 48-h history of irritability, pyrexia and malaise. The child is often unwell, has difficulty in eating and drinking and typically drools. Stomatitis is present, with the gingival tissues in particular becoming red and oedematous. Intraepithelial vesicles appear and rapidly break down to form painful ulcers. Vesicles may form on any part of the oral mucosa, including the skin around the lips. Solitary ulcers are usually small (3 mm) and painful with an erythematous margin, but larger ulcers with irregular margins often result from the coalescence of individual lesions. The disease is self-limiting and the ulcers heal spontaneously without scarring, within 10–14 days.

Diagnosis

- History and clinical features.
- Exfoliative cytology showing the presence of multinucleated giant cells and viral inclusion bodies can be used for rapid diagnosis if laboratory support is at hand.

- Viral antigen can be detected by polymerase chain reaction (PCR) amplification. This can sometimes be useful in early confirmation of the diagnosis.
- Viral culture. Viral culture can take days or weeks to yield a result.
- Viral antibody detection in blood samples during the acute and convalescent phases. A rise in antibody titre can only provide late confirmation of the diagnosis.

Management

- Symptomatic care.
- Encourage oral fluids.
- If oral fluids cannot be taken then hospital admission is mandatory and intravenous fluids must be commenced.
- Analgesics – paracetamol, 15 mg/kg, 4-hourly.
- Mouthwashes for older children – chlorhexidine gluconate, 0.2%, 10 mL 4-hourly. In children over 10 years of age, tetracycline or minocycline mouthwashes may be beneficial, but must be avoided in younger children to prevent possible tooth discolouration.
- In young children with severe ulceration, chlorhexidine can be swabbed over the affected areas with cotton wool swabs. Much of the pain from oral ulceration is probably as a consequence of secondary bacterial infection. Chlorhexidine 0.2% mouthwash has been shown to be beneficial in the management of oral ulceration. A mouthwash containing benzydamine hydrochloride 0.15% and chlorhexidine 0.12% (Difflam C™) may offer some advantages over chlorhexidine alone.
- Topical anaesthetics: lignocaine viscous 2% or lignocaine (Xylocaine™) spray.
  Note: topical anaesthetics are often advocated; however, the effect of a numb mouth in a young child can be more distressing than the pain from the illness and can lead to ulceration from the decrease in sensation and subsequent trauma. In addition, it is often difficult to initiate swallowing with a soft palate that has been anaesthetized.
- Antiviral chemotherapy. Acyclovir oral suspension or intravenously for immunosuppressed patients. This treatment is only worthwhile in the vesicular phase of the infection, i.e. within the first 72 h.
  Note: the use of antiviral medications is contentious and usually reserved for children who are immunocompromised. There is some evidence to suggest, however, that the administration of aciclovir in the first 72 h of the infection may be beneficial.
- Adequate pain control is also required with regular administration of paracetamol.
- Antibiotics are unhelpful.
- Severely affected young children often present dehydrated, being unable to eat or drink. Hospital admission is required for these cases with maintenance intravenous fluids.

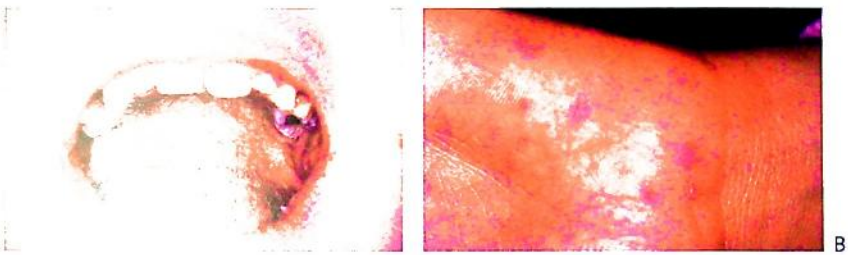


Figure 10.3 Infections caused by Coxsackie group A viruses. (A) Herpangina with characteristic palatal and pharyngeal ulceration and inflammation. (B) Cutaneous lesions in hand, foot and mouth disease.

Clinical tip

An obviously ill child with puffy erythematous gingivae is most likely to have primary herpetic gingivostomatitis.

Herpangina and hand, foot and mouth disease

These infections are caused by the Coxsackie group A viruses. As with primary herpes, both of the above conditions have a prodromal phase of low-grade fever and malaise that may last for several days before the appearance of the vesicles. In herpangina (Figure 10.3A), a cluster of four to five vesicles are usually found on the palate, pillars of the fauces and pharynx, whereas in hand, foot and mouth disease up to 10 vesicles occur at these sites and elsewhere in the mouth, in addition to the hands and feet (Figure 10.3B). The skin lesions appear on the palmar surfaces of the hands and plantar surface of the feet and are surrounded by an erythematous margin. The severity of both diseases is usually milder than primary herpes and healing occurs within 10 days. Both diseases occur in epidemics, mainly affecting children.

Diagnosis

- Clinical appearance and history.
- Known epidemic.
- Viral culture from swab.

Management

- Symptomatic care, as for other viral infections.

Infectious mononucleosis (Figure 10.4A)

- This infection is caused by the Epstein–Barr virus (EBV) and mainly affects older adolescents and young adults. The disease is highly infective and is characterized by malaise, fever, lymphadenopathy and acute pharyngitis. In young children, ulcers and petechiae are often found in the posterior pharynx and soft palate. The disease is self-limiting.

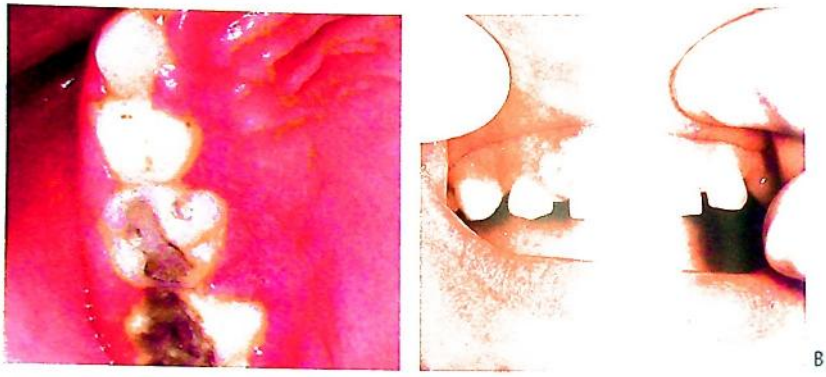


Figure 10.4 (A) Gingival ulceration and stomatitis during an acute episode of infectious mononucleosis. (B) Gingival ulceration in chickenpox infection.

Diagnosis

- History and clinical features.
- Monospot test (Mononucleosis spot test) or Paul–Bunnell agglutination test and atypical monocytes on blood film.

Varicella (Figure 10.4B)

This is a highly contagious virus causing chickenpox in younger subjects and shingles in older individuals. There is a prodromal phase of malaise and fever for 24 h followed by macular eruptions and vesicles. In chickenpox, oral lesions occur in around 50% of cases but only a small number of vesicles occur in the mouth. These lesions may be found anywhere in the mouth in addition to other mucosal sites such as conjunctivae, nose or anus. Healing of oral lesions is uneventful.

Diagnosis

- History and clinical features.

Measles

Measles is a highly contagious paramyxovirus infection, predominately of childhood that presents as an acute febrile illness, a maculopapular rash, keratoconjunctivitis, malaise, cough and the characteristic oral lesions – Koplik's spots. These are small white papules surrounded by an erythematous margin and cover the buccal mucosa. They usually precede the typical measles rash by up to 4 days. The incubation period is 8–12 days with a prodromal phase of malaise, fever, cough and the oral signs. Healing of oral lesions is uneventful; however, some children may suffer other complications of the disease such as pneumonia (1 in 20) or encephalitis (1 in 1000).

Diagnosis

- History and clinical features.

Candidosis

Acute pseudomembranous candidosis

The most common presentation of candidal infection in infants is thrush. White plaques are present, which on removal reveal an erythematous, sometimes haemorrhagic, base. In older children, thrush occurs when children are immunocompromised such as in acquired immune deficiency syndrome (AIDS) or in diabetes, or when prescribed antibiotics, steroids, or during chemotherapy and radiotherapy for malignancies.

White lesions which can be rubbed off leaving a red base are typical of thrush.

Median rhomboid glossitis

This characteristic but uncommon lesion, is a candidosis (rather than a developmental anomaly as was thought for many years) presenting as an erythematous, depapillated well-delineated area on the midline of the dorsal surface of the tongue anterior to the circumvallate papillae, often in children in response to the use of antibiotics.

Diagnosis

Some 50% of children will have *Candida albicans* as a normal commensal, and culture is of little benefit. Smears or scrapings for exfoliative cytology reveal hyphae when disease is present, but the clinical picture may be diagnostic.

Management

- Antifungal medication for 2–4 weeks. Most antifungal treatment is unsuccessful because of poor compliance or instruction by the clinician.
- Amphotericin B lozenges or nystatin drops.
- Fluconazole orally (100 mg daily for 14 days) for cases of mucocutaneous candidosis.
- Systemic antifungal medication may need to be used for children who are immunosuppressed or where the organism does not respond to topical treatment as described above.

Ulcerative and vesiculobullous lesions

Clinicians are often confused regarding the terminology of these lesions. An ulcer is regarded as the localized loss of the full-thickness of the epithelium. Partial thickness loss is termed an erosion. A vesicle is a small fluid-filled blister, while a bulla is a larger blister, generally, measuring >5 mm. Different conditions arise from cleavage of the epithelium at different levels (i.e. intraepithelial or subepithelial) and are important in determining a diagnosis. When these lesions burst, they leave an ulcer. A thorough history noting the number, frequency, duration and site of occurrence is very important.

Differential diagnosis

- Traumatic:
  - Post-mandibular block anaesthesia.
  - Chemical or thermal burns.
  - Riga–Fedé ulceration.
- Infective (see above):
  - Primary herpetic gingivostomatitis.
  - Herpangina.
  - Hand, foot and mouth disease.
  - Infectious mononucleosis.
  - Varicella.
- Others:
  - Recurrent aphthous ulceration.
  - Erythema multiforme.
  - Stevens–Johnson syndrome.
  - Behçet syndrome.
  - Epidermolysis bullosa.
  - Lupus erythematosus.
  - Neutropenic ulceration.
  - Orofacial granulomatosis/Crohn disease.
  - Pemphigus.
  - Drug-induced (chemotherapy) lesions.
  - Lichen planus.

Lip ulceration after mandibular block anaesthesia

This is one of the most common causes of traumatic ulceration. Parents should be warned and children reminded not to bite their lips after mandibular block anaesthesia (Figure 10.5A).

Riga–Fedé ulceration

This is ulceration of the ventral surface of the tongue caused by trauma from continual protrusive and retrusive movements over the lower incisors (Figure 10.5B). Once a common finding in cases of whooping cough, it is now almost exclusively seen in children with cerebral palsy.

Management

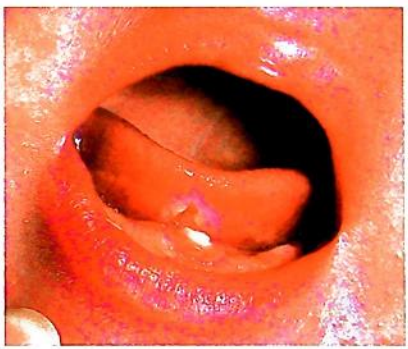
Smoothen sharp incisal edges or place domes of composite resin over the teeth. Rarely, in severe cases, extraction of the teeth might be considered (see also Chapter 13).

Recurrent aphthous ulceration (Figure 10.6)

Recurrent aphthous ulceration (RAU) has been estimated to affect up to 20% of the population. Lesions are classification according to size, duration and severity. Two clinical groups have been described:

- Simple
  - Minor aphthae.
  - Major aphthae (Figure 10.6B).
  - Herpetiform ulceration.

A



B

C

Figure 10.5 (A) Traumatic oral ulceration from biting the lip after a mandibular block injection. (B) Riga–Fedé ulcer on the ventral surface of the tongue arising from rubbing on the solitary mandibular incisor. (C) Chemical burn from an antihistamine tablet placed under the tongue in an adolescent. *(Courtesy Ms Leanne Smith, Uni Newcastle.)*

- Complex
  - This fourth form – complex aphthous stomatitis has recently been described.

Minor aphthae account for the majority of cases, with crops of two to five shallow ulcers measuring up to 5 mm and occurring on non-keratinized mucosa. There is a typical central yellow slough with an erythematous halo. Ulcers heal within 10–14 days without scarring. The cause of RAU is contentious, although it is commonly believed to be precipitated by stress and local trauma. There is some evidence for a genetic basis for the disorder, with an increased incidence of ulceration in children when both parents have RAU. Some studies have suggested that RAU is associated with nutritional deficiency states and so haematological investigation can be helpful. In major aphthae, the keratinized mucosa may also be involved, the ulcers are larger, last longer and heal with scarring.

Complex aphthous stomatitis accounts for less than 5% of cases that are recurrent, with multiple lesions that are extremely painful and are slow to heal. The differences between simple and complex forms are summarized in Table 10.1.

Diagnosis

- History and clinical features.
- Blood tests for full blood count (FBC) and differential white cell count (WCC), iron studies (including serum ferritin), serum vitamin B_{12} levels and folate and red cell folate in particular (if anaemia or latent anaemia suspected).

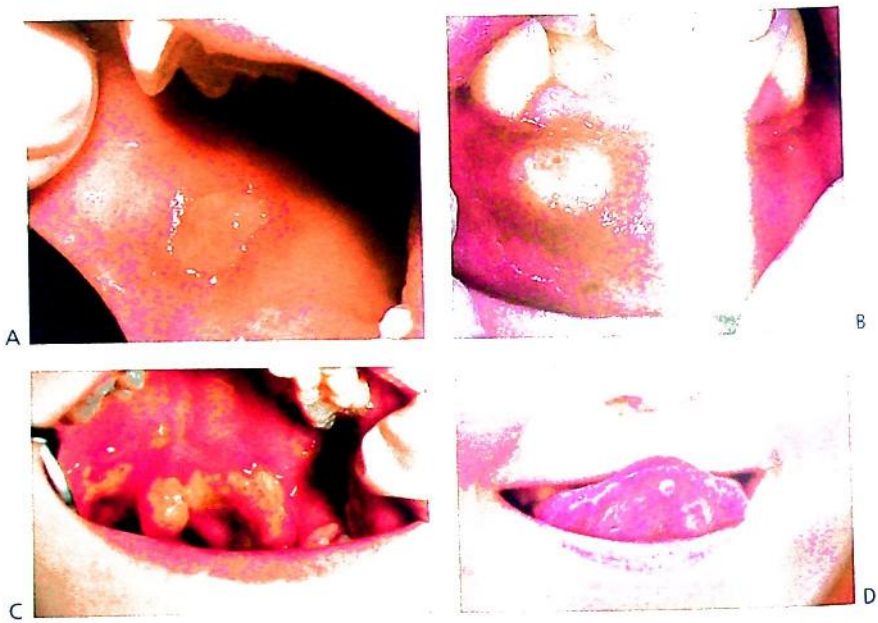


Figure 10.6 (A) Minor recurrent aphthae in an adolescent girl. These lesions were extremely painful. Haematological investigations revealed a low folate level, which when corrected eliminated further ulceration. (B,C) Major recurrent aphthous ulceration is a debilitating condition and heals with scarring. This girl's ulcers were managed with systemic steroids. (D) Vesiculobullous diseases often present with ulceration due to fragility of the preceeding vesicles or bullae. This is a case of ulceration associated with Epidermolysis bullosa.

Management

- Symptomatic care with mouthrinses:
  - Chlorhexidine gluconate 0.2%, 10 mL three times daily.
  - Minocycline mouthwash, 50 mg in 10 mL water three times daily for 4 days for children over 8 years of age.
  - Benzydamine hydrochloride 0.15% and chlorhexidine 0.12% (Difflam C™).
- Topical corticosteroids:
  - Triamcinolone in Orabase™ (although this may be difficult to apply in children).
  - Beclomethasone dipropionate or fluticasone propionate asthma inhalers sprayed onto the ulcers.
  - Betamethasone dipropionate 0.05% (Diprosone OV) ointment.

Table 10.1 Presentation of aphthous stomatitis

	Simple	Complex
Frequency	Episodic; <6 episodes per year	Continuous rather than episodic; >6 episodes per year
Duration	Short-lived	Persistent
Number	Few/limited number of lesions	Many, rather than few lesions
Healing	Rapid healing and generally heal without scarring except for major aphthae	Slower to heal with scarring
Location	Generally confined to non-keratinized mucosa	Can involve both keratinized and non-keratinized mucosa
Pain	Minimal pain/discomfort with minimal disability	Extremely painful and disabling
Geography	Limited to oral mucosa	May have genital involvement Need to exclude Behçet's disease

Modified from Rogers, R.S. 3rd, 1997. Recurrent aphthous stomatitis: clinical characteristics and associated systemic disorders. Seminars in Cutaneous Medicine and Surgery 16, 278–283.

- Systemic corticosteroids only in most severe cases of major aphthous ulceration.
- Herpetiform ulceration seems to respond best to minocycline mouthwashes.

Biopsy should only be considered if there is any doubt about the clinical diagnosis. Haematological investigations should be carried out to exclude anaemia or haematinic deficiency states (when appropriate, replacement can improve the ulceration or bring about resolution). As with all oral ulceration, symptomatic care with appropriate analgesics and antiseptic mouthwashes is appropriate. In those cases of severe recurrent oral ulceration, systemic corticosteroids may be used, although their use is best avoided in children. Minocycline mouthwashes should not be used in children under the age of 8 years to avoid tooth discolouration. Major aphthae tend to occur in older children.

Clinical Hint

Minor aphthae occur in recurrent crops of two to five ulcers, sparing the palate and dorsum of the tongue.

Behçet syndrome

This condition is characterized by recurrent aphthous ulceration together with genital and ocular lesions, although the skin and other systems can also be involved. Lesions may affect other parts of the body. Behçet syndrome can be subdivided into four main types:

- Mucocutaneous form – classical form with involvement of oral and genital mucosa and conjunctiva.

- Arthritic form with arthritis in association with mucocutaneous lesions.
- Neurological form with central nervous system involvement.
- Ocular form with uveitis in addition to oral and genital lesions.

Diagnosis
- History and clinical presentation.

Management
As for recurrent aphthous ulceration, but systemic treatment (corticosteroids) is usually required.

Clinical Hints
- Full blood count including differential white cell count.
- Erythrocyte sedimentation rate (suggestive of inflammatory diseases).
- Vitamin B_{12} and folate.
- Iron studies
  - Total iron binding capacity.
  - Serum iron.
  - Ferritin.
- Antinuclear antibodies (associated with SLE, Sjögren syndrome, scleroderma, rheumatoid arthritis).
- C-reactive protein (suggestive of inflammatory bowel disease).
- Angiotensin converting enzyme (test for sarcoid disease).
- IgA/IgG Anti-gliadin antibodies (test for coeliac disease).

Erythema multiforme, Stevens–Johnson syndrome and toxic epidermal necrolysis

Three conditions exist that present with similar clinical signs and histopathological appearances. Like many conditions, varied nomenclature and misdiagnosis have clouded and confused the diagnosis/es. There is now a view that these are distinct pathological entities and, perhaps importantly, might be initiated by quite distinct aetiological agents. The alternative view is that these disorders represent different presentations of the same basic disorder, distinguished by the severity and extent of the lesions.

Erythema multiforme (von Hebra) (Figure 10.7A–D)
The original description of erythema multiforme was that of a self-limiting but often recurrent and seasonal skin disease with mucosal involvement limited to the oral cavity. The lips are typically ulcerated with blood-staining and crusting. The characteristic macules ('target lesions') occur on the limbs but with less involvement of the trunk or head and neck. These lesions are concentric with an erythematous halo and a central blister. Although the lesions are extremely painful, the course of the illness is benign and healing is uneventful.

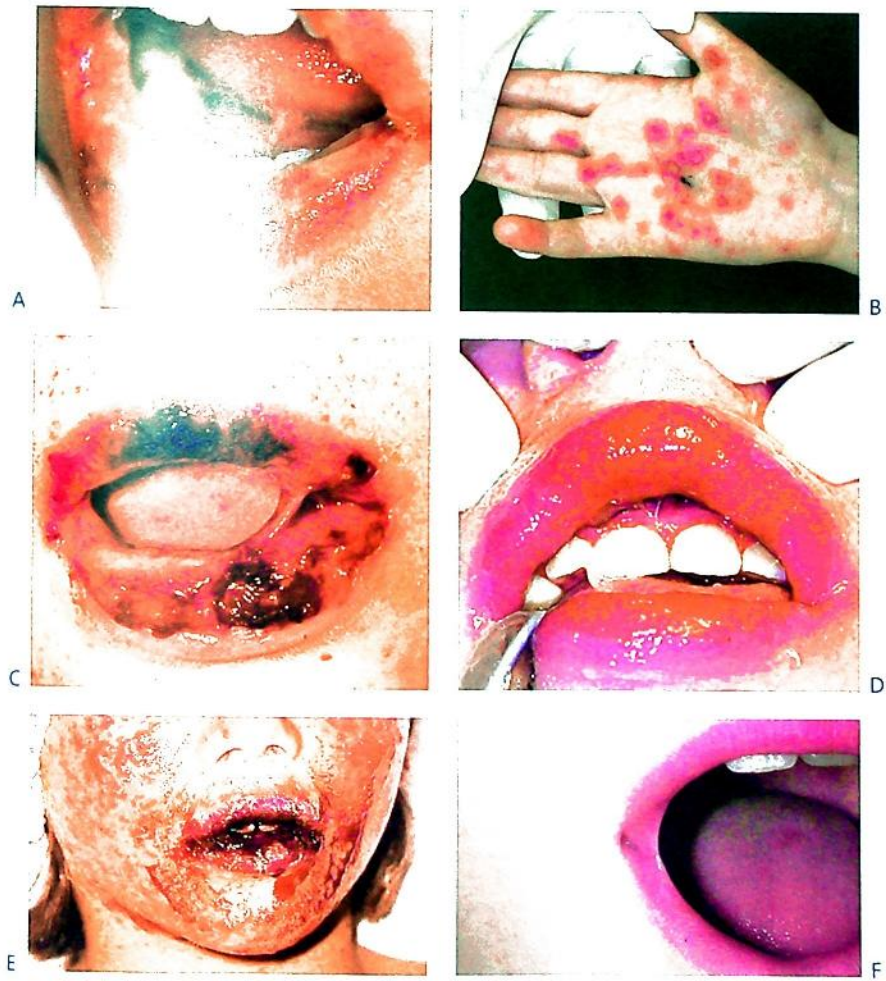


Figure 10.7 (A) Erythema multiforme presenting with pan-stomatitis and severe dehydration. Treatment included rehydration and symptomatic care of the ulceration. (B) Typical target lesions seen in erythema multiforme. (C) Ulceration of the lips may be severe, and resulted in the lips becoming fused together by the slough and crusting in this case. (D) The mouth was debrided under general anaesthesia. (E) Stevens–Johnson syndrome with severe mucocutaneous involvement. This child required admission to the intensive care unit for 3 days. (F) Stricture of the lateral commissure of the lips due to scarring following an episode of Stevens–Johnson syndrome.

Stevens–Johnson syndrome (Figure 10.7E,F)
The condition presents with acute febrile illness, generalized exanthema, lesions involving the oral cavity and a severe purulent conjunctivitis. The skin lesions are more extensive than those of erythema multiforme. Stevens–Johnson syndrome is characterized by vesiculobullous eruptions over the body, in particular the trunk, and severe involvement of multiple mucous membranes including the vulva or penis and conjunctiva. The course of the condition is longer and scarring may occur. Some authors have used the term erythema multiforme major for Stevens–Johnson syndrome, defining the condition as a severe form of erythema multiforme, but this is disputed by others. Although Stevens–Johnson syndrome patients are acutely ill, death is rare.

Toxic epidermal necrolysis (TEN)
Similar to the clinical presentation of Stevens–Johnson syndrome, TEN or Lyell syndrome is a severe, sometimes fatal, bullous drug-induced eruption where sheets of skin are lost. It resembles third-degree burns or staphylococcal scalded skin syndrome. Oral involvement is similar to Stevens–Johnson syndrome. TEN may be misdiagnosed as a severe form of Stevens–Johnson syndrome (given the controversy over nomenclature), but the use of steroids is contraindicated, given an increase in mortality with their use in this disease.

Aetiology
Erythema multiforme is often initiated by herpes simplex reactivation. There is some evidence that Stevens–Johnson syndrome is initiated by a *Mycoplasma pneumoniae* respiratory infection or drug reaction. TEN is drug-induced.

Clinical presentation
A summary of the three conditions is shown in Table 10.2. There is an acute onset of fever, cough, sore throat and malaise, followed by the appearance of the lesions on the body and oral cavity ranging from 2 days to 2 weeks after the onset of symptoms. These break down quickly in the mouth to form ulcers. The most striking feature is the degree of oral mucosal involvement that may lead in all three cases to a pan stomatitis and sloughing of the whole oral mucosa. There is extensive ulceration and crusting around the lips, oral haemorrhage and necrosis of skin and mucosa leading to secondary infection. There may be difficulty in eating and drinking, which complicates the clinical course, and there is usually extreme discomfort from both the skin and oral lesions that may necessitate narcotic analgesics.

Management
If there is a known precipitating factor such as herpes simplex infection, then antivirals such as topical aciclovir can be used in an attempt at prophylaxis. Management is generally symptomatic and supportive. A major problem during the course of the illness is fluid balance and pain, much of which arises from secondary infection of the oral lesions. Debridement of the oral cavity with 0.2% chlorhexidine gluconate or benzydamine hydrochloride and chlorhexidine (Difflam C™) is effective in removing much of the necrotic debris from the mouth. Extensive areas of ulceration tend to be less responsive to chlorhexidine and a minocycline mouthwash may prove more effective. The role of systemic steroids is controversial but they may be necessary in severe cases. Their use in recurrences may obviate the need for hospital admission.

Table 10.2 Differential diagnosis of erythema multiforme (EM), Stevens–Johnson syndrome and toxic epidermal necrolysis

Signs	Erythema multiforme (EM minor)	Stevens–Johnson syndrome (EM major)	Toxic epidermal necrolysis (Lyell syndrome)
Aetiology	HSV reactivation in some cases	HSV reactivation? *Mycoplasma*? Drug-related	Drug-related – trimethoprim and sulfamethoxazole (Bactrim), carbamazepine lamotrigine
Recurrence	75% have multiple recurrences – two to three per year	Usually single but may recur	May recur in response to initiating agent
Target lesions	Yes – typical concentric target lesions on extremities	No – or atypical vesiculobullous eruptions on trunk, head and neck	Skin involvement is so severe that no separate lesions are ever seen
Skin	Raised papules acrally distributed	Multiple tissue involvement usually severe	Severe skin involvement, similar to burns Confluent sloughing of the skin
Mucosa	Crusting and bleeding of lips	Crusting and bleeding of lips Severe multiple mucosal involvement – oral, ocular and genital	Severe multiple mucosal involvement
Course	Within 2 weeks	>2 weeks – Oral lesions may take months	Healing phase will take many months
Management	Short course of high dose systemic steroids Fluid maintenance	Fluid maintenance Debridement of oral lesions Narcotic analgesics/ sedation? Antibiotics for *M. pneumoniae*? Short course of high dose systemic steroids	Similar to burns management Resuscitation and fluid maintenance Narcotic analgesics Intubation and intensive care unit admission
Sequelae	Healing without scarring	May be scarring and oral stricture	Blindness may occur and there is a high mortality
Histopathology	Similar in all three conditions: subepidermal vesicles and bullae		

HSV, herpes simplex virus.

Management also includes:

- Adequate fluid replacement and total parenteral nutrition if required.
- Pain control, which may necessitate the use of narcotics and sedation.

Clinical Hint

Crusted, blood-stained lips are typical of erythema multiforme.

Pemphigus

Pemphigus is an important vesiculo-bullous disease mainly affecting adults; however, children can be also affected. The lesions are intraepithelial and rapidly break down, so that affected individuals are often unaware of blistering, complaining instead of ulceration, mainly affecting the buccal mucosa, palate and lips.

Diagnosis

There may be a positive Nikolsky sign (separation of the superficial epithelial layers from the basal layer produced by rubbing or gentle pressure) and cytological examination can reveal the presence of Tzanck cells. Direct immunofluorescence using frozen sections from an oral biopsy will reveal intercellular immunoglobulin (IgG) deposits in the epithelium that are diagnostic for this disease. Indirect immunofluorescence on blood samples is used to complement diagnosis.

Management

- Systemic corticosteroid therapy in conjunction with steroid-sparing agents.
- Topical corticosteroids are used for oral lesions.
- Antiseptic or minocycline mouthwashes and analgesia as necessary.

All of these mucocutaneous conditions in children heal with scarring and are managed similarly.

Epidermolysis bullosa (Figure 10.6D)

Epidermolysis bullosa is a term used to describe several hereditary vesiculo-bullous disorders of the skin and mucosa. Within the hereditary variants, there are three groups according to the location of skin separation:

- Epidermolysis Bullosa Simplex, non-scarring form, transmitted as an autosomal dominant or sex-linked trait.
- Junctional Epidermolysis Bullosa, with demi-desmosome defect and severe scarring, transmitted as an autosomal recessive trait.
- Dystrophic Epidermolysis Bullosa.

Another form which is not inherited, is termed Epidermolysis bullosa acquisita.

Blisters may form from birth or appear in the first few weeks of life, depending on the form of the disease. Corneal ulceration may also be present and pitting enamel hypoplasia has been reported, mainly in the junctional forms of the disease.

Management

Management is often extremely difficult because of the fragility of the skin and oral mucosa. Intensive preventive dental care is essential to prevent dental caries, combined with treatment of early decay. Supportive care is required with the use of chlorhexidine

gluconate mouthwashes and possibly topical anaesthetics such as lignocaine (Xylocaine viscous™). Fortunately, children may receive dental treatment under general anaesthesia without laryngeal complications. Because of oral stricture, however, access into the mouth is often difficult and in older patients, surgical release of the commissure may be necessary. It is important to cover instruments with copious lubricant; the use of rubber dam is essential.

Systemic lupus erythematosus

Systemic lupus erythematosus is a chronic inflammatory multisystem disease occurring predominantly in young women. The hallmark of systemic lupus erythematosus is the presence of antinuclear antibodies which form circulating immune complexes with DNA. Oral ulceration often occurs in systemic lupus erythematosus and treatment of the condition usually involves systemic steroids.

Orofacial granulomatosis and Crohn disease (Figure 10.8)

Although not primarily an ulcerative condition, oral ulceration may be the presenting sign in orofacial granulomatosis. Orofacial granulomatosis may be confined to the

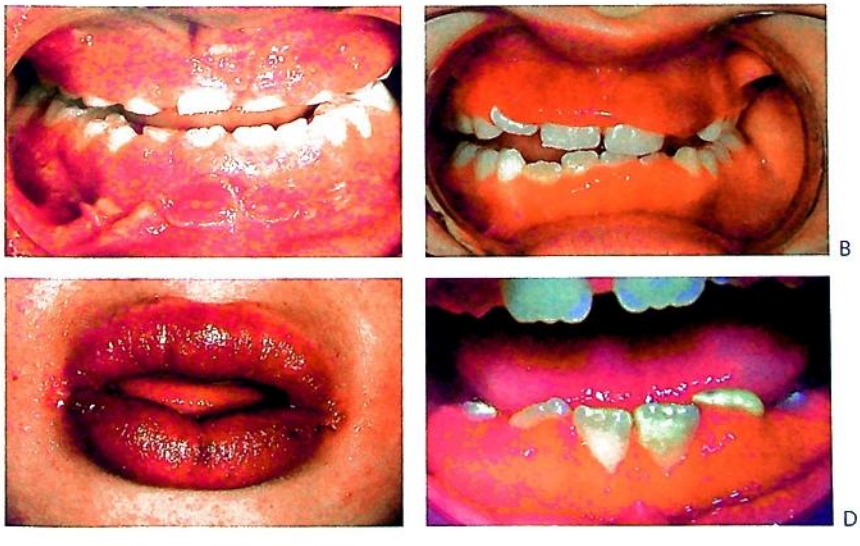


Figure 10.8 Presentations of orofacial granulomatosis/Crohn disease. (A) The intra-oral appearance is pathognomonic with swelling of the gingivae, the patient also had a cobblestone appearance of the buccal mucosa and ulceration of the labial and buccal sulci. (B) A different presentation with bright red swollen gingivae. (C) The patient initially presented with a painless swelling of the lips and had evidence of malabsorption, perianal fissuring and bowel problems, and was diagnosed with Crohn disease. Management was with systemic corticosteroids. (D) Crohn disease. This child has recurrent flare-ups of his disease that is characterized by concurrent deterioration of his oral disease.

orofacial region and precede or be a manifestation of Crohn disease, an inflammatory condition of the gastrointestinal tract, or of sarcoidosis.

Presentation

- Diffuse swelling of the lips and cheeks, often initially recurrent and then becoming persistent.
- Diffusely swollen gingivae (Figure 10.8B).
- Linear ulceration or fissuring of the buccal and labial sulcus. A characteristic 'cobblestone' appearance of the buccal mucosa (Figure 10.8A).
- Polypoid tags of vestibular and retromolar mucosa.
- Children may also present with diarrhoea, failure to thrive, weakness, fatigue, anorexia and perianal fissuring or skin tags as manifestation of Crohn disease.
- Oral changes have been found in between 10% and 25% of patients with Crohn disease and, importantly, their appearance may precede other systemic symptoms.

Diagnosis

- Biopsy of oral lesions. The granulomas may be quite deep within the submucosa and if possible, a minor salivary gland should be included in the specimen.
- Blood tests, including:
  - Full blood count, differential white cell count (to exclude leukaemia), erythrocyte sedimentation rate (ESR) and C-reactive protein.
  - Serum angiotensin-converting enzyme (ACE) level to identify or exclude sarcoidosis.
- Barium studies, endoscopy and biopsy of bowel if Crohn disease is suspected.

Management

- Some cases of orofacial granulomatosis have been shown to be a response to topical medicaments or dietary components. An exclusion diet can be considered to identify food intolerance, in particular cinnamon and benzoates.

Crohn disease

- Prednisolone. Corticosteroids are usually commenced at a high dosage (2–3 mg/kg daily for 6–8 weeks) to gain control of the disease and is then reduced. Clinicians should be aware of the significant adverse effects of high-dose and long-term use of corticosteroids in children.
- Sulfasalazine.
- Dietary management for malabsorption.
- Metronidazole for perianal disease.
- Immunomodulators:
  - Methotrexate, azathioprine.
  - γ-interferon/α-interferon.
  - Thalidomide (anti-TNF).

More recently, methotrexate, budesonide and infliximab (a monoclonal antibody that neutralizes tumour necrosis factor alpha (TNF-α)) have also been used in the management of Crohn disease. Research continues into the use of paratuberculosis therapy (rifabutin, clarithromycin and clofazimine). Thalidomide is used by some clinicians for

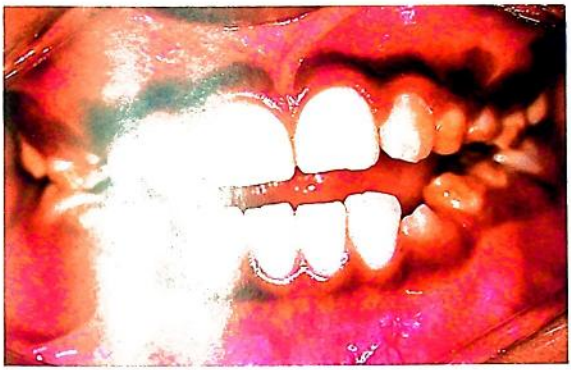


Figure 10.9 Racial pigmentation of the attached gingiva. This should not be confused with heavy metal toxicity which is limited to the marginal gingivae.

refractory adult cases, though this needs to be done with caution and female patients need a full explanation of the possible side-effects.

Melkersson–Rosenthal syndrome

Melkersson–Rosenthal syndrome is regarded by some as a form of orofacial granulomatosis.

Presentation

- Recurrent/persistent orofacial swelling.
- Facial nerve paralysis.
- Fissured (plicated) tongue.

Melkersson–Rosenthal syndrome is a diagnosis sometimes made without this triad – there is controversy regarding its existence or place within the disorders characterized by orofacial granulomata.

Pigmented, vascular and red lesions

If a lesion is red or bluish in colour there should be the suspicion of a vascular lesion. These lesions will blanch on pressure with a glass slide (or end of a test tube if access is difficult) as the blood is removed from the vessels. Melanotic lesions are rare in children (other than racial pigmentation). A characteristic melanin-pigmented oral lesion in young children is the melanotic neuroectodermal tumour of infancy.

Differential diagnosis

- Racial pigmentation (Figure 10.9).
- Vascular lesions:
  - Haemangioma.

- Other vascular malformations.
- Haematoma.
- Petechiae and purpura.
- Hereditary haemorrhagic telangiectasia.
- Sturge–Weber syndrome.

- Pigmented lesions (containing melanin):
  - Melanotic neuroectodermal tumour of infancy.
  - Peutz–Jeghers syndrome.
  - Addison's disease.
- Other red/blue/purple lesions:
  - Giant cell granuloma – peripheral (epulis) or central.
  - Eruption cyst.
  - Langerhans' cell histiocytosis.
  - Geographic tongue.
  - Median rhomboid glossitis.
  - Hereditary mucoepithelial dysplasia.
  - Cyanosis.
  - Heavy metal toxicity.

Lesions of vascular origin

Haemangioma/localized vascular anomaly (Figure 10.10A,B)
Haemangiomas are endothelial hamartomas. Typically present at birth, they may grow with the infant but then may regress with time to disappear by adolescence. As such, they require no treatment other than observation, excepting cosmetic concerns.

Other vascular malformations

Arteriovenous malformations include birthmarks, blood vessel and lymphatic anomalies. These may be life-threatening conditions, which can occasionally present with profound haemorrhage. Arteriovenous malformations have been classified by Kaban and Mulliken (1986) according to their flow characteristics. They are either:

- Low-flow lesions – capillary, venous, lymphatic or combined port-wine stains, Sturge–Weber syndrome.

Or

- High-flow lesions – arterial with arteriovenous fistulae (Figures 10.10C, 10.11). Present with mobile and sometimes painful teeth, a bruit and palpable pulses, bleeding from gingivae and bony involvement.
- Combined lesions – extensive combined venous and arteriovenous malformations.

Diagnosis

- Presentation may be subtle, such as prolonged bleeding from the gingivae after tooth brushing, or alternatively a torrential single episode of haemorrhage.
- Vascular lesions are often warm to touch (although this can be difficult to determine if wearing latex gloves).

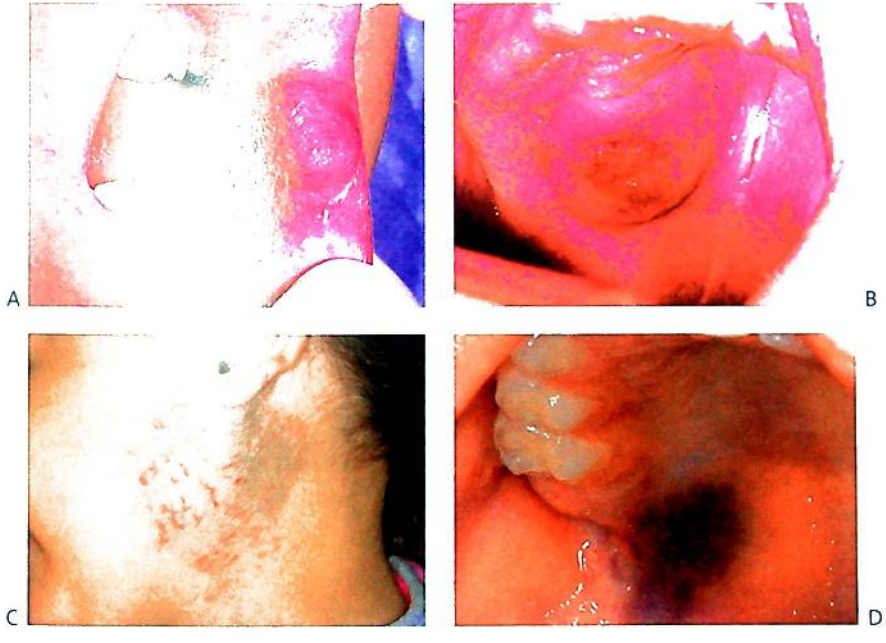


Figure 10.10 (A,B) Different presentations of haemangiomas in infants. (C) A high flow vascular malformation that is warm to touch and a bruit is palpable. (D) It is essential that the correct diagnosis of a vascular malformation is made, as there is a potential for life-threatening haemorrhage.

- Radiographically, there may be enlargement of the periodontal ligament space and a diffuse abnormal trabeculation of the bone.
- A bruit or pulse may be felt over high-flow lesions.
- Teeth may be hypermobile and may have pulsatile movements.
- Facial asymmetry may become apparent as lesions expand.
- Digital subtraction angiography (Figure 10.11C) is required for definitive diagnosis of feeder vessels and the distribution of the lesion.
- Magnetic resonance angiography can be used to aid diagnosis; however, digital subtraction angiography has the advantage that embolization can be performed at the same time.

Management

- Low-flow lesions can be removed by careful surgery with identification and ligation of feeder vessels. Larger lesions can be managed with cryotherapy, laser ablation or injection of sclerosing solutions.

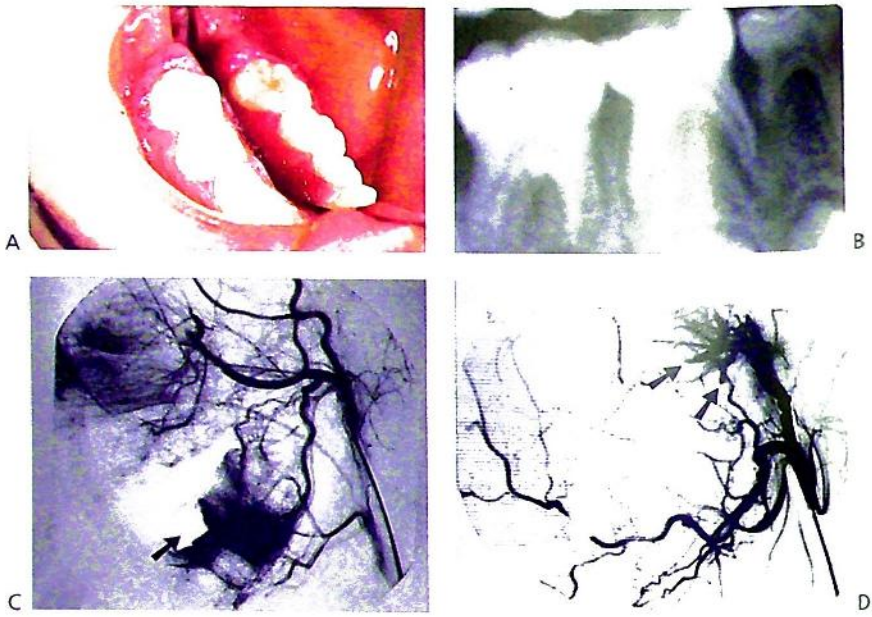


Figure 10.11 A high-flow arteriovenous malformation in the mandible. (A) This child presented with an unusual erythematous enlargement of the gingivae around the mandibular right first permanent molar in addition to mobile second primary molar. (B) Periapical radiograph shows widening of the periodontal ligament space, but there are no indicative changes in the trabecular pattern of the bone. During the biopsy 350 mL (20% of total volume) of blood was lost after extraction of the second primary molar. The haemorrhage was controlled with an iodoform pack (arrowed). (C) Subsequently, an angiogram was ordered. The extent of the lesion inferior and posterior to the pack is seen. (D) The lesion was managed by embolizing the inferior alveolar and maxillary arteries (arrows) and the mandible was resected later.

- High-flow lesions require selective embolization of vessels, but these will generally recur after embolization due to revascularization from contralateral supply and recanalization of the embolized artery. Repeat embolization and resection of the entire lesion is often necessary involving jaw reconstruction.
- If a tooth is accidentally removed and a torrential haemorrhage results, some clinicians suggest that the extracted tooth should be immediately replaced and additional measures used to control the haemorrhage. These rare but potential life-threatening events call for urgent assistance and response.

A

B

C

Figure 10.12 (A) Lymphangioma in the palate. (B) A lesion involving the tongue. (C) A large cystic hygroma that was managed with cryosurgery.

Clinical hint

Vascular lesions blanch on pressure.

Lymphangioma (Figure 10.12)

- Diagnosis of developmental lymph vessel abnormalities must exclude vascular involvement. Surgical excision is only necessary if of functional or aesthetic concern.
- **Cystic hygroma** (Figure 10.12C) is a term used to describe a large lymphangioma involving the tongue, floor of mouth and neck. Expansion of the lesion may cause respiratory obstruction and treatment usually involves multiple resections over time, management with laser ablation or cryosurgery.

Petechiae and purpura

Petechiae are small pinpoint submucosal or subcutaneous haemorrhages. Purpura or ecchymoses present as larger collections of blood. These lesions are usually present in patients with severe bleeding disorders or coagulopathies, leukaemia and other

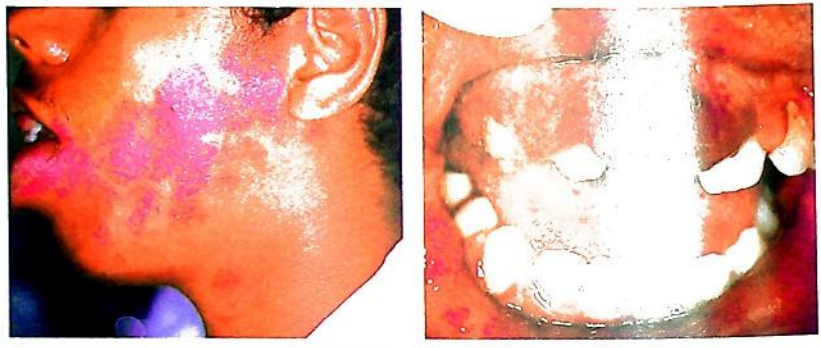


Figure 10.13 (A,B) Sturge–Weber syndrome showing the extent of the capillary vascular malformation in the face which is contiguous with the intra-oral involvement.

conditions such as infective endocarditis. Initially bright red in colour, they will change to a bluish-brown hue with time as the extravasated blood is metabolized.

Hereditary haemorrhagic telangiectasia (Rendu–Osler–Weber disease)

An autosomal dominant disorder presenting as a developmental anomaly of capillaries. Lesions may be small, flat or raised haemorrhagic nodules or spider naevi. Bleeding as a result of involvement of the gastrointestinal tract and respiratory tract can lead to chronic anaemia.

Sturge–Weber syndrome (Figure 10.13)

This syndrome typically presents with:

- Encephalotrigeminal angiomatoses.
- Epilepsy.
- Intellectual disability.
- Calcification of the falx cerebri.
- Vascular lesions involve the leptomeninges and peripheral lesions appear along the distribution of the 5th nerve.

Extraction of teeth within regions of the affected jaws should be performed with caution and only after thorough investigation of the extent of the anomaly, although it has been reported that they are usually uncomplicated (see other vascular malformations above).

Maffucci syndrome

Presents with multiple haemangiomas and enchondromas of the small bones in the hands and feet. Only a small number of cases will manifest with oral lesions, mainly haemangiomas.

Figure 10.14 Eruption cysts may be associated with any tooth. (A) Two eruption cysts associated with the maxillary central incisors. (B) Eruption cyst of the maxillary first permanent molar.

Melanin-containing lesions

Peutz–Jeghers syndrome

An autosomal dominant disorder manifesting as multiple small, pigmented lesions of oral mucosa and circum-oral skin appearing almost like dark freckles. There is an important association with intestinal polyposis coli which requires gastrointestinal investigation.

Red lesions

Eruption cyst or haematoma

Follicular enlargement appearing just prior to eruption of teeth. These lesions tend to be blue-black as they may contain blood. They usually require no treatment unless infected. The parents and child should be reassured and the follicle allowed to rupture spontaneously or it may be surgically opened if infected (Figure 10.14).

Hereditary mucoepithelial dysplasia

A rare disorder where there is a reduced number of desmosomes attaching the epithelial cells to each other. Lens cataracts, corneal lesions leading to blindness, skin keratosis and alopecia are associated with a fiery red mucosa involving both keratinized and non-keratinized mucosa. Diagnosis is confirmed by gingival and/or mucosal biopsy. Transmission electron microscopy is necessary to demonstrate the reduced number of desmosomes and amorphous intracellular inclusions. The oral lesions are usually asymptomatic. Loss of sight is progressive due to corneal vascularization. Corneal grafts are unsuccessful, as they too undergo vascularization.

Geographic tongue (Figure 10.15A)

This condition is also termed glossitis migrans, benign migratory glossitis, erythema migrans or 'wandering rash of the tongue'. It presents as areas of depapillation and

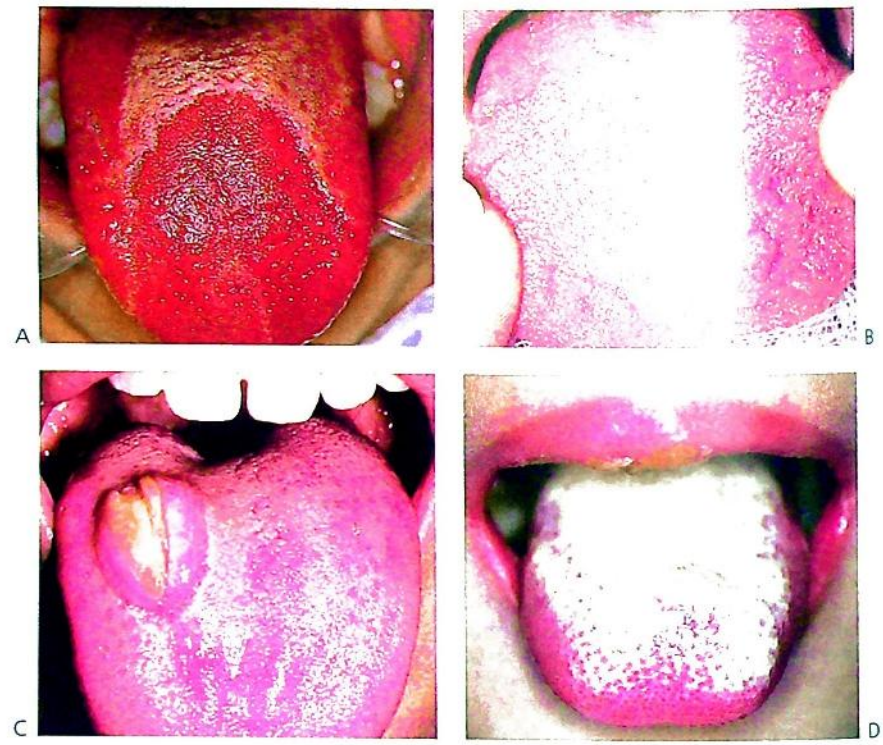


Figure 10.15 Lesions of the tongue. (A) Geographic tongue. (B) Fissured tongue associated with mild geographic tongue. (C) A large swelling (an ulcerated fibroepithelial hyperplasia) on the dorsal surface of the tongue with a central area of ulceration caused by a palatal expansion appliance (quad helix). (D) Acute pseudomembranous candidosis or thrush with typical white plaques on the dorsum of the tongue in an immunocompromised child.

erythema with a heaped 'serpentine-like', keratinized margin on the lateral margins and dorsal surface of the tongue. It can be associated with a fissured tongue. The lesions appear as map-like areas (hence the 'geographic') and may change in their distribution over a period of time (hence the 'migratory'). The areas affected may return to normal and new lesions appear at different sites on the tongue. Sometimes symptomatic, topical corticosteroids may be beneficial for those children in pain.

Fissured tongue (Figure 10.15B)
Also termed plicated tongue, scrotal tongue, fissured tongue or lingua secta. The tongue in these patients is fissured, the fissures being perpendicular to the lateral border. Although this is usually considered a variation of normal, it is a commonly found condition in children with Down syndrome. Some patients with a fissured tongue will also have geographic tongue. Fissuring of the tongue is also a feature of Melkersson–Rosenthal syndrome.

Epulides and exophytic lesions

Differential diagnosis

- Inflammatory hyperplasias:
  - Pyogenic granuloma.
  - Fibrous epulis.
  - Giant cell granuloma – central or peripheral (epulis).
- Congenital epulis of the newborn.
- Squamous papilloma/viral wart.
- Focal epithelial hyperplasia/Heck's disease.
- Condyloma acuminatum.
- Eruption cyst/haematoma.
- Melanotic neuro-ectodermal tumour of infancy.
- Tuberous sclerosis.
- Mucocoele.
- Lymphangioma.

Pyogenic granuloma

This misnamed lesion (i.e. no pus and no granulomas) is actually an ulcerated lobulated capillary haemangioma. Hence, these lesions tend to bleed easily and are covered with a thin fibrin membrane and may recur if not fully excised. They should be completely excised.

Fibrous epulis (Figure 10.16A)

One of the most common epulides that is seen in children resulting from an exuberant fibroepithelial reaction to plaque. Commonly arising from the interdental papillae and covered by epithelium, they range in colour from pink to red to yellow. Those appearing yellow are ulcerated.

Management

Oral hygiene and surgical excision. Lesions may recur, particularly if good oral hygiene is not maintained.

Clinical Hint

Fibrous epulides involve the marginal gingivae.

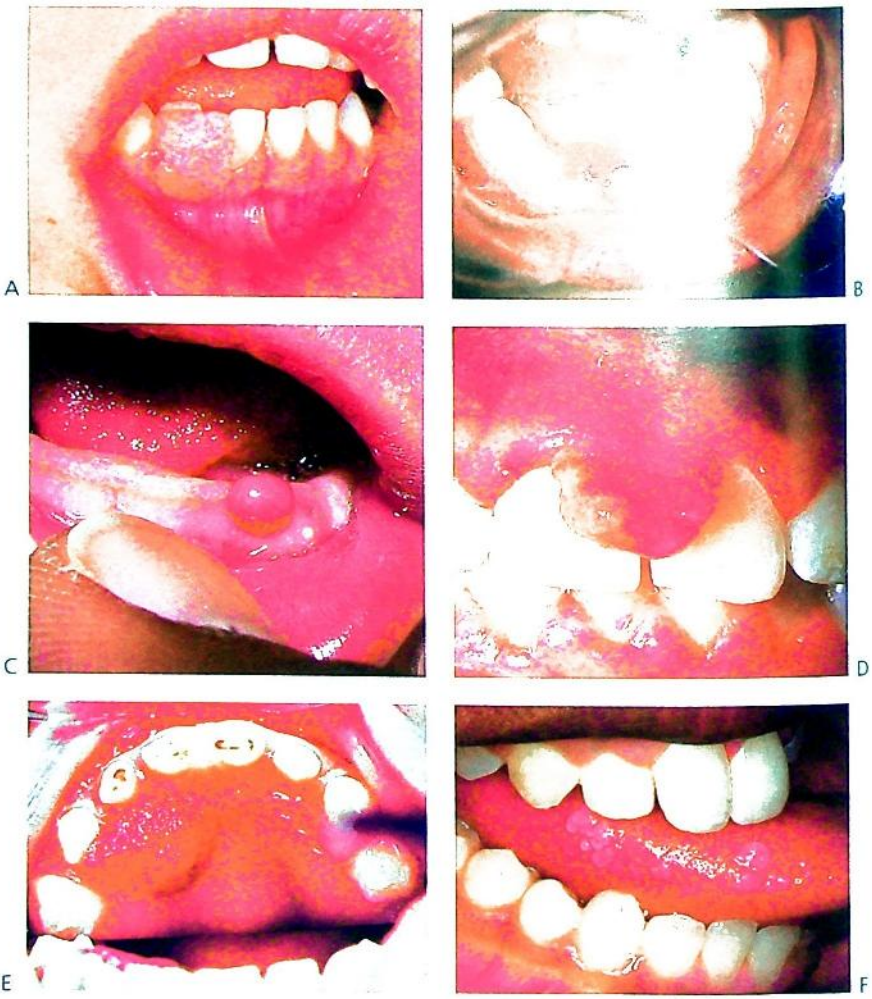


Figure 10.16 Gingival swellings. (A) Fibrous epulis (B) Peripheral giant cell granuloma/giant cell epulis. (C) Congenital epulis. (D) Epulis associated with angiomata in a child with tuberous sclerosis. (E) A papilloma in the palate of a young child. In this case the lesion was associated with viral warts on the extremities, hence this is assumed to be a viral papilloma. (F) Focal epithelial hyperplasia with the characteristic lesions on the slide of the tongue. *(Courtesy Dr Charmaine Hall, Royal Children's Hospital, Melbourne.)*

Giant cell granuloma – central or peripheral (epulis) (Figure 10.16B)

These lesions usually occur in the region of the primary dentition. The colour of these lesions tends to be dark purple. Bone loss of the alveolar crest can sometimes be observed as 'radiographic cupping'. It is important to ensure that there is no intra-osseous component radiographically as in this case the diagnosis would be a central giant cell granuloma. As with all giant cell lesions of the jaws, hyperparathyroidism should be considered in the differential diagnosis. True central lesions are typically aggressive and local resection may be required.

Management

- Surgical excision.
- Haematological investigations for calcium, phosphate, alkaline phosphatase and parathyroid hormone.
- Lesions may regrow if not totally excised.

Squamous papilloma (Figure 10.16E)

A squamous papilloma is a benign neoplasm caused by the human papilloma virus (HPV), presenting as a cauliflower-like growth on the mucosa. The colour of the lesion depends on the degree of keratinization.

Management

Surgical excision, including the stalk and a border of normal tissue.

Viral warts (verruca vulgaris)

This is the cutaneous form of the HPV infection. Lesions may be single or multiple and may appear similar to the papilloma or as the common wart seen on the hands and fingers.

Management

Surgical excision. If multiple lesions are present extra-orally, dermatological management may also be required.

Heck's disease (focal epithelial hyperplasia)

These pale white multiple exophytic lesions, commonly appearing on the side of the tongue or lips are also associated with HPV infection. In some children, there is a genetic predisposition to those who may be affected (types 13 and 32).

Congenital epulis (Figure 10.16C)

The congenital epulis is a rare benign lesion of unknown origin found only in neonates. Lesions are equally distributed between maxillary and mandibular arches and may be multiple in about 10% of reported cases; they are 10 times more common in girls than in boys. It arises from the gingival crest but is thought not to be odontogenic in origin. The swelling is characterized by a proliferation of mesenchymal cells with a granular cytoplasm and is usually pedunculated. There is controversy over the histogenesis of this lesion. The congenital epulis is histologically indistinguishable from the extra-gingival granular cell tumour, which is usually seen on the tongue. Immunohistochemically, the congenital epulis is S100 negative, whereas the granular cell tumour

is positive for both CD68 and S100. It has been suggested that the congenital epulis is a non-neoplastic, perhaps reactive, lesion arising from primitive gingival perivascular mesenchymal cells with the potential for smooth muscle cytodifferentiation.

Alternative terminology
Congenital granular cell tumour, Neumann tumour, granular cell epulis, gingival granular cell tumour.

Management
Lesions often regress with time, although large lesions which interfere with feeding may require surgical excision. Large lesions are sometimes present at birth and may be life-threatening because of respiratory obstruction. The eruption of the primary dentition is unaffected by either surgical or conservative management, and recurrence is uncommon.

Tuberous sclerosis (Figure 10.16D)
Tuberous sclerosis is an autosomal dominant disorder characterized by seizures, mental retardation and adenoma sebaceum of the skin. Epulides or generalized nodular gingival enlargement may be present. Some of these may result from vascular malformations and may bleed profusely when excised. Hypoplasia of the enamel is often observed as surface pitting; this can be demonstrated particularly effectively by the use of disclosing solution.

Gingival enlargements (overgrowth)

Differential diagnosis
- Drug-induced hyperplasia:
  - Phenytoin.
  - Cyclosporin A.
  - Nifedipine.
  - Verapamil.
- Syndromes with gingival enlargement as a presenting feature:
  - Hereditary gingival fibromatosis.
  - Zimmerman–Laband syndrome and other rare, usually autosomal recessive, syndromes.

Phenytoin enlargement (Figure 10.17A,B)
Not all patients taking phenytoin have gingival enlargement. Principally, there is enlargement of the interdental papillae. There may be delayed eruption of teeth because of the bulk of fibrous tissue present and ectopic eruption. Overgrowth has been suggested to result from decreased collagen degradation and phagocytosis, as well as increased collagen synthesis. Withdrawal of the drug will bring about resolution in all but severe cases. Oral hygiene is most important in controlling overgrowth, as there is always a component of plaque-induced gingival enlargement.

Management
- Maintenance of oral hygiene.
- Use of chlorhexidine 0.2% mouthwashes.
- Gingivectomy may be required to allow eruption of teeth or for aesthetics.

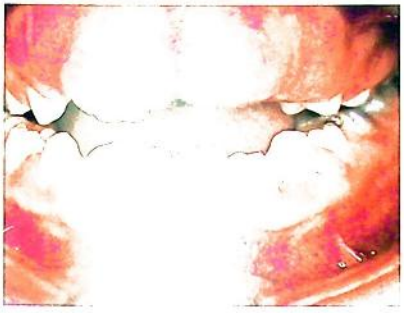
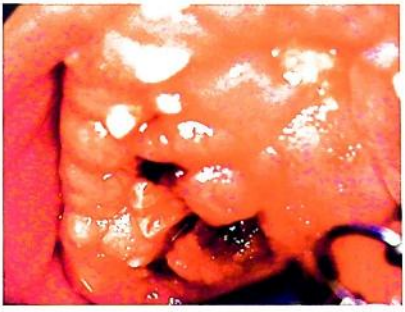
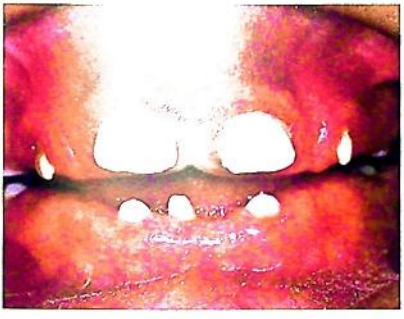
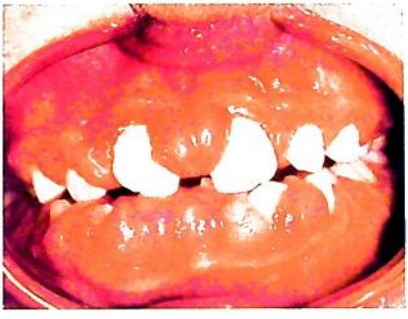
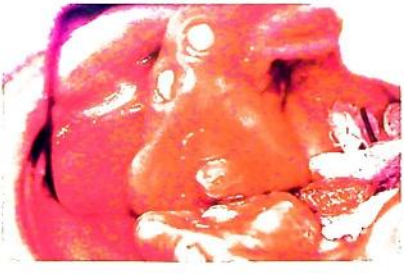






Figure 10.17 (A) Phenytoin-associated gingival enlargement. This initially involves the interdental papillae and then adjacent tissues. (B) The phenytoin-associated gingival enlargement can become so extensive as to cover the whole palate. An acute periodontal abscess was associated with this case. (C) Cyclosporin A-associated gingival enlargement in a child following end-stage renal failure and kidney transplantation. The enamel is malformed due to the effects of the renal disease. (D) Cyclosporin A-associated gingival enlargement in a child following heart transplantation. (E) Hereditary gingival fibromatosis.

Cyclosporine A-associated enlargement (Figure 10.17C,D)

A significant number of children now undergo kidney, liver, heart or combined heart/lung transplantation. The mainstay of immunosuppressive anti-rejection chemotherapy is cyclosporin A. Gingival overgrowth occurs in between 30% and 70% of patients and is not strictly dose-related but may be more severe if the drug is administered at an early age. Individual patients appear to have a threshold below which gingival overgrowth will not occur. Overgrowth appears to be higher in those HLA B37 positive patients and lower in HLA DR1 positive patients.

Nifedipine and verapamil enlargement

Both these drugs are calcium-channel blockers used to control coronary insufficiency and hypertension in adults; their main use in children is to control cyclosporin-induced hypertension after transplantation. An increase in the extra-cellular compartment volume is responsible for enlargement that occurs in addition to the enlargement caused by cyclosporin A, which is invariably used in these patients.

Management

- As with phenytoin enlargement, maintenance of oral hygiene is mandatory.
- Gingivectomy if required.
- In children with severe enlargement, a full mouth procedure may be required. In these cases, periodontal flap procedures are preferable as primary closure can be achieved.

Hereditary gingival fibromatosis (Figure 10.17E)

Gingival enlargement may be a feature of several syndromes, some of which include learning disabilities. These syndromes may occur sporadically or as an autosomal dominant or an autosomal recessive trait.

Management

Gingivectomy or periodontal flap procedures as required to allow tooth eruption and maintain aesthetics. Histopathological examination of the excised tissue may assist in diagnosis of some of the rarer causes of syndromic gingival enlargement (e.g. juvenile hyaline fibromatosis).

Premature exfoliation of primary teeth

Premature loss of primary teeth is a significant diagnostic event. Most conditions that present with early loss are serious and a child presenting with unexplained tooth loss warrants immediate investigation. Teeth may be lost because of metabolic disturbances, severe periodontal disease, connective tissue disorders, neoplasia, loss of alveolar bone support or self-inflicted trauma.

Differential diagnosis

- Neutropenias:
  - Cyclic neutropenia.
  - Congenital agranulocytosis.

- Qualitative neutrophil defects:
  - Prepubertal periodontitis.
  - Juvenile periodontitis.
  - Leucocyte adhesion defect.
  - Papillon–Lefèvre syndrome.
  - Chèdiak–Higashi disease.
  - Acatalasia.
- Metabolic disorders:
  - Hypophosphatasia.
- Connective tissue disorders
  - Ehlers–Danlos syndrome (Types IV and VIII).
  - Erythromelalgia
  - Acrodynia.
  - Scurvy.
- Neoplasia:
  - Langerhans' cell histiocytosis.
  - Acute myeloid leukaemia.
- Self-injury (see Chapter 13):
  - Hereditary sensory neuropathies.
  - Lesch–Nyhan syndrome.
  - Psychotic disorders.

Periodontal disease in children (Figure 10.18D)

Although gingivitis is not uncommon in children, periodontitis with alveolar bone loss is usually a manifestation of a serious underlying immunological deficiency. Two forms of periodontal disease in children, prepubertal periodontitis and juvenile periodontitis, are associated with characteristic bacterial flora including *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia*, *Eikenella corrodens* and *Capnocytophaga sputigena*. The presence of these bacteria is thought to be related to decreased host resistance, specifically neutropenia or neutrophil function defects. Although B-cell defects show few oral changes, altered T-cell function will manifest with severe gingivitis, periodontitis and candidosis.

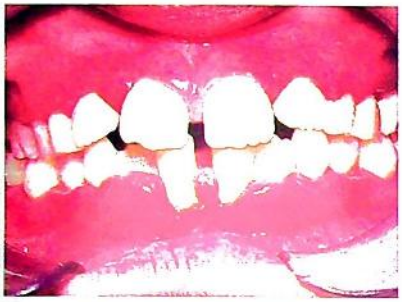
Classification of periodontal diseases

Table 10.3 details the new terminology used to describe the different periodontal diseases. The new terminology for pre-pubertal periodontitis is now *generalized aggressive periodontitis* or *periodontitis associated with systemic disease*. However, in children, it is important to understand that ANY periodontal disease in a young child is associated with some form of immune dysfunction. While any classifications should aid in the description of a particular disease entity, it is essential for clinicians to understand what the different presentations and the pathogenesis of the disease.

Neutropenias and qualitative neutrophil defects

Neutropenia

- Peripheral blood levels <1500/mL.
- Acute forms usually fatal.



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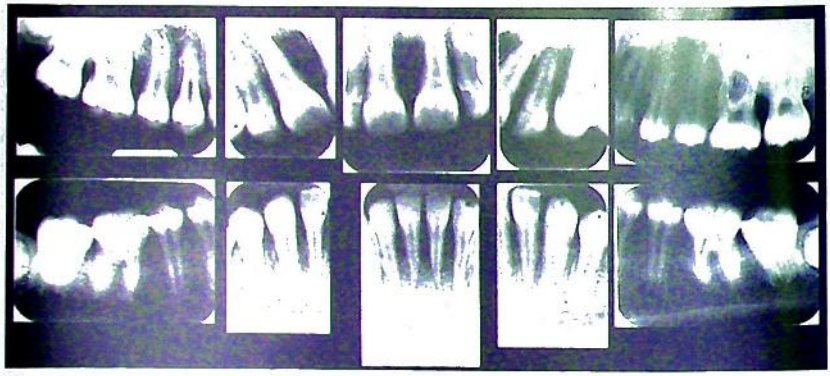

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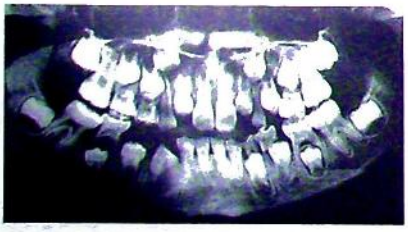

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Figure 10.18 (A) Gross gingival inflammation in an adolescent with cyclic neutropenia. This girl lost most of her primary teeth by the age of 7 years. (B) Periapical radiographic survey showing the extent of bone loss and angular defects in another child with cyclic neutropenia. (C) Severe unexplained palatal ulceration in a child with a leucocyte adhesion defect. The maxillary left incisor exfoliated a short time later. (D) Prepubertal periodontitis, also associated with a leucocyte adhesion defect. It is important to assess normal eruption patterns and to be suspicious of loss of teeth in the absence of caries or other pathology. (E) Acute necrotizing ulcerative gingivitis (ANUG) in a 15-year-old boy, showing the characteristic destruction of the interdental papilla. ANUG is rare in children and only seen in those who are immunocompromised to debilitated.

Table 10.3 Terminology used to classify periodontal diseases

Old terminology	Current terminology
Pre-pubertal periodontitis	Generalized aggressive periodontitis Periodontitis associated with systemic disease
Localized juvenile periodontitis	Localized aggressive periodontitis
Acute necrotizing ulcerative gingivitis (ANUG)	Necrotizing periodontal disease

Research, Science and Therapy Committee, 2003. Periodontal diseases of children and adolescents. American Academy of Periodontology. Journal of Periodontology 74, 1696–1704.

- Chronic forms have indolent progression.
- Cyclic (see below) characterized by recurrent episodes.
- Intermittent as part of Shwachman–Diamond syndrome.

Cyclic neutropenia (Figure 10.18A,B)

In this condition, there is an episodic decrease in the number of neutrophils every 3–4 weeks. Peripheral neutrophil counts usually drop to zero and during this time, the child is extremely susceptible to infection. Recurrent oral ulceration often occurs when cell counts are low. Gingival and periodontal involvement occurs with the emergence of teeth and is progressive.

Management

- Early preventive involvement.
- Dental care through all stages of cycle.
- Chlorhexidine 0.2% mouthwashes or gel.
- Elective extraction of primary teeth may be considered in severe cases.
- In some familial cases, the condition appears to totally regress during adolescence.

Leucocyte adhesion defect (Figure 10.18C,D)

A rare autosomal recessive condition associated with a reduced level of adhesion molecules on peripheral leucocytes resulting in severely reduced resistance to infection. The CD11/CD18 molecules are necessary for effective phagocytosis. Children present with delayed wound healing, persistent severe oral ulceration, cellulitis without pus formation, severe gingival inflammation, periodontitis and premature loss of primary teeth. Also present is a persistently high leucocytosis and reactive marrow, without evidence of leukaemia. One important indicator of this condition is late separation of the umbilical cord after birth.

Diagnosis

Diagnosis is confirmed by examining leucocytes for surface expression of CD11/CD18 markers using immunofluorescence techniques and cytofluorographic analysis.

Management

- Many children succumb to overwhelming infection.
- Granulocyte transfusion and bone marrow transplantation may be effective in some cases.

Papillon–Lefèvre syndrome (Figure 10.19)

An autosomal recessive condition manifesting as hyperkeratosis of the palms and feet and progressive exfoliation of all teeth from periodontal disease. *A. actinomycetemcomitans* has been implicated in the periodontal disease which is associated with a qualitative neutrophil defect and mutations in the lysosomal protease cathepsin C gene on 11q14–21. Primary teeth commence shedding from the time of eruption, with no evidence of root resorption. All primary teeth are usually lost before the permanent teeth erupt, when they in turn are exfoliated.

Diagnosis

- The oral changes and skin lesions are pathognomonic for this condition.
- Selective anaerobic culturing for *A. actinomycetemcomitans* is difficult, and a more reliable alternative is to use an enzyme-linked immunosorbent assay (ELISA) to detect IgG antibodies against this organism.

Management

No treatment is particularly successful. Extraction of any remaining primary teeth before eruption of the permanent teeth has been advocated. Intensive periodontal therapy with metronidazole and chlorhexidine to eliminate or reduce *A. actinomycetemcomitans* may be successful in delaying the inevitable exfoliation of teeth, although the basic neutrophil function defect remains. Treatment of other family members has also been recommended, including pets (especially dogs) if they are found to harbour the bacteria. Several papers have reported the use of vitamin-A derivatives in management that may improve the prognosis. All patients require planned full clearances and dentures to avoid pain and disfigurement. It is important to consider proceeding with extractions soon after the eruption of the permanent dentition to minimize excessive bone loss.

Chédiak–Higashi disease

This is a rare autosomal recessive disorder affecting lysosomal storage and causing a qualitative neutrophil defect. There is defective neutrophil chemotaxis and abnormal degranulation that results in poor intracellular killing. Abnormal B-cell and T-cell function and thrombocytopenia have also been reported. Most children die by 10 years of age because of overwhelming sepsis. Teeth are shed because of severe periodontal disease with rapid alveolar bone loss.

HIV-associated periodontal disease in children

There are few cases documenting periodontal disease in young children. In adolescents, there are reports of acute necrotizing ulcerative gingivitis (ANUG) and a characteristic linear marginal gingival erythema. Excellent oral hygiene and plaque control are essential, combined with supportive therapy including chlorhexidine and metronidazole as required.

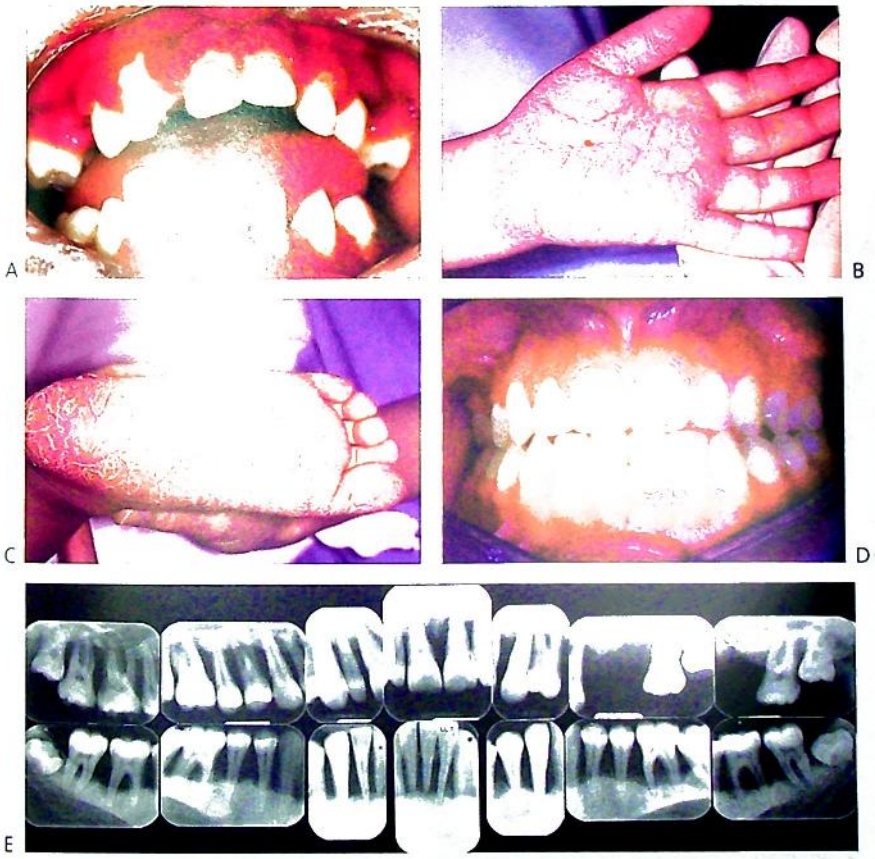


Figure 10.19 Papillon–Lefèvre syndrome. (A) The initial presentation of the child with severe periodontal disease-associated tooth mobility. Many of these teeth exfoliated within 6 months. (B,C) The characteristic appearance of the hands and feet in the same child. (D,E) The permanent dentition in a boy of 17 years, following long-term antibiotic treatment that has failed to improve the prognosis of the dentition.

Langerhans' cell histiocytosis (Figure 10.20)

This condition was previously termed histiocytosis X and included the conditions eosinophilic granuloma, Hand–Schüller–Christian disease and Letterer–Siwe disease. The abnormality in common is a proliferation of histiocytes. Oral lesions characteristically occur in all four quadrants and characteristically affect the tissues overlying or supporting the primary molar teeth. The lesions typically extend forward to the canines, but rarely involve the incisors.

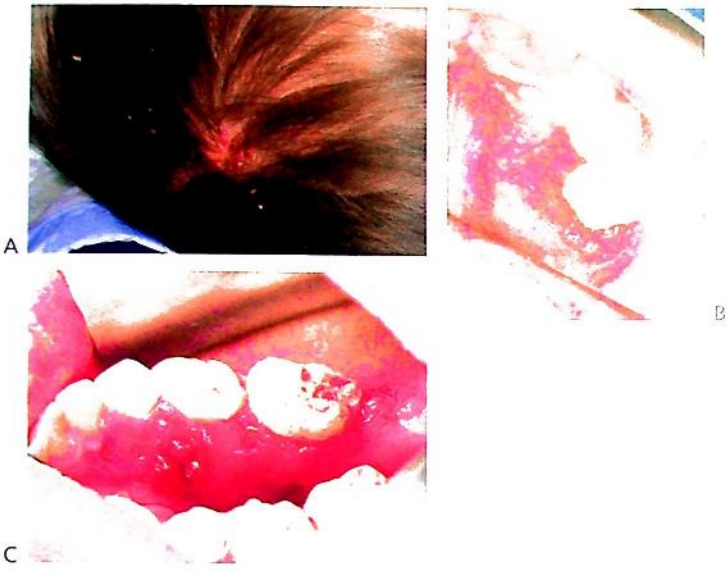


Figure 10.20 (A) Cradle cap-like rash on the head and severe ano-vulval or ano-perineal rash are common presentations of disseminated Langerhans' cell histiocytosis (LCH). LCH characteristically presents intraorally with lesions in all four quadrants (B) and (C). All the posterior teeth were excessively mobile and retained only by soft tissue. Note the perforation of the lesions through the lingual alveolus (C).

Presentation

- Malaise, irritability.
- Anogenital and postauricular rash.
- Diabetes insipidus.
- Premature exposure by alveolar resorption and subsequent loss of primary teeth, especially molars.
- Radiographically, teeth appear to be 'floating in air'.
- Typically, all four quadrants are involved.

Diagnosis

- Biopsy of oral or skin lesions – cells positive for S100 and CD1a.
- Transmission electron microscopy of Langerhans' cells shows characteristic Birbeck granules.

Management

- Excision and curettage of oral lesions and extraction of involved teeth is required to control oral lesions.
- Multiagent chemotherapy is required for disseminated disease and is most effective if commenced early.

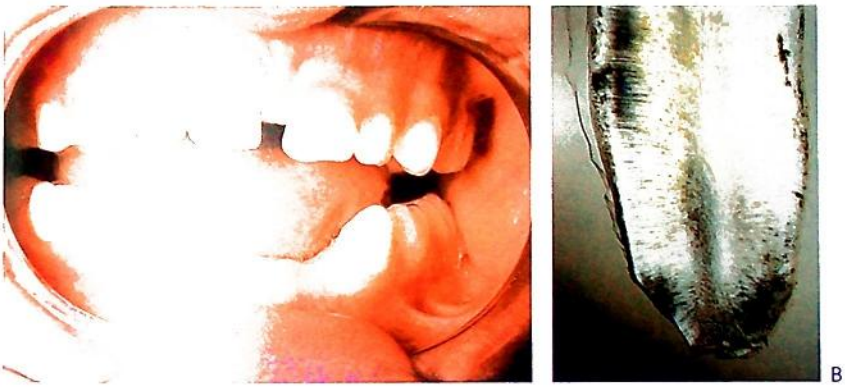


Figure 10.21 (A) Hypophosphatasia presenting with exfoliation of the maxillary and mandibular anterior teeth around 2 years of age. There is minimal gingival inflammation and hard-tissue sections (B) show absence of cementum.

Metabolic disorders

Hypophosphatasia (Figure 10.21)

A decrease in serum alkaline phosphatase and an increase in the urinary excretion of phosphoenolamine (PEA) are pathognomonic for hypophosphatasia. The more usual form is transmitted as an autosomal dominant trait, whereas the autosomal recessive form is invariably lethal. Loss of at least some of the incisor teeth usually occurs before 18 months. Several authors have identified groups of children who manifest only dental changes, namely the early loss of teeth without any rachitic bone changes – the term 'odontohypophosphatasia' has been suggested for these patients but this is inappropriate as the presentation of loss of teeth alone is only one end of the spectrum in the variable expression of this disease. In these children there are less severe changes and we have observed that the permanent dentition can be unaffected.

Diagnosis

- Serum alkaline phosphatase level <90 U/L. The normal range is 80–350 U/L, however, growing children often have levels well in excess of these values (>400 U/L).
- Urinary PEA and serum pyridoxal-5-phosphate (vitamin B_6) tests are required to confirm the diagnosis. A skeletal survey of the long bones is necessary as rachitic changes may be present in severe cases.
- Sections of the exfoliated teeth show abnormal or absent cementum.

Ehlers–Danlos type IV and type VIII

These inborn errors of metabolism present as disorders of collagen formation. Typically, there is hyperextensibility of skin with capillary fragility, bruising of the skin and hypermobility of the joints. Types IV and VIII may present with dental complications, mainly progressive periodontal disease leading to the loss of teeth.

Erythromelalgia
A very rare condition, characterized by sympathetic overactivity, causing an endarteritis and the extremities feeling hot. One case has been reported with loss of primary and permanent teeth at 4 years of age. The child had extreme hypermobility of joints and slept on a tiled floor in the middle of winter because of the heat in her legs. She also had an unexplained tachycardia of 200 beats per minute and presented a diagnostic dilemma for many months. The teeth exfoliated because of necrosis of alveolar bone.

Acrodynia (pink disease)
Mercury toxicity causes alveolar destruction and sequestration. Extremely rare now, although in the past it was not uncommon with the use of teething powders containing mercury.

Acatalasia
Autosomal recessive catalase deficiency in neutrophils leading to periodontal destruction. Extremely rare outside Japan.

Scurvy
Almost unknown today, this nutritional deficiency of vitamin C results in a connective tissue disorder. Tooth loss is due to a failure of proline hydroxylation and consequent reduced collagen synthesis.

Oral pathology in the newborn infant

Differential diagnosis
- Keratin cysts of the newborn:
  - Epstein's pearls.
  - Bohn's nodules.
- Congenital epulis of the newborn.
- Granular cell tumour (granular cell myoblastoma).
- Melanotic neuro-ectodermal tumour of infancy.
- Natal/neonatal teeth.

Cysts in the newborn (Figure 10.22A)
Epstein's pearls
These hard, raised nodules are small keratinizing cysts arising in epithelial remnants trapped along lines of fusion of embryological processes. They appear in the midline of the hard palate, most commonly posteriorly.

Bohn's nodules
These are remnants of the dental lamina and usually occur on the labial or buccal aspect of the maxillary alveolar ridges.

Management
No treatment is required other than reassurance of the parents.

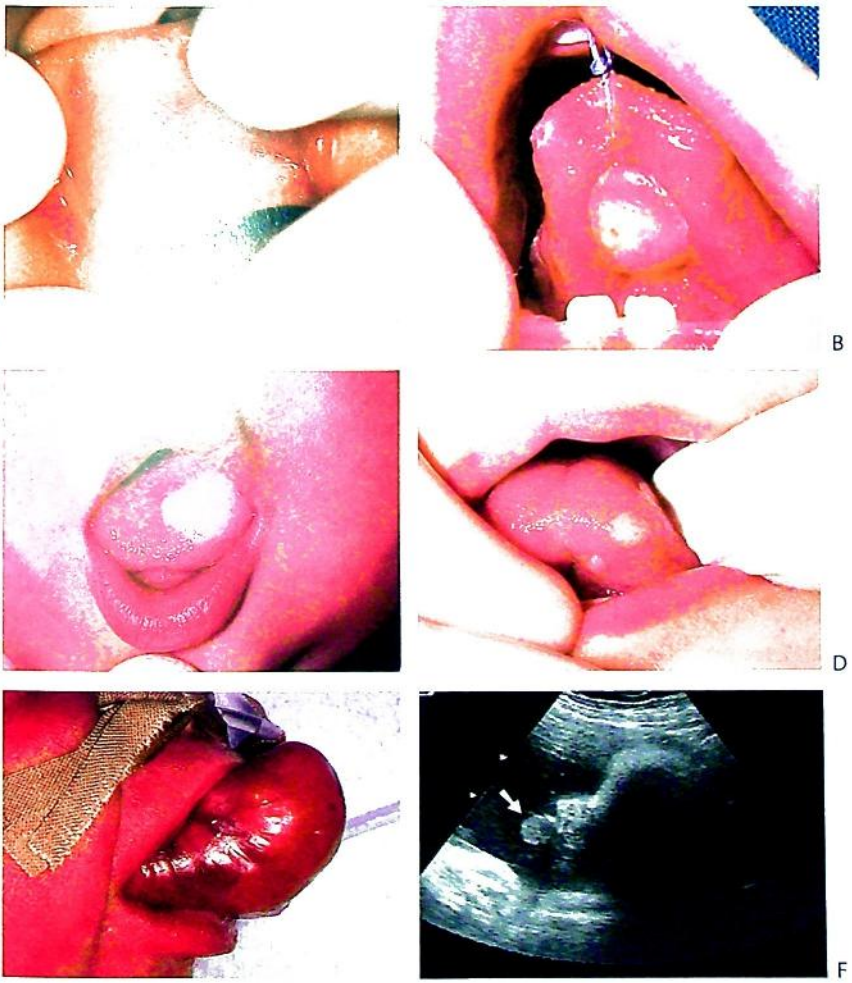


Figure 10.22 Oral pathology in infants. (A) Bohn's nodules in a newborn child (arrow). (B) Fibroepithelial hyperplasia on the ventral surface of the tongue caused by trauma from the erupting mandibular incisors. (C) White sponge naevus of the tongue. (D) Granular cell tumour. (E) Congenital epulis measuring 4 cm in length. (F) The lesion was diagnosed prenatally on ultrasound (arrow).

Melanotic neuroectodermal tumour of infancy

A rare but important paediatric tumour derived from neural crest cells, this occurs predominantly in the maxilla. The condition may be present at birth and all recorded cases have been diagnosed by 4 months of age. Similar to a neuroblastoma, there may be high levels of vanillylmandelic acid (a catecholamine end-product) in the urine. Lesions may be multicentric with close approximation to, but not involving, dental tissues, reflecting their ectomesenchymal origin. Intra-orally, lesions appear as circumscribed swellings that may have the appearance of normal mucosal or have a blue-black hue and may be associated with premature eruption of the primary incisors.

Diagnosis

Computed tomography followed by excisional biopsy because of the extremely rapid growth of the lesion. This condition is usually so unique, at this age and at this site, that diagnosis is not difficult.

Management

- Enucleation of the tumour and involved primary teeth.
- Curettage of the bony floor of the multiple cavities.
- Radiotherapy is contraindicated.
- Recurrences are extremely rare.

Diseases of salivary glands

Differential diagnosis

- Mucocoele.
- Ranula.
- Sialoliths.
- Mumps.
- Autoimmune parotitis.
- Bilateral parotitis associated with bulimia (sialosis).
- Aplasia (or hypoplasia) of major salivary glands.

Mucocoele (Figure 10.23A,B)

Mucous extravasation cyst

The most common mucous cyst in the oral cavity, the mucous extravasation cyst arises from damage to the duct of one of the minor salivary glands in the (lower) lip or cheek. Often caused by lip biting or other minor injuries, mucus builds up in the connective tissue to become surrounded by fibrous tissue. Most mucocoeles are well-circumscribed bluish swellings, although traumatized lesions may have a keratinized surface.

Management

- Some cysts regress spontaneously.
- Surgical excision, ideally together with the associated minor salivary glands.

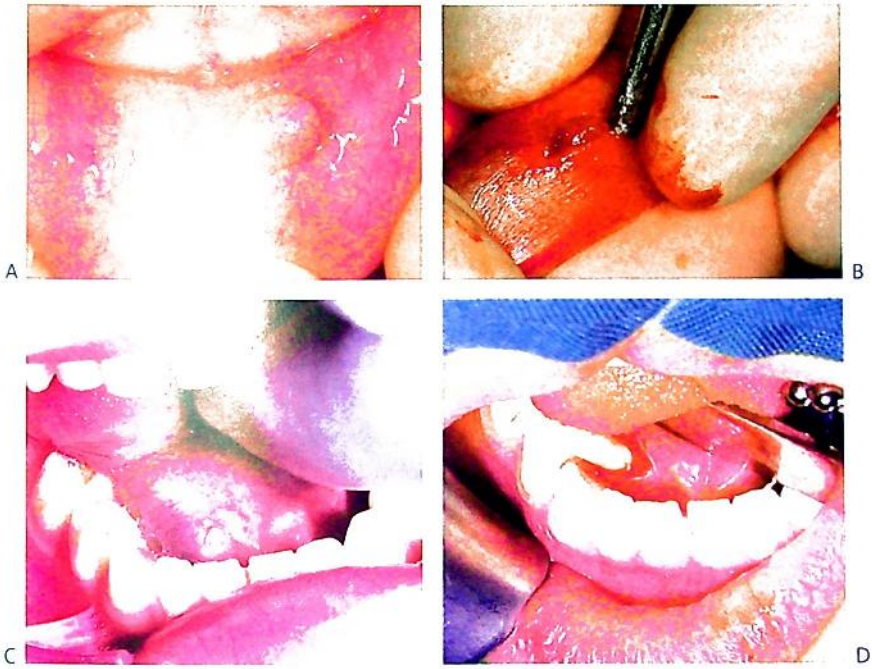


Figure 10.23 (A) Typical presentation of a mucocoele on the lower lip. Most lesions require removal. (B) Semilunar incision with exposure of cyst which may be removed with blunt dissection. (C) A mucous cyst of the floor of the mouth – a ranula. (D) Insertion of a Penrose drain lateral to Wharton's duct to marsupialize the cyst.

Mucous retention cyst

Less common than the mucous extravasation cyst, the retention cyst has a similar or identical clinical appearance but is lined by epithelium. More common in the upper lip and palate, whereas the extravasation cyst is more typical of the lower lip.

Ranula (Figure 10.23C)

A mucous (extravasation) cyst of the floor of the mouth caused by damage to the duct of either the sublingual or submandibular glands. A soft, bluish swelling presents on one side of the floor of the mouth. A plunging ranula occurs when the lesion herniates through the mylohyoid muscle to involve the neck.

Management

- Surgical excision.
- Large lesions may require marsupialization in the first instance.

Sialadenitis

Inflammation of the major salivary glands may result from:

- Viral infection:
  - Mumps or cytomegalovirus infection. Present with bilateral non-suppurative parotitis, usually epidemic.
  - Human immunodeficiency virus (HIV) infection and AIDS; 10–15% of children with AIDS will manifest bilateral parotitis.
- Bacterial infections:
  - Suppurative, usually retrograde infection.
- Autoimmune:
  - Sjögren syndrome (usually seen in older patients).
  - Bilateral autoimmune parotitis. Punctate sialectasis appearance on sialogram.
- Bulimia:
  - A non-tender salivary gland enlargement is a common presentation of bulimia nervosa.
- Chronic sialadenitis:
  - Usually caused by unilateral obstruction of a major salivary gland, either by stricture, epithelial plugging or a sialolith (calculus) causing obstruction and inflammation. Pain occurs during eating and if there is acute exacerbation of infection.

Clinical Hint

A periodontal probe is useful for *gently* exploring the terminal part of a major salivary gland duct. This can identify a small calculus or dislodge an epithelial plug. This should not be attempted in younger and/or anxious patients.

Management

- Massage of the gland/milking of the duct for cases of recurrent epithelial plugging.
- Antibiotics to control infection in the acute phase.
- Removal of sialolith. *Note*: a suture should be passed under the duct behind the sialolith to prevent it being displaced backwards.
- In longstanding cases of obstructive sialadenitis, removal of the gland may be necessary.

Salivary gland tumours

Most tumours of the salivary glands in children are vascular malformations. Pleomorphic adenomas are uncommon. Malignant neoplasms such as mucoepidermoid carcinoma, adenocarcinoma and sarcoma are extremely uncommon and affect mainly older children and adolescents. The parotid is the most common site for such tumours.

Diagnostic imaging of the salivary glands

Radiology

Mandibular occlusal films are useful for imaging salivary calculi in the submandibular duct. These may also be seen on panoramic radiographs, albeit with the mandible superimposed.

Sialography

Used to demonstrate a stricture of the duct and gland architecture.

Computed tomography, magnetic resonance imaging, ultrasound

If neoplasms are suspected. Can be combined with sialography.

Nuclear medicine

Demonstrates salivary gland function. A technetium-99m tracer seeks major protein-secreting exocrine and endocrine glands. The isotope is readily taken up by the major salivary glands. Lemon juice then administered orally to assess function and clearance of the glands.

Aplasia of salivary glands (Figure 10.24)

A number of cases of congenital salivary gland agenesis have been reported. Major salivary gland hypoplasia is an uncommon presentation of a child with gross caries in unusual sites. Caries of the lower anterior teeth should be regarded with suspicion in a young child, as it may indicate aplasia of the submandibular glands. It is uncommon for children to be on medication that will cause severe xerostomia and so aplasia/hypoplasia should always be considered.

Diagnosis

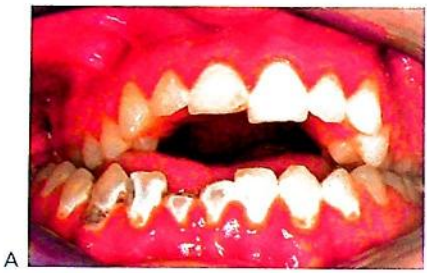
Reduced uptake of technetium pertechnetate.

Differential diagnosis of radiographic pathology in children

The location, size and distribution of radiographic anomalies are important in determining a differential diagnosis. Slowly growing lesions will displace teeth within the jaws, while more aggressive or rapidly growing lesions may resorb teeth. It is also important to have due regard for adjacent structures and the anatomy of where a lesion might derive from and ultimately spread. Space occupying lesions in children are predominately sarcomas rather than carcinomas that are seen in adults.

The position of the lesion and the direction of the displacement of the tooth is important. Observing the radiograph, a line can be drawn through the cemento-enamel junction of the associated teeth. Lesions that arise coronal to this line are generally odontogenic in origin (see Figure 10.25). The tooth tends to be displaced away from the occlusal plane towards the cortical plates. Lesions apical to this line tend to be non-odontogenic. The body of the lesion tends to extend away from the teeth. Some lesions may have different presentations that change over time.

Descriptions of some of the following lesions have been covered in this section, while others will be discussed in other chapters of this book, as referred.



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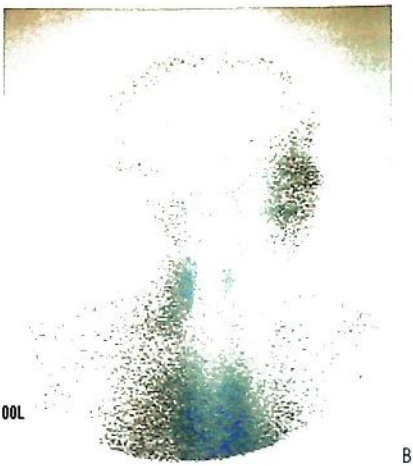

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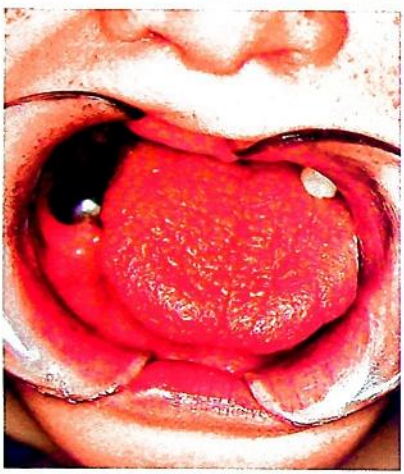


C

Figure 10.24 (A) Gross carious destruction of the mandibular anterior teeth. These are the only affected teeth. (B) The lateral nuclear medicine scan shows normal uptake of tracer in the parotid glands and thyroid but no function in the submandibular gland. A CT scan revealed that only rudimentary glands were present. (C) A 4-year-old boy who, after three procedures under general anaesthesia to treat gross caries in his primary dentition, was subsequently diagnosed with aplasia of his major salivary glands.

Periapical radiolucencies

Radiolucencies in the periapical region are usually associated with pulpal necrosis. All of these lesions tend to be inflammatory in origin but it is important to remember that healing lesions may appear similar to those that are active and sequential radiographs may be required to properly assess progression or healing. It is important to determine the integrity of the lamina dura around the apex of the tooth. Disruption or expansion of the periodontal ligament space indicates pathology.

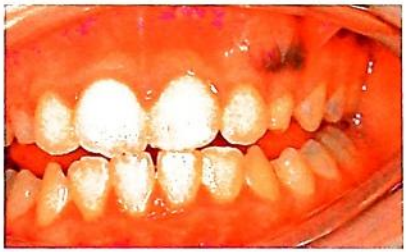
Inflammatory lesions

- Periapical granuloma, abscess, surgical defect, scar.
- Radicular cyst.
- Transient apical breakdown post-luxation of an incisor tooth (see Chapter 9).

Radiolucencies associated with the crowns of teeth

Radiolucencies that are associated with the crowns of unerupted teeth are generally odontogenic.

A



B

Figure 10.25 (A) Line drawn through the cemento-enamel junction of teeth associated with pathology in the jaws. Lesions apical to this line tend to be non-odontogenic in origin with the body of the lesion extending away from the teeth. Those lesions coronal to this line are usually odontogenic in origin. (B) A bluish-coloured swelling of a cyst associated with an unerupted upper left permanent canine. When managing lesions presenting with this colour, it is important to eliminate a lesion of vascular origin.

- Dentigerous cyst (Figure 10.26).

  The dentigerous or follicular cyst is the most common pathological entity associated with unerupted teeth. Some 75% are located in the mandible and usually present as a painless bony expansion and failure of eruption of the associated tooth that may be displaced a significant distance. The cyst enlarges by expansion from the increased hydrostatic pressure within the cavity. In early stages, it may be difficult to distinguish between an expanded follicle or a hyperplastic dental follicle. In some cases, the cyst lining becomes contiguous with the oral epithelium, forming an eruption cyst. The cyst is usually covered by a very thin wall of bone but the cortical plate invariably remains intact. Aspiration typically reveals a straw-coloured fluid containing cholesterol crystals.

  The cyst lining represents a metaplastic change of the reduced enamel epithelium and histologically, is seen as thin, non-keratinized, stratified squamous epithelium but flattened or low cuboidal cells typical of the reduced enamel epithelium may be seen along with islands of odontogenic epithelial rests. Some cysts show inflammatory changes and when there is communication with the oral cavity, the cyst cavity may be infected. The epithelium of a dentigerous cyst may undergo neoplastic transformation. Treatment is by enucleation of marsupialization (Figure 10.26C&D).
- Inflammatory follicular cyst (Figure 10.27) (see Chapter 11).
- Eruption cyst (see Chapter 11).
- Paradental cyst (Figure 10.28).

  The paradental cyst is an inflammatory odontogenic cyst usually seen on the buccal aspect of lower molars. It is thought to arise from the cell rests of Malassez. When there is a communication with the oral environment due to partial

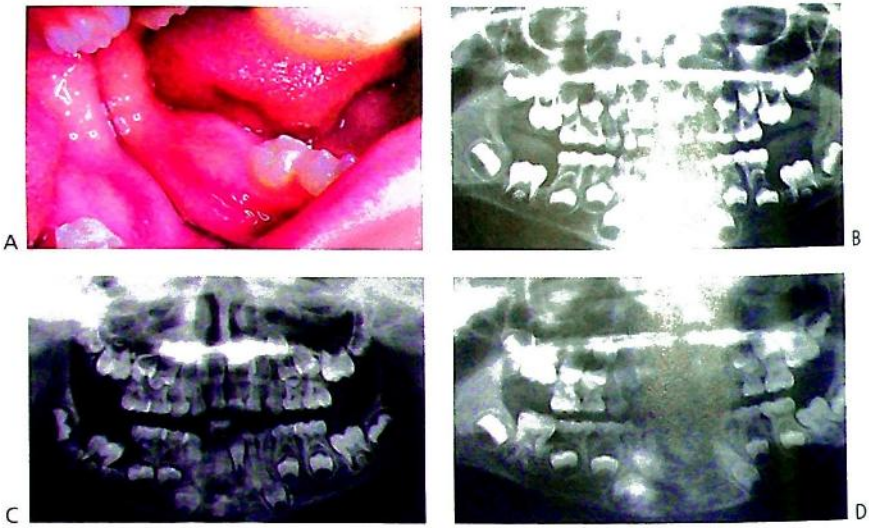


Figure 10.26 An unusual case of bilateral dentigerous cysts associated with the both lower first permanent molars. (A) Uninflamed swelling distal to the second primary molar. (B) Radiographic changes showing expansion of the follicle and resorption of the root of the lower left second primary molar. (C) A large dentigerous cyst around the crown of the tooth 46. This was managed by marsupialization. (D) The appearance 6 months post surgery showing bony infill and eruption of the tooth.

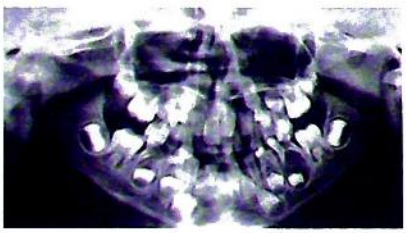


Figure 10.27 Inflammatory follicular cyst. The necrotic lower left second primary molar has caused inflammatory changes in the follicle of the premolar with displacement of this tooth

eruption or pericoronitis, the cyst becomes infected and actinomyces species and other anaerobic microorganisms are frequently found. However, this is not classified as 'actinomycosis', which is a soft tissue lesion with characteristic yellow sulphur granules.

- Odontogenic keratocyst (Keratocystic odontogenic tumour (KCOT)).
  The odontogenic keratocyst (OKC) was re-categorized as a true neoplasm by the WHO in 2005. It is locally aggressive and has a very high risk of recurrence reported to be between 3 to 60%. Approximately 70% of these lesions occur in the mandible with the typical presentation similar to a dentigerous cyst with


A

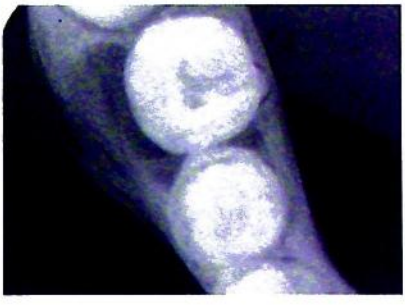

B

Figure 10.28 Paradental cyst. (A) A deep pocket on the buccal of a newly erupted lower right first permanent molar. (B) The true mandibular occlusal film shows the expansion of the buccal cortical plate and the associated bone loss with this cyst.

painless expansion, however, it may appear in association with the crown of an unerupted tooth, as an isolated radiolucency, as a multilocular lesion or as multiple isolated lesions. Radiographically, they have smooth well-demarcated borders but are locally invasive and the cortical plate may be perforated or in the maxilla, the lesion may extend into the antrum. Histologically, there is a parakeratinized stratified squamous epithelial lining with a relatively flat epithelial-mesenchymal junction. The contents of the cyst have been described as caseous or having a yellow, cheese-like consistency and hence aspiration is essential prior to surgical intervention.

Due to the aggressive nature of the OKC, a more radical treatment approach is advised. Recurrence is due to the budding of daughter cysts from the basal layer, an increased mitotic activity of the epithelium and local invasion of surrounding bone. While marsupialization and enucleation with curettage have similar rates of recurrence there is not the surgical morbidity associated with resection. More recently, application of the fixative Carnoy's solution (60% ethanol, 30% chloroform and 10% glacial acetic acid) has shown some promise in reducing recurrence with these lesions.

- Adenomatoid odontogenic tumour (Figure 10.29).

The AOT is a benign slowly-growing unilocular lesion that presents as a well circumscribed radiolucency in maxilla or mandible. There may be islands of calcification within the lesion accounting for either a radiolucent or mixed radiographic presentation. There is contention as to whether this is a true tumour or a hamartoma, as they never recur. Management is by enucleation.

Separate isolated radiolucencies

Odontogenic cysts and tumours

- Ameloblastic fibroma (Figure 10.30) (see also Chapter 11).

The ameloblastic fibroma is a slowly growing, mixed odontogenic tumour containing both epithelial and mesenchymal tissues. If dentine is identified in the

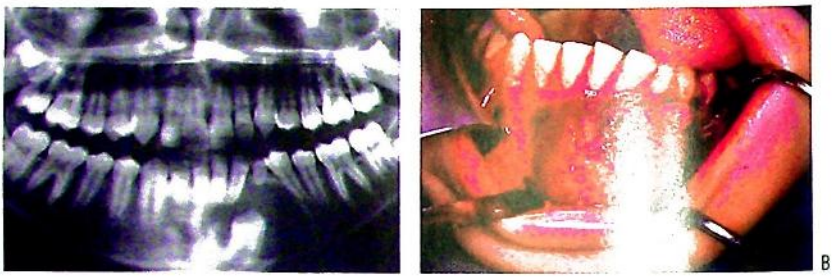


Figure 10.29 Adenomatoid odontogenic tumour. (A) This large cyst associated with the crown of the unerupted tooth 33 has caused displacement of the adjacent incisors and premolars but there is no resorption of the roots. Hence, the lesion is slowly growing. (B) The appearance at surgery.

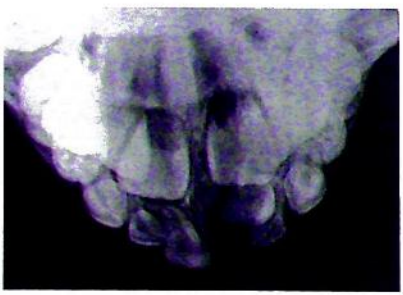


Figure 10.30 Ameloblastic fibroma associated with a maxillary supernumerary tooth. There is enlargement of the follicle surrounding the supernumerary that should arouse suspicion that there is other pathology associated with this tooth, as a dentigerous cyst is uncommonly found with supernumerary teeth in a child of this age.

specimen, then the lesion is classified as an ameloblastic fibro-dentinoma and tends to occur in young children.

There is discussion as to whether there is a continuum of perturbed differentiation from the ameloblastic fibroma, through the ameloblastic fibro-dentinoma, to the ameloblastic fibro-odontoma (see below) and the odontomas. The former two lesions tend to occur at a later age than the latter two and, while all share similar histological features, are considered to form from different mechanisms. This means that while conservative management is appropriate initially, revision may be required as malignant transformation to the ameloblastic fibrosarcoma is commonly seen requiring wide surgical resection.

- Odontogenic Keratocyst or KCOT.

Non-epithelial lined bony cavities

- Solitary bone cyst/traumatic bone cyst/haemorrhagic bone cyst.

These pseudocysts are unlined cavities usually in the mandible, however, there is little evidence that these anomalies form following bleeding in the medullary spaces after trauma. These are asymptomatic and are usually found during

routine radiographic surveys. The lamina dura of adjacent teeth is intact and there is no expansion of the bone and without treatment they probably resolve spontaneously, however, without a definitive diagnosis, the finding of a large radiolucency in the mandible warrants investigation.

- Stafne's bone cyst (cavity).
  While this may have the radiographic appearance of a cyst, this is merely a depression in the lingual aspect of the mandibular ramus and is occupied by the submandibular salivary gland.
- Aneurysmal bone cyst.
  The aneurysmal bone cyst is a rare expansile lesion seen in long bones as well as in the jaws. Of unknown aetiology, it is comprised of fibrous connective tissue interposed between blood-filled spaces. Aspiration yields fresh blood. The cortical plate is expanded but infrequently perforated and may present as a single or multilocular radiolucency.

Non-odontogenic fissural or developmental cysts

This group of cysts arises possibly from remnants of embryonic epithelial tissue, nasopalatine cysts will be lined with stratified squamous epithelium or pseudostratified ciliated columnar epithelium and are commonly observed as heart-shaped radiolucencies posterior to the upper central incisors.

- Incisive canal/nasopalatine duct cyst (Figure 10.31).
- Nasolabial cyst.
- Median palatine cyst.

Others

- Central giant cell granuloma.
  Previously thought to be a reparative process, the central giant cell granuloma is seen much more frequently in females and mainly in the mandible. It is clinically and radiographically identical to the appearance of hyperparathyroidism except that the latter is polyostotic and serum calcium and parathyroid hormone levels are elevated, while phosphorus levels are decreased. Histologically, there is a mass

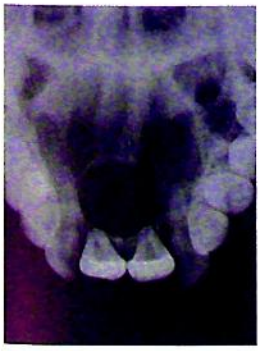


Figure 10.31 Incisive canal cyst. This cyst appears as a well-circumscribed radiolucency in the midline of the anterior palate. Notice the displacement of the central incisors.

of connective tissue with multiple multinucleated osteoclast-like giant cells. More recently, central giant cell lesions have been managed with interferon-α and calcitonin.

- Hyperparathyroidism (see above and Chapter 12).
- Ossifying fibroma.

  This fibro-osseous disease presents as a uni- or polyostotic lesion in either jaw, the radiographic appearance of which is either a radiolucency or mixed depending on the degree of calcification within the lesion. Previously termed the cemento-ossifying fibroma, the lesion is well encapsulated but is more locally aggressive in younger patients. Trabecular juvenile ossifying fibroma affects children and young adolescents, more commonly females, where there is rapid expansion of bone that may involve the cortex.

Multiple or multilocular radiolucencies

These are often termed soap-bubble lesions. Concern should also arise by the presence of any multilocular radiolucency as this represents areas of pathological expansion, bone lysis and invasion.

- Odontogenic keratocyst (Figure 10.32).
- Nevoid basal cell carcinoma syndrome (NBCCS) – OMIM No.109400 (Figure 10.32).

  Also know as Gorlin–Goltz syndrome, NBCCS is transmitted as an autosomal dominant mutation of the PTCH1 gene at 9q22.3–q31 but may be associated also with a 9q deletion. Similar to many autosomal dominant conditions, there is variable expressivity but complete penetrance. The major criteria for a diagnosis of NBCCS include multiple OKC's, basal cell carcinomas, palmar–plantar pits and calcification of the falx cerebri. Other features include rib and vertebral anomalies, medulloblastoma and ophthalmic anomalies. Patients have characteristic facies with frontal and parietal bossing and increased head circumference, hypertelorism and a broad nasal bridge. Patients need to be regularly monitored for the appearance of basal cell carcinomas. The jaw cysts are managed as for other OKC's.

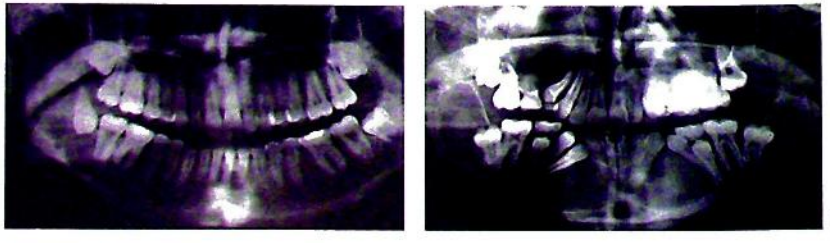


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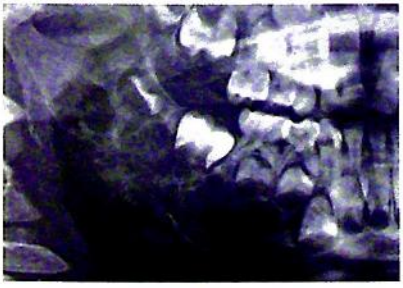
Figure 10.32 (A) Keratocystic odontogenic tumour of the right ramus of mandible in a 16-year-old patient. The presentation of these lesions may be quite variable. They are commonly associated with the crown of an unerupted tooth, but may appear as multiloculated lesions or as a single radiolucency. (B) Gorlin syndrome. Panoramic radiograph of a 15-year-old girl presenting with multiple KCOTs and basal cell carcinomas on her face.

- Central giant cell granuloma/tumour.
- Cherubism (see Chapter 11).
- Hyperparathyroidism (see Chapter 12).
- Langerhans' cell histiocytosis (see above).
- Vascular malformation of jaws (arteriovenous malformation) (see above).
- Odontogenic myxoma.
  Similar in radiographic appearance to the ameloblastoma, the myxoma is a mixed tumour of mesenchymal and odontogenic origin. Occurring mainly from 10 years of age onwards, it presents as a painless swelling, however, adjacent teeth may become loose or exfoliate. Histologically, there is a mass of fibroblast-like cells in a myxoid stroma. While benign, these lesions display local aggressive infiltration requiring wide surgical resection.
- Rare tumours of bone
  - Ewing sarcoma.
  - Desmoplastic fibroma (Figure 10.33A).
- Metastatic tumours (especially rhabdomyosarcoma 10.33B&C, Figure 10.34).



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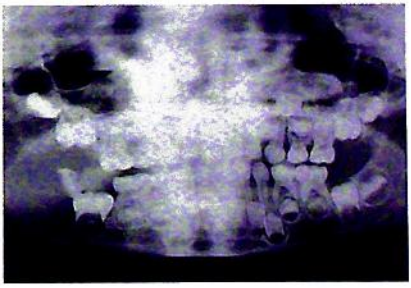

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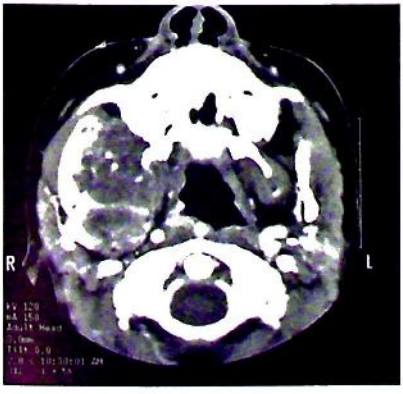
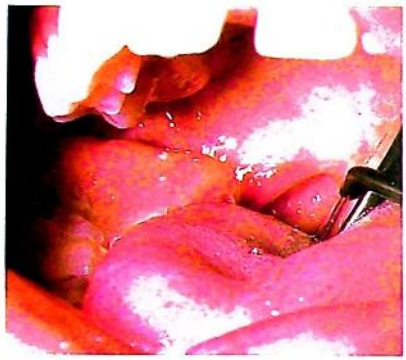


C

Figure 10.33 The diagnosis of a multilocular or soap-bubble lesion must always be treated with concern. These are invasive lesions and prompt referral is necessary. (A) Desmoplastic fibroma of the right mandible. (B) A large rhabdomyosarcoma involving the ramus of the right mandible and infratemporal fossa the extent of which is visible on the CT scan (C).



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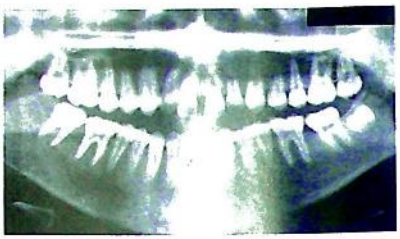

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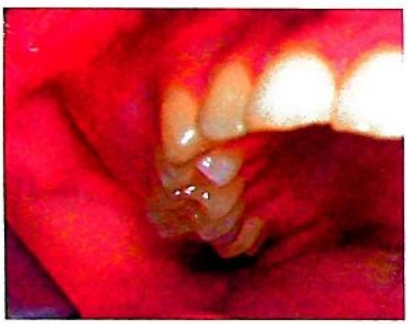
Figure 10.34 (A) A large gingival swelling in the retromolar triangle was associated with a metastatic fibrosarcoma from the abdomen in a 17-year-old boy. Not all such tumours present with multilocular radiolucencies. This patient presented with mobile right lower molars due to metastatic infiltration of the tumour. (B) The panoramic radiograph shows only diffuse changes in the trabecular pattern of bone in the right body and ramus.



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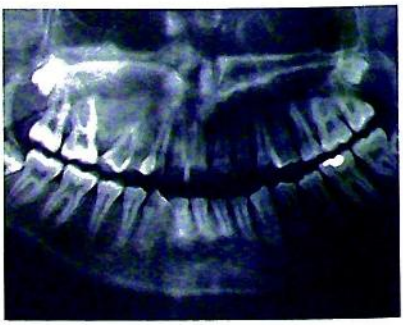

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Figure 10.35 (A) This 16-year-old boy presented unable to fit his sports mouth guard properly due to the painless expansion of bone in his right maxilla. (B) Panoramic radiograph of the bone with the characteristic 'ground glass' appearance.

Generalized bony rarefactions

- Hyperparathyroidism.
- Thalassaemia (see Chapter 12).
- Langerhans' cell histiocytosis.
- Fibrous dysplasia (Figure 10.35).

  Fibrous dysplasia presents as a monostotic or polyostotic lesion where there is abnormal growth and replacement of medullary bone by fibro-osseous tissue. The polyostotic form is more common in childhood. The areas affected are often

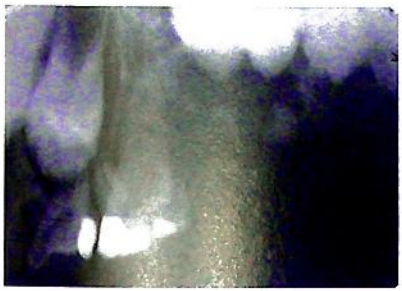

Figure 10.36 Ameloblastic fibro-odontoma. Similar in presentation to the ameloblastic fibroma, this less aggressive lesion typically presents with a mixed radiographic appearance with radiolucencies and opacities.

painful, especially the limbs. Radiographically, the bone appears to have a ground-glass appearance.

McCune–Albright syndrome (OMIM No.174800) is described as the triad of polyostotic fibrous dysplasia, unilateral skin hyperpigmentation (café-au-lait spots) and precocious puberty. There may be generalized endocrine hyperfunction resulting in increased growth hormone, catecholamines (Cushing syndrome), hyperthyroidism and hyperparathyroidism in addition to increased sex hormones. It is caused by a mutation of the GNAS1 gene at 20q13.32. There is no treatment other than bony recontouring in cases of severe disfigurement. Bisphosphonates are often prescribed to relieve pain experienced in the long bones.

- Renal osteodystrophy (see Chapter 12).

Mixed lesions with radiopacities and radiolucencies

- Odontoma (see Chapter 11).
- Ameloblastic fibro-odontoma (Figure 10.36; see also Chapter 11).
  Similar to the ameloblastic fibroma (see above), this benign odontogenic tumour is distinguished by ameloblast-like cells with elements of enamel and dentine present in the lesion. It has been suggested that the ameloblastic fibro-odontoma is an early stage in the development of an odontoma and may be classified as a hamartoma, and so conservative management with enucleation is indicated.
- Calcifying epithelial odontogenic tumour (Pindborg tumour).
  First described in 1955 by Jens Pindborg, this uncommon, benign odontogenic tumour has been termed the Pindborg tumour. It is believed to arise from cells of the stratum intermedium. It is usually unilocular and may be associated with an unerupted tooth. Radiographically, the lesion is well circumscribed with islands of varying degrees of radio-opacity. Histologically, the lesion is comprised of polygonal epithelial cells, interspersed with islands of calcification, and deposits of amyloid-like material. It is has a high rate of recurrence.
- Calcifying odontogenic cyst (Gorlin cyst).
  The Gorlin cyst presents as a single mixed radiolucency in either arch, although may be associated with an odontome, an ameloblastic fibroma or fibro-odontome or even an ameloblastoma. Characteristic of this lesion is the presence of 'ghost cells' in the lining.

- Adenomatoid odontogenic tumour.
- Odontogenic fibroma.
- Ossifying fibroma.
- Fibrous dysplasia (Figure 10.35).
- Garré's osteomyelitis (periostitis ossificans).
  This is a non-suppurative, chronic osteomyelitis caused by a low-grade odontogenic infection in the mandible in children. The radiographic appearance is characteristic showing a proliferative periostitis where new bone is laid down on the periphery of the cortical bone at the angle or lower border of mandible in response to mild infection.
- Osteosarcoma.
  While the average age for the appearance of osteosarcoma is in the 4th decade of life, this rapidly-growing, malignant tumour of bone may affect children. It is primarily an osteolytic lesion displaying a characteristic 'sunray' pattern seen radiographically. It may present with pain and swelling over the area and is managed with chemotherapy and surgery.

Radiopacities in the jaws

- Retained roots.
- Odontomas and supernumerary teeth (see Chapter 11).
- Focal sclerosing osteomyelitis (condensing osteitis).
- Cleidocranial dysplasia (see Chapter 11).
- Gardner syndrome (see Chapter 11).
- Bony exostoses – torus mandibularis or torus palatinus.
- Osteoma.
- Foreign bodies.

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11 Dental anomalies

Michael J Aldred, Angus C Cameron, Nigel M King, Richard P Widmer

Introduction

The diagnosis and management of dental anomalies constitute important areas of paediatric dentistry. Although most dental anomalies present in childhood, many are misdiagnosed or left untreated, perhaps because of lack of experience or because the case is perceived to be 'too difficult'. In some instances, genetic consultation is desirable, not merely to diagnose the condition but also to provide appropriate advice on the prognosis and the risk of recurrence in future generations. In many cases, the presence of an inherited dental disorder in one child would not stop a family from having additional children, but it is important to give parents and the affected children themselves appropriate information on which to base their decisions. Genetic services are usually available at most paediatric hospitals.

In this chapter, reference to particular inherited conditions is made to entries in OMIM (Online Mendelian Inheritance in Man). This online database is a catalogue of genetic disorders developed by Dr Victor McKusick of the Johns Hopkins University and the National Center for Biotechnology Information (see References and further reading, below).

Considerations in the management of dental anomalies

- Informing and supporting the child and parent.
- Establishing a diagnosis.
- Genetic counselling.
- Interdisciplinary formulation of a definitive treatment plan.
- Elimination of pain.
- Restoration of aesthetics.
- Provision of adequate function.
- Maintenance of occlusal vertical dimension.
- Use of intermediate restorations in childhood and adolescence.
- Planning for definitive treatment at an optimal age.

Treatment planning for children with dental anomalies

Treatment planning should be multidisciplinary. Decision-making must involve the child and the parents and should consider the present and future needs and development

of the child. Although children will cope with a range of appliances and treatments during childhood, early adolescence represents a period of social adjustment, as well as the transitional changes in the dentition. It is perhaps the most difficult time in which to formulate a long-term plan. Teenagers are most concerned about aesthetics, yet it may be too early to provide definitive restorations; extensive orthodontic treatment may be required or later orthognathic surgery. In institutions, various teams exist to treatment plan and/or manage these cases and a list is suggested below. Note the involvement of the child's local general dental practitioner.

The team approach

- Paediatric dentist.
- Orthodontist.
- Prosthodontist.
- Surgeon.
- Speech pathologist.
- Clinical psychologist.
- Local general dental practitioner.

It is essential to seek advice from colleagues in the management of children with uncommon dental conditions. Local and international collaboration provides the best opportunities to increase our knowledge and improve the outcomes for these children.

Dental anomalies at different stages of dental development

It is convenient to consider dental anomalies by the development stage at which they arise.

Migration of neural crest cells (ectomesenchyme) into branchial arches

- Duplication of dental arches.

Dental lamina formation stage

Induction and proliferation

- Hypodontia/oligodontia/anodontia (which may be associated with other features of an ectodermal dysplasia).
- Supernumerary teeth.
- Double teeth (geminated or fused teeth).
- Odontomes (complex and compound).
- Odontogenic tumours, particularly the spectrum of ameloblastic fibroma/fibrodentinoma/fibro-odontome (dependent on differentiation and the presence and type of calcification within the lesion).
- Odontogenic keratocysts.

Histodifferentiation

Developmental defects of multiple dental tissues

- Regional odontodysplasia.

Morphodifferentiation

Abnormalities of size and shape

- Macrodontia.
- Microdontia (isolated or as part of a syndrome).
- Invaginated odontome (dens invaginatus).
- Evaginated odontome (dens evaginatus).
- Carabelli trait.
- Talon cusp.
- Hutchinson's incisors and mulberry molars in congenital syphilis.
- Taurodontism.

Matrix deposition

Organic matrix deposition and mineralization

- Enamel:
  - Amelogenesis imperfecta.
  - Chronological enamel hypoplasia.
  - Molar–incisor hypoplasia.
  - Enamel opacities.
  - Fluorosis.
- Dentine:
  - Dentinogenesis imperfecta.
  - Dentinal dysplasia.
  - Vitamin D-resistant rickets.
  - Pre-eruptive intracoronal resorptive lesions.

Eruption and root development

- Premature eruption.
- Natal and neonatal teeth.
- Delayed eruption.
- Ectopic eruption.
- Eruption cyst.
- Transposition of teeth.
- Impactions.
- Arrested root development from systemic illness (or treatment of systemic illness).
- Failure of eruption in amelogenesis imperfecta.
- Failure of eruption in cleidocranial dysplasia.
- Failure of eruption in cherubism.
- Failure of eruption associated with inflammatory follicular cysts.

Formation of dental lamina

Hypodontia

Alternative terminology: Hypodontia, oligodontia, anodontia.

Hypodontia, oligodontia and anodontia are terms that can be interpreted to refer to progressive degrees of missing teeth, though the term hypodontia is preferred

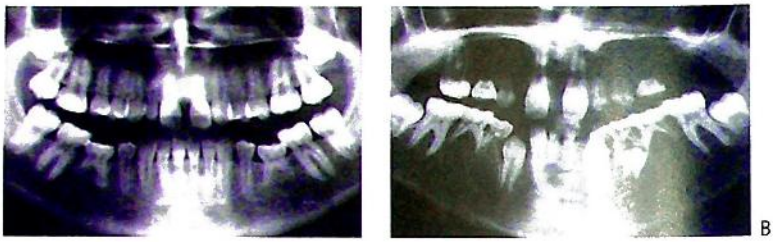


Figure 11.1 (A) The teeth most commonly missing are the last teeth in each series, namely the upper lateral incisors, the second premolars and the third molars. (B) Panoramic radiograph of a boy with autosomal dominant ectodermal dysplasia with absence of both primary and permanent teeth.

because it is inclusive of any number of missing teeth (Figure 11.1A). 'Oligodontia' refers to six or more missing teeth, and 'anodontia' to the complete absence of teeth. It is implicit in all cases that the teeth are missing because of failure of development. The term 'congenitally missing teeth' is a misnomer when applied to the permanent dentition because these teeth do not commence development until after birth (and with regard to the primary dentition one cannot usually determine this clinically at birth); 'partial anodontia' is a nonsense term. Some degree of hypodontia is not uncommon, occurring sporadically or with a hereditary component. The teeth most commonly absent are the last teeth in each series (i.e. the lateral incisor, the second premolar and the third molar). Clinically, it is less important to know how many, but rather which types of tooth are absent. It is particularly unusual for a patient to be missing central incisors, canines or first permanent molars. Multiple missing teeth in a child should lead to investigations to determine if there are other affected family members. The presence of a rudimentary or conical tooth may be associated with the absence of the same tooth on the opposite side of the arch. A common example of this is the peg lateral incisor. Furthermore, that lateral incisor itself may be absent in subsequent generations. Missing teeth are also a manifestation of many syndromes of the head and neck.

Frequency

Primary teeth	~0.1–0.7%	male : female	ratio unknown
Permanent teeth	~2–9%	male : female	1 : 1.4

Third molars > maxillary lateral incisors > second premolars > mandibular central incisors.

Major conditions manifesting hypodontia

Hypodontia is a major clinical feature of over 50 syndromes. These include:

- Ectodermal dysplasias.
- Dento-alveolar clefting.
- Trisomy 21 (Down syndrome).
- Chondroectodermal dysplasia (Ellis–van Creveld syndrome).
- Rieger syndrome.

Figure 11.2 (A) Typical appearance of a boy with X-linked hypohidrotic ectodermal dysplasia (wearing a denture). The skin around the eyes is dry and wrinkled and may be pigmented (not shown here). (B) The hair is fine and sparse and often displays longitudinal grooves on the surface under the scanning electron microscopy.

- Incontinentia pigmenti.
- Oro-facial-digital syndrome.
- William syndrome.
- Craniosynostosis syndromes.

Ectodermal dysplasias

Ectodermal dysplasia describes a group of developmental, often inherited, disorders involving the ectodermally derived structures, i.e. the hair, teeth, nails, skin and sweat glands. The most common is the X-linked hypohidrotic form (OMIM 305100, EDA1, Xq12-q13.1; short arm of X chromosome). In this condition the usual presentation is a male child with:

- Multiple missing teeth (Figure 11.1B).
- Fine, sparse hair (Figure 11.2A,B).
- Dry skin (Figure 11.2A).

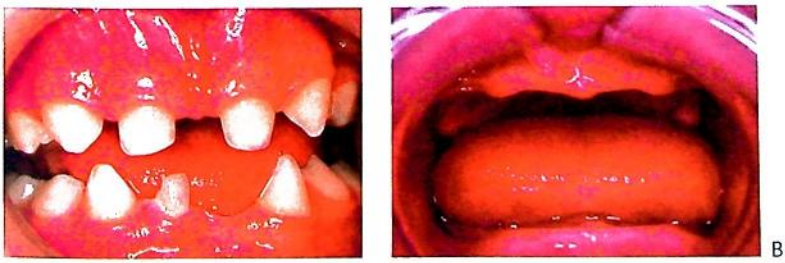
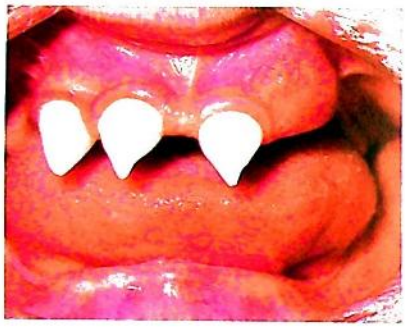


Figure 11.3 (A) This child is a heterozygous female with the X-linked form of ectodermal dysplasia and is less severely affected than her brother who has anodontia (B).



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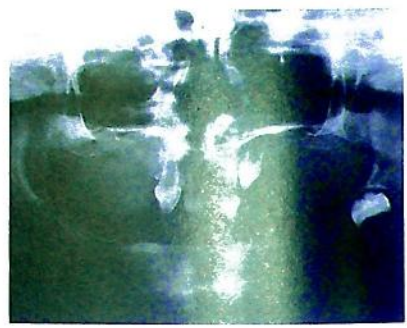

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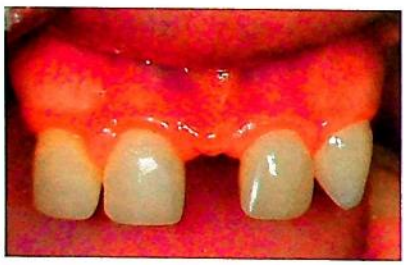

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Figure 11.4 (A,B) Typical intra-oral and radiographic presentations in a boy with ectodermal dysplasia with multiple missing teeth; the teeth that are present are small and conical in shape. In the regions where teeth are absent, alveolar bone does not develop. (C) Composite resin build-ups of the conical teeth have provided improvement in the aesthetics; however, the problem of the diastema remains, given that there are only a few teeth for orthodontic anchorage.

- Maxillary hypoplasia.
- Eversion of the lips.
- Circum oral and orbital pigmentation.

Teeth are small and conical, often with a large anterior diastema (Figure 11.4). Heterozygous females are often identified by dental examination and their manifestations may be limited to a single missing tooth or to a peg lateral incisor (see the Lyon hypothesis, below).

In the group of ectodermal dysplasias, autosomal dominant and recessive modes of inheritance are also seen. In such families, there will not be such a striking difference in the degree of the disorder between males and females compared with X-linked hypohidrotic ectodermal dysplasia (Figures 11.2A, 11.3). Mutations in the MSX1 gene (4p16.1) have been identified in families with missing third molars and second premolars with or without clefting, as well as in families with tooth-nail (Witkop) syndrome. PAX9 (14q12–q13) gene mutations have been found in other families with autosomal dominant missing teeth. More genes implicated in missing teeth and other anomalies continue to be identified.

In some countries, dental care (including prevention, orthodontics and prosthetics) for affected children may be provided under government-funded schemes.

Management

The aim of treatment is to provide adequate function, maintain the vertical dimension and restore aesthetic appearance. Ideally, for social reasons, treatment should begin

at around 2–3 years of age. A first step is often the placement of composite restorations to mask the 'fang-like' appearance of the caniniform anterior teeth (Figure 11.4A). There is often considerable parental pressure to 'normalize' the appearance and later, steps may involve the provision of dentures to reduce the likelihood of teasing, often at about the time that the child starts school. This can begin as soon as the child allows adequate impressions to be taken. Often, however, the first denture is initially worn in the pocket(!), but as the child grows, there is often a desire to have a more ordinary appearance. With encouragement and positive reinforcement, most children will soon try their new appliances.

Treatment planning for children with hypodontia

Treatment planning should be multidisciplinary and should consider the present and future needs and development of the child, while being cognizant of the concerns of the individual and parents.

Treatment options

- Acid-etch retained, composite resin build-ups of conical teeth (Figure 11.6).
- Composite resin or bonded orthodontic buttons can also be added to provide undercuts for denture clasps and retainers.
- Partial dentures: conventional or overdentures (Figure 11.7).
- Surgical exposure of impacted teeth.
- Orthodontic management of spaces.
- Laboratory-fabricated composite resin veneers, crowns and bridges.
- Osseointegrated implants (usually after the cessation of growth).

Clinical Hints — Provision of dentures for young children

Generally, children can tolerate dentures well, nevertheless, provision of the upper denture before the lower may be one way of increasing acceptance. The aim is for these children to be wearing appliances that give them a dentition similar to their peers, to enhance their self-esteem and promote normal speech development and masticatory function by the time they are at kindergarten or primary school. Dentures need to be re-made at regular intervals and a same-age model from an unaffected child should be used as a template for the occlusion.

- Use fast-setting alginate impression material or a bite-registration elastomeric material and sit the child upright with the head forwards.
- Use Adams' clasps on molars and ball retainers between upper canines and first molars.
- Use overdentures when there are multiple missing teeth and/or irregular spacing.
- Daily fluoride mouthrinses should be used with overdentures.
- Resilient or soft liners aid retention.
- Make dentures with irregular, or partly erupted, teeth during the mixed dentition stage.
- Long school holidays are often a good time for the provision of new dentures.

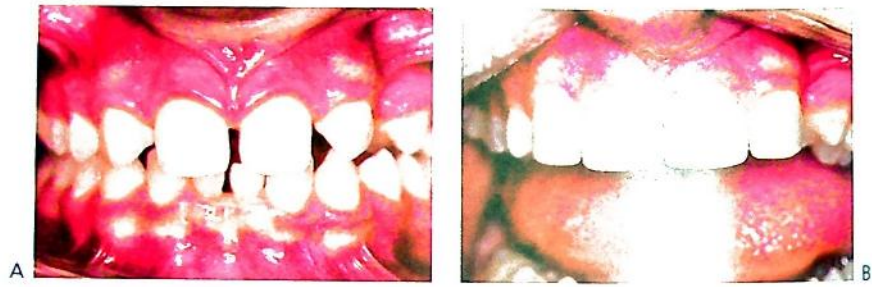


Figure 11.5 (A,B) Closure of an anterior diastema and reshaping of the canines with composite resin in a child with absence of the upper lateral incisors. The successful masking of upper canines to appear like lateral incisors is dependent very much on their size.

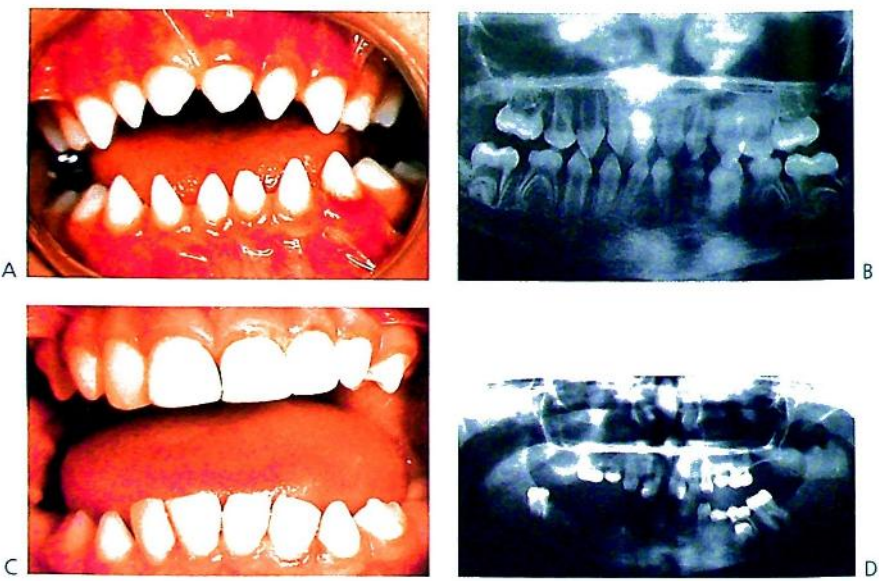
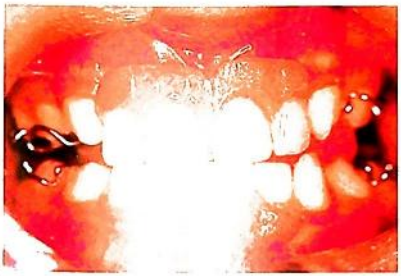


Figure 11.6 (A,B) Conical primary teeth are often associated with missing permanent teeth. This child had an autosomal recessive form of ectodermal dysplasia and was missing almost all of the permanent teeth. (C) These teeth have been built up with composite resin strip crowns. (D) Radiographic appearance of the same child at 15 years of age. Most of the primary teeth have exfoliated even in the absence of a permanent successor. There has also been loss of bone in the region of the tuberosity due to pneumatization of the sinus that will complicate implant placement.



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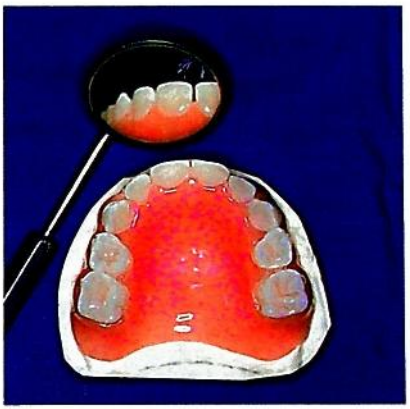

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Figure 11.7 (A–C) Dentures for young children with ectodermal dysplasia. (A) Young children tolerate dentures extremely well and Adams' cribs and ball retainers provide retention around primary molars. In this case, an overdenture covers two conical, widely spaced incisors. (B) A full upper denture for a child of 30 months will require periodic relining and re-making as the child grows. (C) Stock prosthetic teeth are sometimes difficult to obtain but paediatric denture teeth may be made freehand from acrylic and the palate can be customised.

The Lyon hypothesis (X chromosome inactivation)

During cellular differentiation, one of the two X chromosomes in each female somatic cell is inactivated. This means that in families with X-linked disorders, approximately 50% of the cells of heterozygous females will express the mutant gene disorder, whereas the remainder will express the normal gene. In the tissues affected by the condition, such females have a mosaic of affected and normal cells. This is of particular importance in X-linked forms of conditions such as haemophilia, hypohidrotic ectodermal dysplasia, vitamin D-resistant rickets and amelogenesis imperfecta. Thus, heterozygous females with X-linked hypohidrotic ectodermal dysplasia may have missing teeth, although they are invariably less severely affected than males. Similarly, in haemophilia A, heterozygous females do not usually have a clinical bleeding abnormality but this can occur if lyonization is severely skewed so that there is a preponderance of cells producing factor VIII under control of the mutant gene.

Dentoalveolar clefting

In patients affected by dentoalveolar clefting, disruption of the dental lamina at that site, there may be abnormal cellular induction or proliferation. This may give rise to

either missing teeth, usually the maxillary lateral incisor, and/or supernumerary teeth adjacent to the cleft. However, it is extremely rare for the canine tooth to be affected in the same way.

Solitary median maxillary central incisor syndrome

(OMIM 147250)

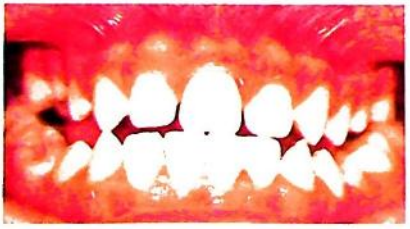
Solitary median maxillary central incisor syndrome (SMMCI) (Figure 11.8) is very rare. It presents with a midline symmetrical maxillary central incisor. The condition may also be associated with other midline disturbances such as cleft palate, choanal stenosis or atresia, imperforate anus or umbilical hernia and is probably part of the spectrum of the holoprosencephaly malformation complex. Of importance in some cases is the association with hypoplasia of the sella turcica, pituitary dysfunction, growth hormone deficiency and subsequent short stature. The syndrome is usually diagnosed on the basis of the dental manifestations. A mutation in the SHH gene (7q36) has been identified in one family but it is probable that there is genetic heterogeneity in the condition.

Ultimately, management of the dental anomaly is by orthodontic and prosthodontic therapy, determined by space considerations. In most cases, the single central incisor is moved to one side of the midline with either creation of space for a prosthodontic replacement, or the adjacent lateral incisors are recontoured.

Osseointegrated implants in children

There has been much controversy about the timing of placement of osseointegrated implants in young children. To date, there has been only limited published material about early placement and any long-term consequences. It is generally understood that implants act similarly to ankylosed teeth and do not move occlusally with the growing bone around adjacent natural teeth. Recent animal research has confirmed that most fixtures do become osseointegrated in growing jaws; however, there was

Figure 11.8 (A) Solitary median maxillary central incisor syndrome presenting with a symmetrical incisor in the midline. This child had a mild growth hormone deficiency, with his height on the 10th centile. (B) Periapical radiograph of the same patient in the primary dentition showing the single primary and permanent central incisors.



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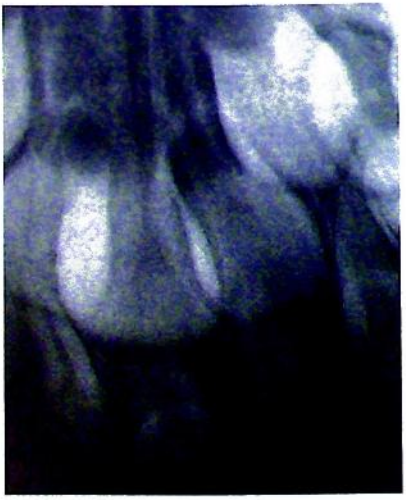

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no evidence from this research that the fixtures behaved like normal teeth during development. In the mandible, the fixtures came to lie lingual to the natural teeth; in the maxilla, they came to lie palatal and superior to the adjacent teeth and did not follow the normal downwards and forwards growth of this bone. This latter point is important when considering the placement of implants in the anterior maxilla. Furthermore, placement of fixtures retarded alveolar growth locally and changed the eruptive path of distally positioned tooth buds. Implants should, in most cases, not be considered before the cessation of growth. It should be noted, however, that in children with conditions such as ectodermal dysplasia, alveolar bone does not develop where teeth are not present. Consequently, it may be considered appropriate, particularly where there are multiple missing teeth, to place implants much earlier in these children than in those with a normal alveolus. Recent research suggests that in cases of anodontia, implants are best placed in the mandibular canine region at around 8–10 years of age (which is after the period of maximal mandibular transverse growth) to facilitate lower denture construction.

Disorders of proliferation

Supernumerary teeth (Figure 11.9)

- Supernumerary teeth arising as a result of budding of the dental lamina can occur sporadically or be inherited, as in cleidocranial dysplasia.
- The shape may resemble a tooth of the normal series (a supplemental tooth), in which case it can be incisiform, caniniform or molariform; otherwise it may be conical or tuberculate.
- Most often present as a result of failure of eruption of one or more permanent teeth. Usually appear as conical or tuberculate forms.
- Supernumerary teeth have been considered to be manifestations of a separate dentition (occurring between the primary and permanent dentitions), and consequently it may be possible to predict when and where supernumeraries may form (Jensen & Kreiborg 1990).

Alternative terminology

Mesiodens (a term restricted to supernumerary teeth in the midline of the maxilla), paramolar, distomolar, hyperdontia, polydontism, supplemental teeth.

Frequency

Primary teeth	~0.3–0.8%	male : female	ratio unknown
Permanent teeth	~1.0–3.5%	male : female	1 : 0.4

- Some 98% occur in the maxilla, 75% of which are mesiodens.

Diagnosis

- Failed or eruption disturbance of permanent tooth (Figure 11.9B).
- Routine radiographic survey.
- As part of a syndrome such as cleidocranial dysplasia (Figure 11.10).

Management

- Conical teeth often erupt and are easily extracted (Figure 11.9A).
- Tuberculate and/or inverted conical teeth require surgical removal ((Figure 11.9D)) as early as possible to allow uninhibited eruption of the permanent teeth.

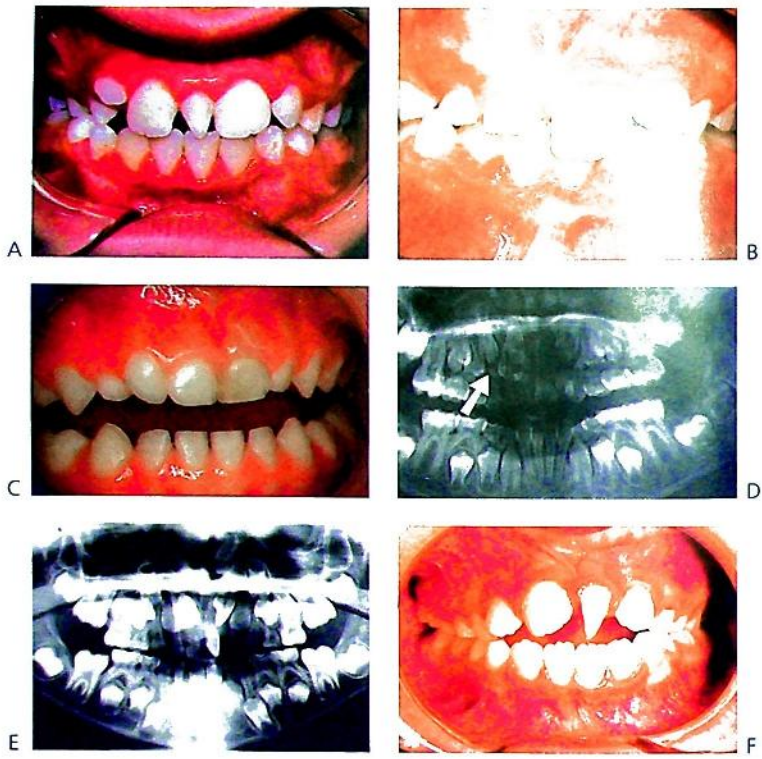


Figure 11.9 Common presentation of supernumerary teeth. (A) Conical teeth often erupt, except when inverted. (B) The late eruption of a permanent central incisor is most commonly caused by a supernumerary tooth. (C) Supplemental upper primary lateral incisor. (D) A panoramic radiograph is useful in determining the vertical orientation of the extra tooth (arrowed) and the degree of displacement of the permanent central incisor. In this case, after removal of the supernumerary, an upper denture was used as a space maintainer and the impacted tooth subsequently erupted into a normal position. (E,F) Dependent on the degree of displacement, given adequate space, most impacted incisors will normally erupt once an obstruction such as a supernumerary is removed. The rotation can be corrected later.

- It is essential to localize the position of the tooth to be removed *prior to* surgery. Periapical films using a tube-shift technique can be used to locate the tooth, however this is always open to errors and misinterpretation. Panoramic and standard maxillary occlusal films may be used in the same way.
- Digital imaging techniques using cone-beam tomography (CBCT) provide high definition, 3-dimensional imaging of the head and neck with much reduced radiation exposure than traditional computed tomography (CT) (see Figure 11.19, below).

- During surgical removal, care should be taken to avoid disturbing the developing permanent teeth.
- Before 10 years of age: if the unerupted central incisor is correctly aligned the treatment of choice is to remove the supernumerary surgically and allow normal eruption of the permanent tooth. Gingival exposure may be required later because of surgical scar formation that can inhibit final soft-tissue emergence. Some authorities recommend the simultaneous removal of primary canines to counteract this tendency. Inverted supernumeraries can be removed less traumatically if surgery is performed early, however, this needs to be done with caution to avoid damage to the adjacent tooth germs.
- After 10 years of age, or if the central incisor is malaligned: surgical exposure with or without bonding of orthodontic brackets or chains and subsequent traction may be required (Figure 11.11).

Clinical

- Most maxillary supernumerary teeth are best removed surgically via a palatal approach. The only exceptions are those that are inverted, conical in shape and positioned between the roots of the central incisors. Usually the crown is found lying adjacent to the anterior nasal spine and is best approached via a labial flap.
- Supernumerary teeth may be also found in the midline of the palate as far posteriorly as the line of the first molars. These appear to be placed very high in the maxilla on radiographs but are usually quite superficial and simple to remove with a wide palatal flap.

Cleidocranial dysplasia (Figure 11.10) (OMIM 119600)
This condition has an autosomal dominant mode of inheritance, with a high frequency of spontaneous mutations. The condition has been mapped to 6p21 with mutations found in the CBFA1 gene.

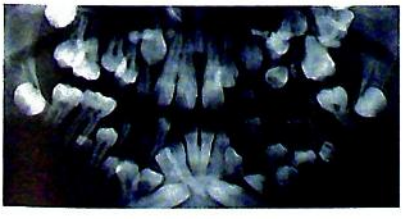
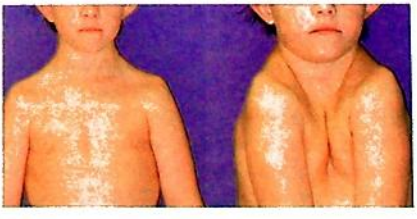
A   B

Figure 11.10 (A) case of cleidocranial dysplasia with 18 supernumerary teeth. (B) A boy with cleidocranial dysplasia showing the characteristic absence of the clavicles.

Figure 11.11 Surgical exposure and bonding of a gold chain to a central incisor which was impacted by a supernumerary tooth. (A) Elevation of the labial and palatal flaps and removal of the supernumerary. (B) Acid-etch applied to the labial surface of the upper left central incisor (C) Rinsing the acid etch gel from the tooth. (D) The appearance of the etch pattern on the prepared tooth. (E) Bonding of a gold chain attachment to the labial surface of the incisor. (F) The flap is closed and the chain is sutured to the gingiva with surgical nylon. The chain will be attached to an archwire and orthodontic traction will be applied to orthodontically align the tooth.

Manifestations

- Short stature.
- Aplasia or hypoplasia of one or both clavicles (Figure 11.10B).
- Delayed ossification of fontanelles and sutures.
- Frontal bossing.
- Hypertelorism and maxillary hypoplasia.
- Wormian bones in cranial sutures.
- Multiple supernumerary teeth (Figure 11.10A).
- Delayed eruption of teeth.
- Dentigerous cyst formation.
- Absent or altered cellular cementum.

Management

- Early diagnosis and documentation.
- Planned removal of non-resorbing primary teeth.
- Surgical removal of supernumerary teeth.
- Surgical exposure of permanent teeth.
- Orthodontic alignment and consideration of orthognathic surgery when growth complete.

Note that the simple extraction of a primary tooth will frequently guarantee the eruption of the impacted permanent tooth. A two-stage surgical procedure is usually required with an attachment placed on the permanent tooth followed by orthodontic traction. The first procedure involves exposure of the anterior segments with removal of the anterior primary teeth and any supernumeraries that may be present. The permanent teeth are surgically exposed, either with primary apically repositioned flaps or with bonded gold chains attached for orthodontic traction. The anterior teeth are then aligned orthodontically. The second stage involves extraction of the primary molars, surgical removal of remaining supernumerary teeth and exposure of the premolars and molars in the buccal segments. Definitive orthodontic therapy follows; orthognathic surgery may be required in cases with severe skeletal Class III malocclusion. Treatment obviously extends over many years and clinicians should be aware of the potential problems relating to the child's compliance and the need for multiple surgical procedures.

Cherubism (OMIM 118400)

Cherubism is an autosomal dominant condition caused by mutations in the SH3BP2 gene at 4p16.3.

Patients may present in childhood with facial swelling and/or failure of eruption of teeth, typically the mandibular molars. Radiographs will reveal multilocular radiolucencies, typically involving the angles of the mandible (Figure 11.12). A biopsy will reveal multinucleate giant cells in a fibrous tissue stroma. Developing teeth in the affected area tend to be displaced and fail to erupt at the normal time. The maxillae can also be affected, as can the ribs. The facial swelling reflects the involvement of the underlying bone. In some patients the sclera in the lower part of the eyes may be exposed to give the cherubic or heavenward gaze that gives the condition its name. In some cases, there is no discernible facial swelling and the condition is identified as a result of routine radiographic studies such as for orthodontic treatment planning, or because of delayed eruption of teeth.

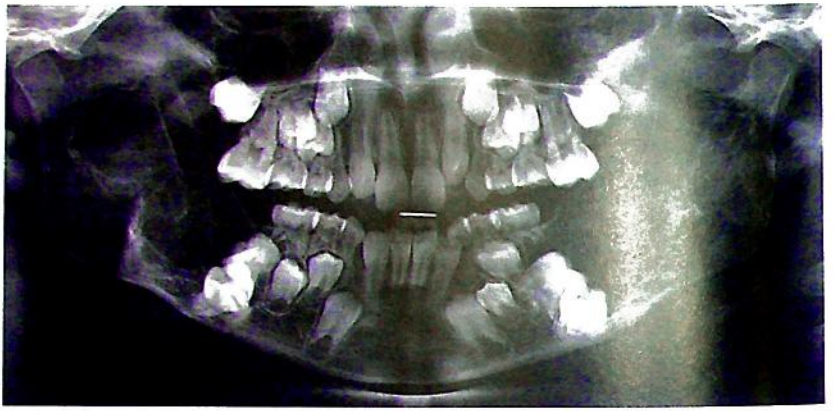


Figure 11.12 Dental panoramic radiograph showing almost symmetrical multilocular radiolucencies in the angles of the mandible of an 8-year-old boy. The displacement of developing molars and delayed eruption of teeth is usually seen in cherubism.

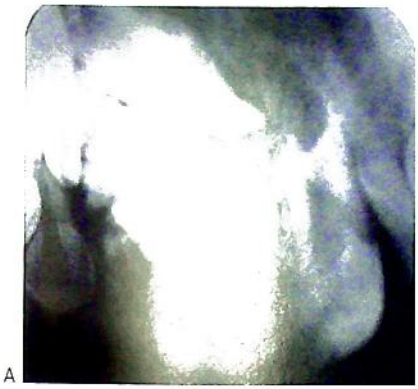
The condition progresses into adolescence and then tends to resolve, so that by the 3rd or 4th decade radiographic changes may no longer be found. In some families more affected males than females may be identified – this is a result of reduced penetrance in females and needs to be taken into account in genetic counselling. A subset of patients with cherubism is more severely affected with the multilocular radiolucencies affecting the whole of the mandible and maxillae. In mildly affected cases regular review may be all that is necessary, in more severely affected cases surgical reduction may be considered if the patient is distressed by their appearance.

Inflammatory follicular cysts

Some children may present with failure of eruption of a mandibular premolar associated with a radiolucency involving the roots of the primary molar and crown of the unerupted premolar (see Figure 7.2B and Figure 10.27). There is controversy as to whether such cases are due to radicular cyst formation associated with the roots of the primary tooth (which is considered by some to be a rare occurrence) or dentigerous cyst formation around the crown of the premolar. The common characteristics of such cases tend to be:

- Prior endodontic treatment of the primary molar.
- A radiolucency involving the roots of the primary molar and crown of the permanent successor.
- Displacement of the permanent successor away from the alveolar crest.

Histopathological examination tends to show intense acute and chronic inflammation of the curetted tissue which is lined by hyperplastic stratified squamous epithelium. Such cases have been designated 'inflammatory follicular cysts', with persistent inflammation from the endodontically treated primary molar leading to an inflammatory enlargement of the follicle of the underlying permanent tooth.



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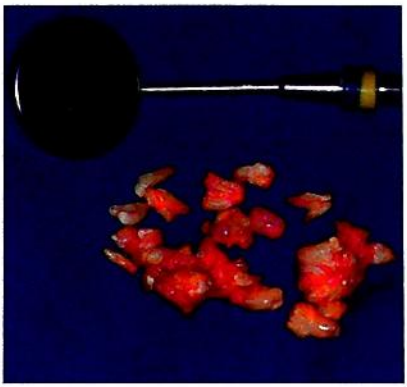

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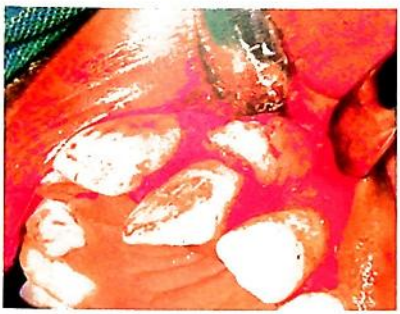

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Figure 11.13 Odontomes. (A) Compound odontome with multiple denticles causing displacement of the maxillary right central incisor. (B) Macroscopic specimen of a compound odontome from the anterior maxilla showing the numerous denticles surrounded by a well-defined capsule. (C) Complex odontome following elevation of a labial flap.

Odontomes (Figure 11.13)

Odontomes occur because of disordered differentiation and often present because of failure of eruption of a permanent tooth. In compound odontomes, multiplex of irregular denticles are found in a circumscribed soft-tissue stroma. Complex odontomes are disordered lesions with a discrete, haphazard mass of calcified tissue containing all dental elements. There is either a normal complement of teeth or the odontome replaces a tooth of the normal series.

Management

- Surgical enucleation.
- Depending on the time of diagnosis, permanent teeth may be ectopically positioned and may require surgical exposure and orthodontic alignment.

Odontogenic tumours (see Chapter 10)

The ameloblastic fibroma, fibrodentinoma and fibro-odontome are uncommon benign odontogenic mixed tumours. All are seen as altered differentiation of the tooth bud: in an ameloblastic fibroma no hard tissue is formed, in an ameloblastic fibrodentinoma only dentine-like tissue is recognizable and in an ameloblastic fibro-odontome enamel is also formed. The lesions tend to be well demarcated.

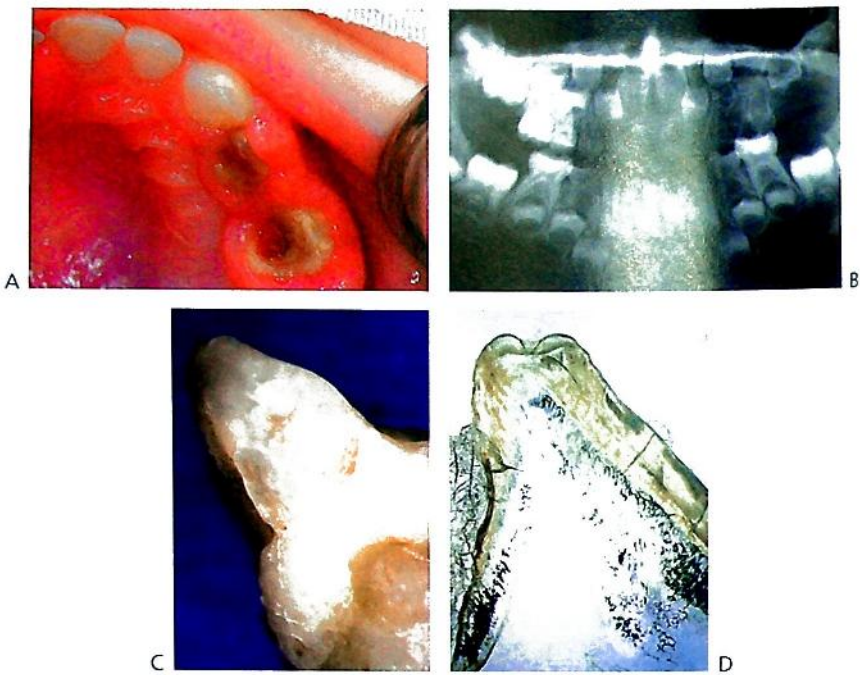


Figure 11.14 (A) Regional odontodysplasia presenting with abscessed primary molars in the maxillary left quadrant, soon after eruption. (B) The panoramic radiograph shows involvement of all the teeth in this quadrant including the permanent teeth. (C) Grossly abnormal enamel in an affected tooth and (D) the hard-tissue section demonstrates the disruption of odontogenesis. (Courtesy Dr N Pai, Sydney, Australia.)

Management

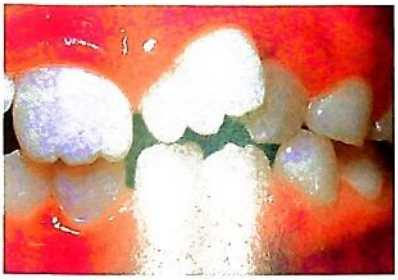
- Surgical enucleation.
- Follow-up of erupting permanent dentition if teeth are displaced by the lesion.

Odontogenic keratocysts (see Chapter 10)

Odontogenic keratocysts may arise in place of a tooth of the normal series or from the dental lamina in addition to a normal complement of teeth. They constitute 5–15% of odontogenic cysts.

Regional odontodysplasia (Figure 11.14)

Regional odontodysplasia is a sporadic defect in tooth formation with segmental involvement, usually localized to one, or part of one quadrant, but it may cross the midline to affect the contralateral central incisor. All dental tissues are involved in a bizarre dysplasia with hypoplastic teeth which are slow to erupt and which typically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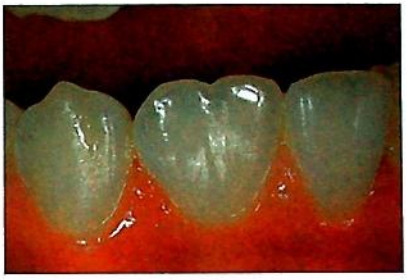
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Figure 11.15 Morphological anomalies. (A) Mamelons, which are variations of normal anatomy. (B) Double tooth involving the right mandibular incisor, probably caused by fusion of the lateral and central incisor tooth germs.

radiographically show a ghost-like appearance. The aetiology of the condition is unclear.

- Usually presents initially with abscessed primary teeth before or soon after eruption.
- Some cases are associated with superficial vascular anomalies.

Alternative terminology
Ghost teeth.

Management

- In spite of attempts to restore teeth with stainless-steel crowns or composite resin, most affected teeth require extraction. Permanent successors of affected primary teeth are invariably affected, though sometimes to a lesser degree. There is no justification for bony excision at the time of tooth removal. There are reports of successful autologous tooth transplantation into the sites of removal of affected teeth.
- Partial dentures are required to restore the lost teeth.
- Implants may be appropriate.

Abnormalities of morphology

Macrodontia (Figure 11.16)

- Any tooth larger than normal for that particular tooth type.
- True macrodontia involving the whole dentition is extremely rare. More commonly, single teeth are abnormally large because of an isolated disturbance of development.

Aetiology

- Unknown for a single tooth, but generalized macrodontia may be caused by a hormonal imbalance, as this has been described in pituitary gigantism. It should be remembered that an illusion of generalized macrodontia will occur if the jaws are small relative to the size of the teeth.

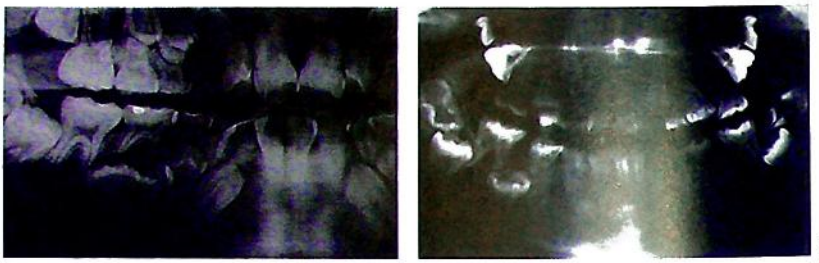


Figure 11.16 Macrodontia. (A) Isolated macrodontia in a mandibular second premolar tooth. (B) Generalized macrodontia associated with KBG syndrome. These children present with intellectual disabilities, broad facies, short stature and skeletal abnormalities.

- May also be associated with hemifacial hyperplasia.
- True macrodontia should not be confused with the fusion or gemination of adjacent tooth units or a supernumerary to form a single tooth.
- Generalized macrodontia is also associated with KBG syndrome (the initials are taken from the surnames of the families first reported with the condition). These children present with short stature, intellectual disability, skeletal abnormalities, syndactyly, a broad face with microcephaly and other facial anomalies (Figure 11.16B).

Alternative terminology
Megadontia, megalodontia and gigantism.

Frequency
Primary dentition	Unknown
Permanent dentition	~1.1%

More common in males.

Management
- Stripping to reduce tooth size; however, usually only a small change can be achieved.
- Can be combined with composite resin build-up of the antimere if only one tooth affected.
- Extraction and replacement by a prosthesis.
- Aesthetic adjustment of an isolated macrodont tooth by incisal edge 'notching' and the generation of a labial groove to break up light reflections may be helpful in some cases.

Microdontia
- One or more teeth that are smaller than normal for the tooth type.
- The most common form of microdontia affects only one or possibly two teeth; it is much rarer in the primary than in the permanent dentition.
- This anomaly most often affects the maxillary third molars and lateral incisors. It is noteworthy that the affected teeth are usually the ones that are also most often missing.
- Supernumerary teeth are frequently microdont.
- Patients with ectodermal dysplasia often present with microdontia.

True generalized microdontia
All of the teeth are of a normal morphological form but they are smaller than normal teeth. This condition is exceedingly rare but can occur in pituitary dwarfism.

Generalized relative microdontia
The teeth are of normal size but appear relatively small with respect to the jaws that are larger than normal.

Alternative terminology
Peg-shaped laterals.

Frequency
Most data are available only for maxillary lateral incisors.
Primary dentition <0.5%
Permanent dentition ~2.0% (maxillary lateral incisors)

More common in females.

Management
- Composite resin or (eventually) porcelain veneers to improve shape.
- The profile of the tooth is narrower at the gingival margin than a normal-sized tooth (emergence profile), and there is therefore a limit to how large the tooth can be enlarged with a restoration, without producing an overhang in the gingival region or an unsightly interdental shadow.
- Orthodontic alignment and extraction of the tooth may be required and other techniques such as autotransplantation and implants should also be considered.

Clinical latest

In patients with missing teeth, the central incisors are often conical in form. When closing an anterior diastema, it is often preferable to add composite to the distal aspect of the crown rather than the mesial. The diastema can be closed orthodontically to avoid a 'flared' appearance of the tooth crown that tends to look artificial. A more vertical mesial proximal surface and the addition of composite to the distal surface give a better appearance with a more normal distal angle and arch form.

Double tooth (Figure 11.17)

This anomaly is manifest as a structure resembling two teeth that have been joined together. In the anterior region, the anomalous tooth usually has a groove on (at least) the labial surface and a notch in the incisal edge. Although rarer in the posterior region, the cuspal morphology can be suggestive of two teeth that are joined together. Radiographs are necessary to determine if there is a union of the pulp chambers, and even then it may be speculative. If the 'double tooth' is present together with a normal complement of teeth in the same quadrant then it is presumed to have arisen as a result of gemination; if the number of teeth is reduced then fusion of tooth germs is assumed. If teeth have been extracted or exfoliated, the use of the neutral term 'double tooth' avoids the need to arbitrarily decide if this is due to gemination or fusion.

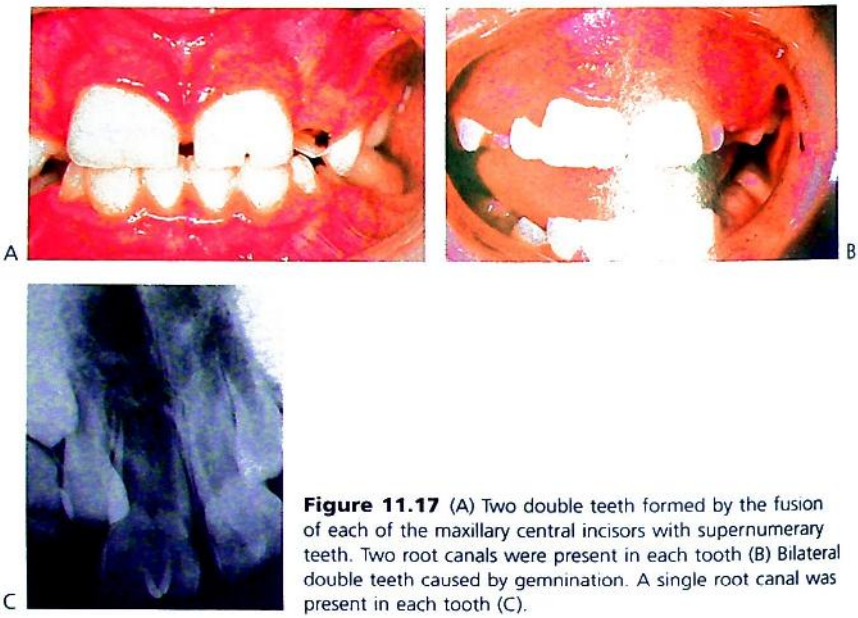


Figure 11.17 (A) Two double teeth formed by the fusion of each of the maxillary central incisors with supernumerary teeth. Two root canals were present in each tooth (B) Bilateral double teeth caused by gemnination. A single root canal was present in each tooth (C).

Alternative terminology
Fusion, gemination, connation, schizodontia, dichotomy.

Frequency
Primary dentition	~2.5%
Permanent dentition	~0.2%

Fusion, gemination or a 'double tooth' in the primary dentition should alert the clinician to the possibility of the same condition in the permanent dentition.

Fusion
Joining of two teeth of the normal series or a normal tooth and a supernumerary tooth by pulp and dentine. Two canals are usually present. The tooth has arisen from two tooth germs and so the number of teeth in the dentition is normally reduced by one unit. If, however, the normal tooth is fused to a supernumerary, the number of teeth in the arch will be normal. This fusion is assumed to occur between normal and supernumerary teeth because of the close proximity of the tooth buds.

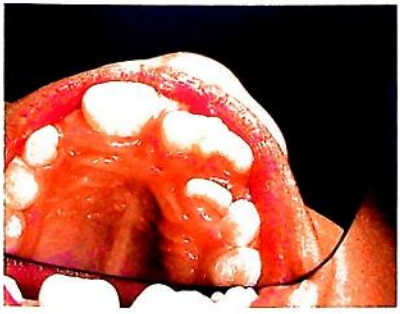
Gemination
Budding of a second tooth from a single tooth germ. Usually one root canal is present.

Management
- The central groove on the labial and palatal surfaces of a double tooth is prone to dental caries; therefore early application of a fissure sealant is recommended.

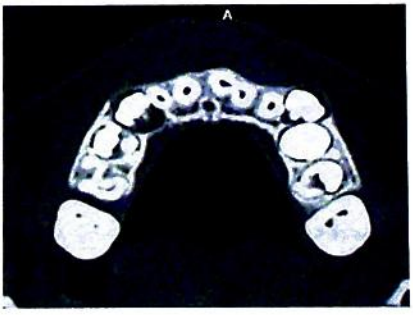
- In the permanent dentition, surgical separation of fused teeth may be possible with subsequent orthodontic alignment and restorative treatment as needed to reshape the crown. The lack of cementum on the cut surface of the root may result in a periodontal defect.
- Reshaping or reduction of a double tooth with a single canal (geminated tooth) may be attempted by modifying the appearance of the labial groove and the use of composite resin but is often impossible and extraction may be the only alternative. Orthodontic treatment and/or prosthetic replacement is then required. Implants may be an option for adolescents.
- Deliberate extraction and surgical separation outside the mouth with replantation might also be considered, although this is not always successful because of resorption subsequent to reimplantation.

Clinical [illegible] geminated teeth

Large geminated teeth present difficult management issues. It is essential to diagnose whether a single canal or separate canals are present. Plain radiographs are often of little benefit, especially when the abnormal tooth is in the central incisor region and there is superimposition of the lateral incisor which erupts palatal to the double tooth due to a lack of space. CT scans (Figure 11.18) and (better still) cone beam tomography (Figure 11.19) can be helpful to determine the morphology of the root canal.



A



B

C

Figure 11.18 (A) It is often very difficult to determine the root canal anatomy of double teeth when using periapical films due to superimposition of the lateral incisor that has usually erupted palatally. CT can be used to determine the true internal anatomy of the root canal. (B) Apically, it appears that there are two distinct canals; however, a more coronal section shows that there is communication between the canals. (C) This contraindicates surgical separation of the roots. The diagnostic value of this radiography must be weighed against the radiation exposure.

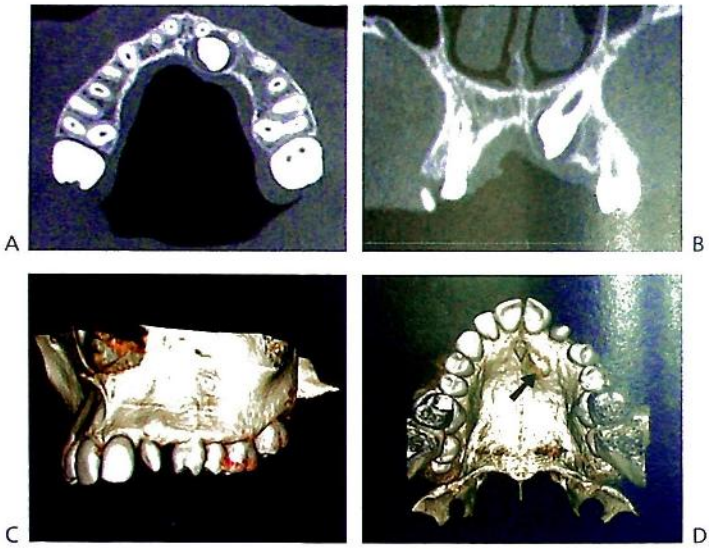


Figure 11.19 Further use of 3-dimensional CT to determine positioning and morphology of teeth. In these projections, the upper left canine is impacted. (A) The axial view shows the horizontal position in the palate in relation to the peg lateral incisor. (B) A coronal section details the root and the close association with the antrum. (C,D) 3-dimensional reconstructions are invaluable to the surgeon to determine the exact position in the mouth and the proximity of related structures.

Concrescence

Joining of two teeth, one of which may be a supernumerary, by cementum. Concrescence most commonly affects maxillary second and third permanent molars in older adults. Apart from when involving supernumeraries, this condition is rarely seen in children.

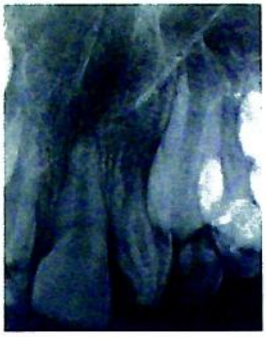
Dens invaginatus (Figures 11.20, 21)

Maxillary lateral incisors may have a developmental invagination of the cingulum pit with often only a thin hard-tissue barrier between the oral cavity and the pulp. Pulp necrosis often occurs soon after eruption of the affected tooth and may lead to a canine fossa abscess or cellulitis. This anomaly may occur in other teeth such as the maxillary central incisors and canines.

Alternative terminology

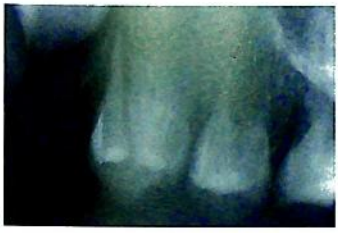
Invaginated odontome, dens in dente (used to describe the extreme variant, but is a misnomer), dilated odontome.

A

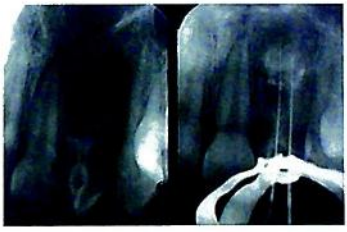


B

Figure 11.20 (A,B) Maxillary canine fossa cellulitis from an infected dens invaginatus. (B) Because of root canal morphology and the severity of the infection the tooth was removed. The patient required hospital admission, with high-dose intravenous antibiotics and surgical drainage of the abscess under general anaesthesia.



A



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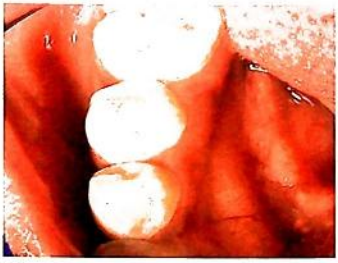

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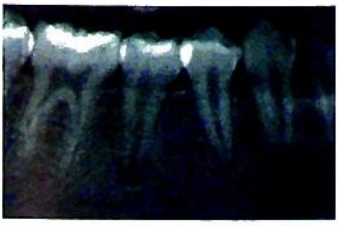
D



E

Figure 11.21 (A) Dens invaginatus in a maxillary first premolar tooth. (B) Ultimately, the prognosis is related to the ability to adequately instrument and obturate the canals of these teeth. Temporary endodontic dressings can be placed in such teeth to relieve symptoms and treat infection but it is almost impossible to obturate such canals. (C) Dens evaginatus. (D) Dens evaginatus where the tubercle has fractured off and the tooth has become necrotic. This child presented in acute pain with a facial cellulitis. The radiograph (E) shows the periapical area.

Frequency

Primary dentition	~0.1%
Permanent dentition	~4%

More common in males.

Management

- If newly erupted, the palatal fissures should be sealed as a preventive measure.
- If caries is evident, then place an acid-etched retained composite resin restoration with minimal preparation.
- If symptomatic and the root canal morphology is favourable, endodontic treatment can be undertaken.
- If the internal anatomy is complex and the root canal is not negotiable then, in the event of infection, extraction is necessary. The presence of this anomaly should be carefully considered during orthodontic treatment planning.
- The same tooth on the opposite side should be carefully assessed for the same problem.

Dens evaginatus (Figure 11.21C)

- An enamel-covered tubercle usually projecting from the occlusal surface of a premolar tooth.
- Usually bilateral and more common in the mandible.
- There is evidence of pulp tissue within the tubercle in ~50% of cases.
- Radiographs may show occlusal extension of the pulp chamber.

Alternative terminology

Leong's premolar, tuberculated premolar, axial core type odontome, occlusal enamel pearl, composite dilated odontome, cone-shaped supernumerary cusp, evaginated odontome, interstitial cusp.

Frequency

Primary dentition	Almost unknown
Permanent dentition	~4% (Almost exclusively in people of Asian extraction)

More common in females.

Management

- The tubercle can easily fracture because of occlusal interference, therefore grinding of the tubercle followed by fissure sealing can be of assistance. An alternative prophylactic measure is to support the sides of the tubercle with composite resin and then to recontour the occlusal surface to produce a central ridge. Ideally, this should be performed before the tooth comes into complete occlusion.
- If fractured or subject to attrition, pulp exposure occurs frequently. Because this exposure occurs soon after eruption, the apex of the tooth is often open and the long-term prognosis is less certain. Extraction of the tooth may be considered after orthodontic consultation. If the tooth is to be retained, a calcium hydroxide dressing is appropriate with an apexification procedure (see Chapter 9) to stabilize the tooth if orthodontic therapy is to commence later (and subsequent definitive endodontic

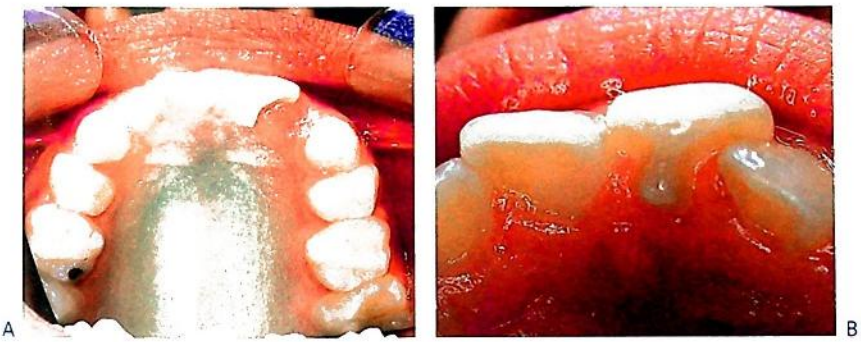


Figure 11.22 (A) Talon cusp. (B) T-cingulum. The cingulum cusp has pulp horns and removal of the cusp will often result in an exposure of the pulp.

treatment). More recently, revascularization has been proposed in the management of these teeth.

- If diagnosed early, an elective (Cvek) pulpotomy can be performed in an attempt to allow normal root formation.

Talon cusp (Figure 11.22)

This is a horn-like projection of the cingulum of the maxillary incisor teeth. It may reach the incisal edge of the tooth.

Alternative terminology

T-cingulum, Y-shaped cingulum.

Frequency

Primary dentition	Almost unknown
Permanent dentition	~1–2%

Management

- If there is no interference with the occlusion, no treatment is required.
- Fissure sealants to prevent caries in the grooves between the various parts of the tooth.
- If occlusal interference is present, small progressive reduction of enamel only to avoid pulp exposure, or elective pulpotomy, to allow root completion.

Taurodontism

Used to describe a molar tooth with a pulp chamber that is vertically enlarged at the expense of the roots. The distance from the cemento-enamel junction to the furcation of the root may be greater than the distance from the furcation to the apices. The tooth, therefore, has a long body and short roots, with a tendency towards a single root or apical displacement of the furcation. The anomaly appears to be caused by delay or failure of invagination of Hertwig's epithelial root sheath. Taurodontism may have a genetic basis. Several syndromes and conditions such as ectodermal dysplasias,

X-chromosome aneuploidies and some families with (autosomal dominant) amelogenesis imperfecta have this anomaly. The appearance may also be reflected in single-rooted teeth, with the pulp canals being wider than usual.

Frequency
Uncommon rather than rare. Enlarged pulp chambers may also be seen in:
- X-linked vitamin D-resistant rickets (hypophosphataemic rickets).
- Vitamin D-dependent rickets.
- Hypophosphatasia.
- Dentinogenesis imperfecta (some cases).
- Regional odontodysplasia.
- Klinefelter syndrome.
- Shell teeth.

Congenital syphilis
Although now very rare in most parts of the world, congenital syphilis presents with several important diagnostic dental manifestations. Both primary and permanent incisors have tapering crowns and central notching of the incisal edge. This tapering or screwdriver-like appearance is important in the differential diagnosis, as there are other causes of non-syphilitic notching of the incisal edge (e.g. trauma). This screwdriver morphology is also seen in Nance–Horan syndrome. The crowns of the molar teeth have a 'cobbled' or 'mulberry' appearance in congenital syphilis.

Developmental defects of enamel
Developmental defects of enamel can be acquired or inherited.

Chronological disturbances
Any severe systemic event during the development of the teeth (i.e. from 3 months *in-utero* to 20 years of age) may result in some dental abnormality. Many of these anomalies are subclinical and can only be observed in hard-tissue sections as changes in the incremental deposition lines. The neonatal line is manifest in all primary teeth, but unless there is a severe physiological disturbance or fetal distress the disturbance may not be clinically evident. Different teeth will show defects at different levels of the crown depending on the stage of crown formation at the time the disturbance occurred. The resulting enamel may be reduced in quantity (hypoplasia) and/or quality (usually hypomineralization).

A defect is described as localized when one or more teeth are affected, in an asymmetrical way, and generalized when there is a symmetrical disturbance on teeth of the same type on both left and right sides (and in both maxillary and mandibular teeth).

More than 100 aetiological agents have been reported to cause developmental defects of enamel. Those causing localized defects are listed in Table 11.1 and those causing generalized defects are listed in Table 11.2.

Developmental defects of enamel can be considered according to their clinical appearance:
- Discolouration.
- Opacity.

Table 11.1 Aetiological agents shown to produce developmental defects of enamel with a localized distribution

Acute osteomyelitis	**Gunshot wounds to jaws**
Acute trauma to primary teeth	Irradiation
Ankylosis	Jaw fracture
Cleft palate	Laryngoscopy
Congenital epulis	Periapical infection of primary teeth
Electrical burn to mouth	Periodontal ligament injection
Extraction of primary teeth	

In general, the aims of management are to treat associated pathology and pain, provide adequate aesthetic appeal, maintain occlusal function and maintain the vertical dimension.

Tooth discolouration

Tooth discolouration may be extrinsic or intrinsic in nature. Extrinsic staining is superficial and occurs after tooth eruption. Intrinsic discolouration may result from a developmental defect of enamel or internal staining of the tooth (Figure 11.23). Although such internal staining is manifest as a change in tooth colour, the intrinsic defect may affect the dentine primarily or exclusively. See Table 11.3 for the differential diagnosis of tooth discolouration.

Opacity

Opacities result from a defect in the quality of the enamel, affecting the translucency of the tissue. Hypomineralization results in a change in the porosity of the enamel, causing opacity. This may be located below the enamel surface, which otherwise remains intact.

Fluorosis (Figure 11.23C, see also Chapter 5)

In its mildest forms, fluorosis is manifest as hypomineralization of the enamel, leading to opacities. These can range from tiny white flecks to confluent opacities throughout the enamel, making the crown totally lacking in translucency. Hypoplasia occurs at higher concentrations of fluoride. When the tooth first erupts, the surface of even the most severely affected enamel may be intact; however, with wear, areas of enamel are lost and stains are taken up into the porosities. At 1 ppm of fluoride in public water supplies, up to 10% of the population will show very mild opacities attributable to fluorosis (though this may depend on individual water consumption); interestingly, this seems to be a minimum value and the proportion of opacities increases as fluoride levels either fall below 1 ppm or rise above 2 ppm. Severely affected cases may require microabrasion or restoration with composite resin, either in a localized or a more generalized manner, or porcelain veneers. Many opacities are incorrectly labelled as fluorosis without adequate justification or investigation of the patient's fluoride history.

Management of stains and opacities

- Extrinsic stains can be removed with abrasives.
- Mild discolouration may be improved using peroxide-based bleaching agents.
- Intrinsic stains, if superficial, may be removed with microabrasion techniques.

Table 11.2 Environmental aetiological agents shown to produce developmental defects of enamel and discolouration with a generalized distribution

Prenatal	Perinatal	Postnatal	
Anaemia	Bile duct defects	Adrenal hyperfunction	Lead intoxication
Cardiac disease	Breech presentation	Cytotoxic medications	Measles
Congenital allergies	Caesarean section	Bulbar poliomyelitis	Mumps
Congenital syphilis	Erythroblastosis fetalis	Candida-endocrinopathy syndrome	Nephrotic syndrome
Cytomegalovirus	Haemolytic disorder	Chickenpox (varicella)	Neurological disorders
Diabetes	Hepatitis	Cholera	Otitis media
Fluoride	Intrapartum haemorrhage	Congenital cardiac disease	Pneumonia
Hypoxia	Low birth weight	Diphtheria	Pseudohypothyroidism
Pregnancy toxaemia	Neonatal asphyxia	Encephalitis	Renal dysfunction
Malnutrition	Neonatal hypocalcaemia	Fluoride	Scarlet fever
Renal disease	Placenta praevia	Gastrointestinal disturbances	Sickle cell anaemia
Rubella	Prematurity	Hyperpituitarism	Small pox
Stress	Prolonged labour	Hyperthyroidism	Stress
Thalidomide	Respiratory distress syndrome	Hypogonadism	Tetracyclines
Urinary tract infection	Tetanus	Hypoparathyroidism	Tuberculosis
Vitamin A deficiency	Tetracyclines	Hypothyroidism	Typhus
Vitamin D deficiency	Traumatic birth injuries	Intestinal lymphangiectasia	Vitamin A deficiency
	Twinning		Vitamin C deficiency
			Vitamin D deficiency
			Vitamin D intoxication

Table 11.3 Causes of tooth discolouration

Colour	Aetiology	Comments
Extrinsic discolouration		
Green	Chromogenic bacteria	Usually cervical and gingival areas
Yellow	Bile pigments from gingival crevicular fluid	Biliary atresia and jaundice
Black-brown	Ferrous sulphate Chromogenic bacteria	Iron supplementation Arrested caries
Intrinsic discolouration with localized staining on one or several teeth		
Yellow/brown	Developmental defects	Usually after trauma or infection
White	Developmental defects	Subsurface decalcification in permanent teeth, after trauma or infection
Pink	Internal resorption	Seen before exfoliation or after trauma
Grey/Black	Amalgam staining	Leakage of old amalgam restorations causing discolouration at the periphery
Chronological staining of dentition		
Bright yellow	Tetracycline	Unoxidized fluorophore, seen in newly erupted teeth
Yellow/ grey-brown	Tetracycline	Erupted teeth, oxidized fluorophore (UV light)
Yellow-brown	Systemic illness	Developmental defect of enamel affecting all teeth forming during illness
Generalized intrinsic staining of teeth, either single of complete dentition		
Grey-brown	Necrotic tooth	Usually after trauma
Yellow-brown to dark yellow	Amelogenesis imperfecta	Both dentitions are affected
Green-blue	Hyperbilirubinaemia	Seen in children with end-stage liver disease and premature infants
Blue-brown (opalescent)	Dentinogenesis imperfecta	Uniformly affected teeth, may be associated with osteogenesis imperfecta
Red-brown	Congenital porphyria	All teeth affected
White	Fluorosis/non-fluorotic	Usually only permanent dentition

Microabrasion

It must be understood that microabrasion techniques involve removal of the surface opaque layer of enamel. The opaque but often bright white layer of enamel is removed and children and parents are often disappointed at the appearance of the normal 'yellow' colour of the permanent crown. It is important to make an initial decision

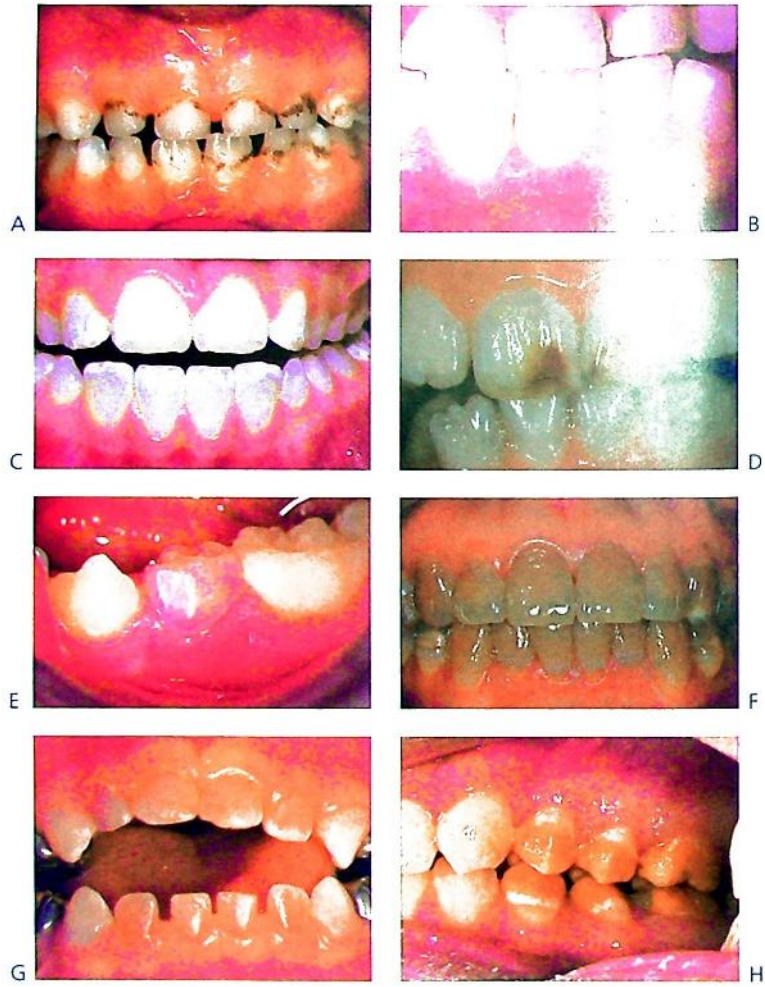


Figure 11.23 Tooth discolouration. (A) Brown-black superficial staining from chromogenic bacteria. (B) Localized enamel opacity caused by the root apex of a traumatized primary incisor. (C) Fluorosis – uniform opacity-type defects throughout the crown. Some of the hypomineralized enamel has been lost on the incisal edge revealing normal enamel underneath. (D) Brown discolouration due to incorporation of blood pigments into the enamel following trauma to the primary dentition. (E) Pink discolouration caused internal resorption of the tooth 74. (F) Tetracycline staining in a child from South-east Asia. Tetracycline containing liquid preparations are no longer routinely prescribed for children, however, tetracyclines may be included in some homoeopathic preparations in some countries. (G) Blue-brown appearance of dentinogenesis imperfecta. (H) Chronological discolouration of an unknown aetiology. There is a precise pattern to the hypomineralization and appearance of the posterior teeth, however, the anterior teeth, in particular, the canines that are developing at the same age are unaffected.

whether to attempt to alter the entire dentition to this darker colour, or to continue with the piecemeal adjustment of darker areas to match the overall 'paper-white' appearance. In present-day society, the latter decision may prove to be satisfactory or even desirable. Acid-based techniques may create more porosity in the enamel, which may accumulate more stains over time.

Hydrochloric acid – 18% (with or without the use of pumice)

Rubber dam should always be used and must be secured by ligation around individual teeth and sealing with copal-ether varnish or a proprietary sealer. Orabase® paste applied to the gingival margin prior to dam placement can be used to protect the soft tissues from any acid leakage. An aqueous slurry of sodium bicarbonate may be laid on the dam around the teeth to neutralize any inadvertent excess acid. An hydrochloric acid/pumice slurry is applied to the affected area using a slowly rotating rubber cup for 10 s only, and then rinsed thoroughly with water. Application is repeated a maximum of 10 times. This technique is potentially destructive to enamel and soft tissues, and must be used with caution.

Alternatives

- Abrasion with a mixture of pumice and 37% phosphoric acid (etchant).
- Polishing labial surfaces with a multi-fluted tungsten-carbide bur.
- Application of a 2% neutral sodium fluoride, followed after a short time by polishing with fine polishing paste (non-coloured toothpaste is suitable) in a rotating rubber cup is advisable after use of each of these techniques.
- Recent research has shown that mild fluorosis can also be remineralized and the opacity reduced by casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) or casein phosphopeptide-amorphous calcium phosphate fluoride (CPP-ACPF). The enamel should be pretreated with sodium hypochlorite to denature any residual protein.

Deep intrinsic stains require removal of the affected enamel and rebuilding, usually with composite resin. Although localized marks may be dealt with by this method, treatment using composite resin or porcelain as full-face veneers, or crowns, should ideally be delayed in adolescents until the gingival attachment is established at the cemento-enamel junction. The longevity of hybrid composite resins has improved substantially, along with their colour stability, strength and translucency. These materials may be placed quickly and more cost-effectively than porcelain and other complex restorations such as crowns. Modern materials generally provide for densely white shades that make matching possible. It is important always to keep treatment options open. Involvement in contact sports may be another reason for delaying placement of complex restorations.

Enamel hypoplasia (Figure 11.24)

A defect in quantity that causes an altered contour of the surface of the enamel. This is usually caused by initial failure of the deposition of enamel protein, but the same clinical effect could also result if there is a mineralization defect that leads to loss of enamel substance after eruption. In the former case, the enamel is often hard and glassy, in the latter it will usually pit on probing. In some trauma cases, tissue may be lost after formation and is not regarded as a true hypoplasia. Examples of hypoplastic defects following trauma are shown in Figure 9.18.

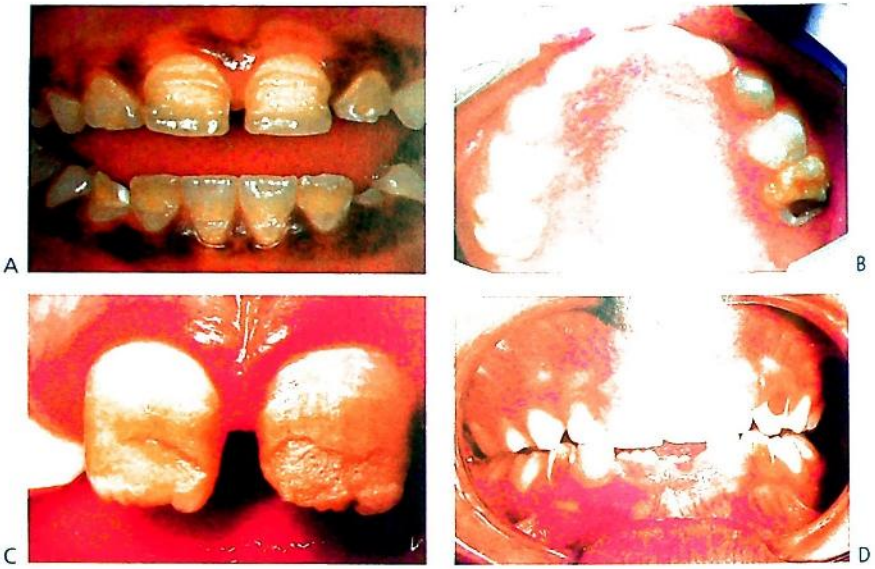


Figure 11.24 (A) Chronological enamel hypoplasia after a childhood illness from 11 months up to around 18 months of age. The incisal edges of the maxillary lateral incisors are affected. Fortunately, there is little hypomineralization of the teeth, making the dentition more easily restored with composite resin. (B) Chronological hypoplasia may affect the primary dentition, this child with a child suffered fetal distress and meconium aspiration at delivery. It is possible that the tips of the cusps of the first permanent molars are similarly affected. (C) Another form of chronological hypoplasia. Note that there is normal enamel at the cervical region and the primary tooth is not affected. (D) Localized hypoplasia of primary canines. These anomalies present as defects on the labial surface of primary canine teeth and often become carious. A minute area of hypoplasia is visible on the maxillary right canine, and all the other canine teeth are carious, while the remainder of the teeth are caries free.

Management

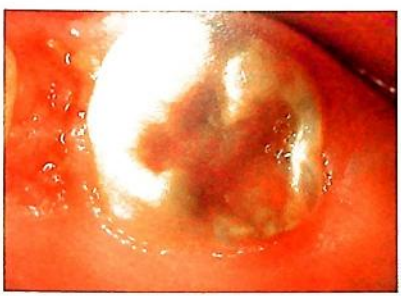
- Localized hypoplastic defects may be restored with composite resin. Pitting defects may need initial localized debris or stain removal with either rotary instruments or amine peroxide bleaching systems.
- It is important to maintain posterior support, and stainless steel crowns may be required to restore grossly hypoplastic molars. These teeth may be exquisitely sensitive to thermal and osmotic stimuli and treatment is made difficult by an inability to achieve good isolation of teeth that are only partially erupted. Glass ionomers may be used temporarily to restore hypoplastic occlusal defects and prevent caries.
- A realistic assessment of the likely longevity of affected first molars is important from an early age. Consideration should be given to the elective loss of these teeth

as part of an occlusal developmental plan for the child. A pre-assessment accompanied by orthodontic advice not later than 8 years of age is recommended.

- Complex restorative treatment involving onlays, veneers and crowns should generally be delayed until late adolescence but the selective use of metal, adhesively retained onlays may provide a long-term solution in some molar cases.

Molar–incisor hypomineralization (Figure 11.25)

Molar–incisor hypomineralization (MIH) is a condition that, although recognized as a clinical entity for some time, is still a subject of considerable study. It presents as a qualitative change in enamel that, initially, is of normal thickness, ranging from localized opacity through opacity with discolouration and obvious poor quality to post-eruptive enamel breakdown. The cervical enamel appears to be normal in most affect first permanent molars. One or more first permanent molars may be affected in a



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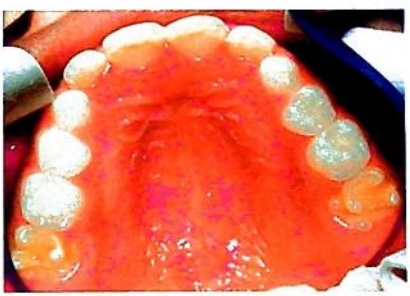

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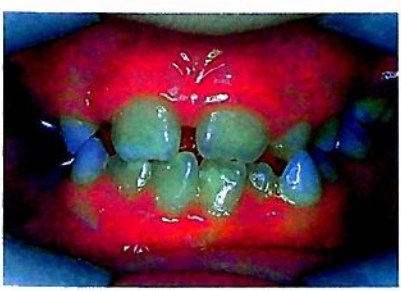

C

Figure 11.25 Molar incisor hypomineralization. Teeth may be variably affected in the one mouth. (A) Severely affected upper first permanent molar with loss of enamel and hypomineralization. (B) An otherwise intact dentition shows typical MIH. The incisors (palatal surface) are less severely affected than the molars. (C) Hypomineralization of the incisors associated with MIH and rampant caries in the primary dentition. Note that the crowns of the second primary molars and first permanent molars form at similar times, and this may explain the high rate of caries seen in the primary dentition in some of these patients. (Courtesy Dr E Alcaino.)

quasi-chronological but inconsistent manner, together with (usually lesser) effects on one or more incisors. The presentation is puzzling since as few as one molar, or as many as all four may be affected. Often affected teeth are extremely sensitive, and this is often an indicator to poor ability to gain complete anaesthesia of the affected teeth. Use of low viscosity GIC sealants soon after eruption and also remineralizing agents such as CPP-ACP can help decrease sensitivity and reduce post-eruptive breakdown.

Many possible aetiological factors, especially those related to childhood disease and maternal illness during the 3rd trimester, have been suggested. Research is under way in many centres to define the aetiological factors, since a knowledge of these would permit early preventive and restorative interventions. The prevalence ranges widely, however, it is between 10% and 15% in many communities worldwide; with around 5% of the population being severely affected. It is believed that MIH may account for a large proportion of the restorative needs of children in many communities, and therefore is an important public health issue.

A familial tendency to the condition has been recognized by some authors. Irrespective of the exact aetiology of MIH, it is important to recognize that this condition represents a chronological disturbance in tooth formation between birth and 24 months of age. Enamel hypomineralization can affect any teeth in the permanent dentition, however, there is a very low prevalence compared with the first molars. It also affects second primary molars, with a prevalence of approximately 6%.

Management

- The ideal restorative approach for these cases has yet to be determined however if intracoronal restorations are planned, composite resin should be used. Some research indicates that pre-treating the enamel with 5% sodium hypochlorite after etching increases bond-strengths significantly. Stainless steel crowns are an option for severely affected teeth, however it needs to be explained to the child and parents that this is an intermediate phase of treatment, and further restorative work will be required at maturity. Trimming the length of the stainless steel crown for partly erupted first permanent molars so as to obtain a fit just apical to the maximum convexity of the crown makes such placement easier in many cases.
- There is a clear association between repeated, well-meaning attempts by practitioners to place 'minimal', adhesive or other treatments for these molars, without adequate local anaesthesia, and a real antipathy to dental treatment on the part of the affected children. Local analgesia should be used for the treatment of these cases but it should be noted that even under these conditions pain control will not always be adequate and treatment may be compromised. General anaesthesia is often required to provide high quality dental care for these children.

Amelogenesis imperfecta

The term amelogenesis imperfecta is usually applied to inherited defects of the enamel of both primary and permanent teeth (Figures 11.26–29). Although the definition implies a family history, for practical purposes it seems reasonable to extend this to include sporadic cases and also to those cases where the enamel defects are associated

Always consider the long-term implications of your treatment.

- Is it preferable to extract the first molar at 8–9 years of age and allow the second molar to migrate mesially? (See Chapter 14.)
- What impact will the treatment plan have on the future behaviour of the child?
- Many of these teeth are extremely sensitive – will local anaesthesia be effective?
- Once a stainless steel crown has been placed – what are the treatment options at age 20 years? A clear record of the state of the underlying tooth at crown placement is essential for the later restorative dentist.
- Does it matter if a third molar is not present – does this affect your decision?
- Is it preferable to leave a child free of disease – but also free of any restoration that will require multiple replacements throughout life?

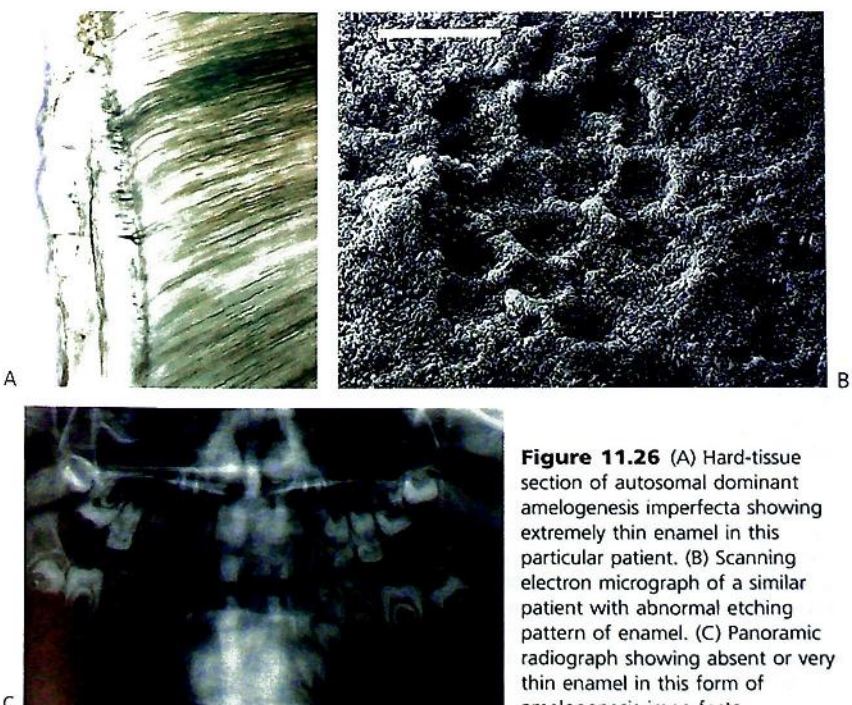

Figure 11.26 (A) Hard-tissue section of autosomal dominant amelogenesis imperfecta showing extremely thin enamel in this particular patient. (B) Scanning electron micrograph of a similar patient with abnormal etching pattern of enamel. (C) Panoramic radiograph showing absent or very thin enamel in this form of amelogenesis imperfecta.

with extra-oral features, as found in some syndromes (i.e. focal dermal hypoplasia or the trichodento-osseous syndrome).

Frequency examples

- Estimated 1:14000 in the USA.
- Up to 1:800 in northern Sweden.

Few studies of prevalence have been carried out and there may be marked differences according to the gene pool involved.

Diagnosis

- Based on a combination of the mode of inheritance and clinical and radiographic appearances.

Amelogenesis imperfecta variants with Mendelian inheritance

- X-linked (Xp22.3-p22.1) (OMIM 301200): The AMELX gene on the short arm of the X chromosome which codes for amelogenin, the major enamel matrix protein, has been shown to be mutated in several families with X-linked amelogenesis imperfecta.
- X-linked (Xq22-q28) (OMIM 301201): Another locus for X-linked amelogenesis imperfecta has been identified on the long arm of the X chromosome.
- Autosomal dominant (OMIM 104500): A number of genes in the 4q11–q21 region appear to be implicated in causing autosomal dominant amelogenesis imperfecta in some families. Mutations in the enamelin gene, which maps to the same region, have been identified. Families with autosomal dominant amelogenesis imperfecta with taurodontism (ADAIT) as part of the trichodentoosseous (TDO) syndrome have mutations in the DLX3 gene; one family with ADAIT without other features of TDO has also been found to have a mutation in the same gene, whereas in other ADAIT families mutations in the DLX3 gene have been excluded. With time, it is expected that this area will be better understood, as it is probable that as yet unknown genes are involved in the disorder in some families.
- Autosomal recessive (OMIM 204650): Mutations in the matrix metalloproteinase-20 (MMP-20) or kallikrein-4 (KLK-4) gene appear to cause autosomal recessive amelogenesis imperfecta.
- Sporadic cases.

Phenotypes

Phenotypes range from markedly hypoplastic (thin) enamel (Figure 11.27) (either uniformly with spacing between adjacent teeth or irregularly giving rise to pits or grooves) to varying degrees of hypomineralization (poorly formed enamel) with altered colour and translucency (Figure 11.28). In many cases, both hypoplasia and hypomineralization are seen together. The colour of the teeth is presumed to reflect the degree of hypomineralization of the enamel – the darker the colour the more severe the degree of hypomineralization.

In X-linked amelogenesis imperfecta (Figure 11.29), females exhibit vertical bands of altered enamel (manifesting lyonization; see Lyon hypothesis, above). There may be vertical grooves (because of hypoplasia) and/or vertical bands of enamel of altered

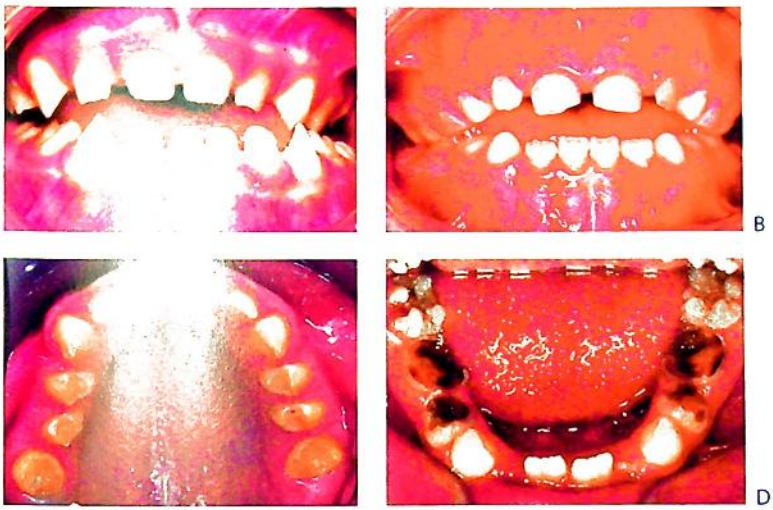

Figure 11.27 Different forms of predominately hypoplastic amelogenesis imperfect (AI). (A) Autosomal recessive with a rough hypoplastic phenotype. (B) Autosomal dominant with smooth hypoplasia with a marked anterior open bite. Note the open contact points in these two cases. (C) Hypoplastic AI teeth are yellow-brown with spacing between the teeth. (D) Rough or grossly pitted forms may be susceptible to caries as they are extremely difficult to keep clean.

colour or lucency (because of hypomineralization) or a combination of the two. In such families, there will be no male-to-male transmission, whereas the heterozygous females may pass on the trait to children of either sex.

In some patients affected by amelogenesis imperfecta, one or more teeth fail to erupt, presumably due to a more severe disturbance of the enamel organ, and may undergo resorption of their crowns. In some cases (up to 50%), a skeletal anterior open bite is seen.

Classification of amelogenesis imperfecta

There has been great controversy and confusion created with different nomenclature and classifications. Indeed, up to 14 different forms of the condition are described in some texts. It should be noted that all of these different manifestations are based on the clinical and radiographic appearance. It is essential that diagnosis and classification be based on the mode of inheritance and phenotype. Understanding the mode of inheritance is essential for genetic counselling. However, setting aside questions of inheritance for our present purposes, from a clinical treatment planning perspective, two clinically distinct basic forms are considered here.

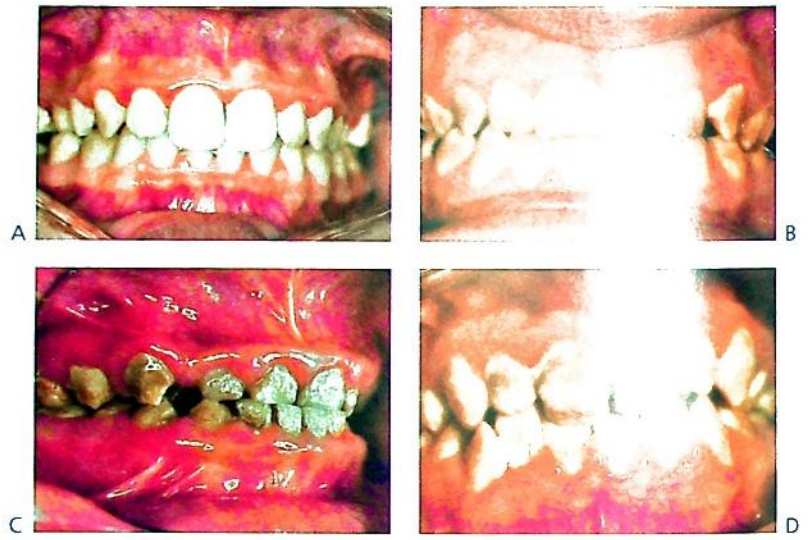

Figure 11.28 Different forms of predominately hypomineralized AI. (A–D) All these cases are characterized by varying degrees of hypomineralization. The enamel is so soft that it can be removed with an excavator. Note the discolouration and gross build-up of calculus on all tooth surfaces.

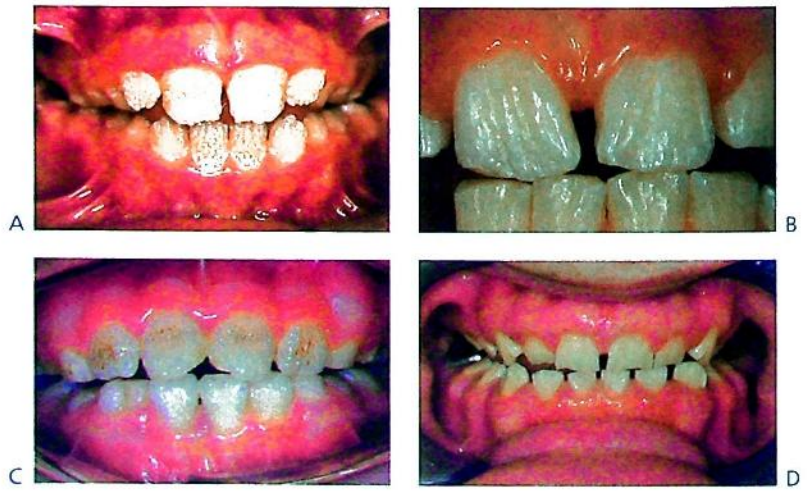

Figure 11.29 Different forms of AI with X-linked inheritance. (A,B) Typical appearance of vertical hypoplastic grooves in females. These represent enamel derived from different clones of ameloblasts that have undergone lyonization (X chromosome inactivation). (C) Pitting hypoplastic AI represents another clinical variety of X-linked AI. (D) X-linked AI in a male. All the enamel is affected uniformly.

Predominantly/exclusively hypoplastic forms (Figure 11.27)

- Thin enamel.
- Lack of contact points between teeth.
- Enamel may be rough, smooth, or randomly pitted.
- Heterozygous females with X-linked amelogenesis imperfecta manifest lyonization (see above) with vertical banding of normal and abnormal enamel.
- Teeth may be delayed in eruption.
- Unerupted teeth may undergo resorption.
- Anterior open bite associated in about 50% of cases.
- Radiographically, it may be difficult to distinguish enamel from dentine if the former is extremely thin.

Predominantly/exclusively hypomineralized forms (Figure 11.28)

- May be normal thickness of enamel, at least initially.
- Yellow to brown in colour.
- Enamel may be softer than normal, tends to chip and can be penetrated with an explorer. In severely hypomineralized cases, the enamel may be scraped away with a scaler.
- Teeth may erupt with enamel of normal thickness but it can be quickly lost, exposing highly sensitive dentine.
- Large masses of supragingival calculus may be present.
- Radiographically, can be difficult to distinguish between enamel and dentine because of decreased degree of mineralization of enamel.
- Unerupted teeth may undergo resorption; radiographic review is needed to monitor this.

Management

- Appropriate diagnosis, taking into account the mode of inheritance and phenotype.
- Continued commitment to and support of both children and families. These are disfiguring, painful conditions and children may be badly teased by their peers.
- Offer genetic counselling if appropriate.
- Early orthodontic assessment.
- Preservation of molar teeth with full coverage restorations to maintain vertical dimension.
- Overdentures may be an option in children with small, hypoplastic teeth (Figure 11.30C,D).
- Stainless steel crowns or gold onlays on molars (Figures 11.31 & 11.32) or laboratory-made composite resin crowns may be useful.
- Care is required when trial fitting crowns, because defective enamel can be easily scraped or flaked off the tooth in some cases.
- Composite resin veneers over anterior teeth for aesthetics. It is possible to bond composite resin successfully to hypoplastic and hypomineralized enamel (Figure 11.30A&B).
- Adequate margins may be difficult to achieve because of the poor quality of the enamel (Figure 11.32A).

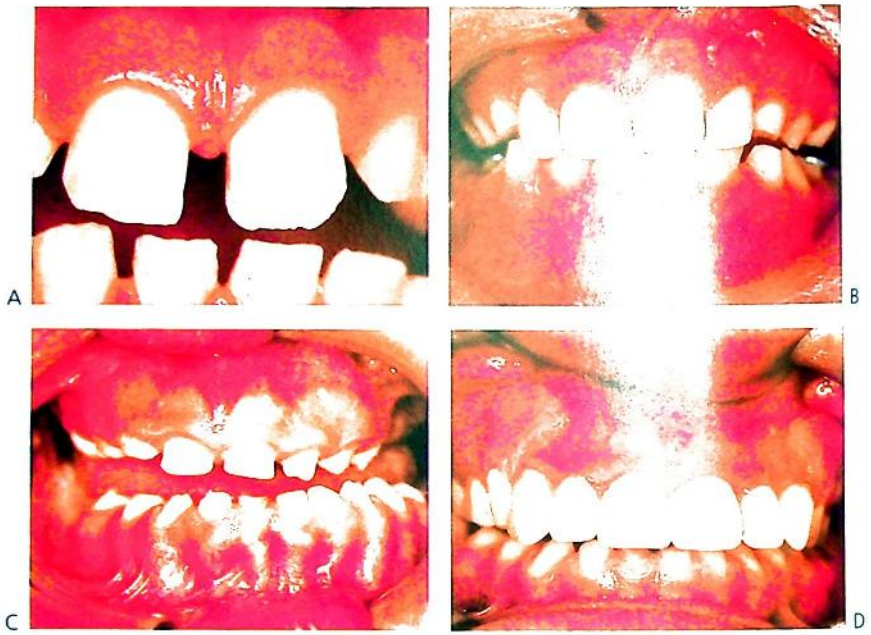

Figure 11.30 Different management options for amelogenesis imperfecta. (A,B) Composite bonding in a case with rough hypoplasia of the enamel. Etching times should be slightly longer than usual; however, the roughness of the enamel surface aids in mechanical retention. (C,D) A patient with autosomal recessive dystrophic epidermolysis bullosa with enamel defects. The posterior teeth failed to erupt and underwent spontaneous replacement resorption within the alveolus. Because of the small crown length and the prominent alveolus, an overdenture without a labial flange was constructed.

- Ideally, delay definitive treatment with porcelain and precious metals until late adolescence. However, some middle-aged patients have commented that, had they known that their dentition was going to 'fail' at that stage, they would have preferred their practitioner to have been less 'conservative' in their teenage years and to have provided more conventional restorative care. Two points arise:
  - Modern composite resins have improved greatly and 'adolescent' treatment now is hopefully more aesthetic and longer lasting than previously.
  - There is evidence of a clear association between these conditions and lack of self-esteem. It is as important here as anywhere in dentistry to treat the whole patient, and not only the teeth.
- Orthodontic and possible orthognathic surgery to correct anterior open bite in hypoplastic forms.

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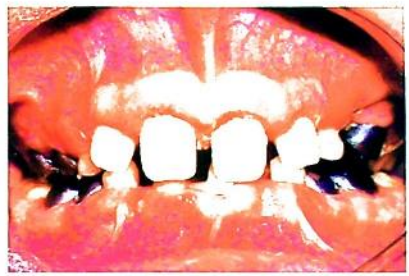

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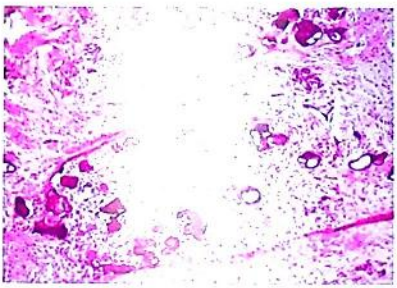
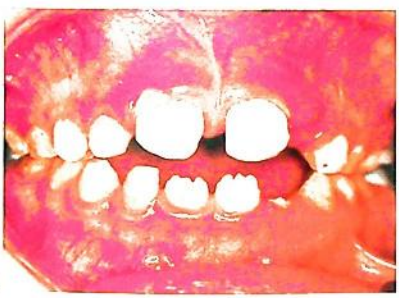


C

Figure 11.31 (A,B) A case of autosomal dominant amelogenesis imperfecta with failure of eruption of the anterior teeth. Many of the posterior teeth were unerupted and undergoing resorption. Initial surgical exposure of the anterior segments did not aid their complete eruption. The gingivae contained small islands of calcification which may be a significant factor in the failure of eruption (C). Periodontal surgery with apically repositioned flaps was used to fully expose the crowns.



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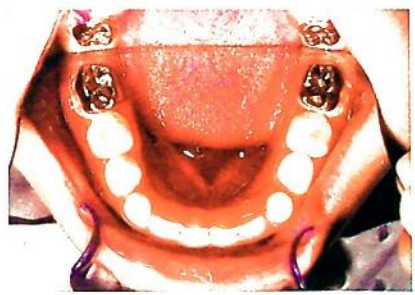

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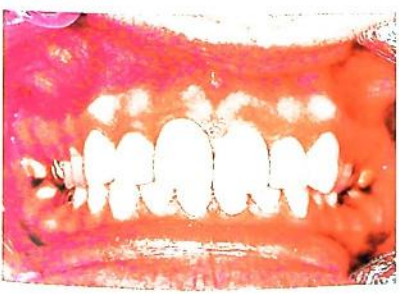

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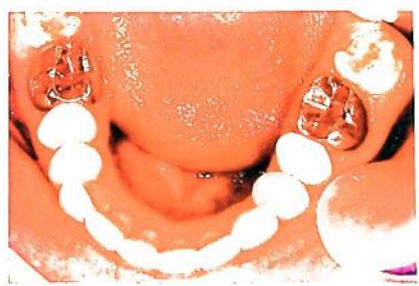


D

Figure 11.32 (A) Not all cases using composite resin are successful. With progressive eruption of the teeth it is difficult for the patient to keep the gingival margins clean and the restoration may therefore fail. (B) Cast gold onlays are useful to protect the occlusal surfaces. No preparation of the crown was performed. Onlays were cemented with composite luting cement. (C,D) Use of gold onlays and indirect composite resin veneers on anterior teeth and onlays on premolars. These composite restorations can provide good aesthetics during adolescence.

Clinical Hints

- Acid-etch composite resin seems to bond more successfully to hypoplastic enamel than to hypomineralized enamel.
- In severely affected dentitions, it is preferable to place stainless steel crowns on primary molar teeth very early (e.g. at around 3–4 years of age) to preserve the vertical dimension and allow maximal eruption of the first permanent molar.
- Cast metal (precious or base-metal) onlays on suitable permanent posterior teeth have the best long-term clinical success.
- Regular radiographic examination is required to detect early caries.

Disorders of dentine

Dentinogenesis imperfecta (OMIM 125490) (Figure 11.33)

Dentinogenesis imperfecta is an inherited disorder of dentine, which may or may not be associated with osteogenesis imperfecta. The term 'hereditary opalescent dentine' is sometimes used for the isolated condition. Both osteogenesis imperfecta and dentinogenesis imperfecta are transmitted as autosomal dominant traits and are clinically indistinguishable dentally, although they have a different genetic basis. Osteogenesis imperfecta is caused by mutations in the type I collagen genes and dentinogenesis imperfecta to mutations in the dentine sialophosphoprotein I gene. Some individuals and families with osteogenesis imperfecta may have clinical evidence of dentinogenesis imperfecta but in other families there may be variable expression of the trait. Within these families, some individuals may have abnormal dentine, while others are clinically unaffected as far as the teeth are concerned. However, because of the same collagen defect, all such children with osteogenesis imperfecta may have abnormal dentine, albeit at a subclinical level. The possibility of osteogenesis imperfecta should be considered in children presenting with dentinogenesis imperfecta and investigated by measurement of bone density if necessary. The presence of blue sclera or a history of bone fractures should alert the clinician to osteogenesis imperfecta.

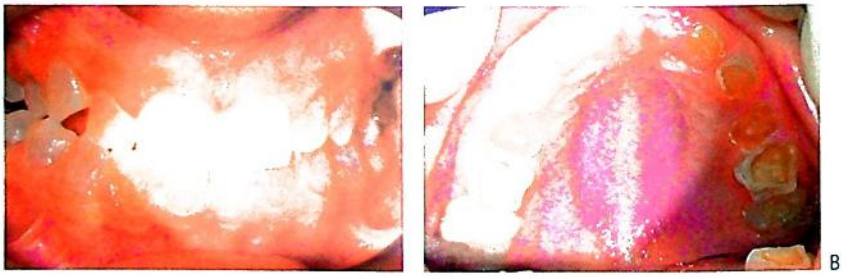


Figure 11.33 Manifestations of dentinogenesis imperfecta. (A) Dentinogenesis imperfecta associated with osteogenesis imperfecta. Dark discolouration of the crowns which appear normal in size and shape. (B) Severe attrition in the primary dentition in a case of dentinogenesis imperfecta.

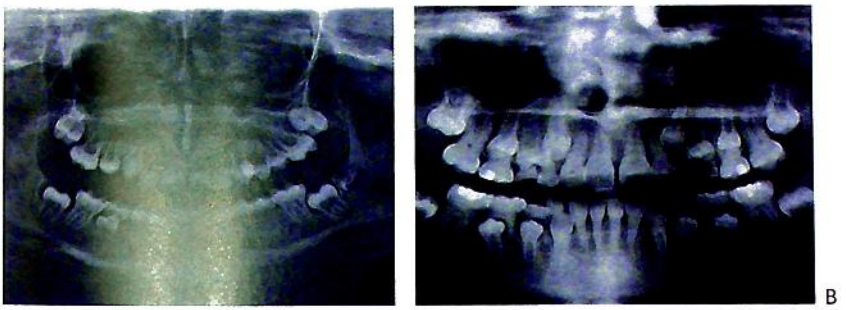


Figure 11.34 (A) Radiographic manifestations of dentinogenesis imperfecta showing short, bulbous crowns with wide-open root canals. (B) With further development, these teeth undergo pulpal canal obliteration, however, periapical pathology is fortunately rare.

Dental manifestations

- Amber, grey to purple-bluish discolouration or opalescence (Figure 11.33).
- Pulpal obliteration (Figure 11.34B).
- Relatively bulbous crowns.
- Short, narrow roots.
- Enamel may be lost after tooth eruption, exposing the soft dentine, which rapidly wears. This is probably due to inherent weakness in the dentine rather than because of an enamel defect or abnormality at the dentinoenamel junction.
- Mantle dentine appears normal.
- Circumpulpal dentine has poorly formed dentine with abnormal direction of tubules. Small soft-tissue inclusions represent remnants of pulpal tissue.

Management

- Preservation of the vertical dimension of the occlusion.
- Continued commitment to and support of both children and families, providing adequate aesthetics and function through childhood and adolescence.
- Protection of posterior teeth from attrition using full coverage restorations.
- Provision of aesthetic appeal.
- Stainless steel crowns for posterior teeth.
- Initially composite resin to build up anterior teeth, possibly followed later by porcelain crowns. (These teeth will remain or even become increasingly brittle throughout life. Conventional crowns requiring tooth preparation may never be the treatment of choice, but see above, under 'Amelogenesis imperfecta'.)
- Overdentures or even full dentures may be required in severe cases.

We have followed cases over many years into adulthood. The initial optimism over retaining these teeth for a life-time has been tempered by the eventual failure of complex restorative work and the loss of teeth in early adulthood. Clinicians must be

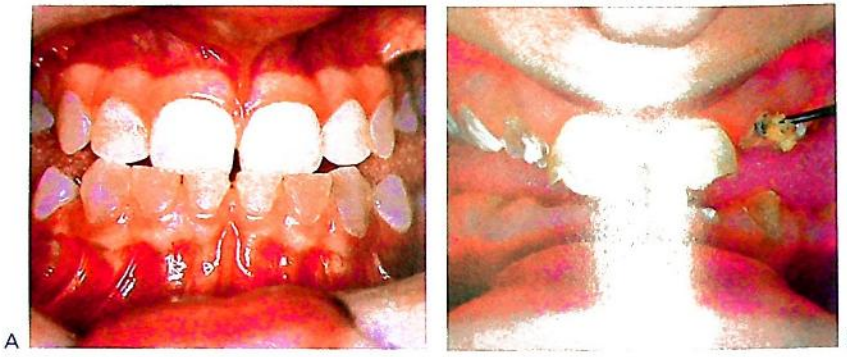


Figure 11.35 (A) It appears initially that the permanent dentition may not be as severely affected as the primary dentition in osteogenesis imperfecta. In this child, the severe skeletal Class III malocclusion and the posterior open bite required a surgical solution. (B) Clinical photograph of the same case at 30 years of age shows the destruction of the dentition over time resulting in the eventual loss of all of her teeth.

sensitive to the implications of long-term failure and the aesthetic, functional and indeed financial legacy with which the patient is left.

Osteogenesis imperfecta (OMIM 166200)

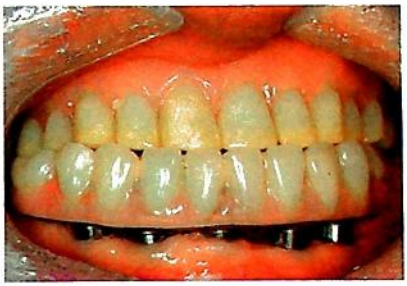
Osteogenesis imperfecta is caused by mutations in the collagen type I genes on chromosomes 7 (7q22.1) and 17 (17q21.3-q22). Previous classifications listed several variants of osteogenesis imperfecta with either autosomal dominant or autosomal recessive modes of inheritance. More recently it has been realized that most or all cases represent autosomal dominant osteogenesis imperfecta with variable expression. The essential features are:

- Bone fragility (Figure 11.36).
- Blue sclera.
- Progressive hearing loss.
- Dentinal changes.

Dentinal dysplasia – radicular dentinal dysplasia (Shields type I DD) (OMIM 125400)

- Originally described as rootless teeth, this appears to be a distinct entity from dentinogenesis imperfecta. Both dentitions are equally affected. The teeth may be lost early due to periapical infection or spontaneous exfoliation caused by the short roots.
- Autosomal dominant transmission.
- Teeth with very short or absent roots but clinically normal crowns (Figure 11.37).
- Total or partial obliteration of radicular pulp prior to eruption but with demilune of coronal pulp shown on the radiographs of molar teeth.
- Mantle and coronal dentine are histologically normal.


A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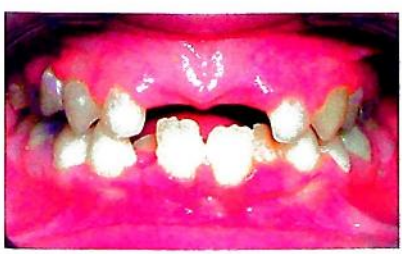
C

Figure 11.36 (A) The 'Ransford Loop' used to stabilize the vertebral column in a child with type IV osteogenesis imperfecta and basilar compression. This life-saving procedure is performed by an anterior approach through the pharynx, splitting the palate and sectioning the odontoid process of C2. The vertebral column is then wired to the occipital bone. This child initially presented with trigeminal neuralgia caused by pressure from C2 on the pons. (B) In spite of the bone pathology, osseointegrated implants can be successfully placed in patients with osteogenesis imperfecta. The same patient as in Figure 11.36A was rehabilitated with implant supported dentures following his surgery. (C) The panoramic radiograph shows the survival of the implants at 9-year follow-up.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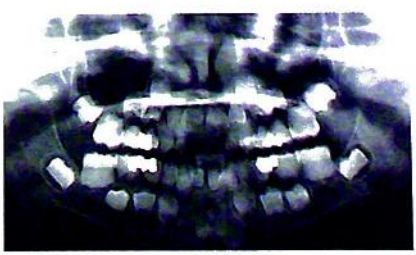
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Figure 11.37 Radicular dentinal dysplasia. (A) The general appearance of the teeth is relatively normal. (B) While it appears that there is complete absence of the pulp chamber, a small horizontal band of pulp is evident at the beginning of root formation that is quite abnormal in appearance. (Courtesy Prof M-C Maniere, Strasbourg, France.)

Management

- In spite of excellent preventive care, these affected teeth are commonly lost due to loss of enamel, pulp necrosis or periodontal disease.
- Prophylactic stainless steel crowns.
- Endodontic therapy may be successful if there is minimal pulpal obliteration.
- Long-term prognosis for the dentition is poor.

Dentinal dysplasia – coronal dentinal dysplasia (Shields type II DD) (OMIM 125420)

- The consensus now is that this is a variant of dentinogenesis imperfecta rather than a distinct entity. The primary teeth have a typical amber discolouration and undergo tooth wear associated with loss of the enamel and the appearance of 'shell teeth' radiographically.
- Normal crown and root form.
- Varying degrees of pulp canal obliteration.
- Altered pulp morphology resembling a 'thistle-shaped' pulp chamber.
- Intrapulpal calcifications (pulp stones).

Management

Similar to the management of dentinogenesis imperfecta, however, some authors suggest that no treatment is required, as there are few sequelae. If there is enamel loss in the primary dentition, then full coverage restorations should be placed (stainless steel crowns). If the permanent dentition is clinically normal, then no special care may be needed.

Different classification of dentine anomalies

Many texts describe up to four different forms of dentinogenesis imperfecta. All these dentine anomalies are autosomal dominant in inheritance. Dentinogenesis imperfecta has been mapped to 4q13–21. Linkage studies of families with coronal dentine dysplasia (Shields type II DD) have shown that the candidate mutation occurs in a region on 4q that overlaps the most likely location of the dentinogenesis imperfecta locus. Further, a similar locus has been determined to that of dentinogenesis imperfecta Shields type III (Brandywine isolate – OMIM 125500). These results suggest that dentinogenesis imperfecta (Shields type II), Shields type III and DD type II (coronal dentinal dysplasia) are allelic or the result of mutations in tightly-linked genes. Subsequent studies have identified that the mutated gene in all of these phenotypes in the DSPP gene. Radicular dentinal dysplasia may be a separate entity.

X-linked vitamin D-resistant rickets (OMIM 307800) (Figure 11.38)

- Also termed X-linked rickets or hereditary hypophosphataemic rickets is due to a defect in a gene located at Xp22.
- X-linked disorder with rachitic changes in long bones associated with a failure of distal tubular reabsorption of phosphate in the kidneys. The rickets is unresponsive to vitamin D.
- Short stature.
- Bowing of the legs.

Figure 11.38 (A) X-linked vitamin D-resistant rickets presenting with multiple abscessed teeth in the absence of caries. (B) Under polarized light, the hard-tissue section demonstrates globular dentine and a pulp horn that extends to the dentinoenamel junction, resulting in early exposure caused by attrition and subsequently pulpal necrosis.

- Males severely affected, females may show milder features (typically short stature with bowing of legs), often not affecting the teeth.
- Low serum phosphate.
- Elevated alkaline phosphatase.

Dental manifestations

- Attrition of incisal and occlusal enamel exposes elongated pulp horns, which often extend up to the dentinoenamel junction.
- Males (and some females), typically present with multiple abscesses in the absence of dental caries.
- Large pulp chambers and delayed apical root closure.
- May be reduced radiodensity of dentine on radiographs. Enamel may be spared or show some evidence of hypoplasia and/or hypomineralization.
- Patients may have repeated orthopaedic procedures and indwelling mechanics to promote bone straightening or lengthening. The avoidance of sepsis is essential. Treatment planning should include collaboration with orthopaedic colleagues and may demand the regrettable removal of infected teeth at times of particular infection risk.

Pre-eruptive intracoronal resorptive defects (Figure 11.39)

These defects are dentine lesions found on unerupted teeth, usually detected on routine dental radiographs. They have often erroneously been referred to as 'pre-eruptive caries' or 'dentine cysts'. They are often located adjacent to the dentinoenamel junction in the occlusal aspect of the crown. There is evidence that these defects develop as a result of coronal resorption. On opening into the lesion, it is often empty or filled with an amorphous tissue comprising small particles of tubular dentine and crystalline material. Resorptive cells such as osteoclasts and macrophages may be

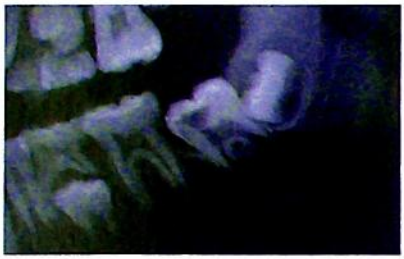


Figure 11.39 Pre-eruptive intracoronal resorptive defect in an unerupted mandibular second molar.

found. When the tooth erupts, the lesion is likely to be rapidly colonized by oral flora and the lesion becomes similar to a carious lesion.

Management

The cavity should be restored conservatively. It has been proposed that these lesions may be responsible for many of the lesions that are clinically undetected in molar teeth that progress to rapid carious breakdown and, ultimately, loss of the tooth.

Dental effects of prematurity and low birth weight

Normal birth weight for gestational age	>2500 g
Low birth weight	>1500–2500 g
Very low birth weight	<1500 g
Extremely low birth weight	<1000 g

Problems in extreme prematurity

- Hyaline membrane disease and respiratory insufficiency.
- Hyperbilirubinaemia (Figure 11.40D).
- Necrotizing enterocolitis.
- Cerebral intraventricular haemorrhage.
- Oxygen retinopathy.

The limiting factor in survival is based on lung development and infants weighing less than 400 g at birth or those born before 24 weeks, rarely survive. Hyaline membrane disease is now treated with synthetic surfactant, although very young babies often develop pneumothoraces caused by the prematurity and fragility of the alveoli. Cerebral intraventricular bleeding and necrotizing enterocolitis with the resulting sepsis are common causes of mortality and morbidity. Surviving children may be left with problems of growth retardation, delayed cognitive development and a range of other abnormalities.

Dental implications

- Hypoglycaemia.
- Hypocalcaemia with reactive pseudohyperparathyroidism.
- Hyperbilirubinaemia, causing intrinsic staining of the teeth.

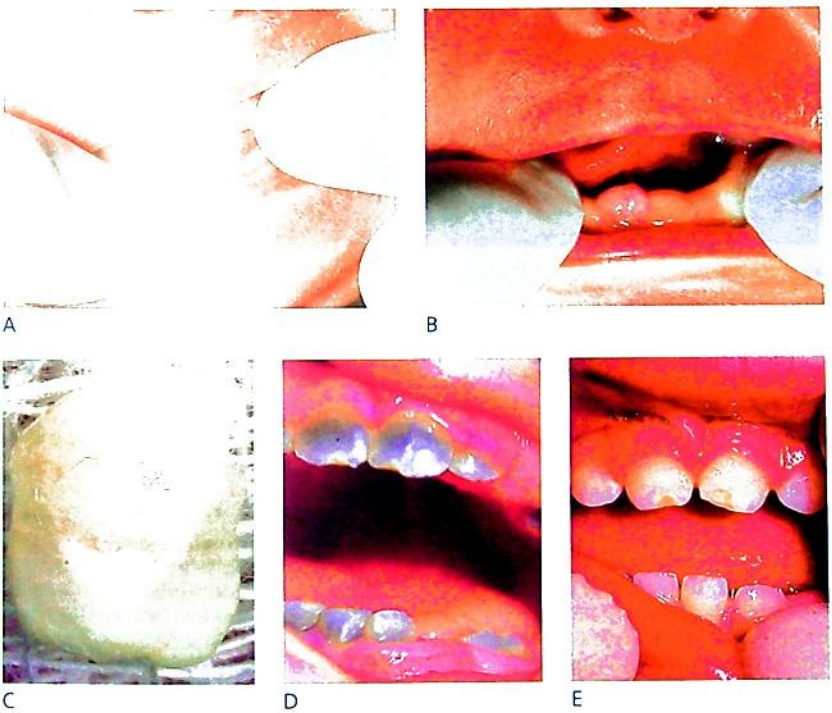


Figure 11.40 (A) Natal teeth in a 36-week premature infant. The teeth were extremely loose and the teeth were removed. (B) A newborn infant with two mandibular incisors soon to erupt. The reduced enamel epithelium has fused with the gingivae and teeth will probably erupt within a few days. (C) A neonatal tooth from a 27-week premature infant. Note the extent of the crown formation is consistent with the chronological age. (D,E) Dental effects of prematurity: hyperbilirubinaemia staining of the enamel (D) and hypoplastic defects on the incisal edges of the central incisors caused by laryngoscopy (E).

- Intubation trauma, causing enamel hypoplasia/hypocalcification. The maxillary central incisors are most often affected (Figure 11.40E). If the baby is intubated orally, palatal grooving may occur. Tooth eruption may be delayed, although it is often normal for the 'corrected' age after adjustment for prematurity.
- Chronological opacities or hypoplasia.

Disorders of eruption (Figure 11.41)

Eruption of teeth is not always correlated with somatic development. Children with growth disturbances may exhibit delayed eruption or the delay may be due to other

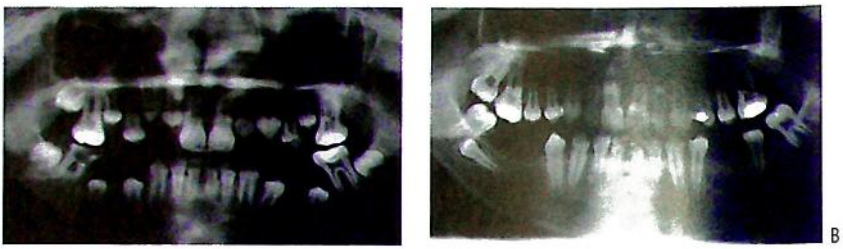


Figure 11.41 Teeth require guidance for normal eruption. (A) In this case the mandibular right first permanent molar was removed because of gross caries and pulpal necrosis. (B) While the second molar has drifted slightly mesially, the second premolar has rotated and drifted distally and impacted against the second molar. Had the second primary molar not been removed it is unlikely that this premolar would have drifted.

causes such as gingival overgrowth due to medication such as phenytoin. More importantly, premature exfoliation of teeth is invariably associated with severe systemic disease (see Chapter 10) and requires investigation.

Delayed eruption of the primary dentition requires no treatment other than determining that all teeth are present. It is uncommon for children to require surgical exposure of the teeth in infancy. Parents should be reassured that there is considerable variability in the eruption of teeth (plus or minus 6 months for primary teeth, plus or minus 1 year for permanent teeth). In the permanent dentition, delayed eruption beyond this range should be investigated for the presence of supernumeraries and other pathology. Although the actual timing of tooth eruption is variable, evidence of progress in tooth crown and root development and the eruption sequence are of much more relevance. In contrast, the failure of eruption of a contralateral tooth more than 6 months after the appearance of its partner requires investigation.

Natal and neonatal teeth (Figure 11.40)

A natal tooth is present at birth, while a neonatal tooth is one that erupts within 30 days of birth. In almost all cases, this is simply the early eruption of a normal primary incisor tooth. The development of this tooth is consistent with the expected stage of development of a primary incisor at birth (i.e. only five-sixths of the crown is formed without any root being present). This lack of root development accounts for the mobility of the tooth. Babies with posterior natal teeth should be carefully investigated for other systemic conditions that may be associated with syndromes or other diseases.

Management

- The most important point to consider is whether the nursing mother can adequately establish breast-feeding. If either the nipples or the ventral surface of the infant's tongue are being traumatized, the tooth should be removed.
- If the tooth is not excessively mobile it should be retained as it can become firm with time as the root continues to develop.
- If the tooth is excessively mobile, then it may spontaneously exfoliate; however, because of the theoretical risk of aspiration or ingestion it should be electively removed.

- Always protect the airway when removing these teeth by placing a gauze in the back of the mouth. The teeth are easily dislodged or dropped. A pair of Spencer Wells forceps or similar will provide a firm grip on the tooth to be removed.
- Check the medical history for significant jaundice, which may predispose to postoperative bleeding.

- If tooth removal is indicated, care should be taken to extract the entire tooth, as the crown only may be removed leaving behind the pulpal tissue. If this is the case, the dentine and a root will form subsequently; the root will then require removal at a later date.
- The permanent teeth should be unaffected by extraction of the primary tooth.

Infraoccluded (submerged) primary molars (Figure 11.42)

It is quite common for primary molars to become infraoccluded, however, it is controversial as to whether all teeth are truly ankylosed. The mechanism by which this occurs is unknown, but it has been suggested that a cessation of normal primary root resorption may stimulate healing and then ankylosis as the bone remodels. Some teeth never appear in the mouth and this infraocclusion may result from a failure or partial failure of teeth eruption, particularly secondary primary molars. If ankylosis occurs post-eruption, the tooth will appear to submerge into the alveolus (in fact, the tooth remains stationary while the alveolar bone grows around it and adjacent teeth erupt). The timing of the removal of an ankylosed tooth is based on the position of the first permanent molar and the extent of the resorption of the primary tooth.

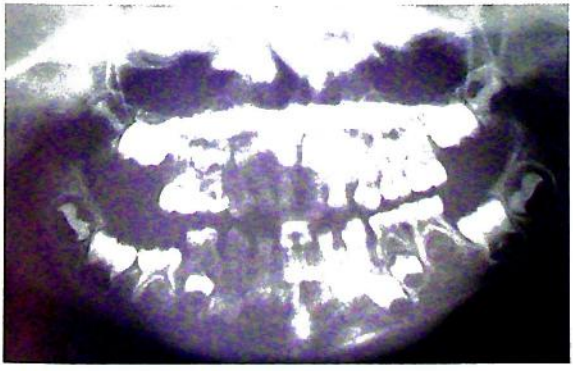


Figure 11.42 Failure of eruption of the mandibular right second primary molar. It is questionable whether these teeth are truly ankylosed. It is important to wait until the first permanent molar has erupted before surgical removal to avoid impaction of the second premolar. These teeth are often difficult to remove, especially if there is space loss and they should therefore be sectioned and elevated to minimize excessive bone loss.

Management

- If there is radiographic evidence of resorption of the roots, then removal should be delayed, as the vast majority of these teeth will exfoliate normally, because the tooth is probably not ankylosed.
- Orthodontic consultation (see Chapter 14).

Clinical Hints – Submerged teeth

- Submerged primary molars are difficult to remove intact surgically and there may be significant comorbidity associated with surgery.
- Where space has been lost due to migration or tipping of the first permanent molar, consider orthodontic uprighting in the first instance. Space can be maintained to await normal root resorption or facilitate more conservative surgical removal later.

- If the premolar is congenitally absent, early removal of the submerging primary molar might be indicated.
- Surgical removal of the ankylosed tooth is to be avoided before eruption of the first permanent molar as this latter tooth will migrate mesially and space loss is likely to occur together with impaction of the second premolar. Once the permanent tooth has erupted, the primary tooth may be removed and a space maintainer inserted.
- Retain space and use orthodontic treatment to align the permanent molar as required.

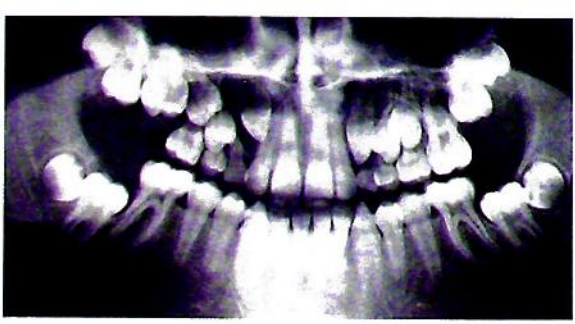
A   B

Figure 11.43 (A,B) Failure of eruption of first permanent molars is not uncommon. Surgical exposure of the crown may be sufficient to allow these teeth to erupt. Note that root development will continue in spite of a failure of tooth eruption and deviation of the roots will occur when the roots reach the cortical bone, in this case the antral floor. This root deviation may not be evident on radiographs, especially in the maxilla, and extraction of these teeth may prove extremely difficult.

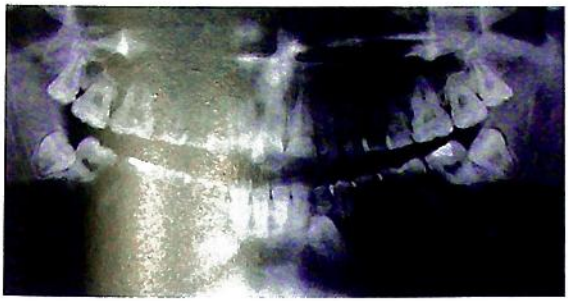


Figure 11.44 Arrested root development in a child who developed Stevens–Johnson syndrome at 10 years of age. Root development ceased at that time (probably as a result of the treatment as much as the disorder itself) and all teeth except the third molars were affected. It is interesting that these molars, which were not undergoing calcification, were unaffected.

Failure of eruption of first permanent molars (Figure 11.43)

Failure of eruption of a first permanent molar is an uncommon finding, however, no one has yet been able to explain the mechanism as to why these teeth do not erupt. During surgical intervention it is invariably noted that these teeth are not ankylosed.

Root development (Figure 11.44)

Just as enamel can be affected by systemic illness, so too can root development be delayed, altered or arrested by systemic disease. This is most commonly seen when radiotherapy causes shortening and tapering of the roots of premolars (see Chapter 10). Excessive orthodontic forces may also cause root resorption.

Dental age (maturity) determination

The paediatric dentist is often asked to help in age assessment, either at necropsy or for orphaned children. It is important to take into consideration ethnicity and variation in somatic growth potential.

Tooth emergence may not be as important as tooth crown calcification and root development. The most widely used and accepted method is that developed by Demirjian (1978), based on the panoramic radiographic appearance of tooth calcification at different ages.

Although there remains little doubt that peak height velocity, skeletal development and sexual maturation are associated, dental development seems to be independent of general somatic development.

Loss of tooth structure

- Attrition:
  - From wear of one or more teeth in one arch against one or more teeth in the opposing arch.

- Erosion:
  - Exogenous from diet, habits or environment.
  - Gastro-oesophageal reflux.
  - Bulimia.
- Abrasion.
- Exogenous tooth substance loss from diet, habits or environment.

Enamel erosion

The prevalence of erosion in children and adolescents has been reported recently as very high, with over half of 14-year-olds in a UK population having moderate erosion, with an increased prevalence seen in lower socioeconomic groups. The aetiology of erosion in children and adolescents is varied and it has been suggested that the increased consumption of fruit juices and carbonated drinks is the most important factor, with the sale of soft drinks increasing by 56% over the past decade.

The erosive potential of acidic drinks is related to:

- Titratable acidity (TA).
- pKa.
- Type of acid.
- Calcium chelation ability.
- Method and temperature of consumption.

Carbonated soft drinks contain carbonic acid and often organic acids (commonly citric acid) are added to improve taste and 'mouth feel'. Citrate ions strongly chelate calcium in both acidic and basic environments decreasing the amount of free ionic calcium available in both saliva and at the enamel surface and thereby enhancing demineralization. The erosive potential of 'diet' soft drinks is similar to that of sugared drinks; however, their potential to increase caries risk is decreased markedly. The method of drinking can also affect the extent of erosion, with the decrease in intraoral pH becoming greater as the beverage is held in the mouth or is drunk by 'long sipping'; when the beverage is gulped, intra-oral pH does not decrease significantly.

The long-term and short-term consequences of dental erosion are marked, with the need for extensive and costly dental care and potential loss of teeth. The concomitant dental sensitivity can be severe and debilitating. It has been shown that even a few intakes of acidic drinks on a regular basis may be associated with considerable dental erosion. It is important to question the child and parent(s) carefully as to total family usage of such drinks (fruit squashes, fresh fruit juices, particularly citrus juices, carbonated drinks and colas) in the first instance. The taking of study models and the institution of an exclusion diet for 3 months may show a diagnostic 'tide mark' of unattacked tooth substance at a later review.

Prevention of erosion

- Cessation or restriction of exposure to the aetiological factor.
- Modification of beverage erosivity seems to have the most future potential for reducing tooth structure loss. Recent research has concentrated on the addition of calcium and/or phosphate and pH alteration of soft drinks.
- Families are bombarded with advice on healthy eating. They may find advice to limit intake of fruit juices confusing and careful explanation is required.

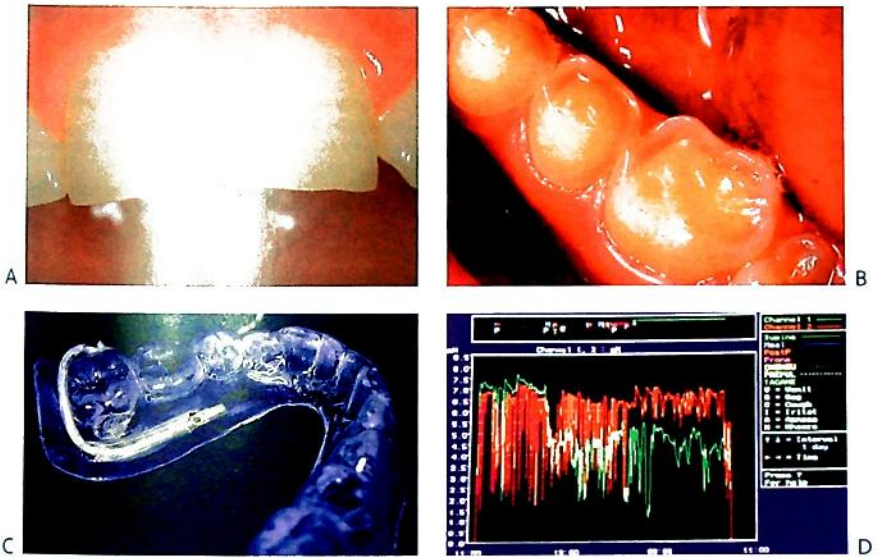


Figure 11.45 (A) Enamel erosion with asymptomatic gastro-oesophageal reflux. The first presentation of this child to a dentist was because of the erosion. Note the smooth, almost glassy appearance of the incisors. (B) Severe erosion in a 12-year-old boy showing the outline of enamel with exposed dentine. (C) A mouthguard containing a micro pH probe was worn in the mouth for a 24-hour period to measure the intra-oral pH. Another is placed in the lower oesophageal sphincter. (D) In some children an intra-oral pH of <1 has been recorded.

Gastro-oesophageal reflux (Figure 11.45A)

When loss of enamel by erosion cannot be explained by dietary factors, reflux must be considered. Children with reflux will show enamel erosion, which is a smooth loss of tooth structure, characteristically with any restorations standing proud. Some children have undiagnosed, asymptomatic reflux that presents first with enamel erosion.

Diagnosis

- Barium swallows may not demonstrate reflux.
- 24-hour pH manometry is required to assess the extent of reflux (Figure 11.38D).

Management

- Diagnosis and treatment of reflux condition before definitive restoration of the teeth.
- Histamine blockers (H_2 antagonists) such as ranitidine and cimetidine.
- Anti-emetics (prokinetic agents) such as metoclopramide.
- Composite resin, stainless steel crowns, glass ionomer cement coverings or onlays over the posterior teeth.
- Onlays on posterior teeth to protect occlusion and maintain vertical height.
- Professional application of fluorides. Nocturnal mouth guards with fluoride toothpaste as mechanical barriers against acid attack and fluoride to promote remineralization.

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12 Medically compromised children

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Introduction

Comprehensive dental care of a medically compromised child requires consideration of their underlying systemic condition and coordination of their dental treatment with their medical consultant. Although dental problems are common in this group, their oral health is overlooked frequently by the medical profession. The term used to identify this particular group, 'medically compromised children', has been replaced recently by the more general term 'children with special needs'. However, the older term is still relevant because it reminds the dentist that these children often have medical conditions that can affect dental treatment or that they can present with specific oral manifestations of a systemic disease. This chapter discusses the common paediatric medical conditions that require consideration in the provision of optimal dental treatment. The prevention of oral diseases is important in children with chronic medical problems (Figure 12.1), as oral complications can severely compromise a child's medical management and overall prognosis.

Cardiology

Congenital heart disease

Congenital heart disease (CHD) has an incidence of approximately 8–10 cases per 1000 live births and represents the largest group of paediatric cardiovascular diseases. Although most lesions occur individually, several form major components of syndromes or chromosomal disorders such as Down syndrome (trisomy 21) (see Figure 12.2A) and Turner syndrome (45, X chromosome) with over 40% of children being affected. However, in the majority of cases, no cause can be determined and a multifactorial aetiology is often assumed. Known risk factors associated with CHD include maternal rubella, diabetes, alcoholism, irradiation and drugs such as thalidomide, phenytoin sodium (Dilantin) and warfarin sodium (Coumadin). Turbulent blood flow is caused by structural abnormalities of the heart anatomy and presents clinically as an audible murmur. The degree of clinical morbidity is determined by the haemodynamics of the lesion. Congenital heart disease can be classified into acyanotic (shunt or stenotic) and cyanotic lesions depending on clinical presentation. Eight common conditions account for 85% of all cases.

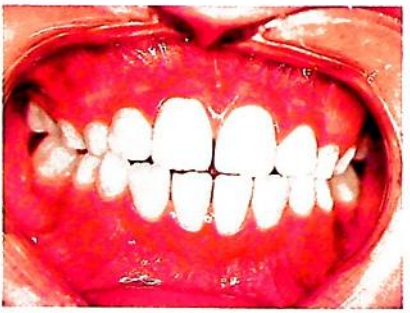
Acyanotic conditions

The acyanotic group of conditions is characterized by a connection between the systemic and pulmonary circulations or a stenosis (narrowing) of either circulation. Infants

A

B

Figure 12.1 (A) Children with medical problems may have conditions with a dental manifestation. This child has osteogenesis imperfecta – or they may present with a medical comorbidity that complicates their general dental care. (B) Not all children with medical problems require hospital admission, although treatment of such patients is often challenging. Mobile dental equipment is invaluable in providing quality dental care. The majority of hospital inpatients are treated in the dental surgery and usually only those who may be in traction or in intensive care units require bedside treatment.



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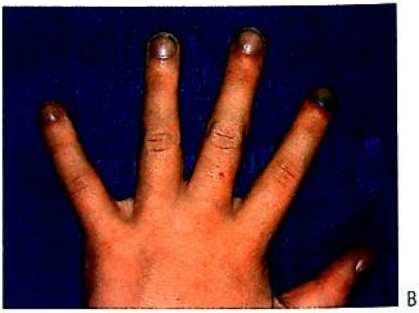


B

Figure 12.2 (A) Intra-oral photograph of a child with complex cyanotic cardiac disease associated with trisomy 21. Note the cyanosis of the gingival tissues. Of importance is to note that he has no dental disease. His oral hygiene is exceptional and there is no caries. Treatment would have been extremely difficult due to his intellectual disability. (B) Clubbing of the fingers associated with complex cyanotic cardiac disease. The nail beds also show cyanosis.

often present with feeding difficulties, breathlessness and failure to thrive. Shunts are from the left to right. The most common anomalies and their specific sites are:

- **Atrial septal defect** (ASD) – usually located near foramen ovale.
- **Ventricular septal defect** (VSD) – in the membranous septum of the ventricular wall.
- **Patent ductus arteriosus** (PDA) – caused by failure of closure of the ductus connecting the pulmonary artery with the aorta (normally closes soon after birth). If cardiac failure develops the infant is digitalized and prescribed diuretics if necessary. Hospitalization, oxygen, nasogastric tube feeding and antibiotic therapy for chest infection may also be required. If the lesion does not close spontaneously, surgery to reduce torrential pulmonary flow or repair of the defect is indicated.

Acyanotic defects with obstruction include:

- **Coarctation or localized constriction of the aorta** – usually in the area related to the insertion of the ductus.
- **Aortic stenosis** or narrowing of the aortic central orifice – due to fusion of the aortic valve cusps.
- **Pulmonary stenosis** – due to narrowing of the pulmonary valve which may also involve the pulmonary arteries.

Cyanotic conditions (Figure 12.2)

All cyanotic conditions exhibit right-to-left shunting of desaturated blood. Cyanotic defects become clinically evident when 50 g/L of desaturated haemoglobin is present in peripheral arterial blood. Infants with mild cyanosis may be pink at rest but become very blue during crying or physical exertion. Children with cyanotic defects are at significant risk for desaturation during general anaesthesia and preoperative consultation with the paediatric cardiologist and anaesthetist is essential.

The most common cyanotic lesions are:

- **Tetralogy of Fallot** – which includes a VSD, pulmonary stenosis at valve or subvalve levels, a large overriding aorta and right ventricular hypertrophy.
- **Transposition of great vessels** – when the aorta exits the heart from the right side and the pulmonary artery exits from the left. Although the internal heart anatomy is normal, the systemic blood circulation cannot be resaturated with oxygen and immediate management of transposition by opening the ductus arteriosus and corrective surgery is required.
- **Eisenmenger syndrome** – this refers to cyanosis from any right-to-left shunt caused by increased pulmonary resistance through a VSD or PDA.
- **Tricuspid atresia** – due to absent tricuspid valve and may present with an absent right ventricle and pulmonary valve. The pulmonary circulation is maintained through a PDA in the neonate.
- **Pulmonary atresia** – which is similar to tricuspid atresia except that the tricuspid valve is patent.

Other cardiovascular diseases

Other common paediatric cardiovascular disorders include cardiomyopathies such as myocardial disease and pericardial disease, cardiac arrhythmia, infective endocarditis

and rheumatic heart disease (RHD). Both CHD and RHD can predispose the internal lining of the heart to bacterial or fungal infection (infective endocarditis) and lead to the formation of friable vegetations of blood cells and organisms. Vegetations may embolize and cause renal, pulmonary or myocardial infarcts or cerebrovascular accidents. *Streptococcus viridans*, a common commensal organism in the oral cavity, is most frequently responsible for chronic infective endocarditis, whereas *Staphylococcus aureus* is often implicated in the acute fulminating form of infective endocarditis.

Dental management

Several important principles need to be followed when managing children with cardiac disease. Transient bacteraemia can occur following invasive dental procedures and potentially cause infective endocarditis in a susceptible patient. Therefore, all children with CHD or previous RHD require antibiotic prophylaxis to reduce the risk of infective endocarditis (see Appendix E). Those children who have been previously taking long-term antibiotics should be prescribed an alternative medication as per the protocol to avoid development of resistant oral organisms. In addition, a preoperative oral antiseptic mouthwash, such as 0.2% chlorhexidine gluconate, is recommended to reduce the oral bacterial counts.

Children with CHD have a higher prevalence of enamel anomalies in the primary dentition and concomitant risk of early childhood caries. Some cardiac medications may contain up to 30% sucrose and dietary prescription with high-caloric supplements (Polyjoule) further potentiate caries risk. Meticulous oral hygiene and preventive dental care, such as fissure sealants and topical fluoride therapy is recommended to reduce the risk of dental caries in susceptible children.

Dental disease in children with cardiac disorders can seriously complicate their medical management. Children with advanced cardiovascular disease should receive only palliative dental care until their medical condition has been stabilized. Aggressive treatment of pulpally involved primary teeth is recommended. Pulpotomy or pulpectomy is contraindicated in these children due to the possibility of subsequent chronic bacteraemia. Although routine treatment in the dental surgery environment is possible, it is often preferable to manage children with multiple carious teeth under general anaesthesia in the hospital environment. This protocol allows completion of treatment with one invasive procedure and negates the risk of infective endocarditis with further operative procedures. If multiple visits are planned, there is a need to prescribe alternative antibiotics or wait for a month between appointments to reduce bacterial resistance.

A thorough preoperative assessment of the child's regular medication (including anticoagulants, antiarrhythmics, and antihypertensives) is essential to avoid any potential drug interactions during treatment. There is no contraindication to the use of vasoconstrictors in local anaesthetic solutions. If conscious sedation is used, vital signs and oxygen saturation during the procedure should be carefully monitored. Avoid the use of electrosurgery, electronic pulp testers and ultrasonic cleaning devices in children with cardiac pacemakers, in case of potential interference. Some common impediments are non-compliance with oral hygiene and dietary advice, postoperative infection and bleeding.

Haematology

Disorders of haemostasis

Primary haemostasis is initiated after injury to a blood vessel with the formation of a primary platelet plug. This process is mediated by interactions between the platelets and coagulation factors in the plasma and the vessel wall. Secondary haemostasis or coagulation is also triggered by the initial injury and reaches its greatest intensity after the primary platelet plug is formed. Fibrin deposition provides the framework for the formation of a stable blood clot.

Prolonged bleeding can occur when either phase of haemostasis is disturbed. The clinical manifestations of a haemostasis disorder vary depending on the phase affected. Defects in primary haemostasis generally result in bleeding from the skin or mucosal surfaces, with the development of petechiae and purpura (ecchymoses). These disorders include von Willebrand's disease as well as defects in platelet function. In contrast, defects in secondary haemostasis, such as haemophilia, lead to bleeding that tends to be more deep-seated in muscles and joints. In both disorders, uncontrolled prolonged oral bleeding can occur from innocuous insults such as a tongue laceration or cheek biting.

Children with haemostasis disorders can be identified from a thorough medical history, examination and laboratory tests. Questions should reveal episodes of spontaneous bleeding or bruising; the occurrence of prolonged bleeding in other family members and prescription of anticoagulant medication. A physical examination of skin (unusual areas of bruising on the chest or back or bruising from lying on a toy), joints and oral mucosa should be undertaken for evidence of petechiae, ecchymoses and haematoma. If a haemostasis disorder is suspected, referral to a haematologist is recommended for evaluation and laboratory blood tests.

Laboratory tests

- **PFA 100** (platelet function analysis) may be used as a screening test for von Willebrand's disease and platelet dysfunction. If it is prolonged, specific testing for these disorders may be required.
- **Full blood count** (FBC) is required to determine platelet levels (normal range $150–400 \times 10^9$/L) and platelet function tests may be necessary in selected cases. Adequate haemostasis can usually be maintained by no less than 40×10^9 platelets/L.
- Coagulation tests include **prothrombin time** (PT) that is a test of the extrinsic coagulation pathway (normal range 11–17 s) and **activated partial thromboplastin time** (APTT), which is a test of the intrinsic coagulation pathway (normal range 24–38 s). Test results greater than 2 s compared with control values should be considered abnormal.

Classification

Vascular disorders

Vascular disorders are characterized by increased capillary fragility and include the purpuras, hereditary haemorrhagic telangiectasia, haemangiomas, vitamin C deficiency, Henoch Schönlein purpura and connective tissue disorders such as Ehlers–Danlos syndrome.

Platelet disorders

Platelet disorders can be either a deficiency (thrombocytopenia) or dysfunction.

Thrombocytopenia

Thrombocytopenia is defined as a platelet count $<150\times10^9$/L. Clinical signs and symptoms associated with decreased platelet counts are as follows:

- $<75\times10^9$/L – May exhibit post-surgical haemorrhage.
- $<25\times10^9$/L – Spontaneous haemorrhage, easy bruising.
- $<15\times10^9$/L – Petechiae appear on the skin.
- $<5\times10^9$/L – Oral petechiae, submucosal and mucosal bleeding.

Thrombocytopenia may occur as an isolated entity of unknown cause (idiopathic thrombocytopenic purpura, ITP), as a result of marrow suppression by drugs or from other haematological diseases such as aplastic anaemia. Marrow replacement by neoplastic cells in haematological malignancies will also result in thrombocytopenia. Children undergoing chemotherapy will have decreased platelet counts.

Thrombocytosis

Thrombocytosis is an increased number of platelets ($>500\times10^9$/L) and may be associated with prolonged bleeding due to abnormal platelet function. Myeloproliferative disorders may present with thrombocytosis.

Platelet function disorders

These may be congenital or acquired. The most common cause of acquired platelet dysfunction is the use of non-steroidal anti-inflammatory drugs (e.g. aspirin). Administration of cyclo-oxygenase inhibitors will result in blockage of the production of thromboxane A_2 for the life of the platelet (7–9 days). This results in a decrease in platelet aggregation. Some metabolic diseases such as Gaucher's disease also manifest as defects of platelet function.

A decrease in the number of platelets or platelet dysfunction will result in failure of initial clot formation. Children with thrombocytopenia will bleed immediately after trauma or surgery, unlike those with haemophilia, who usually start to bleed 4 h after the incident. The most common oral manifestations are petechiae and ecchymoses. There may also be spontaneous gingival bleeding and prolonged episodes of bleeding after minor trauma or tooth brushing.

Inherited coagulation disorders

Coagulation disorders result from a decrease in the amount of particular plasma factors in the coagulation cascade. The most common disorders are haemophilia A and von Willebrand's disease, both manifesting a decrease in factor VIII levels. The factor VIII is produced by endothelial cells and is composed of two portions. The largest part of the molecule is the von Willebrand's factor and is responsible for initial platelet aggregation. The factor VIII part of the complex and factor IX are responsible for activation of factor X in the intrinsic pathway of the coagulation cascade. Other disorders of coagulation include vitamin K deficiency, liver disease and disseminated intravascular coagulation usually from overwhelming (Gram-negative) infection.

Coagulation disorders are classified according to the defective plasma factor; the most common conditions are factor VIII (haemophilia A) and factor IX (haemophilia B or Christmas disease). von Willebrand's disease occupies a unique position in that both platelet and factor VIII activity is decreased, therefore both bleeding time and APTT are prolonged.

Haemophilia A

This is inherited as an X-linked recessive disorder with deficiency of factor VIII, and occurs 1 in 10000 live male births. Spontaneous mutation occurs in 30% of cases. The disease is classified as:

- Severe (<1% factor VIII) – with spontaneous bleeding into joints and muscles.
- Moderate (2–5% factor VIII) – with less severe bleeding usually following minor trauma.
- Mild (5–25% factor VIII) – which may not manifest until middle-age following significant trauma or surgery.

Factor VIII assay is usually performed after the initial diagnosis of a coagulopathy. Affected children and their families require considerable medical support and may have an indwelling central line for regular factor VIII concentrate infusion.

Haemophilia B or Christmas disease

This disease has clinical features similar to factor VIII deficiency. It is also inherited as an X-linked recessive gene and results in prolongation of the APTT. It is diagnosed by specific assay of factor IX.

von Willebrand's disease

von Willebrand's disease is inherited as an autosomal dominant trait (gene locus 12p13). The most common clinical manifestations include epistaxis and gingival and gastrointestinal bleeding. von Willebrand factor is found in the plasma, platelets, megakaryocytes and endothelial cells and circulates as a major component of the factor VIII molecule complex. This disease is divided into various subtypes, based on the platelet and plasma multimeric structure of the von Willebrand factor.

Dental management

Dental management of children with suspected haemostasis disorders should begin with screening laboratory tests. If tests are abnormal, haematological consultation is required for a definitive diagnosis. Invasive dental procedures should be performed only after the extent of the problem has been determined. Extractions must never be performed without first consulting the haematologist. It is preferable to have platelet levels $>80\times10^9$/L before extractions. Endodontic procedures may be preferable to extractions in order to avoid the need for platelet transfusion.

Dental procedures

- Use an atraumatic technique. In the event that oral surgery is necessary, a sound surgical technique to minimize trauma and local measures to control bleeding such as careful atraumatic suturing and socket dressings are mandatory.
- Maxillary infiltration anaesthesia can generally be administered slowly without pre-treatment with platelet or factor replacement. However, if the infiltration injection is into loose connective tissue or a highly vascular area, then factor replacement to achieve 40% activity levels is recommended.
- Avoid mandibular block injections as these may be complicated by dissecting haematoma and airway obstruction. In the absence of suitable factor replacement, intra-periodontal injections may be used, but with great caution. The anaesthetic solution is placed under moderate pressure along the four axial surfaces of the tooth by inserting the needle into the gingival sulcus and the periodontal ligament space.

- Nitrous oxide sedation can be effective for restorative procedures with the need for local anaesthesia; however, care must be taken when placing matrix bands.
- Use rubber dam to protect the soft tissues.
- Endodontic treatment can be safely carried out without factor cover.
- Periodontal treatment with deep scaling and subgingival curettage requires factor replacement.
- Multiple extractions require hospital admission and haematological work-up in conjunction with the haematology team.

Medical management

Haemophilia A

- All children and most adult patients are treated with recombinant factor VIII. A small number of adults continue to receive either recombinant or purified plasma derived factor VIII.
- Severe haemorrhage is treated to 100% replacement, although minor bleeds can be controlled with partial replacement between 30% and 50%.
- Minor trauma may also be life-threatening, especially with intracerebral bleeds.
- Some patients will form antibodies (inhibitors) to factor VIII, severely complicating medical management.
- A single unit of factor VIII concentrate per kg will raise blood levels by approximately 2% and has a half-life of 10–12 h.

Haemophilia B

- Same as for haemophilia A but infused with either recombinant (monoclonal) factor IX or highly purified plasma derived factor IX.

von Willebrand's disease

- Type I may be treated with 1-deamino (8-D-arginine) vasopressin (DDAVP).
- Types II and III require treatment with purified plasma derived factor VIII concentrate (which contains both factor VIII and von Willebrand's factor).
- Avoid platelet transfusions, if possible, due to the development of antiplatelet antibodies and the risk of transmission of viral diseases such as hepatitis B and C.

Other factor replacements

- Recombinant activated factor VII for haemophilia A with inhibitors and genetic deficiency of factor VII.

Antifibrinolytics

- Tranexamic acid (Cyklokapron).

1-Deamino (8-D-arginine) vasopressin (DDAVP)

- Can be used for people with mild haemophilia and those with von Willebrand's disease.
- Can result in an up to two-fold release of factor VIII from endothelial cells – this is adequate if levels of factor VIII are >10% and the patient is responsive to DDAVP.

Post-surgical administration of antifibrinolytic agents such as tranexamic acid (Cyklokapron) 25 mg/kg loading dose and 15–20 mg/kg three times daily for 5–7 days is helpful in preventing clot lysis. During the time that antifibrinolytics are given, the parent and child should be instructed not to use straws, metal utensils, pacifiers or baby bottle teats.

Characteristically, haemophilia bleeds are delayed 12–24 h, as primary haemostasis is not impaired, and local pressure has little effect. It is worth noting that mild haemophilia can go undiagnosed. The APTT is not sensitive to detect mild deficiencies of FVIIIc and levels of FVIIIc 25–30 IU/dL can be associated with a normal APTT. In addition, FVIIIc values in mild haemophilia are temporarily increased (as occurs in unaffected persons) by stress, exercise and bleeding. If there is a convincing history of a bleeding tendency always do a specific factor assay even if the initial screening tests are normal.

The normal regimen for DDAVP is 0.3 μg/kg intravenous infusion over 1 h before surgery followed by tranexamic acid 15–20 mg/kg orally every 8 h for 7 days. After 9–12 h, if the FVIIIc levels are still low (50–60%), then the original dose of DDAVP may be repeated. If repeated doses are planned or required it is important to fluid restrict the patient and monitor electrolytes. Repeated doses of DDAVP may cause fluid retention and hyponatraemia. This regimen is useful in von Willebrand's disease and children on renal dialysis.

Clinical

Questions commonly asked by parents are:

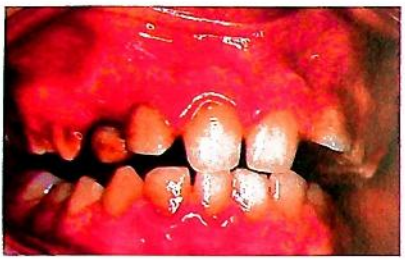
- Will my child's teeth erupt normally? Usually yes, but there is often more bleeding from a traumatized operculum that may require active intervention.
- Will my child's teeth fall out normally? Usually yes, unless continually traumatized, there is normally no abnormal bleeding associated with exfoliating primary teeth. However, if there is prolonged mobility and oozing occurs, then extraction may be necessary under appropriate factor cover to reduce the risk of persistent bleeding (Figure 12.3).
- Can a child with a bleeding disorder have orthodontic treatment? Yes, provided extractions are performed after appropriate consultation with the haematologist and there is vigilant maintenance of the appliances.

Anticoagulant therapy

Management of children on anticoagulant therapy needs special consideration. Anticoagulants are usually prescribed for children with valvular heart disease and prosthetic valves to reduce the risk of remobilization. If extractions or surgery are required, it is necessary to decrease the clotting times to facilitate adequate coagulation but not to such an extent so as to cause emboli or clotting around the valves. The dental management of these children is also complicated by their congenital cardiac defect and antibiotics are required for prophylaxis against infective endocarditis.

Therapeutic drugs used

- Oral warfarin sodium (Coumadin):
  - Vitamin K antagonist depleting factors II, VII, IX and X.
  - Usually 3–4 days are required for full anticoagulation onset and its efficacy is assessed by PT level (factor VII levels).



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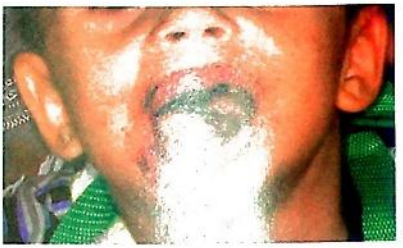


B

Figure 12.3 (A) Gingival haemorrhage around an exfoliating maxillary right primary canine in a child with Christmas disease (factor IX deficiency). Normally, exfoliation of primary teeth is not of major concern and bleeding is locally controllable. (B) A boy with haemophilia presenting following minor trauma to the labial frenum. Note the poorly formed clot in the mouth and continued oozing after several days.

- Heparin sodium (Heparin):
  - Shorter acting and has an immediate onset (inhibits factors IX, X and XII).
  - Can be administered either subcutaneously using a low-molecular-weight derivative or intravenously under the supervision of a paediatric haematologist.
- Enoxaparin sodium (Clexane):
  - Low-molecular-weight heparin which inhibits factor Xa and thrombin.
  - Usually administered subcutaneously.

Children on anticoagulant therapy should stop taking warfarin 3–5 days prior to the surgery date. In those in whom there is a significant risk for thrombosis with subtherapeutic warfarin level, parenteral anticoagulation may be necessary. This is generally achieved with enoxaparin sodium (Clexane) 1.5 mg/kg subcutaneously once daily (mane) via Insuflon. This drug is omitted on the morning of surgery. With the use of this regimen, the child may be admitted to hospital on the day of dental surgery. Warfarin is recommenced in normal dose on the evening of surgery. If further enoxaparin sodium prophylaxis is required, it should be given the morning after surgery and continued until the PT and international normalized ratio (INR) are therapeutic. Monitoring of enoxaparin sodium is rarely required. In emergency situations with prolonged bleeding from oral wounds post-surgery, following recommencement of warfarin, FFP (fresh frozen plasma) may also be of benefit.

Local haemostatic measures

- Application of topical thrombin (Avitene).
- Packing of the socket with microfibrillar collagen haemostat (MCH or CollaTape), oxidized regenerated cellulose (Gelfoam or Surgicel).
- Suturing of attached gingivae to maintain pressure.
- Splints or stomo-adhesive bandages may also be of benefit.
- There have been recent reports of the efficacy of 'fibrin glue' in the management of coagulopathies, but its use on moist oral mucosa is limited.

Management of oral haemorrhage

Unexpected bleeding from the oral cavity can occur at any time. There may have been a slow ooze for several days or, in the other extreme, there may be a significant sudden oral bleed. Such bleeding can occur without warning and may not be associated with any prior investigative or operative work. As well, haemorrhage from the mouth can occur following such routine procedures as biopsy, restorative work or tooth extraction.

The initial management of such cases involves identifying the exact site of haemorrhage, controlling the bleeding and then preventing a recurrence. In the cases of haemorrhage from the mouth that has not been associated with any dental procedure, clinicians should take an accurate history of the bleeding, the duration, lost volume and any causative factors. Abnormal bleeding may occur around an erupting tooth, from an exfoliating tooth site or may be associated with physical and sexual child abuse or congenital vascular anomalies such as arteriovenous malformations. The possibility of a childhood malignancy should also be considered.

In cases of oral haemorrhage following dental procedures, the following steps should be taken (it is important to prevent or minimize bleeding in the first instance):

- A sensible limitation of surgical trauma.
- Digital compression of the alveolus after tooth extraction.
- Packing of the socket with a resorbable gel.
- Adequate suturing of extraction sites to help reduce postoperative complications.
- Pressure application to the surgical site with gauze packs.
- Construction of a removable splint is recommended following more extensive surgery.
- Written postoperative instructions to ensure adequate rest, avoidance of hard foods and early mouth rinsing.
- Prescription of non-aspirin medication are necessary to avoid any parent misunderstanding.

In cases of severe uncontrollable haemorrhage following tooth extraction that can occur due to arteriovenous malformations, remember that the best method of controlling the bleeding is to replant the extracted tooth back into the socket and suture it well.

Red cell disorders

Anaemia

Anaemia is considered to be present if the haemoglobin level falls below 100 g/L. The cause of anaemia in children may be due to blood loss, iron, folate and vitamin B_{12} deficiency, bone marrow failure, haemolysis of red blood cells or anaemia of chronic disorders. It is usually an incidental finding in the routine dental management of children. A full blood count (FBC) is usually ordered when children present with pallor, lethargy, fever, bruising, undiagnosed systemic or oral pathology after major trauma associated with excessive blood loss or on work-up before surgery for other medical conditions. When unexpected anaemia is discovered, follow-up by the paediatrician is required.

Haemolytic anaemia

Acute haemolytic disease of the newborn or erythroblastosis fetalis is caused by ABO incompatibility and Rhesus (Rh) iso-immunization. There will be discolouration of those primary teeth that are calcifying at the time of birth. The cusp tips of the first permanent molars may also be affected. A yellow-green staining is most commonly seen as a result of high levels of unconjugated bilirubin.

Glucose 6-phosphate dehydrogenase (G6PD)

G6PD deficiency also results in acute haemolytic anaemia when the child is exposed to certain drugs (sulphonamides, chloramphenicol, aspirin, antimalarials) or infection (hepatitis).

Aplastic anaemia

Aplastic anaemia is defined as a decrease or absence of haemopoiesis in the bone marrow that is not due to marrow involvement or recognized disease process. FBC and bone marrow aspirate confirms the diagnosis. Bone marrow transplantation is the treatment of choice for moderate to severe aplastic anaemia.

Haemoglobinopathies

Thalassaemia

The haemoglobinopathies are a group of genetic disorders involving the globin chains of the haemoglobin (Hb) complex. These diseases comprise two main groups: the structural haemoglobinopathies, resulting in abnormal globins (HbE, HbS) and the thalassaemias. The thalassaemias represent a group of autosomal recessive disorders, common in patients from the Mediterranean, North Africa, the Middle East, India and Central Asia, expressing mutations of genes responsible for the production of any of the haemoglobin chains.

Haemoglobin is a tetrameric protein comprising four globin protein subunits. Adult blood contains haemoglobin A (HbA), comprised of two α-chains and two β-chains and a small amount of haemoglobin A_2 (HbA_2) comprised of two α-chains and two δ-chains. Children also produce fetal haemoglobin (HbF – two α-chains and two γ-chains), which has a much higher oxygen affinity. Fetal haemoglobin levels decrease after 6 months of age from around 70% at birth to trace amounts in adulthood.

α-Thalassaemia – is caused by deletions or mutations of the four alpha globin genes on chromosome 16. One to four genes may be affected, resulting in a relative overproduction of β-chains. Homozygous α-zero thalassaemia (four alpha globin genes deleted) is incompatible with life, while carriers (1–2 gene deletions) have no clinical symptoms. Children with HbH disease (three alpha genes deleted/abnormal) may have mild anaemia or a transfusion dependent anaemia.

β-Thalassaemia – Of more clinical significance is homozygous β-thalassaemia major (Cooley's anaemia). Due to the absence of the β-chain, there is a compensatory increased production of HbA_2 and HbF. As erythropoiesis is inadequate, the bone marrow is reactive and there is compensatory intermedullary haemopoiesis in the maxilla and diploe of the skull. There may be severe haemolytic anaemia with marked hepatosplenomegaly and failure to thrive. Those children with sickle/β-thalassaemia show evidence of vascular thrombosis with ischaemia to organs, especially bones.

Due to maxillary and zygomatic overgrowth there is often a severe Class II Division 1 malocclusion with separation of teeth and widening of the periodontal ligament space. Lateral skull radiographs demonstrate a 'hair on end' appearance. Children are

given regular packed red cell hypertransfusions until the haemoglobin rises to 140–150 g/L and desferrioxamine, an iron-chelating agent, to increase iron excretion. When excessive haemosiderosis in the spleen adds significantly to the haemolysis rate, elective splenectomy is performed.

Sickle cell disease

This is different from the other haemoglobinopathies in that the red blood cells are more susceptible to haemolysis and have difficulty passing through small blood vessels causing infarcts and ischaemia of organs and bone. These patients are usually asymptomatic unless subjected to low oxygen concentrations and this may be an issue when a general anaesthetic is required. Blood transfusions, analgesics, antimicrobials, adequate hydration and other life-supportive measures are necessary.

Dental management

Consultation with the child's haematologist prior to treatment is essential to arrange haematological preparation and transfusion. It is important to schedule dental treatment shortly after blood transfusions and provide antibiotic prophylaxis, especially if the child has had splenectomy. Avoid elective treatment if haemoglobin level is <100 g/L. Minimize stress that might compromise the child's ability to oxygenate the tissue adequately. Respiratory depressants should be avoided and additional oxygenation during conscious sedation or general anaesthesia is desirable along with the use of pulse oximetry. Local anaesthesia is not contraindicated but the use of prilocaine (Citanest) is not advised due to the formation of methaemoglobin. Vasoconstrictors in the standard dose are not contraindicated. Orthodontic treatment may be undertaken but teeth will move quickly through the bone and relapse will most likely occur.

Immunodeficiency

Immunodeficiency may be caused by quantitative or qualitative defects in neutrophils, primary immunodeficiencies, involving T cells, B cells, complement or combined defects and secondary immunodeficiency or acquired disorders.

Qualitative neutrophil disorders

Chemotactic disorders

- Chediak–Higashi syndrome.
- Lazy leukocyte syndrome.
- Leukocyte adhesion defects (Figure 12.4).

Phagocytic disorders

- Agammaglobulinaemia.
- Chronic granulomatous disease.

Quantitative neutrophil disorders

Neutropenia

This is defined as $<1.8\times10^9$ cells/L. Life-threatening sepsis is associated with a level of neutrophils $<0.5\times10^9$ cells/L. Neutropenia can occur in the following situations:

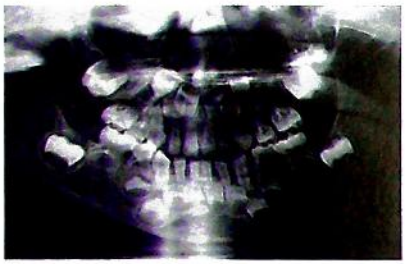


Figure 12.4 Leukocyte adhesion defect in a 4-year-old manifesting as severe periodontal disease and marginal bone loss. The maxillary and mandibular anterior teeth exfoliated within months of this radiograph being taken.

- Infiltration of bone marrow by neoplastic cells.
- After administration of cytotoxic drugs used for treatment of childhood malignancy.
- Cyclic neutropenia (21–28 day cycling).
- Agranulocytosis.
- Nutritional: protein-calorie malnutrition, vitamin B_{12} deficiency, copper deficiency.
- Viral induced neutropenia.
- Aplastic anaemia.
- Drug-induced neutropenia.
- Pseudo neutropenia: usually mild and spontaneously resolves.

Primary immunodeficiencies

B-cell defects

- Selective IgA deficiency.
- Agammaglobulinaemia.

T-cell defects

- Di George syndrome with thymic aplasia.
- Chronic mucocutaneous candidiasis.

Secondary or acquired immunodeficiencies

Secondary or acquired immunodeficiencies include those conditions acquired during childhood, such as:

- Human immunodeficiency virus (HIV) infection.
- Drug-induced immunodeficiency (cytotoxics, corticosteroids, cyclosporin A, tacrolimus).

These can also occur in children who have undergone bone marrow transplantation and radiotherapy (radiotherapy-induced immunodeficiency).

Combined immunodeficiencies

- Severe combined immunodeficiency.
- Wiskott–Aldrich syndrome.
- Ataxia telangiectasia.

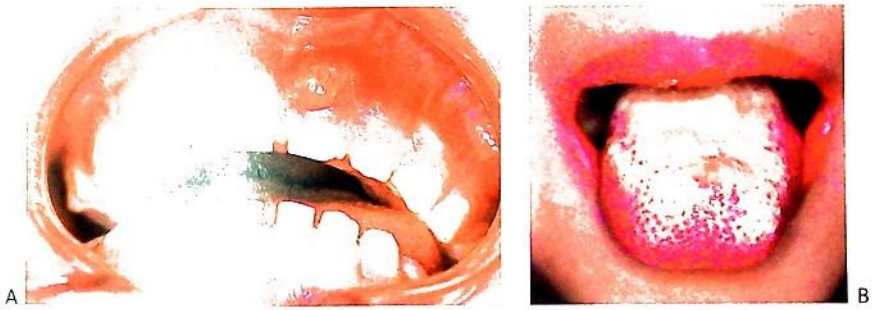


Figure 12.5 Two manifestations of immunodeficiency. (A) Abscess formation above the maxillary right primary lateral incisor tooth after administration of high-dose steroids for asthma, in an area that was previously quiescent. (B) Candidal infection of the tongue in an immunocompromised child.

Dental implications

Both neutrophil and T-cell-mediated immunodeficiencies predispose the child to infection by compromising the host defence system. Opportunistic organisms that do not usually cause disease in a healthy child can proliferate in the oral cavity of the immunodeficient host. Common oral manifestations seen are:

- Acute pseudomembranous candidiasis (Figure 12.5B).
- Severe gingivitis.
- Generalized prepubertal periodontitis (Figure 12.4).
- Gingivostomatitis.
- Recurrent aphthous ulceration.
- Recurrent herpes simplex (HSV) infection.
- Premature exfoliation of primary teeth.

Generally, B-cell deficiencies exhibit fewer oral complications but are often associated with chronic bacterial infections such as pneumonia, otitis media and skin lesions.

Dental management

Regular review of the developing dentition, gingivae and mucosa and the institution of a preventive programme are essential for maintenance of healthy hard and soft tissues. Elimination of any potential oral focus of infection during the course of medical treatment is the primary objective.

The underlying deficiency must be fully assessed and the likelihood of oral complications endangering the child's medical status should be evaluated. An individual risk–benefit assessment of any oral lesion must be considered with regard to the overall management plan.

A decision whether to extract or maintain carious teeth and exfoliating primary teeth must be based on the worst case scenario during the immunodeficiency period. If a carious lesion cannot be stabilized with an adequate interim restoration, then extraction is the preferred treatment. In a case being prepared for bone marrow

transplantation, all mobile primary teeth should be removed at least 2 weeks prior to the conditioning phase.

Thorough dental scaling and prophylaxis and the provision of custom trays for delivery of medication (antiseptic or fluoride gels) prior to commencement of head and neck radiotherapy is also recommended to prevent oral sepsis and radiation-induced dental caries.

Prophylactic antimicrobials specific for commensal oral organisms determined from culture and sensitivity tests are indicated during the course of medical treatment. Biopsy specimens may assist the diagnostic process. The antimicrobial protocol may include appropriate antibiotics (amoxicillin trihydrate and ampicillin, vancomycin), acyclovir sodium if HSV-positive, ganciclovir if cytomegalovirus-positive, antifungals (topical nystatin and amphotericin B) and twice daily 0.2% chlorhexidine gluconate (Curasept) mouthwashes during the active therapy phase.

Acquired immunodeficiency syndrome (AIDS)/HIV

HIV infection has been identified in increasing numbers of children with otherwise unexplained immune deficiency and opportunistic infections of the type found in adults with acquired immune deficiency syndrome (AIDS). For the limited purposes of epidemiological surveillance, the Centers for Disease Control (CDC) characterizes a case of paediatric HIV infection as a reliably diagnosed disease in children that is at least moderately indicative of underlying cellular immunodeficiency, and with which no known cause of underlying cellular immunodeficiency or any other reduced resistance is reported to be associated.

Transmission

The main transmission media are body fluid, such as blood and semen. Saliva contains low and inconsistent levels of the HIV virus and is unlikely to provide a significant mode of transmission. Consequently, the two major routes of transmission in children are vertical (from an infected mother) and from blood products, with children with haemophilia being most at risk. Vertical transmission rates are up to 39% and occur before, during or after birth. Infection from breast-feeding may be up to 29%.

Risk factors

- The risk factors for paediatric HIV infection vary depending on the age group.
- Most children with AIDS are under 5 years of age.
- The primary risk factors are perinatal.
- Infants born to women who are intravenous drug users or who have bisexual partners comprise the largest group.
- About one-third of the infants weigh less than 2500 g at birth and are small for gestational age. Of these babies, 25–30% of children develop AIDS in the first year of life.
- The presenting pattern of encephalopathy varies with age and significant growth failure occurs in early infancy.

Figure 12.6 Severe oral ulceration in a child in the terminal stages of HIV/AIDS. The ulceration was most likely due to disseminated HSV infection, to which the child succumbed 7 days after this photograph was taken.

Serodiagnosis and immune function

The screening ELISA test for HIV antibodies is liable to give false negatives and any apparently positive results must be confirmed by the western blot assay. Antigen assays are far more reliable, but a failure to detect virus or antigen in a young antibody-positive child does not exclude infection. A positive virus or antigen test is likely to indicate infection. Due to the long incubation period and the limitations of medical history and serodiagnosis, it must be assumed that all blood derivatives may be infectious.

The human immunodeficiency virus attaches to the CD4 variant of the T4 helper lymphocyte and remains within infected cells throughout their life, being transmitted to other cells mainly by cell-to-cell contact. Other cells that may be affected include macrophages, and possibly endothelial, neuroglial, epithelial and dendritic cells. The principal effect of HIV infection on the immune system is depletion of CD4 lymphocytes (helper cells), which results in a drop in the absolute CD4 count and a reversal of the CD4/CD8 ratio. These are immune indicators of disease progression.

Oral manifestations (Figure 12.6)

Oral lesions are often early warning signs of HIV infection. Common disorders may manifest in different ways in the presence of HIV. In children, the most common lesions are:

Candidosis

The most common oral lesion in HIV infection is acute pseudomembranous candidiasis. It is an early lesion and suggests the presence of other opportunistic infections. The severity of the candida infection may be related to the T4/T8 ratio and occurs when CD4 counts are <300/mL. Oesophageal candidiasis occurs when CD4 counts drop below 100/mL. Fungal infections can be related to reduced salivary flow and S-IgA. It responds well to treatment with systemic antifungals and an improvement in oral hygiene.

Ulceration

Recurrent herpes simplex infections are frequent and are typically intra-oral and circum-oral. Other parts of the body may also be affected. Aphthous-type ulcers are persistent and very common in children. Treatment is palliative with adequate hydration, analgesia and the use of Diflamm-C mouth rinses.

Atypical gingivitis

HIV-related gingivitis manifests as red erythematous gingival tissues and can extend to the free gingival margin. There is often spontaneous gingival haemorrhage and petechiae within the gingival margin, either localized or generalized. Consideration must be given to a fungal component. Treatment involves improved tooth brushing and flossing and the use of daily 0.2% chlorhexidine gluconate (Curasept) mouthwashes and gels.

Salivary gland enlargement

Parotitis or HIV associated parotid gland disease (HIV-PGD) occurs more frequently in paediatric than in adult patients and is similar to the presentation of mumps. It may be unilateral or bilateral and results in xerostomia and pain. Reduced salivary flow may lead to pseudomembranous candidiasis and dental caries. There have been mixed results with the use of antibiotics and glucocorticosteroids in treating this condition. Artificial saliva substitutes or oral lubricants can alleviate the xerostomia.

Hairy leukoplakia

This is uncommon in children, with only a few reported child cases. It occurs predominantly on the lateral border of the tongue and occasionally on the buccal mucosa and the soft palate.

HIV-related periodontitis

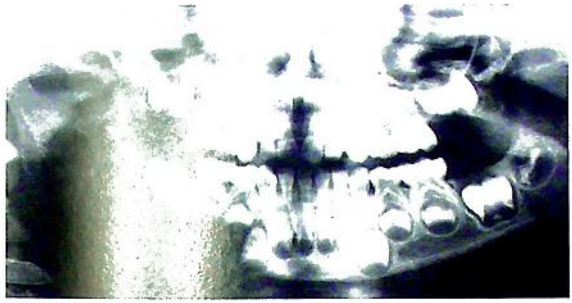
HIV-related periodontitis presents with deep pain and spontaneous bleeding, interproximal necrosis and cratering, and intense erythema more severe than acute necrotizing ulcerative gingivitis (ANUG). HIV periodontitis appears more frequently in HIV-infected patients who have reduced T4/T8 ratios and symptomatic opportunistic infection. Organisms such as black-pigmented bacteroides and Gram-positive bacilli, which are similar to those found in adult periodontitis, have been identified in HIV periodontitis.

Kaposi's sarcoma

Uncommon in children and adolescents. The lesion mainly affects the palate, and also the gingivae and the tongue. Treatment is by chemotherapy, radiotherapy or laser excision.

Outcomes

Primary colonization by commensal organisms rather than reactivation of opportunistic infections usually occurs (cytomegalovirus, retinitis and toxoplasmosis are rare). Bacterial infections are also rare, although *Streptococcus pneumoniae* and *Haemophilus influenzae* are common respiratory complications. Kaposi's sarcoma is seen infrequently but lymphomas (especially with central nervous system (CNS) involvement) can occur. The progression of disease process can vary and in many instances, oral and physical symptoms do not often present for years after infection with the immunodeficiency virus. Lymphocytic interstitial pneumonitis is frequently the cause of death for children with AIDS, but is often asymptomatic. There have been major advances in the management of HIV/AIDS with antiretroviral medications and consequently, many children may lead a normal and effective life.



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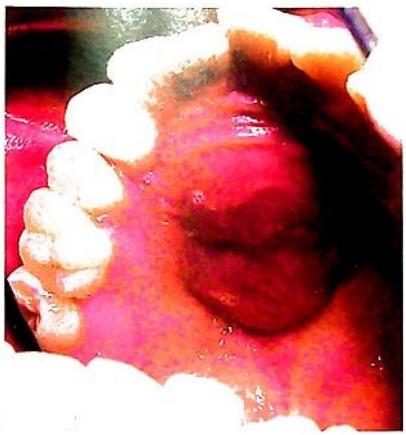

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Figure 12.7
Neoplasms may arise as primary lesions within the jaws, invade from local tumours or may seed as metastases from distant primaries. (A) The panoramic radiograph shows an extensive primary neoplasm of the right mandible involving the infratemporal fossa. Histologically, this lesion was a desmoplastic fibroma and required a hemimandibulectomy. (B) Presentation of a lymphoma in the palate of a 15-year-old girl. The lesion was asymptomatic and the child had merely presented for a routine check-up.

Oncology

Childhood cancer accounts for about 1% of all cancer cases in the population. In Australia, the annual incidence of malignant tumours in children under 15 years is approximately 11 per 100 000 children. Approximately 600–700 children between birth and 15 years of age develop cancer each year. Whereas most adult cancers are carcinomas with strong aetiological associations, childhood cancers are a wide range of different histological types of tumour with less aetiological connection.

The incidence, either in childhood cancer as a whole or in individual types of cancer, varies little from one country to the next and no racial group is exempt. Among more than 50 types of childhood cancers, the most common forms include leukaemias, lymphomas, CNS tumours, primary sarcomas of bone (Figure 12.7A) and soft tissues, Ewing sarcoma, Wilms' tumours, neuroblastomas and retinoblastomas. Acute leukaemias and tumours of the CNS account for approximately one-half of all childhood malignancies. Multimodal therapy (chemotherapy, radiotherapy and surgery) has resulted in an overall 5-year survival rate for childhood cancer of approximately 70%.

Leukaemia

Leukaemia is a heterogeneous group of haematological malignancies caused by clonal proliferation of primitive white blood cells.

Acute lymphoblastic leukaemia (ALL)

- Accounts for 80–85% of acute childhood leukaemias.
- Defined by the presence of more than 25% lymphoblasts in the bone marrow.
- Therapy is tailored to the risk of relapse dependent on cytogenetic markers and includes a combination of induction chemotherapy, CNS prophylaxis and maintenance chemotherapy for approximately 2 years' duration.
- Intrathecal therapy (commonly methotrexate) has been used to replace cranial irradiation.
- Cure rates for standard risk ALL are now over 90–92% on current protocols. If relapse occurs 40–50% can be cured with chemotherapy and/or haematopoietic stem cell transplantation.
- Prognosis depends on age of onset, initial white cell count, cytogenetic abnormalities and other features.
- Bone marrow transplantation is reserved for very high risk or patients with relapse.

Acute myeloid leukaemia (AML)

- Accounts for 15–20% of acute childhood leukaemias.
- In this disease, the bone marrow is infiltrated with primitive myeloid cells, classified by their morphological appearance (FAB subtypes M1–M7). The clinical features of AML are similar to other leukaemias.
- AML with monocytic morphology (M4/M5) can manifest gingival infiltration and promyelocytic morphology (M3) is associated with disseminated intravascular coagulation.
- Induction chemotherapy therapy is often followed by early allogenic bone marrow transplantation for high-risk patients.
- The cure rate is approximately 60% with modern therapy.

Chronic myeloid leukaemia (CML)

- Rare in childhood and accounts for less than 5% of leukaemic cases.
- Two types: one identical to adult CML and characterized by the presence of the Philadelphia chromosome (Ph) in malignant cells; the second or juvenile form (JCML) occurs earlier in infancy with a rapid course, infection, haemorrhage and poor survival rate.
- Bone marrow aspirate reveals granulocytic proliferation without an excess of lymphoblasts.
- The chronic phase of this disease is now effectively treated with specific Bcr-Abl tyrosine kinase inhibitors (imatinib or dasatinib), which can lead to remission lasting for years.
- Allogeneic bone marrow transplantation remains the only definitive curative therapy but is now generally reserved for patients relapsing on tyrosine kinase inhibitors or for children who have an HLA-matched sibling.

Clinical features of childhood leukaemia

- Fatigue and weight loss.
- Anaemia.
- Purpura.
- Infections and unexplained febrile episodes.
- Marked hepatosplenomegaly and lymphadenopathy.

Investigations

- FBC shows anaemia, neutropenia, thrombocytopenia and leukocytosis with circulating lymphoblasts.
- Bone marrow aspirate is required to confirm diagnosis.
- Lumbar puncture to exclude CNS involvement.

Problems in medical management

The main problems in medical management are bone marrow suppression initially at diagnosis due to malignant infiltration and later due to chemotherapy, subsequent anaemia, infection and mucosal ulceration and bleeding. Infection in the immunocompromised child is a life-threatening condition and may be due to bacteria, viruses, fungi or parasites. Broad-spectrum triple antibiotic treatment is usually required. Disease relapse may occur in the marrow, CNS or other organs (e.g. testes).

Solid tumours in childhood

Brain tumours

- These are most frequent solid tumours of childhood.
- Approximately 70% are gliomas, mostly low-grade astrocytomas or medulloblastoma.
- More than 50% of paediatric intracranial tumours occur in the posterior cranial fossa region. Surgical excision combined where possible with chemotherapy and radiotherapy is the standard approach to treatment.
- Chemotherapy can be used to delay or avoid cranial radiotherapy in infants.
- The overall survival rate is approximately 60% at 10 years.

Non-Hodgkin's lymphoma (Figure 12.7B)

- Arises from neoplastic B or T lymphocytes in lymph nodes and lymphoreticular tissue.
- The primary tumour may be abdominal (B cell) or in the mediastinum (T cell).
- Tumour spread is usually local or to bone marrow and CNS.
- The primary mode of therapy is chemotherapy.
- 90% cure rate for localized tumours.

Wilms' tumour

- Occurs in the kidney around 3–4 years of age.
- Usually presents as an asymptomatic abdominal mass.
- Often associated with aniridia and other congenital anomalies.
- The tumour responds well to combined therapy: chemotherapy with or without radiotherapy to reduce the tumour mass and surgical removal depending on disease stage. Commonly lung, hepatic and skeletal metastases occur.

Neuroblastoma

- Arises from neural crest cells anywhere along the sympathetic chain.
- Most common site is abdominal, either in the adrenal gland or paraspinal ganglia. Other sites include thorax, neck or pelvis.
- Tumour spread to lymph nodes, bone marrow, liver or subcutaneous tissues.
- Diagnosis is confirmed by raised levels of urinary catecholamines and tissue biopsy.
- Prognosis depends on patient age at diagnosis, tumour stage and biological features of the tumour, especially presence of amplification of the n-myc gene. Children with high-risk disease (approximately 50% of cases) have 25% survival rates even with aggressive chemotherapy, surgery, radiation and autologous bone marrow transplantation.

Rhabdomyosarcoma

- Arises from embryonal mesenchymal tissue with potential for differentiation to skeletal (striated) muscle.
- Children often present with a painless, usually rapidly enlarging subcutaneous lump, almost anywhere in the body.
- Common sites include head and neck, genitourinary tract and extremities.
- Large lesions in the head and neck invade bone and jaw lesions are quite common in advanced cases.
- Treatment involves surgery with adjuvant chemotherapy and radiotherapy.
- Prognosis is influenced by site, subtype of rhabdomyosarcoma, and stage at diagnosis.

Hodgkin's disease

- Lymphoid malignancy characterized by presence of Reed–Sternberg cells in the tumour.
- Usually affects teenagers and young adults.
- Presents most commonly as a painless enlargement of the lower cervical or mediastinal lymph nodes accompanied by unexplained fever and weight loss.
- Excellent response (cure rate approaches 90% for low stage disease) to chemotherapy.
- Radiotherapy is often required for more advanced disease.

Retinoblastoma

- Tumour of the retinoblasts in children under 5 years of age.
- Strong hereditary component.
- Diagnosis is usually a white or yellow pupillary reflex (normally red reflex).
- Treatment often requires enucleation of the globe and post-surgical radiotherapy. Occasionally adjunct chemotherapy is also required.

Osteosarcoma

- Rare malignant tumour of bone, mostly in the metaphyseal region of long bones, with the distal femur being the most common site.
- Teenagers are the most common age group affected.

- Frequently metastasizes to the lung and requires wide resection of primary tumour plus multi-agent chemotherapy.

Ewing's sarcoma
- Malignant tumour of bone in teenagers, commonly involving the midshaft of long bones, although any bone may be involved.
- Occurs most commonly in the proximal femur or pelvis and is characterized by densely packed small round cells.
- Treatment involves surgery, chemotherapy and local irradiation.
- The prognosis worsens with pelvic primary or metastatic disease.

Langerhans' cell histiocytosis (see Chapter 8)
- A rare neoplasm similar to ALL, often presenting with eczematous, purpuric rash on the hands, scalp and trunk.
- Osteolytic lesions of the skull and mandible can occur and premature exfoliation of primary teeth has been reported.
- Prognosis depends on the extent of disease at diagnosis and the progression of lesions.

Dental management

Close collaboration between the child's oncologist and the paediatric dentist is essential when planning appropriate dental care. At the time of diagnosis and during the initial stages of chemotherapy, dental care should be provided by the paediatric dentist at the hospital. Once the child has achieved remission, or has successfully completed chemotherapy, routine dental care can often be provided by the child's own dentist.

Where dental treatment is needed prior to or during chemotherapy, planning with the oncology team is essential. If extractions are required, a FBC including differential white cells and platelets is necessary. If the platelet count is $<30\times10^9$/L, then platelet infusion is indicated and antifibrinolytic agents (doses similar to management of haemostatic disorders) may be helpful. As with immunocompromised children, if the neutrophil count is $<1.8\times10^9$/L, specific antimicrobial prophylaxis should be administered. As many children have been receiving systemic corticosteroids, the possibility of adrenocortical suppression should be considered and additional steroid cover provided as appropriate.

Elective dental treatment should be delayed until the child is in remission or on maintenance chemotherapy. Children in full remission can be treated as normal for most routine dental treatment, although an FBC is prudent if an invasive procedure is planned. Pulpal therapy of primary teeth during the induction and consolidation phase of chemotherapy is contraindicated. When pulpal therapy of permanent teeth is needed, the risk of bacteraemia and potential septicaemia must be weighed against the potential benefits.

Oral hygiene and prevention

It is important to maintain meticulous oral hygiene by using a soft toothbrush during chemotherapy. Four times daily 0.2% chlorhexidine gluconate mouthwashes or gel application to the mucosa helps reduce the symptoms of mucositis and topical and systemic antifungal agents (nystatin or fluconazole) help prevent candidiasis during

immunosuppression. Topical lidocaine hydrochloride (Xylocaine Viscous 2%) is helpful during acute episodes of mucositis prior to eating (if possible) or drinking. Prophylactic parenteral antibiotics and antiviral medications, if indicated, are always given during febrile episodes and periods of severe neutropenia to prevent further medical complications.

Immediate oro-dental effects of childhood neoplasia and treatment

Dramatic advances in the treatment of childhood cancer in the past three decades have led to the long-term cure of 70% of the children diagnosed today. Since about 1 in 600 children develop cancer before the age of 15 years, almost 1 young adult in every 1000 will be a long-term survivor of childhood cancer.

As the number of survivors of a variety of paediatric cancers increases, the oro-dental sequelae of effective medical treatment in these patients are emerging. These effects are unique because of the impairment of active growth and development during the cancer therapy. Other late effects include short stature, growth hormone deficiency, cognitive defects, secondary malignancy. Adverse sequelae caused by the cancer treatment can be grouped into postsurgical, post-radiotherapy, post-chemotherapy and combined effects.

Post-surgery

Surgical removal of a solid tumour in the oral cavity can cause:

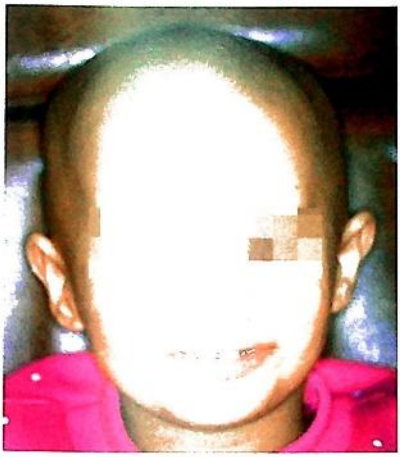
- Disfigurement (temporary or permanent) (see Figure 12.8A).
- Loss of teeth and function.
- Stenosis and paraesthesia.

Post-radiotherapy (Figure 12.8B)

Radiotherapy produces an initial mucosal inflammation that is often followed by surface sloughing and ulceration (mucositis). The extent of inflammation depends on the location and dosage of radiotherapy and whether fractionated versus whole-dose radiation is used. The most common symptoms following cranial irradiation are oral pain and difficulty in eating and drinking, which are most severe 10–14 days following commencement of radiotherapy. The mucositis usually resolves in another 2–3 weeks after radiotherapy.

When radiotherapy involves the major salivary glands, xerostomia frequently occurs within a few days producing a viscous, acidic saliva. Loss or alteration of taste (hypo- or dysgeusia) may also occur prompting the patient to change to a softer, more cariogenic diet to alleviate soreness and dryness of the oral cavity. This is probably the major factor in the aetiology of rapid dental caries that has been reported in these patients if they are not given adequate preventive therapy. Radiation-induced dental caries has a distinctive generalized cervical pattern and sometimes the complete dentition can be destroyed in a relatively short period.

Progressive endarteritis is a complication that can occur in irradiated bone and can lead to osteoradionecrosis. The mandible is particularly prone to this complication and if such an area of dead bone should become infected following dental extraction, a refractory osteomyelitis may ensue. Endarteritis may also cause fibrosis in the masticatory muscles and subsequent trismus.



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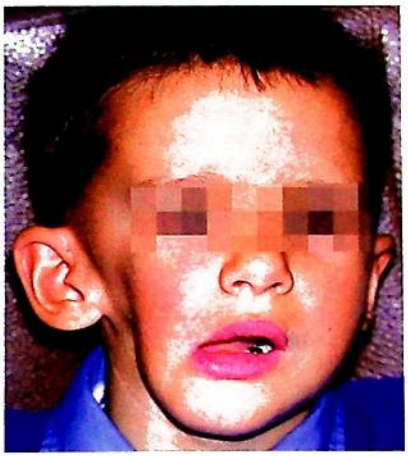

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Figure 12.8 (A) A child in remission from acute lymphoblastic leukaemia with typical alopecia resulting from chemotherapy. (B) Late effects of surgery and radiotherapy for a rhabdomyosarcoma of the right mandible involving the parotid, neck and infratemporal fossa. This child underwent a hemimandibulectomy and radical neck dissection, followed by reconstruction with a free vascular rib graft. Note the limited oral opening and the facial deformity. Access for restorative work on the carious molars was extremely difficult. Caries resulted from reduced salivary flow after removal of the parotid gland.

Chemotherapy

The cytotoxic drugs used during chemotherapy can cause damage to several organs:

- Liver.
- Kidney.
- Intestine.
- Germ cells of the testes and ovaries.
- Lung.
- Heart.
- Brain.

Direct stomatotoxicity is caused by the cytotoxic action of the chemotherapeutic agents on oral mucosal cells leading to inflammation, thinning and ulceration of the mucosa (mucositis). Saliva function may also be diminished although this response has not been reported as common in children. These problems are commonly encountered in the induction and consolidation phases of chemotherapy when relative high doses of multi-agent therapy are employed. Recent case reports suggest that the incidence and severity of stomatotoxicity is reduced with the concomitant administration of granulocyte colony-stimulating factor (G-CSF) during chemotherapy.

The effects of chemotherapy and radiotherapy appear to be synergistic. Since craniofacial and dental development have not been completed until the adolescent period, it is not surprising that dental late effects are commonly found in survivors of

childhood cancer. Chronic problems involving target tissues lead to impairment of growth and development of hard and soft tissues, which may result in orofacial asymmetry, xerostomia, dental caries, trismus and a variety of dental abnormalities. Generally, the nature and degree of these complications vary widely and depend on several factors including the type and location of malignancy, the age of the patient, total dosage and timing of chemotherapeutic agents, and the initial oral health status and the level of dental care before, during and after therapy.

Late oro-dental effects of childhood neoplasia and treatment (Figure 12.9)

The majority of those children for whom oncology treatment results in a stable remission can expect to follow a healthy life. Recurrence of the original malignancy may occur although the likelihood of this becomes increasingly remote as time passes. Consequently, successfully treated paediatric oncology patients are never 'discharged', their health being regularly monitored throughout their life.

With the exception of those children treated with radiotherapy to the orofacial region, the majority of children are no more prone to dental and periodontal disease than the well child, and often exhibit excellent oral health. However, long-term oro-dental effects of radiotherapy can influence dental management.

Growth disturbances

Following head and neck radiotherapy, facial growth can be impaired and alterations to developing teeth can occur. Children younger than 5 years of age are affected more severely than older patients. An altered craniofacial growth pattern with diminished mandibular growth, is often associated with a field of irradiation that includes a portion of ascending ramus and the entire condyle of the mandible. Dental effects may include:

- Incomplete calcification.
- Enamel hypoplasia.
- Arrested or altered root development, and premature closure of the root apices can complicate permanent tooth development.
- Microdontia and agenesis of teeth are also common (Figure 12.9A).

The exact nature and extent of damage depends on the stage of dental development and the timing and dosage of irradiation. The lack of specificity of cytotoxic agents in terms of differentiating neoplastic cells from metabolically active normal cells, such as ameloblasts and odontoblasts, can result in abnormalities of dental development. Microdontia, enlarged pulp chambers, shortening, thinning and blunting of the root apex and delayed tooth eruption have been frequently reported in children receiving chemotherapy. Enamel opacities, hypocalcification and a high rate of dental caries have also been reported in several studies, however, it remains unclear whether these findings are due to direct alteration of enamel formation or maturation or to alterations in the oral environment (saliva and flora), diet and home care often observed among young patients on chemotherapy.

Xerostomia

Cranial irradiation can also irreversibly damage the acini cells of the major salivary glands and xerostomia can occur in children. This condition is often transient due to the lower dosage of radiation used and greater regenerative capacity of the exocrine cells in children. Generally, there is a lower incidence of radiation-induced dental caries

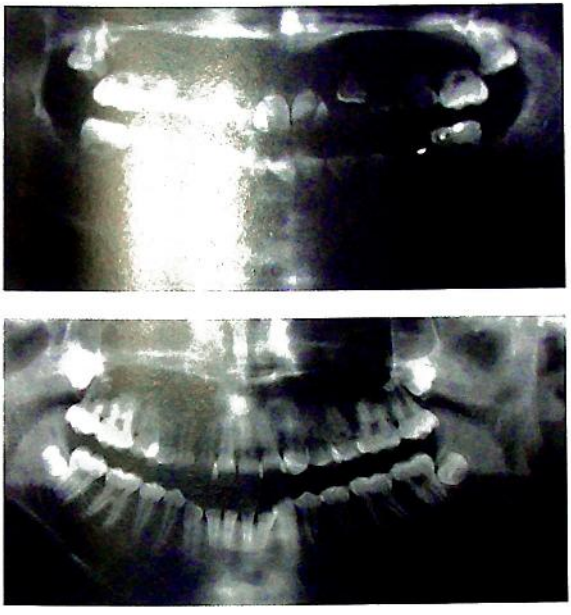


Figure 12.9 Effects of radiation to the head and neck. A set of identical twins, the first of which (A) had acute lymphoblastic leukaemia diagnosed at 18 months. She relapsed during the first remission and received a bone marrow transplant and total body irradiation (TBI). The comparison with her sister (B) at 15 years of age is dramatic. There is agenesis of some permanent teeth, arrested root development of the incisors and first permanent molars, and microdontia.

in children compared with adults. Regular nightly fluoride mouth rinsing is required to prevent enamel demineralization during this critical period.

Other less common effects

Epidermal and mucosal changes include skin hyperpigmentation, cutaneous telangiectasia, subcutaneous tissue atrophy and permanent thinning or loss of hair. Disturbances of intellectual, endocrine and germ cell development have also been reported following cranial irradiation. However, the mean age of dental maturation in children following cranial irradiation is within the normal range.

Since most craniofacial tumours are treated by combined chemotherapy and head and neck irradiation, it is difficult to know the exact effect of each treatment. In general, late oro-dental effects are more severe in patients who receive a higher-dosage treatment either with chemotherapy or radiotherapy. Dental aberrations are more severe and extensive in patients younger than 6 years of age due to the immature development of the permanent teeth. Total body irradiation in bone marrow transplantation appears to increase the risk of disturbance to dental development.

Complications associated with bone marrow transplantation

Almost all children undergoing bone marrow transplantation develop the typical oral mucosal changes of ulceration, keratinization and erythema that develops in 4–14 days post-transplantation. Mucosal atrophy is also frequently associated with ulceration between 1 and 3 weeks after bone marrow transplantation. During this period, oral pain is often severe with many patients requiring narcotic analgesia. The use of keratinocyte growth factor (palifermin) has been demonstrated to reduce this complication in adults undergoing autologous transplantation and paediatric studies of this promising treatment are in progress.

As mentioned previously, oral infection with *Candida albicans*, herpes simplex, cytomegalovirus and varicella zoster are the major infective agents seen in children undergoing bone marrow transplantation, if inadequate prophylaxis is given. These conditions can be life-threatening if not treated aggressively at diagnosis.

Oral manifestations of defective haemostasis are common but seldom serious and include mucosal bleeding or crusting of the lips and gingival oozing.

Graft-versus-host disease (GVHD)

This condition occurs when transplanted T cells recognize the host tissues as foreign. GVHD is a major problem following bone marrow transplantation with clinical manifestations in up to half of patients. The acute form of GVHD tends to occur within weeks of bone marrow transplantation, with signs of fever, rash, diarrhoea and abnormal liver function leading to jaundice. Chronic GVHD may follow some months later and is characterized by lichenoid or scleroderma-like changes of the skin, keratogingivitis, abnormal liver function, pulmonary insufficiency and intestinal problems. Oral manifestations of GVHD vary with the severity of the conditions but often include:

- Mild oral mucosal erythema.
- Painful desquamative gingivitis.
- Angular cheilitis.
- Loss of lingual papillae.
- Lichenoid patches of the buccal mucosa.
- Striae on the buccal mucosa with subsequent restriction of mouth opening.
- Xerostomia.
- Mucosal atrophy, erosion and ulcerations.

Management of chronic GVHD necessitates a multidisciplinary approach. The goal of treatment entails effective care as well as minimizing toxicity and relapse. Long-term systemic immunosuppression with prednisone and other agents is often needed. Recently, extracorporeal photopheresis has been known to produce disease remission. Topical treatments can be effective such as dexamethasone mouthwashes, Biotene moisturizing drops, Difflam-C, and sodium bicarbonate diluted in water helps with disturbances in taste (dysgeusia). Careful attention to oral health with close communication with the treating medical team is needed to give the best outcomes.

Bisphosphonate-related osteonecrosis of the jaw (BRONJ)

Osteonecrosis of the jaw is a well-described complication of bisphosphonate therapy in adults and has not yet manifested as a disorder in children. While there have been

no reported cases of bisphosphonate-related osteonecrosis (BRONJ) in children, there has been a significant increase in the use of these drugs in the management of children with connective tissue disorders and decreased bone density including:

- Congenital osteoporosis – osteogenesis imperfecta.
- Secondary osteoporosis – immobility, steroid induced.
- Focal orthopaedic conditions – avascular necrosis (AVN), Perthes' disease; fracture non-union; Ilizarov limb lengthening; bone cysts.
- Other bone disorders – fibrous dysplasia, idiopathic juvenile osteoporosis.

There is an increased association of BRONJ with any invasive dental procedure such as extractions. While the risks for children are unknown, clinicians should be aware of this potentially destructive condition. There is no concurrence regarding the optimal bisphosphonate drug in children, dosage or duration of treatment. The more serious side-effects linked to bisphosphonates in adults such as uveitis, thrombocytopenia or oesophageal or oral ulcerations are rare in children. Potent bisphosphonates have established therapeutic half-lives of over 30 years, so there may be long term, a low to very low, residual risk of BRONJ in these patients.

Adolescents and young adults requiring bisphosphonate therapy, particularly in the setting of malignancy may also be at increased risk of BRONJ and caution needs to be applied in the undertaking of extractions and oral surgery in this select patient group.

Current management of established BRONJ

Stage 1 conservative:	Chlorhexidine 0.12% t.d.s.
Stage 2 conservative + symptomatic:	Chlorhexidine 0.12% t.d.s.; antibiotics
Stage 3 conservative + surgery:	As above + sequestrectomy/resection

Prevention of BRONJ in children requiring invasive dental procedures

Pre-bisphosphonate therapy: dental evaluation with radiographic screening, within 3 months of starting treatment.

Management for children requiring dental interventions:

- If possible stop bisphosphonate 3 months prior and for 3–6 months after procedure.
- Prophylactic antibiotics 10 days prior to and after procedure.
- Gentle handling of dental tissue and consider primary closure of wounds.
- Chlorhexidine mouthwash 10 days prior to and after procedure.
- Regular monitoring until complete healing.

Dental follow-up for children receiving bisphosphonates: 6-monthly dental reviews with 1–2-yearly radiographic survey as required.

Nephrology

Renal disorders

Renal diseases are classified as acute, chronic, acquired or congenital conditions.

Acute renal failure
- Results in a sudden onset of impaired renal function and perfusion.
- Can occur subsequent to septicaemia, dehydration, severe burns and blood loss, glomerulonephritis, pyelonephritis, tumour lysis and ureteric obstruction.

Chronic renal disease
The most common chronic conditions affecting the kidneys are:
- Ureteric reflux causing reflux nephropathy or hypoplasia.
- Obstructive uropathy.
- Glomerulosclerosis.
- Medullary cystic disease.
- Systemic lupus erythematosus.
- Cystinosis.

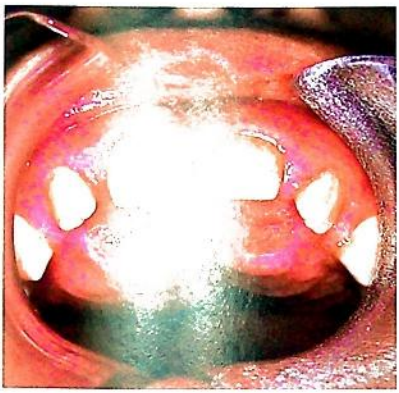
End-stage renal failure (Figure 12.10)
- Leads to a progressive drop in glomerular filtration rate that results in hypertension, fluid retention and build-up of metabolites that are not excreted normally.
- Medical management is directed toward prevention of fluid and electrolyte imbalance, restriction of proteinuria, correction of hypoalbuminaemia, hypocalcaemia, hyperphosphataemia and control of anaemia and hypertension.
- In children with severe renal failure, drug treatment is often inadequate and artificial filtration by either peritoneal dialysis or haemodialysis becomes necessary.

Acquired conditions
- Urinary tract infections, usually from coliform bacteria from the intestinal tract and cystitis (bladder infection).
- Acute glomerulonephritis usually accompanies β-haemolytic streptococcal infections and often resolves with antibiotic therapy in most children. However, 3–4% may develop post-infection chronic renal failure and subsequently need dialysis.

Medical complications can be overcome with successful renal transplantation, which is now the preferred treatment of choice for children with end-stage renal failure. Despite the restricted availability of donor organs, renal transplantation has a high success rate. Complications associated with immunosuppression due to cyclosporin (Sandimmune) and prednisone therapy to prevent organ rejection need to be considered in dental management. The most common oral manifestations following renal transplant are gingival hyperplasia (Figure 12.10B) and opportunistic infection from commensal oral flora.

Dental implications

Impaired renal function can result in several oral manifestations including:
- Uraemic stomatitis: as a result of high levels of urea due to breakdown of ammonia.
- Oral ulceration.
- Dysgeusia.
- Pallor or petechiae and ecchymosis of oral mucosa.
- Xerostomia.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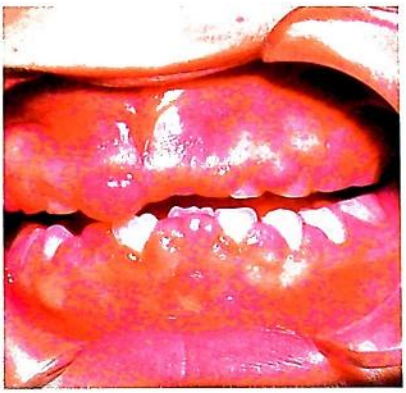
B

Figure 12.10 (A) Severe renal osteodystrophy in a child in end-stage renal failure. There has been gross expansion of the maxilla in an attempt to produce red blood cells because of failure of erythropoiesis. This is similar to events in β-thalassaemia. (B) Gingival overgrowth due to cyclosporin A treatment after kidney transplantation. The teeth are also hypoplastic and small because of renal disease in infancy.

- Intrinsic and extrinsic tooth staining.
- Excessive supragingival calculus due to increased salivary urea and phosphate levels.
- Enamel hypoplasia and hypocalcification.
- Delayed dental development.
- Dental pulp mineralization.

Uraemic stomatitis may develop when the serum urea level is >300 mg/mL. It occurs as ulcerated or non-ulcerative forms involving the tongue and buccal mucosa predominantly. Both forms have a tendency to bleed and are susceptible to secondary infection by oral commensal organisms.

Renal osteodystrophy (Figure 12.9A)

Lytic lesions of the mandible or maxilla, known as Brown's tumours, can also occur in severe renal failure due to secondary hyperparathyroidism. Histologically, these lesions are similar to giant cell tumours and usually resolve following correction of hypocalcaemia and hyperphosphataemia with vitamin D metabolites. Hypocalcaemia occurs due to increased phosphate retention and decreased calcium absorption. Active calcium absorption from the gut depends on the presence of the active metabolite, 25-hydroxy-cholecalciferol (vitamin D_3). However, vitamin D metabolism is impaired due to failure of the hydroxylation of 25-hydroxy-cholecalciferol to 1,25-dihydroxy-cholecalciferol in the diseased kidney. In an attempt to raise serum calcium there is a secondary hyperparathyroidism and calcium is removed from bone stores giving rise

to the characteristic radiographic appearance of renal osteodystrophy. Other dental manifestations include demineralization, decreased trabeculation, loss of lamina dura, macrognathia, tooth mobility, malocclusion, enamel hypoplasia and pulp stones.

Dental management

The observed dental changes depend on the time of onset of renal disease. Those teeth calcifying during renal failure will exhibit chronological hypoplasia or hypomineralization of the enamel and dentine. Developing teeth are often stained green or brown due to the incorporation of blood products such as unconjugated bilirubin or haemosiderin, respectively. Caries is often minimal in these children, possibly due to urea metabolites in the saliva, but supragingival calculus formation is markedly increased, even when oral hygiene is adequate.

- Consultation with a renal physician or nephrologist is often required before these children can receive routine dental treatment.
- Children with acute renal conditions should have elective dental treatment postponed until restoration of their renal function.
- Emergency or palliative care is only indicated following pre-treatment screening for elevated bleeding time or APTT.
- Extraction of pulpally involved primary teeth is the preferred treatment option due to the risk of chronic bacteraemia following pulpotomy or pulpectomy.
- Symptomatic patients with proteinuria or on long-term steroid therapy are best managed in the hospital environment where blood pressure and fluid balance can be monitored before treatment. Fluids and electrolytes in such children can be adjusted by the nephrologist and steroid supplementation can be given prior to general anaesthesia or a major dental procedure.

Dialysis

Children receiving dialysis often exhibit somatic growth retardation, and are pale and anaemic on presentation. They also have a bleeding tendency due to increased capillary fragility and thrombocytopenia. In addition, children on haemodialysis receive anticoagulation with intravenous heparin, and can experience other complications such as infection of the port site and increased risk of hepatitis. In children receiving peritoneal dialysis, complications can occur with catheter placement including peritonitis and exit-site infections. However, peritoneal dialysis is easier to manage in children, requiring less time for the fluid exchange, less restriction of food and fluid intake, and fewer haemodynamic problems compared with haemodialysis.

- Children on haemodialysis and anticoagulant therapy can be successfully managed with pretreatment DDAVP and antibiotic prophylaxis to prevent infection of the access device.
- Any dental treatment, especially extractions, should be performed the day after dialysis when the heparin is no longer active (heparin half-life is 4 h but residual effects can occur for 24 h).
- Sockets should be packed with a haemostatic agent and sutured well. Platelet transfusions are to be avoided if possible.
- Children on continuous ambulatory peritoneal dialysis can be managed more conservatively.

Drug interactions

Drug interactions can occur in children with end-stage renal failure who are managed with long-term antihypertensives and steroids. Medications that are metabolized in the kidney or nephrotoxic should be avoided in children with renal insufficiency. These include:

- Cefalotin (cephalothin).
- Paracetamol (acetaminophen).
- Non-steroidal anti-inflammatory agents.
- Tetracycline.

Dentists should also be aware that renal excretion of drugs is also impaired and their half-life may be extended. However, if children are adequately haemodialysed, it may be necessary to increase dosage of drugs to obtain the necessary pharmacological effect. Adjustment of a drug dosage as well as timing of intake should be made in consultation with the child's nephrologist.

Renal transplantation

A pre-transplant dental assessment is essential to reduce the risk of oral complications following organ transplant and immunosuppression. The presence of dental caries and oral infections will necessitate delay in transplantation until all potential foci of infection are eliminated. Comprehensive dental treatment and institution of a rigorous preventive programme are recommended prior to transplant to reduce the risk of subsequent oral diseases. Antibiotic prophylaxis as per the protocol for prevention of infective endocarditis is essential prior to invasive surgical procedures.

Gastroenterology

Hepatic and biliary disorders

Biliary atresia

In this condition, there is congenital obliteration or hypoplasia of the bile ducts, resulting in biliary cirrhosis and portal hypertension. In severe cases, transplantation is necessary (Figure 12.11).

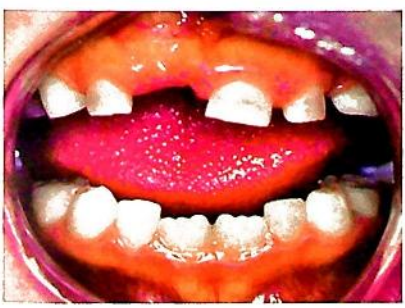

Figure 12.11 Yellow colouration of the gingival tissues in a child with jaundice. This photograph was taken just prior to the child undergoing a liver transplantation due to biliary atresia.

α_1-Antitrypsin deficiency

- Deficiency in hepatic secretion of α_1-globulin.
- Leads to progressive hepatomegaly and cirrhosis.
- Treated by liver transplantation.

Liver function tests

- Full blood count and coagulation profile should be routinely tested.
- Alanine aminotransferase (ALT): 7–47 U/L.
- Alkaline phosphatase: 60–391 U/L.
- γ-Glutamyl transpeptidase: 5–43 U/L.
- Total bilirubin.
- Albumin (a decrease may reflect impaired protein synthesis).

Dental implications

- Coagulation problems are of major concern due to the reduction in production of vitamin-K-dependent clotting factors (II, VII, IX, X).
- Patients are immunocompromised. Children are usually mildly anaemic due to destruction of red blood cells. High levels of circulating red-blood-cell degradation products may become incorporated in developing enamel. High levels of unconjugated bilirubin will cause green developmental staining of enamel.

Dental management

- Consultation with paediatric gastroenterologist and haematologist.
- Aggressive management of caries with extraction of suspect teeth, especially prior to transplantation.
- Coagulopathies are usually managed with fresh frozen plasma to replace deficient clotting factors.
- Antibiotic prophylaxis required.

Hepatitis A (infectious hepatitis)

Transmission period is very short (3 weeks). The important point to note is that there is no carrier state. If a patient is hepatitis-A positive treatment should be delayed for at least 4 weeks.

Hepatitis B (serum hepatitis)

Interpretation of hepatitis B test results

- If the HBsAg test is positive the blood is automatically tested for the 'e' antigen. If 'e' is negative, the patient is a chronic healthy carrier.
- If the 'e' antigen is positive the patient is a chronic active carrier.

Chronic healthy carrier (HBsAg +ve, e –ve)

- The degree of infectivity of these patients, although significant, is thought to be less than that of chronic active carriers. The liver function test for this patient should be normal.

Chronic active carrier (HBsAg +ve, e +ve)

- The chronic active carrier has active viral replication and is very infective. These patients have active liver disease, and their liver function may be abnormal.
- It is important to liaise directly with the patient's doctor in the first instance when planning dental treatment and to do so again at regular intervals while the patient is under the care of the paediatric dental unit.

Liver function tests

These are useful in chronic active carriers. Liver function tests should be considered in chronic healthy carriers (HBsAg +ve, e –ve), if recently diagnosed or just recently become negative for HBsAg, and in patients who have just recovered from hepatitis A.

Hepatitis C (non-A, non-B hepatitis)

The hepatitis C virus (HCV) is the cause of what was previously termed non-A, non-B hepatitis. Patients with antibodies to HCV are chronic carriers and are potentially infectious. They are usually asymptomatic although their liver enzymes may be intermittently abnormal. Transmission of HCV is primarily by blood or blood products; however, the virus can be detected, by polymerase chain reaction (PCR), in the saliva of chronic carriers.

Liver transplantation

Children with end-stage liver disease and doubtful prognosis are candidates for liver transplantation. Unfortunately, there is an acute shortage of organs for transplantation and many children succumb to their illness before a donor organ is found. Complete oral and radiographic evaluation is essential prior to transplantation. Any potential foci of oral infection, including exfoliating primary teeth should be eliminated before transplantation can proceed. Teeth with large carious lesions, even if not pulpally involved, should be extracted.

Blood transfusion with packed red cells or partial exchange transfusion with coagulation factors is often required. Prophylactic antibiotic therapy before surgical procedures should be instituted. The main management problem with children undergoing liver transplantation is that of immunodeficiency.

Immunosuppressive therapy includes various corticosteroids and cyclosporin. Cyclosporin is both nephrotoxic and hepatotoxic and can cause hypertension. However, most children are more easily treated post-transplantation due to the cure of their original disease. This is especially true of children with end-stage hepatic failure.

Clinical hint

Gingival overgrowth and candidiasis can result from cyclosporin and nifedipine therapy. Meticulous oral hygiene should be instituted and gingivectomy considered to assist development of the permanent teeth following transplantation.

Oesophageal disorders

Neuromuscular control of the lower oesophageal sphincter is inadequate in some children resulting in chronic gastro-oesophageal reflux disease (GORD). The exact pathophysiology of GORD is unknown, although gastric motor abnormalities characterized by delayed gastric emptying have been observed in some children. Regurgitation is not always clinically obvious or symptomatic and 24-hour oesophageal pH monitoring may be necessary to demonstrate significant GORD. Symptomatic children are given oral metoclopramide hydrochloride (Maxolon) to prevent reflux, whereas in children with concurrent oesophagitis, a histamine H_2 antagonist such as cimetidine is helpful. Surgical treatment is required if medical therapy fails.

Increasingly, paediatric dentists may be the first professionals to observe the chronic effects of undiagnosed GORD on the primary dentition, namely severe dental erosion of the canine and molar teeth. If a diagnosis of GORD is suspected, prompt referral to a gastroenterologist is recommended for endoscopy and a pH monitor. Fluoride mouth rinses and varnishes help in remineralization. Comprehensive dental treatment with stainless steel crowns is usually required to restore the lost vertical dimension.

Inflammatory bowel disease (see Chapter 8)

Commonly, 'chronic inflammatory bowel disease' entails ulcerative colitis and Crohn disease that may represent ends of a spectrum of tissue reaction to a common agent. Establishing an aetiological agent is difficult. Both conditions present as a chronic inflammatory process of the gastrointestinal tract with acute exacerbations. Of particular interest to the dentist, is the association with orofacial granulomatosis that sometimes precedes the onset of ulcerative colitis by 1–2 years.

Oral changes reported in some children include linear ulceration or fissuring of the buccal and labial mucosa, diffuse swelling of the lips, angular cheilitis and diffusely swollen erythematous gingivae, chronic granulomatous lesions. These lesions may improve with sulfasalazine (Salazopyrin) therapy but usually reappear on the buccal mucosa periodically.

Endocrinology

Diabetes mellitus

Type 1 or insulin-dependent diabetes mellitus (TIDM) is the most common form of diabetes in children. Approximately 1 in 1200 children between the ages of 5 and 18 years have the disease. Type 1 diabetes results from autoimmune destruction of the insulin-producing β cells in the pancreas, which is thought to be triggered by viral or toxic insults to the pancreas in the child genetically predisposed to developing the disorder. The goal of treatment is to maintain blood glucose levels in a range that prevents the development of short-term and long-term complications. The target blood glucose ranges are 5–12 mmol/L in children less than 5 years of age, 4–10 mmol/L for middle childhood and 4–8 mmol/L thereafter.

Management protocols

While there are individual variations, contemporary management generally involves administration of a long-acting insulin once or twice per day supplemented with short-acting insulin at meals. For those children with insulin pumps, a subcutaneous

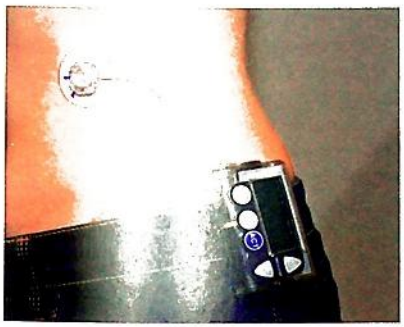

Figure 12.12 An insulin pump showing the position of the subcutaneous canula, usually sited in the abdomen. The pump can be programmed to deliver a basal level of insulin and bolus doses as required with meals mimicking the function of the pancreas.

continuous infusion of short acting insulin maintains a (variable, but programmed) basal level throughout the day. Additional bolus doses are given with meals (Figure 12.12).

Pancreatic islet cell transplantation and stem cell research raises the possibility for cure for juvenile diabetes and is currently under critical investigation.

Relatives of patients with diabetes are 2.5 times more likely to develop the disease than the population at large. Presenting symptoms include:

- Polydipsia.
- Polyuria.
- Weight loss.
- Secondary enuresis.
- Lethargy.
- Recurrent infections and candidiasis.
- Diabetic ketoacidosis.

A diagnosis of diabetes is made when fasting blood glucose level above 7.7 mmol/L is recorded or a random blood glucose level >11.0 mmol/L is recorded. Occasionally, children will have diabetes diagnosed following an abnormal oral glucose tolerance test.

Dental implications

- Periodontal disease is the most consistent oral finding in children with diabetes and is more common if control of blood glucose levels is suboptimal. These children exhibit increased alveolar bone resorption and inflammatory gingival changes. This may mimic the clinical manifestation of chronic generalized juvenile periodontitis.
- Xerostomia and recurrent intraoral abscesses may also be present in severe cases.
- Enamel hypocalcification and hypoplasia along with reduced salivary flow can predispose these patients to an increased risk of early childhood caries.
- Altered oral flora changes can occur with an increase in *Candida albicans*, haemolytic streptococci and staphylococci.

Dental management

- Children with well-controlled diabetes can receive dental treatment in the usual way, except when a general anaesthetic is required. The best measure of control is the level of glycosylated haemoglobin (HbA_{1c}). The target for most children is a value of <6.5–7.0% (≤53 mmol/mol). For routine dental appointments, the child should eat a normal meal prior to the dental procedure although a glucose source should always be available to treat hypoglycaemia.
- Fasting before a general anaesthetic requires careful blood glucose monitoring and adjustment of insulin doses during the fasting period. This is to prevent extremes of hyper- and hypoglycaemia. Insulin doses and the possibility of intravenous fluid therapy should be discussed with the child's treating paediatrician/endocrinologist.
- Post-surgical healing can be delayed, particularly if blood glucose control is sub-optimal and oral sepsis can be an additional risk.
- It is often possible to manage many T1DM children requiring general anaesthesia in day-stay facilities, provided that children can begin taking fluids shortly after their procedure. Other children may require admission to a paediatric hospital, with a dextrose and insulin infusion set-up to maintain BGL levels and avoid complications during the fasting and postoperative periods. It is obviously essential to liaise with the treating endocrinologist.
- Prophylactic antibiotic therapy is recommended prior to surgical procedures.

Pituitary disorders

Hypopituitarism

Hypopituitarism (complete or partial) may be either congenital or secondary to pituitary or hypothalamic disease (tumours, infections, trauma or after exposure to ionizing radiation). These children need to be managed in conjunction with a paediatric endocrinologist to prevent potentially serious complications of pituitary hormone deficiency.

Isolated growth hormone (GH) deficiency is the most common pituitary hormone deficiency, but evolution of other deficiencies may take place over time. Treatment consists of supplemental GH that stimulates the differentiation of epiphyseal growth plate precursor cells and induces clonal expansion of cartilage cells. Timely detection and treatment of GH deficiency is essential to minimize the growth disturbance.

Dental implications

- It is important to ensure that any child with ACTH deficiency having general anaesthesia, be given stress doses of hydrocortisone (100 mg/m^2) as an IV injection at the onset of anaesthesia. Until the child has recovered from the surgery, stress doses of hydrocortisone will be required. The exact dose and route of administration will vary depending on the situation. An endocrinologist should always be consulted prior to any procedure requiring a GA in these children to guide the hydrocortisone therapy.
- Hypopituitarism can decrease linear facial measurements (particularly in the posterior facia height) and linear cranial base measurements.
- Children often present with an open bite accompanied by the typical immature hypopituitary facies.

- Somatic skeletal development is consistently more retarded than craniofacial development, although tooth eruption and root formation can be delayed or incomplete.
- Early orthodontic assessment is required to monitor orofacial growth and development.

Pituitary hormone excess

Gigantism results from primary hypersecretion of growth hormone during childhood. Gigantism is extremely rare in children and is associated with pituitary tumours and McCune–Albright syndrome.

Dental implications

- Precocious and accelerated development of the craniofacial skeleton.
- Prognathism.
- Accelerated dental development and eruption.
- Enlarged crenated tongue, and coarse facial features.
- Radiographically, there is a marked thickening of the cranium and cortical bone of the mandible.
- Osseous structures exhibit overdevelopment with poor maturation and bone quality (osteoporosis) and hypercementosis of tooth roots is common.

Dental management

Dental management of patients with pituitary disorders focuses mainly on the management of the associated craniofacial malformations. Treatment needs to be planned carefully and coordinated in a multidisciplinary setting. No contraindications exist for comprehensive dental healthcare but the treating endocrinologist must be consulted prior to any invasive treatment or general anaesthesia.

Thyroid disorders

Hypothyroidism

Thyroid hormone deficiency (hypothyroidism) is most often due to a primary disorder of the thyroid gland and less commonly secondary to hypothalamic and/or pituitary insufficiency. Primary hypothyroidism may be either congenital or acquired (thyroiditis, after radiation exposure or surgical excision). Untreated congenital hypothyroidism leading to severe developmental delay (Cretinism) is rare in those countries where neonatal testing is performed (TSH with or without fT4) and treatment with synthetic thyroxine started in the first 1–2 weeks of life. Congenital hypothyroidism can be attributed to aplasia, hypoplasia or maldescent of the thyroid gland, abnormal thyroid hormone production (dyshormonogenesis) or prenatal iodide deficiency. Juvenile hypothyroidism can stem from autoimmune thyroiditis, thyroidectomy, thyroid irradiation, infection or medication.

Although hypothyroid changes can include: growth failure, diminished physical activity, decreased circulation, poor muscle tone, speech disorders, delayed mental development and craniofacial manifestations, these changes are obviously dependent on the age of onset of the disease, the degree of hypothyroidism and the timing of diagnosis and treatment. Most hypothyroid children have very few clinical features, despite a quite severe deficiency state.

Dental implications of untreated or suboptimally treated hypothyroidism

- Decreased vertical facial growth.
- Decreased cranial base length and flexure, maxillary protrusion, and open bite with typical immature facial patterns.
- Delayed eruption of teeth and increased spacing between both primary and permanent teeth.
- Developmental anomalies such as enamel hypoplasia have been reported.
- Mouth breathing and dysgeusia (altered taste).

Hyperthyroidism

Hyperthyroidism can occur due to autoimmune hyperstimulation of the gland (Grave's disease or Hashimoto's thyroiditis), inflammatory disorders (subacute thyroiditis), iodine excess or rarely neoplasm. In children, the most common causes are Graves' disease, and thyroiditis. Thyrotoxicosis is more common in females and is most likely to appear between 8 and 16 years of age and is usually associated with a goiter.

Changes associated with hypothyroidism are largely related to hyperactivity of the sympathetic nervous system. Typical clinical manifestations include nervousness, poor school concentration with deteriorating performance, muscle weakness, heat intolerance, loss of weight despite increased appetite, insomnia, tachycardia, palpitations, marked perspiration and gastrointestinal disturbances. In addition, ocular abnormalities such as eyelid lag, exophthalmos and widening of the palpebral fissures may be seen in some cases.

Dental implications

- Accelerated growth and development of the craniofacial complex and skeleton.
- Precocious eruption of teeth.
- Increased susceptibility to caries and periodontal disease, burning mouth syndrome.
- Osteopenia or poor bone mass accrual.
- Typical orofacial changes are increased vertical facial height with anterior open bite and mild mandibular prognathism.

Dental management

The principal concern in children with thyroid disorders is the increased risk associated with general anaesthesia. The hypothyroid patient is at risk of extreme delay to wake normally after general anaesthetic.

If a child with inadequately controlled hyperthyroidism is anaesthetized, there is a risk of precipitating thyroid storm, a very dangerous condition of tachycardia, acute onset of fever, with possible cardiac failure and death. The untreated patient is also at risk from oral infection or surgical procedures as thyroid crisis may be precipitated.

Rarely, intercurrent infections may precipitate significant worsening of hyperthyroidism. As such, oral infections should be treated aggressively and thyroid function (TSH and fT4) monitored. Rare side-effects of antithyroid drugs include parotitis and agranulocytosis, which predispose the patient to bleeding episodes, ulceronecrotic lesions and chronic oral infections.

In general, a child with well-controlled thyroid disease can undergo dental management similar to any other child. Where possible, thyroid function should be normalized

prior to dental intervention. In the unusual situation where this is not possible, close collaboration with a paediatric endocrinologist is recommended.

Parathyroid disorders

Hypoparathyroidism

Hypoparathyroidism results from structural or functional deficiencies in the parathyroid glands. In children, it most commonly presents as hypocalcaemia in the neonatal period in association with complex disorders, e.g. Di George syndrome and CATCH22 disorders. During childhood it may result from autoimmune destruction of the parathyroid gland, after parathyroidectomy for gland over-activity in association with multiple endocrine neoplasia or after inadvertent parathyroidectomy at the time of thyroidectomy.

Treatment focuses on maintaining near normal serum calcium by calcium and calcitriol supplementation. Trials are underway to supplement with recombinant PTH. If untreated, hypocalcaemia can lead to neuromuscular excitability, tetany, muscle weakness, lethargy and fatigue. Associated syndromes have a variety of clinical features including cardiovascular dysfunction, epilepsy, ectodermal defects, immune dysfunction and craniofacial manifestations.

Dental implications of untreated hypoparathyroidism

- Circumoral paraesthesia and spasm of the facial muscles has been reported in severe hypocalcaemia.
- Hypoplasias of enamel, wide pulp chambers, pulp calcifications.
- Hypodontia and root anomalies are common clinical findings.
- Tooth eruption can be markedly delayed or arrested.
- Increased risk of acute and chronic oral candidiasis has also been reported due to associated immune dysfunction.

Pseudohypoparathyroidism (PHP)

This is a heterogeneous disorder that has at its core, resistance to PTH action. Inheritance of PHP is classically an X-linked dominant disease in which there is resistance to the effect of PTH with resultant increase in PTH but low serum calcium. It is characterized by low serum calcium, high phosphate levels and high PTH levels. If inherited from the mother, the child will have typical physical features of short stature, round face and short hands (Albright's hereditary osteodystrophy), but if inherited from the father, biochemical changes will only be present.

Dental implications

- Round full facies with short neck.
- Delayed or incomplete eruption of teeth.
- Enamel hypoplasia.
- Short tooth roots.

Hyperparathyroidism

Excessive production of parathyroid hormone may result from a primary defect in the gland (adenoma, hyperplasia, hypertrophy) or secondarily as a compensatory phenomenon, usually correcting hypocalcaemic states due to rickets or from chronic renal disease. Tertiary hyperparathyroidism (autonomous hyperparathyroidism) can occur following prolonged and uncontrolled secondary hyperparathyroidism. Primary and

tertiary disease results in hypercalcaemia and hypercalcuria, muscle weakness, abdominal pain and constipation, polyuria, polydipsia, kidney stones and bone loss. In infants and young children, there may be failure to thrive, poor feeding and muscular hypotonia.

The bony lesions are rarely seen in children but include brown tumours, so-called because they contain areas of haemorrhage, an abundance of multinucleated giant cells, fibroblasts and haemosiderin. Hypophosphataemia can lead to rickets in children and osteomalacia in adults. Generalized osteoporosis with cortical resorption is the most common bone lesion in adults and radiographic signs can also include multiple rarefactions, loss of typical trabeculation, ground-glass appearance and metastatic calcifications.

Dental implications

- Increasing tooth mobility.
- Severe malocclusion and drifting of teeth with no apparent pathological periodontal pocketing have been reported.
- Radiographic changes with metastatic soft-tissue calcifications, periapical radiolucencies, root resorption, loss of lamina dura, and generalized loss of radiodensity.
- Dental abnormalities associated with underlying disorder if secondary or tertiary hyperparathyroidism.

Dental management

Generally, routine dental treatment involves no treatment modifications provided there are no associated medical complications present. Pitting enamel hypoplasia and failure of tooth eruption may occur. Addison's disease (adrenal insufficiency), on the basis of autoimmunity, may accompany hypoparathyroidism and thus the child may be at risk from stressful procedures such as oral surgery and general anaesthesia.

Hypo- and hypercalcaemia may be associated with cardiac arrhythmias that increase the risk of cardiac arrest during general anaesthesia. Risk of pathological fracture in advanced cases of hyperparathyroidism should be a consideration during oral surgical procedures. Splinting of mobile teeth is a useful adjunct to prevent further drifting following stabilization of the dentition.

Adrenal gland disorders

Adrenal glands have two endocrine functions, located within the cortical and medullary areas. The adrenal cortex produces three major classes of steroid hormones: glucocorticoids, mineralocorticoids, and sex hormones. Glucocorticoids (cortisol) have an important role in carbohydrate, fat and protein metabolism, assist in the maintenance of normal blood pressure and protect the body against stresses of various types. Mineralocorticoids (aldosterone) help maintain salt and water balance through their action on the kidney. Adrenal sex hormones help complement the actions of the gonadal steroids in the development of sexual characteristics and reproductive capability.

Adrenal insufficiency

The major problems associated with adrenal hypofunction are the result of glucocorticoid and mineralocorticoid deficiency. Primary adrenal insufficiency (Addison's disease) is a chronic condition characterized by:

- Anorexia.
- Weight loss, vomiting.
- Salt craving.
- Nausea.
- Weakness.
- Skin hyperpigmentation.

There are low blood cortisol levels, and an acute adrenal crisis (hypotension and collapse, electrolyte disturbance and hypoglycaemia) can be precipitated by a relatively minor stress. This is a medical emergency, and management is with immediate steroid supplementation. Secondary adrenal insufficiency is caused either by prolonged administration of steroids resulting in the suppression of endogenous cortisol or by a central lack of adrenocortical stimulating hormone (ACTH). Children do not usually present with obvious clinical signs unless stressed and an adrenal crisis can occur without warning.

Congenital adrenal hyperplasia

Congenital adrenal hyperplasia (CAH) is the most common congenital cause of adrenal insufficiency. CAH arises when there is an enzymatic block in the complex biosynthetic pathways from cholesterol to cortisol and mineralocorticoid. Females usually present in the neonatal period with ambiguous genitalia. Untreated, CAH is fatal with babies becoming desperately unwell – vomiting, electrolyte disturbance, hypoglycaemia and shock. Management includes immediate and long-term replacement of cortisol, together with restoring electrolyte balance with mineralocorticoid replacement therapy.

Adrenocortical hyperfunction

Cushing syndrome refers to a clinical condition characterized by manifestations of adrenocortical hyperfunction, with physical features of growth failure together with weight gain, 'moon facies', truncal obesity, formation of striae, hirsutism and poor wound healing. It is caused by a tumour in the pituitary gland secreting ACTH (Cushing's disease) or in the adrenal, in which case treatment requires removal of the lesion surgically. It may also be iatrogenic or secondary to treatment of inflammatory or immune diseases with high-dose glucocorticoids.

Dental management of adrenal insufficiency

It is important to confirm the diagnosis and medical management with the endocrinologist before commencement of any dental treatment. Any child with adrenal insufficiency must have a plan for stress glucocorticoid therapy prior to undertaking dental treatment. Failure to do so may result in serious medial complications for the child. An assessment of the potential for adrenal suppression can be considered from the duration and dosage of previous corticosteroid treatment but if a child has used steroids for more than 3–4 weeks, there is a risk for adrenal suppression and advice should be sought prior to treatment as to the necessity of giving steroid cover during any procedure. Supplemental steroids should be given following medical consultation and proportional to the degree of adrenal suppression and the perceived stress of the dental procedure. It is beyond the scope of this chapter to outline the various treatment strategies that best suit an individual patient. As such, individual specialist paediatric endocrine consultation is required.

Neurology

Febrile convulsion

This is a term used to describe a child under 5 years of age who has a seizure in response to a febrile illness. It usually occurs with body temperatures over 38°C and when no other cause can be determined. Recurrent, non-febrile seizures are termed as epilepsy.

Epilepsy

Epilepsy is the most common disorder in paediatric neurology and the predominant aetiologies are birth injury and congenital abnormalities.

Convulsions can be classified as:

- Generalized, either tonic–clonic (grand mal) or absence (petit mal).
- Focal (partial), either simple or complex.

Lennox–Gastaut syndrome is an intractable form of severe epilepsy with clinical features of head-dropping attacks, atypical petit mal and brief tonic–clonic seizure usually at night.

Modern treatment is usually restricted to the use of one anticonvulsant medication and slowly increasing the dose to achieve therapeutic blood levels and minimizing side-effects. About 70% of children do very well, with only minimal problems, even if treatment is long term. However, drowsiness, ataxia, excessive salivation, hyperactivity and aggressive behaviour are common complications of anticonvulsant therapy. Gradual reduction of multiple medications has a beneficial effect in terms of increased alertness, and a reduced number of seizures. Phenytoin sodium (Dilantin) is still probably the most effective drug for grand mal seizures but the cosmetic side-effects such as hirsutism and gingival hypertrophy have reduced its use in favour of carbamazepine (Tegretol).

Dental implications

The major oro-dental concerns with epileptic children are gingival enlargement (Figure 12.11) and the precipitation of a seizure in the dental surgery.

Dental management

Management of gingival hypertrophy (Figure 12.9B) is dependent on oral hygiene and dental development at diagnosis. In the permanent dentition, full mouth gingivectomy may be required, but gingival overgrowth will recur if oral hygiene is not optimal. Maintenance of adequate oral hygiene may be especially difficult in children with additional intellectual disability and is highly dependent on the motivation and skill of the parents and caregivers. Battery-operated plaque removers with small circular heads are beneficial in the maintenance of good oral hygiene in the more intractable cases. The use of daily chlorhexidine-containing gels is effective in reducing the inflammatory component of the gingival overgrowth. It is important to always keep the interests of the child in mind, particularly with regard to aggressive surgical treatment that may not benefit the child in the long term.

The general goal of dental management is the avoidance of a seizure. It is important to know the type of epilepsy and any precipitating factors, medications and dosage, compliance and degree of seizure control before commencing treatment. In addition,

drug interactions with anticonvulsants are common and their half-life and blood levels can be increased substantially. Consultation with the child's neurologist is essential before commencement of treatment.

The following management protocol is recommended for prevention and control of seizures in the dental surgery:

- Reduce stress to the child by behavioural management and conscious sedation techniques.
- Reduce direct overhead lighting, particularly for the photosensitive form of epilepsy.
- Avoid seizure-promoting medications, such as CNS stimulants and local anaesthetics containing adrenaline (epinephrine).
- Emergency drugs such as oxygen, intravenous or rectal diazepam (Valium) and intravenous phenobarbital sodium should be readily available.
- Pre-arranged transfer to a paediatric hospital, in case required.

General anaesthesia is preferable in children with poor seizure control as the abnormal neural activity is completely ablated during the procedure. Dental trauma is an obvious consequence in the child with frequent, poorly controlled seizures. Removable appliances are contraindicated in an epileptic child due to potential airway obstruction.

Respiratory disease

Asthma

Australia has one of the highest rates of childhood asthma in the world, with 1 in 5 children and 1 in 7 adolescents affected. It is a respiratory condition characterized by increased responsiveness of the airways to a wide variety of stimuli, leading to widespread narrowing of the airways resulting in symptoms of dyspnoea, wheezing and coughing. Precipitating factors include emotional stress, exercise, cold air, viral respiratory infections, air pollution and aspirin. The condition is reversible, either spontaneously or as a result of bronchodilator therapy. Currently, there is an emphasis on prophylactic medications to prevent episodes rather than simply treating acute attacks.

Bronchodilators include β_2-adrenergic drugs such as salbutamol sulphate (Ventolin) and theophylline (Nuelin). Preventive agents include disodium cromoglycate (Intal) and oral corticosteroids such as prednisone and inhaled corticosteroids such as beclomethasone dipropionate (Beclovent) and salmeterol xinafoate (Seretide).

Dental implications

- The major concern to the paediatric dentist is the exacerbation of an acute attack in the dental surgery.
- Avoid any known precipitating factors prior to dental treatment and ensure that the child has the appropriate medication (inhaler) for emergency use if an asthmatic attack occurs during dental treatment.
- Some bronchodilator and corticosteroid medications may cause extrinsic staining of the teeth due to changes in oral flora and may also predispose to oral candidiasis.
- Children on high-dose corticosteroids (>1600 μg/day) may be immunocompromised and may also require additional supplemental corticosteroids on the day of dental treatment due to adrenal suppression.

- Children are often mouth breathers causing gingivitis and swelling of anterior gingival tissues.
- Disturbances in taste, xerostomia and gastro-oesophageal reflux due to β-2-agonist that may also contribute to tooth surface loss, are other concerns.

Dental management

- Regular dental prophylaxis may be necessary if extrinsic staining is present.
- The use of inhalers with a spacer device minimizes medication deposits in the oral cavity and oropharynx, thus allowing more of the particles to be inhaled into the lung.
- The use of saliva substitutes can improve xerostomic effects. Xylitol-containing products reduce the acidogenic potential of plaque.
- Advise parents to brush their child's teeth following administration of medication or use an aqueous mouth rinse if oral hygiene is not possible, particularly during the night to reduce the risk of dental erosion and counteract the acidic pH of the medication.
- Children who have been admitted to hospital with frequent episodic asthma within the past 12 months or those managed with high-dose oral corticosteroids are not suitable for day surgery and should be admitted preoperatively to be adequately prepared by the paediatric respiratory team.
- Avoidance of adrenaline-containing local anaesthetic solutions is recommended due to their potential adrenergic effects if injected intravenously.
- There are no known contraindications to the use of nitrous oxide or sedative doses of benzodiazepines or antihistamines for asthmatic children.

Cystic fibrosis

This multisystem disorder is an autosomal recessive condition affecting all mucus-secreting exocrine glands of the body, especially the respiratory and gastrointestinal systems. The incidence is 1 in 2500 and the heterozygote frequency is estimated to be 1 in 25 in the Caucasian population. Cystic fibrosis is characterized by chronic respiratory obstruction and infection, intestinal malabsorption and growth retardation. Sexual development is often delayed. Although the disease is currently incurable, aggressive symptomatic treatment has improved survival and quality of life in recent years. Long-term complications include sinusitis, pneumothorax, osteopenia, diabetes, liver disease with problems in bleeding and drug metabolism. The major cause of death is respiratory failure from recurrent chest infections.

Children were previously treated with broad-spectrum tetracyclines and often exhibited classic intrinsic staining and enamel hypoplasia of the permanent dentition. However, in Australia, this practice stopped in the early 1970s and the incidence of enamel anomalies has decreased dramatically in recent years. Increased and altered rates of salivary flow seem not to predispose to dental caries, possibly due to long-term use of antibiotics.

Dental management

- Dental management should be based on optimal prevention, including strict oral hygiene and diet control. Routine dental treatment can be undertaken provided pulmonary involvement is stable and well controlled with medication.

- Local anaesthetics with vasoconstrictors are not contraindicated.
- Long appointments should be avoided. However, the use of conscious sedation or general anaesthesia must be carefully discussed with the respiratory paediatrician and only used when routine pain management is not possible.
- Physiotherapy and prophylactic antibiotic treatment is required both preoperatively and postoperatively in cases of severe pulmonary involvement.
- Although, life expectancy has increased with current medical therapies, there should be a rational and pragmatic approach to treatment planning and expectations.

Tuberculosis

Tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis* that involves granulomatous inflammation and caseating necrosis in the lung tissues. The most common presentation in children is malaise, anorexia and weight loss, fever and cough with signs of cervical lymphadenitis. Children are managed with long-term antibiotic treatment specific to the infectious organism. Chemoprophylaxis is given to other family members and contacts, to prevent spread of the disease. The bacille Calmette–Guérin (BCG) vaccine is used routinely in communities with a high prevalence of tuberculosis.

Dental treatment should not be performed until the diagnosis and causative agent have been identified and only then be performed following consultation with the respiratory physician.

Genetics and dysmorphology

Diagnosis

Although individually uncommon, many children with genetic disorders will present to the paediatric dentist with specific dental anomalies associated with their condition or medical problems that complicate their dental management. Never assume that all conditions will have been diagnosed before they present, as many children are often diagnosed as having a significant genetic disorder quite late in childhood, either because the condition has late manifestations or because features have simply been missed. When taking a history, it is always useful to draw a simple three-generation family pedigree (Figure 12.13).

Many disorders do not follow Mendelian inheritance patterns, but are clearly of 'familial' or hereditary nature. Many important and common conditions fall into this group; they are often not single entities but causally heterogeneous and are seen as the end result of multiple gene effects against a variable environmental background (multifactorial or polygenic), e.g. cleft lip and palate.

One in 170 live-born births has a major chromosomal abnormality. Chromosomal abnormalities, particularly those involving imbalance of the autosomes, usually result in developmental delay and dysmorphic features and often include multi-system anomalies. These include numerical abnormalities (trisomy 21 or Down syndrome) and structural abnormalities such as segmental deletions (cri-du-chat syndrome), duplications and unbalanced translocations. Numerical abnormalities of the sex chromosomes are better tolerated than those of the autosomes and include conditions such as Turner syndrome (45, X) in females and Klinefelter syndrome (47, XXY) in males. Standard

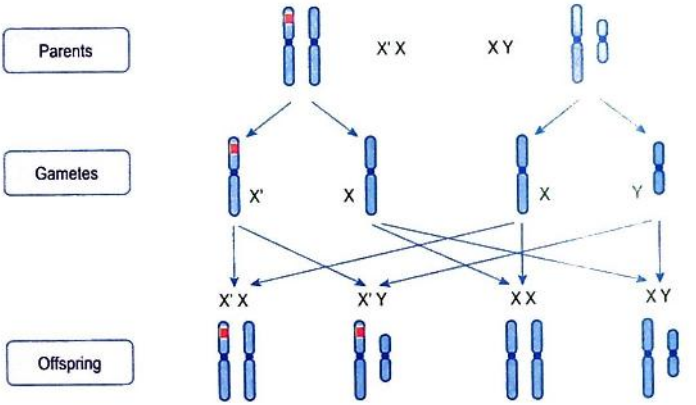

Figure 12.13 Calculation of risk in an X-linked recessive disorder. For a carrier mother (illustrated), there is a 50% chance that sons will be affected and a 50% chance that daughters will be carriers; for an affected father, no sons will be affected but all daughters will be carriers.

cytogenetic karyotyping is now being replaced by DNA-based chromosome microarrays (CMA) that can detect many more significant submicroscopic deletions and duplications (pathological copy number variations). Many other genetic disorders are caused by mutations *within* genes, which cannot be detected by karyotype or CMA analysis, but may be able to be confirmed by targeted gene testing after a syndrome is recognized (Table 12.1).

The first step in making a diagnosis is the recognition that the child is dysmorphic or unusual looking. It is the pattern of dysmorphism and associated malformations rather than a single feature that aids diagnosis. Accurate diagnosis is the key to prognosis, management and sometimes the underlying genetic cause of the disorder. It also helps the parents, as it removes the anxiety of uncertain aetiology and prognosis.

The diagnostic procedure involves history, clinical examination and laboratory investigations. Based on the above information, children can be divided into three basic groups:

- Fetal environmental syndromes (teratogens, compression).
- Developmental anomalies.
- Genetic syndromes.

Referral to a genetic clinic is recommended for all children with multiple anomalies or dysmorphic features of unknown cause. Syndrome diagnosis aids (available by paid subscription) such as POSSUM (www.possum.net.au) or the London Dysmorphology

Table 12.1 Classification of genetic abnormalities

	Defect	Examples
Chromosomal structure	Aneuploidies (abnormal number of chromosomes)	Down syndrome, trisomy 21 Klinefelter syndrome (XXY) Turner syndrome (45,X)
	Chromosomal deletions, duplications and translocations	18q- (deletion of part of long arm of chromosome 18)
Single gene defects	Autosomal dominant	Cleidocranial dysplasia Gorlin syndrome Osteogenesis imperfecta
	Autosomal recessive	Cystic fibrosis
	X-linked recessive	Haemophilia A, B Hypohidrotic ectodermal dysplasia
Polygenic disorders	Multiple minor gene abnormalities interacting with environmental influences	Cleft palate Diabetes mellitus Spina bifida Schizophrenia

Adapted from Jones, K L., 1997. Smith's recognizable patterns of human malformation, fifth ed. WB Saunders, Philadelphia.

Databases (www.lmdatabases.com/) can assist. Laboratory investigations include radiographic survey, chromosomal or microarray analysis, biochemical screening of urine or blood or cultured fibroblasts for specific enzymatic or protein deficiencies if a metabolic disorder is suspected. Referral to a genetics clinic is usually recommended to further elaborate a diagnosis and to assist with specialized genetic testing and genetic counselling.

It is not the intention of this section to detail those anomalies with a major craniofacial or orofacial manifestation, of which dental clinicians should be aware. The reader is directed to texts and online sources that more comprehensively cover these conditions (see References and further reading, below).

Terms used in morphogenesis

Sequence

A pattern of multiple anomalies arising from a single structural defect or event; previously termed anomalad. They usually originate early in development with single problems that create secondary anomalies and manifest with multiple defects at birth or later. These sequences may be divided into three basic groups:

Malformation sequence

- Poor formation of tissue.
- May be single or multiple but are primary structural abnormalities.
- Poor prognosis for normal growth in the areas affected.
- Recurrence rate of 1–5%.
- Example: cleft palate.

Malformations may present as:

- Accessory tissue (e.g. polydactyly, preauricular skin tags).
- Hamartomas (e.g. haemangioma/lymphangioma).
- Incomplete morphogenesis:
  - Agenesis (e.g. salivary gland agenesis).
  - Hypoplasia (e.g. enamel hypoplasia in amelogenesis imperfecta).
  - Incomplete septation (e.g. ventral septal defect, VSD).
  - Incomplete migration of neuroectoderm cells (e.g. Di George association).
- Failure of cell death (apoptosis) (e.g. spina bifida, syndactyly).

Deformation sequence

- Unusual forces acting on normal tissues, usually from abnormal intrauterine pressures.
- Good prognosis for normal growth and a low risk of recurrence.
- Examples are:
  - (Some) isolated talipes.
  - Micrognathia.
  - Torticollis.

Disruption sequence

- Destruction or breakdown of normal tissue, which may result from vascular, infective or physical causes.
- Low risk of recurrence, poor prognosis for normal growth in affected areas.
- Examples:
  - Deafness from congenital rubella.
  - Facial clefting from amniotic bands.
  - Hemifacial microsomia (from stapedial artery haemorrhage).

Syndrome

- A recognizable pattern of malformation.
- Multiple defects of related pathogenesis, arising directly from a single cause (genetic or acquired).

Association

- Non-random occurrence of several morphologic defects not identified as a syndrome or sequence.
- Low risk of recurrence.
- Example: VATER association (**V** vertebral anomalies, **A** anal atresia, **T-E** tracheo-esophageal fistula ± oesophageal atresia, **R** radial and renal dysplasia).

Risk of recurrence in genetic disorders

Autosomal dominant

There is a 50% risk to each offspring of a single affected parent. Many dominant conditions have variable expression and so the manifestations may be increased or reduced compared with the parent. New dominant genetic conditions can also occur.

Autosomal recessive

Carriers do not express the trait. If both parents are carriers, the risk of the child being affected (homozygous; the trait will be expressed) is 25%; the risk of being a carrier

is 50% and the chance of neither being a carrier nor affected is 25%. If one parent is a carrier, there is a 50% chance of the child being a carrier.

X-linked (sex-linked)

- 50% risk of transmission from female carriers to sons (who would then be affected) or daughters (who would then be a carrier).
- No male-to-male transmission from affected fathers to sons, but all daughters will be carriers.

The terms 'X-linked recessive' and 'X-linked dominant' are used to describe sex-linked conditions with an altered frequency of phenotypic expression. Males usually have only one X chromosome; males with an X-linked abnormality are described as hemizygous for the trait, and will be affected. Females usually have two X-chromosomes; female carriers of an X-linked trait are heterozygous. In X-linked recessive disorders, female carriers can be unaffected or affected (manifesting carrier) but the latter are usually much less severely affected than males. Rarely, females are homozygous for an X-linked trait, so will be affected as severely as a hemizygous male. 'X-linked dominant' traits manifest in females and males, but males are often more severely affected.

The degree of phenotypic expression in heterozygous females is determined by the pattern of X inactivation (Lyonization, see Chapter 11) in each tissue. For example, some female carriers of haemophilia A will show a measurable (but subclinical) reduction in factor VIII; and those that carry X-linked hypohidrotic ectodermal dysplasia may also show some phenotypic variation in the dentition such as microdontia and oligodontia, but not to the same extent as in hemizygous males. The markedly increased frequency of females seen with some 'X-linked dominant' conditions, such as incontinentia pigmenti or focal dermal hypoplasia (Goltz–Gorlin syndrome) is explained by male lethality for these mutations in most hemizygous males.

Prenatal tests for genetic disorders

Ultrasound

Ultrasound has become a routine investigation for most pregnancies. It is non-invasive to the mother and the fetus and there are many anomalies that may be diagnosed by this technique. Common ultrasound techniques include the first trimester nuchal translucency measurement (a screening examination for Down syndrome risk) and the mid-trimester fetal morphology scan.

Amniocentesis

This is the sampling of cells from amniotic fluid at around 15–18 weeks. A number of tests can be performed including:

- Karyotyping.
- Sex determination.
- DNA diagnosis.
- Enzyme assays.

Chorionic villus sampling

This test is performed earlier than amniocentesis, at around 11–13 weeks. Similar tests are performed to those done with amniocentesis, although it has the advantage of earlier diagnosis.

Preimplantation genetic diagnosis (PGD)

This is DNA testing of cells biopsied from IVF embryos. This is an option increasingly chosen by couples where genetic risk is high, abortion is not an acceptable option and targeted testing is possible (prior work-up is required).

Population screenings

- 1st trimester combined maternal serum screening and NT scan for Down syndrome risk.
- Mid-trimester maternal serum 'triple test' for Down syndrome risk.
- Mid-trimester maternal α-fetoprotein levels for neural tube defects.

Postnatal tests

Chromosome analysis (cytogenetic) or chromosomal microarrays (DNA-based)

For any baby with dysmorphic features or multiple abnormalities.

DNA analysis

For specific disorders; direct mutation detection in selected gene(s).

Neonatal screening tests

A range of newborn screening tests is undertaken in each country, but the tests offered vary significantly between centres. Commonly performed tests include those for:

- Phenylketonuria.
- Cystic fibrosis.
- Congenital hypothyroidism.
- Galactosaemia.
- Thalassaemia.
- Sickle cell disease.
- Aminoacidopathies.
- Organic acidaemias.
- Fatty acid oxidation defects.

Genetic counselling

Genetic counselling is the process of providing diagnostic assessment, information and support to families or individuals who have, or are at increased risk for, birth defects, chromosomal abnormalities or a variety of inherited conditions. Genetic counselling specialists include clinical geneticists and genetic counsellors. Clinical geneticists provide medical diagnostic assessment and investigation, interpretation of information about the disorder, analysis of inheritance patterns and risks of recurrence and review of available management options with the family. Genetic counsellors take a major role in providing information and supportive counselling to individuals and families, including facilitation of access to resources such as genetic support groups or other community or state support services.

Dental management

The aim of dental management should be a part of a team approach in the care of the child. The overall aim is to reduce the handicapping consequences of the condition.

Restorative and surgical management of the specific dental disability (e.g. enamel hypoplasia or hypodontia) should be addressed with a long-term comprehensive management plan. This must involve several dental disciplines such as orthodontics, periodontics, oral surgery and restorative dentistry, working together to coordinate a treatment plan. Overall success in management is measured by the total rehabilitation of the child and family.

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Endocarditis

See Appendix E.

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13 Children with special needs

Neeta Prabhu, Wendy J Bellis, Angus C Cameron

Introduction

Although the oral health of people with disabilities is similar to the rest of the population, it is generally accepted that many persons with disabilities have extensive treatment needs, which for a variety of reasons, are not adequately met. Throughout the world, the standards of oral healthcare for this population have failed to achieve what would normally be expected for those without a disability.

What is special care dentistry?

It is a discipline targeted to meet the needs of individuals with a variety of limitations that require more than just routine dental care.

A disability may be:

- Intellectual.
- Physical.
- Developmental.
- Emotional (psychological or behavioural).
- Medical comorbidity (covered in the previous chapter).

Barriers to care and philosophies of management

Access to dentistry is often influenced by:

- The attitudes and willingness within the dental team to treat special needs children.
- A perception that the clinician may not have the skills or have the facilities to manage their care.
- Financial considerations.
- Physical access and transport barriers.
- Problems of self-image.
- Issues relating to consent for treatment.

The successful management of these children depends fundamentally on the dentist's ability to:

- Establish a rapport and form a partnership with the patient, family and the carer.
- Clearly understand the condition of the child whom they are treating and.
- Use appropriate behaviour management techniques based on the level of the patient's understanding.

Consent

Providing dental care for people with cognitive impairment who are unable to consent to treatment can raise ethical and legal problems for the practitioner. There is variation in the practice of consent ranging from the ability of an individual to legally consent to their own treatment to the delegation of authority to their parents, caregivers or a guardianship board. Because these ethical predicaments are not obscure, healthcare professionals who routinely care for such patients must complement their clinical skills with their ability to recognize and clearly address these legal responsibilities.

Where are special needs children to be managed?

General dentists have often expressed concerns about a lack of adequate training in appropriately managing these patients in practice, leading to an increase in the number of referrals to the tertiary hospitals. While there has been an overall strategy of integration and normalization of these individuals into mainstream society, unfortunately, most have become reliant on the already stretched hospital-based healthcare system leading to extended waiting times. It must be emphasized that children with special needs require dental appointments that are tailored to make best use of their abilities. The majority of children can be managed successfully in a general practice setting with appropriate training of the dental team. All of the required preventive and maintenance programmes and much of the restorative work can be performed under local anaesthesia and/or sedation. However, there will always be a cohort for whom dental treatment under general anaesthesia is the only alternative. This incurs high costs and has its own problems of access and additional risks and should be recommended only when all other forms of behaviour management have failed or are clearly inappropriate. Additionally, the patient's ability for oral health maintenance postoperatively must always be factored in the treatment planning process to avoid the misuse of these expensive facilities.

There is no doubt that the provision of care for many children with disabilities is challenging. Clinicians should be aware of their own limitations and should consider who and where the child is best managed.

Prevention

The best means of establishing good oral health is by a combination of early contact with dental services and increasing the awareness of regular dental check-ups. Many studies have demonstrated that certain groups of people with disabilities can be instructed in oral hygiene measures if sufficient encouragement and motivation was provided. It is important to introduce these measures from an early age and clinicians should not be deterred from providing comprehensive preventive programmes.

Attention deficit hyperactivity disorder (ADHD)

Attention deficit hyperactivity disorder (ADHD) is a common developmental disorder affecting about 3–5% of the population. The term ADHD is currently used to describe a range of children with varying functional difficulties, but who share the feature of poorly sustained attention. The exact causes remain unknown, however most theories indicate abnormalities in the brain function that are mostly genetic in origin.

Features of ADHD

- Boys are affected much more commonly than girls.
- They are characterized by developmentally inappropriate degrees of impulsivity, inattention and often hyperactivity.
- The symptoms arise in early childhood, usually well before school entry and are present in all settings.
- Some children are extremely impulsive, some aggressive, others quiet and restless. Many have low self-esteem.
- Comorbidities include developmental language disorders, anxiety, oppositional-defiant behaviours, fine motor and coordination difficulties and specific learning disabilities.
- Virtually all children with ADHD have deficits in short-term auditory memory.

Assessment

The assessment of a child for the diagnosis of ADHD requires a number of essential components including:

- Detailed developmental history.
- Physical, neurological and neurodevelopmental examination.
- Detailed standardized behaviour rating scale data from at least two sources, usually school and home and psychometric testing (e.g. Conners' Parent and Teacher Rating Scale; ADD-H Comprehensive Teacher's Rating Scale; Child Behaviour Checklist).

Management

Management of the child with ADHD involves three broad approaches:

- Behavioural.
- Educational.
- Pharmacological.

Many other approaches are commonly applied to these children, including dietary modification, 'natural' or complementary therapies of diverse types and behavioural optometry, however, there is little evidence to support the broad use of any of these interventions, though some individuals report benefits.

Pharmacological management

Psychostimulant medication is the principal pharmacological therapy for ADHD. The two stimulants most commonly prescribed are methylphenidate (*Ritalin*) and dexamphetamine.

- Onset of behavioural effect is usually noticeable within 30–60 min of ingestion.
- Significant clinical improvements in approximately 75% of correctly diagnosed children.
- Common oral side-effects include dry mouth. Some of the medications can cause adverse interactions with drugs commonly used in dentistry, e.g. local anaesthetics and therefore monitoring vital signs during dental treatment is essential.
- Other medications sometimes used in ADHD include the antihypertensive drug clonidine, antidepressants (selective serotonin re-uptake inhibitors, reversible monoamine oxidase inhibitors, and tricyclics) and occasionally neuroleptics.

Dental implications

The visit to a dentist is likely to raise anxiety levels in any child and indeed their parents. In a child with ADHD, this anxiety may manifest in overexcited behaviour and many parents worry about the effect of their child's behaviour on others. They have become accustomed to failure having taken their 'difficult' child to dentists only to be told that it is not possible to provide treatment/care. This may result in either an excessively protective/embarrassed parent with constant apologies on behalf of the child or else an overly firm parent exerting inappropriate, heavy-handed disciplinary actions throughout the encounter. In either situation, the child's behaviour is likely to be reactive towards the parent, thus precluding the establishment of a successful relationship with the dental practitioner.

Successful management of these children may be facilitated using similar strategies to those employed in other disabilities.

- It is important that the patient and the parent are managed positively and with confidence. By creating an atmosphere of confidence, the parental anxiety is often alleviated allowing the child and the dentist to establish a relationship in a more relaxed environment. Similarly, a gentle but firm approach will convey to the child a confidence and structure to the situation within which it is easier for them to conform.
- It is useful for the practitioner to have an understanding of the current management strategies being employed by the family at home and in school and to adopt these techniques in order to maximize success in the dental clinic. For example if a child is used to raising their hand prior to speaking, it is useful for the dentist to employ the same strategy. Clear instructions should be given to the child maintaining eye contact throughout and taking care not to over burden the short-term memory. Such instructions need to be given at a time when the child is not distracted by other activities in the dental surgery.
- The use of the tell-show-do method of behaviour direction has been shown to have value in the management of children with ADHD. Praise and encouragement have an important role in the management of these children and good behaviour should be reinforced and rewarded.

Management strategies

- The current medication scheme should be discussed with both the parents and the prescribing practitioner. It may be helpful to either change the dose or the timing of medication to optimize the action at the time of the dental visit. There is also some suggestion that morning appointments may be more successful, however this may be related to the timing of medication rather than anything else.
- A preventive approach is essential. Tooth brushing and controlling diet both require concentration, motivation and understanding, all of which can be problematic for the child with ADHD. Tooth-brushing charts for the child to take home and mark off daily are more likely to be successful than verbal instructions to brush daily.
- Repetition is important in building up self-confidence in the child.
- Multiple short visits have a higher chance of success than a few, prolonged visits.
- Inhalation sedation can be a particularly useful adjunct to non-pharmacological behaviour management techniques.

- Finally, it is important to realize that oral health is only one of many priorities for the family of a child with ADHD, and the multiple demands made of the parents need to be weighed against the need for dental care. Again, it is important to realize that many of these children are already struggling to master other life skills.

Autistic spectrum disorder

Autism or autistic spectrum disorder (ASD) is defined as a severe developmental disorder characterized by the classic triad of impairments:

- Impaired communication.
- Impaired socialization.
- Repetitive and restricted patterns of behaviour.

ASD is polygenic in origin, however, there are still aspects of the aetiology that are not fully understood. Approximately 50% of affected children also have moderate to severe learning difficulties and there may be other comorbidities such as Fragile X, Rett syndrome, tuberous sclerosis, PKU and epilepsy. As ASD is predominantly a genetic condition, there may be other family members who are affected, including the parents themselves.

Frequency

- Approximately 1% of the population.
- Male : female approximately 4 : 1.

Asperger syndrome

Asperger syndrome is a form of autism. These children have fewer problems with speaking and are often of average or above average, intelligence. They do not usually have the accompanying learning disability associated with autism, but they may have specific learning difficulties.

Problems associated with the dental treatment of a child with ASD

The dental management of children with autism can be a huge challenge for the paediatric dentist, mainly because of the child's behaviour and their impaired communication.

Impaired communication

- Children may have limited speech and language.
- Augmentative communication aids such as Makaton or Pictorial Exchange Communication System (PECS) may be required once the degree of learning disability is ascertained.
- Impaired reciprocal social interaction and lack of eye contact is common. Children with ASD do not understand humour.
- Children are unable to imagine what someone else is feeling ('theory of mind').

Impaired behaviour

- Behaviour may be erratic, disruptive and difficult to predict.
- Both the child and parents may be highly anxious about a visit to the dentist.

- The child may be resistant to change, especially in a new and unfamiliar environment and may show signs of self-injurious behaviours.
- Some children may have a persistent occupation with objects like buttons, beads, etc. (sensory stimulus) and can engage in repetitive body movements.
- Remember, in most cases, conventional behaviour management techniques do not seem to apply.
- Some patients may exhibit ADHD-type traits.

Sensory problems
Many children with ASD have problems with sensory processing and consequently may be hyper- or hyposensitive to sights, sounds, smells and tastes in their environment. They may have an elevated pain threshold and are also known to restrict their diet to certain foods only. Sensory overload and anxiety can result in extreme behaviours such as 'meltdown'.

Medication
Many medications may cause xerostomia and some may not be sugar-free in some countries.

Trauma
Injury to anterior teeth is not uncommon due to the association with epilepsy and dyspraxia.

Late diagnosis
Difficulties and delays in confirming the diagnosis of ASD often results in a delay in accessing early preventive dental care.

Problems with therapies
Linked to the huge number of proposed therapies, there may be dietary restrictions and limitations imposed on specific dental materials. Confectionery may be being used for the reinforcement of good behaviour and as part of a behavioural approach.

Clinical management

Dentists are one of the few professionals who we permit to enter our personal space. Many people find this uncomfortable but understand that the dentist needs to be so close in order to examine teeth. For children with ASD, this close proximity may well be extremely distressing.

Therefore, prevention is the key element to managing these children. Local anaesthesia and inhalation sedation is limited to the higher functioning children with autism, where their communication is not severely compromised.

General anaesthesia and deep sedation are the most frequently used approaches, especially for those children who are young and present with extensive disease. Such treatment should obviously be definitive and comprehensive, including preventive as well as curative elements.

Important tips for management
- Make contact with the families as soon as possible to encourage early access to services through the local child health networks and development teams.

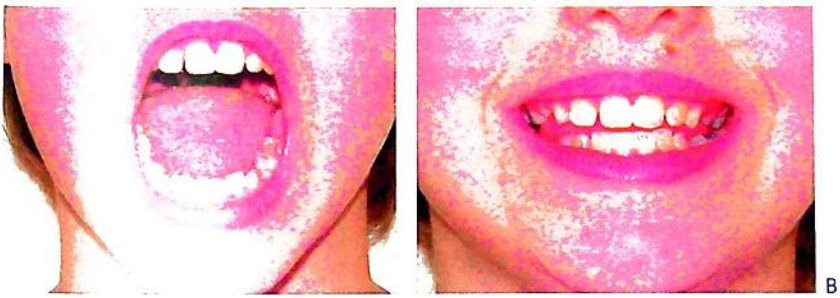


Figure 13.1 Encouraging echolalic children to copy expressions can aid examination and access to the oral cavity. (A) The 'AHHHH' sound helps open the mouth, while the sound 'EEEEEEE' (B) helps with access to the anterior teeth.

- Send out a pre-appointment questionnaire-style letter and an information leaflet.
- Familiarize yourself with the different communication aids that the child may be using. Photographs or images can be put together in the form of a storyline/social story, so that the child is prepared for the dental visit. End the social story (book) with a 'reward picture'. This helps to reduce a build-up of anxiety by making events more predictable for the child.
- Establishing the behaviour of tooth brushing as early as possible is extremely important for these children, not only for oral health and fluoride delivery but also, it is the most successful way of initiating a dental examination.
- Utilize behavioural approaches such as Applied Behavioural Analysis to establish patterns of behaviour around tooth brushing and also to teach the child to accept a dental examination.
- It may not be the actual brushing that the child dislikes, it may be the taste of the paste and the oral sensation. Explore other flavours and low foaming options.
- Some echolalic children (automatic repetition of vocalizations) are able to copy words and expressions, and if this applies to the treating dentist, then the parents can be taught to encourage the child to say 'AHHHH'. This not only helps the parent to brush, it allows the dentist to examine the teeth visually, and also facilitates examination of the pharynx by the child's medical practitioner. The sound 'EEEEEEE' can help display the upper anterior gingival margins, that are sometimes difficult to access (Figure 13.1).
- Actively look for evidence of trauma because of the association with epilepsy or self-injurious behaviours.
- Frequent visits to the dental setting will provide opportunities to learn about the child and give preventive support ('Hello visits').
- Dietary advice must be specific to each individual child. If dietary reinforcers are being used, encourage the use of low-sugar safe snacks and consider the use of sugar-free confectionery.

- Establish time indicators. It is important to help the child realize that this experience does have a time limit. Visual or auditory timers (e.g. sand timers, buzzers, watch alarms), will help them understand this and also to monitor the length of the experience.

Maximizing communication with autistic spectrum children

- Position yourself so that the child can see you.
- Get their attention by using the child's usual name at the beginning of the sentence.
- Use simple language without jokes, sarcasm or jargon.
- Use a minimum of social language and avoid 'Childrenese'.
- Speak slowly to allow information to be processed.
- Limit any background noises in the surgery and use the same staff and a secluded dental surgery if possible.
- Positive re-enforcement of desired behaviour should be 'celebrated' so that it is repeated. If the patient gets aggressive, maintain an unresponsive facial expression and use a calm tone.

Paediatric dentists who care for a large number of these children need to have a full understanding of the nature of ASD and the specific issues around their care. It is important to remain flexible in order to meet the challenges which these children can present in the dental environment.

Developmental disabilities and intellectual disabilities

Developmental disabilities are described as differences in neurological-based functions that have their onset before birth, or during childhood, and are associated with long-term difficulties. People with intellectual disability have an IQ of <70, deficits in adaptive functioning and an onset before 18 years of age. The term developmental disability includes all people with an intellectual disability; however, not all people with a developmental disability have an intellectual disability. For example, children with cerebral palsy and autism have a developmental disability, but not all of them will be intellectually disabled.

Tips for management

- The first appointment is often one in which to familiarize both the dentist with the child's condition and the child with the dental environment.
- Consultation with the family and caregivers helps in finding out the patient's likes, dislikes and behaviour patterns.
- Determine each individual's level of communication; do not treat them as a 'homogenous group'. Be aware of your body language (non-verbal communication). Do not patronize but share the same social courtesies.
- Always allow extra time for your patients to familiarize; keep consistency with staff if possible. Short early morning appointments are preferable.

- Allow time for introduction of new concepts; prior explanation (announce each step) has better acceptance with patients as well as parents and caregivers.
- Repeat instructions when needed; offer praise and reinforce good behaviour. Be sensitive to your patient's gestures. Ask direct closed Yes/No questions.
- Developmental delay is a broad term covering children with a range of medical conditions and syndromes. It is essential that obscure syndromes be researched before performing treatment. Photocopy relevant information for the child's file.
- Support of the parent or caregivers is extremely important in reinforcing and administering preventive advice, oral hygiene practices and diet modification. Consultation with the school or institution may be required to modify diet.

Problems associated with intellectual disabilities

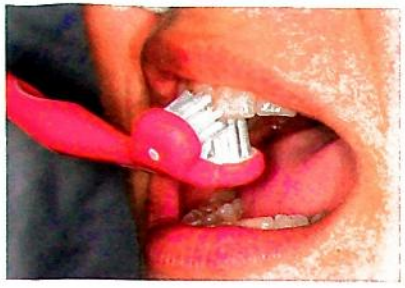
Management of poor plaque control

Patients with intellectual disabilities require assistance to maintain adequate oral hygiene to prevent gingivitis and periodontal disease. Carers should be trained in techniques to deliver oral care in a safe and effective manner. With some patients that may be tactile-defensive, referral to a speech pathologist for an oral desensitization programme prior to commencement of any oral hygiene programmes may be beneficial. These programmes include vibration and extra-oral massage to treat tactile-defensive behaviour. The upper front teeth and gums are the most sensitive regions and therefore avoiding these areas until after complete desensitization of the oral cavity will assist in increasing compliance with tooth brushing.

Adjuncts to oral care

- When brushing, the parent or carer should stand behind and above the child whenever practicable to facilitate control of the head and the brush. This also aids better visual access. Other positions might include swaddling very young children; brushing while still in the wheelchair/feeding chair or sitting on the floor (Figure 13.2).

Figure 13.2 Sitting on the floor, supporting the child from behind facilitates tooth brushing and oral hygiene for infants and young children with disabilities.



A

B

Figure 13.3 (A) Three-headed toothbrush. (B) A range of toothbrushes designed to facilitate improved brushing for patients with disabilities.

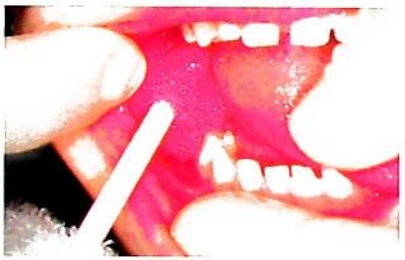


Figure 13.4 Foam tooth swabs are useful in children with disability but also in those children undergoing chemotherapy using sodium bicarbonate or chlorhexidine mouthwashes.

- A flexible '3-headed toothbrush' simultaneously brushes the gums and teeth making it easier for those with limited dexterity. It covers the entire tooth surface and is ideal for use in individuals with short tolerance or attention span. Other toothbrushes with large handles assist patients with disabilities (Figure 13.3).
- Although electric toothbrushes have smaller heads and are easy to use, they run the risk of breaking or splitting inside the mouth and should be used with caution.
- Foam oral swabs (Figure 13.4) help to gently remove debris from the mouth in between brushing. They can be dipped into warm water, mouthwash or sodium bicarbonate (0.2%), however, they should not be substituted for regular tooth brushing.

Malocclusion

There is a higher incidence of hypotonicity and hypertonicity of oral musculature in people with intellectual disabilities. These patients may also have unusual oral habits such as tongue thrusting, which creates malocclusions. Many patients with intellectual disabilities will be able to manage conventional orthodontics with an appropriate level of support including with the use of relative analgesia or sedation. However, for those patients with challenging behaviours, conventional orthodontics may not be possible. An orthodontist may consider interceptive orthodontic measures that might reduce

the degree of malocclusion and the need for appliance wear. Early referral and consultation is beneficial for all children with a disability, who are developing a malocclusion in the mixed dentition stage.

Management of tooth-wear in the patients with an intellectual disability

Study models should be taken at the earliest signs of tooth-wear to establish the rate of tooth-wear over time. The causes of the tooth-wear should be established and if possible, eliminated. Gastro-oesophageal reflux is common in people with developmental disabilities and must be addressed by appropriate referral to gastroenterology. The incidence of tooth grinding is also higher in this population and should be addressed where possible by appropriate means.

Only treat the tooth-wear restoratively if there is:

- Uncontrolled tooth-wear over time.
- Loss of vitality or risk of loss of vitality.
- Aesthetic issues.
- Functional issues.

The restorative treatment of choice is overlaying of worn teeth using an indirect composite resin material with minimal tooth preparation. Two treatment sessions using sedation will be required for many patients with developmental disabilities to adequately take impressions and maintain isolation for cementation procedures.

Tooth grinding

Many parents and caregivers seek dental consultation because of tooth grinding and the worry or associated dental damage it can cause. It can be a social problem for families, teachers and caregivers and consideration should be given as to whether or not the behaviour has other implications such as attention seeking in changed family circumstances. Tooth grinding is either physiological or pathological. It is quite commonly seen in individuals with neuromuscular and learning difficulties.

Physiological tooth grinding (Figure 13.5)

- Often occurs during times of concentration or at night during sleep, although it may occur at any time.
- Begins early during the development of the primary dentition usually once the primary first molars erupt.
- Usually diminishes once the primary teeth have exfoliated.

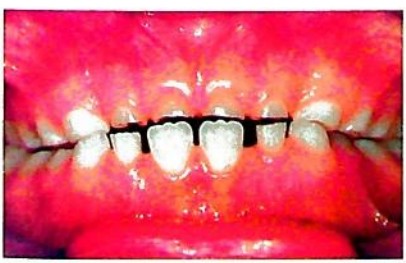


Figure 13.5 Physiological tooth-wear can be quite extensive. Pulp exposure is uncommon, however, it is important to monitor the rate of tooth loss with serial photographs or, if possible, study models.

- No treatment is usually required other than parental reassurance.
- Use of soft or hard acrylic splints is indicated to protect the teeth, however, if the wear is excessive threatening pulp exposure, then restorations using stainless steel crowns or extractions are indicated.
- Unusual to reflect any generalized systemic condition and dental anthropologists regard this grinding as a phenomenon of 'tooth sharpening' termed 'thegosis'.

Pathological tooth grinding

- The amount of wear exceeds that which is felt to occur normally. Children may lose up to half the crown length in upper anterior teeth. Extensive enamel loss with wear facets and exposed dentine is unusual in posterior teeth.
- Often seen in children with underlying neurological disorders or medical problems such as Down syndrome, cerebral palsy or head injury. It has been hypothesized that tooth grinding in these patients stimulates endorphin production and is perceived to be a pleasurable activity.
- An increase in grinding intensity in these children may reflect other pathology such as otitis, salivary gland infection or generalized pain elsewhere in the body.

Management of tooth grinding

- If there has been extensive loss of tooth structure in the primary dentition, it will be essential to monitor any changes in the first permanent molars. Treatment may involve the placement of stainless steel crowns on the second primary molars. This will not only protect the permanent teeth but preserve the vertical dimension of occlusion and tends to decrease grinding.
- Tooth grinding that is associated with self-mutilation of the soft tissues is extremely difficult to manage and some strategies have been discussed in Chapter 8.
- It must be noted that when, in the more severe cases, extractions of permanent teeth are contemplated, eventually, all teeth will probably be lost. For those cases of intractable grinding and self-mutilation, the removal of only a few (anterior) teeth invariably leads to removal of all teeth in the arch.
- It is also important to identify other intrinsic or extrinsic factors such as reflux or an erosive diet that would contribute to further tooth surface loss.

Self-mutilation (Figure 13.6)

A number of conditions exist which present with self-mutilation:

- Hereditary sensory neuropathies (congenital insensitivity to pain syndrome).
- Lesch–Nyhan syndrome (hypoxanthine guanine phosphoribosyltransferase deficiency).
- Hereditary neuropathies are rare inherited disorders affecting the number and distribution of small myelinated and unmyelinated nerve fibres. Most categories in classification systems arise from the varied clinical presentations – terms used have included congenital indifference or insensitivity to pain, dysautonomia, sensory anaesthesia, painless whitlows of the fingers and recurrent plantar ulcers with osteomyelitis.

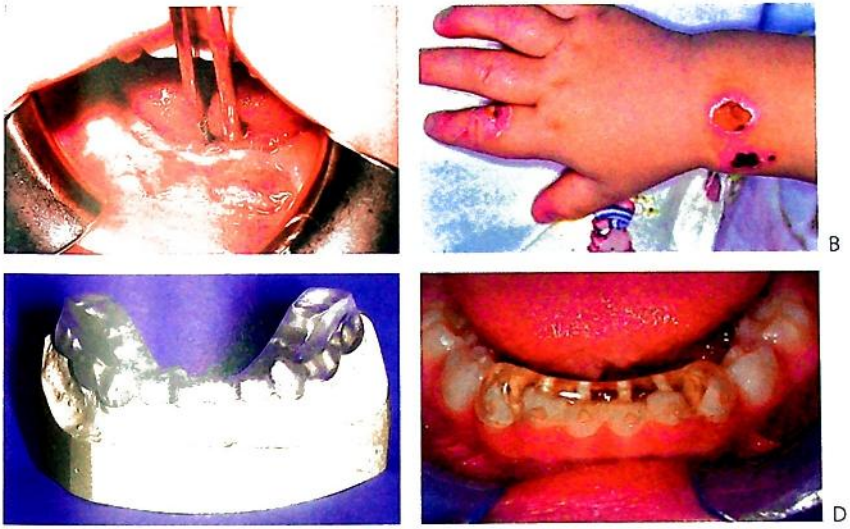


Figure 13.6 (A) Self-mutilation in a child with a peripheral sensory neuropathy. This child presented with exfoliation of the anterior teeth. She was investigated for many of the conditions described above until it was discovered that she herself was pulling out her teeth. Having no sensory nerve endings, she could feel no pain. (B) Finger-biting can also be a manifestation of neuropathies. (C) An appliance to prevent self-injury. All cases are different and an appliance that is successful in one patient may not prove to be appropriate in another. (D) A lower acrylic splint to cover the teeth and prevent tongue biting. The holes on the labial aid in retention of the cement.

The term 'indifference to pain' in these cases, is a misnomer in that indifference implies a cerebral inattention or cognitive dysfunction. Those patients with 'indifference' correctly receive painful stimuli but fail to react in the usual defensive manner by withdrawal. In those patients with 'insensitivity to pain', the deep tendon reflexes are preserved, as these are controlled by large-diameter myelinated fibres. The lack of pain perception is due to a true peripheral neuropathy.

Diagnosis

The diagnosis of these conditions is often made by exclusion and by careful observation of the child. It is not uncommon for many months to pass before a correct diagnosis is made and, in the absence of other pathologies, parents or caregivers may incorrectly be suspected of child abuse or Munchausen syndrome by proxy. Because of an inability to recognize or feel pain, these children may avulse teeth and inflict extensive trauma to the gingivae, tongue or mucosa with their fingers or

by biting and chewing. Self-inflicted ulceration (factitious ulceration) may also occur as a habit (akin to nail biting) but may also be a manifestation of psychological disorders.

Management

- Selective grinding of tooth cusps or 'dome' build-ups of the occlusal table with composite resin to produce a smooth surface.
- Acrylic splints or cast silver splints to prevent gross laceration of the tongue or fingers.
- Extraction of teeth may be required as a last option in severe cases.

Initial management in young children often necessitates restraint to prevent these children from injuring themselves. Even for the most vigilant parents, constant supervision is impossible and invariably, these children will continue to sustain injuries despite the best care. The involvement of occupational therapists is invaluable to support parents and institute protective measures in the home such as the use of padded clothing, arm splints, helmets and other protective devices. Where lacerations to the tongue and other soft tissues occur, mouth guards and other appliances which prevent the teeth from occluding are required. Lower appliances are generally more suitable than those placed in the upper arch. In severe cases where the mutilation is intractable, botulinum toxin A (Botox) has been used to selectively paralyse the major mandibular elevator muscles (medial pterygoid and masseter).

Prognosis

The prognosis for most children with peripheral sensory neuropathies is poor and, in one case managed by one of the authors, the child died of an undiagnosed pneumonia before 3 years of age. Children tend to have repeated hospital admissions, fractures of long bones, injuries to the extremities and recurrent chronic infections. This pattern of repeated traumatic injuries is characterized in one such patient:

- Premature loss of all lower anterior primary teeth.
- Chronic ulceration of the lower alveolus.
- Second degree burn to right forearm from a radiator.
- Fracture of left humerus (during hospital admission) with subsequent multifocal osteomyelitis.
- Fracture of left condyle and mandibular symphysis.
- Death from respiratory sepsis at 2 years of age.

Cerebral palsy

The cerebral palsies are a heterogeneous group of static encephalopathies that have in common, a disorder of posture and movement. The motor disability is permanent and the clinical manifestations are variable. Cerebral palsy can be simply classified into:

- Spastic (hemiplegia, paraplegia and quadriplegia).
- Dyskinetic (choreoathetoid and dystonic).
- Ataxia.
- Mixed.

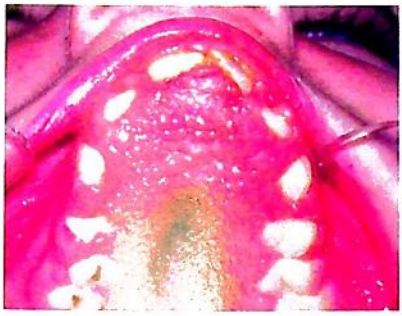


Figure 13.7 Severe phenytoin gingival enlargement, candidosis and papillary hyperplasia in the palate of a child with cerebral palsy. The hypertonicity of the oral musculature has caused the protrusion of the anterior teeth and an orthopaedic compression of the maxilla.

Adverse prenatal and perinatal events that affect the brain account for the known causes of cerebral palsy, although most causes are unknown.

The cognitive ability of a child with cerebral palsy cannot be quickly determined. Time is required with these children to assess their physical and mental abilities. Many patients with cerebral palsy have no cognitive impairment at all and may use a form of verbal communication that requires an electronic aid and operator patience. It is often not necessary to change voice tone or level of language when addressing these children.

Maxillary protrusion and generalized anterior tooth spacing are common sequelae due to abnormal orofacial neuromuscular tone (Figure 13.7). Tongue thrust, dribbling, mouth breathing, and perioral sensitivity are also common clinical presentations. Dental caries and periodontal disease may be severe due to neglect and following surgery to reposition the major saliva gland ducts to reduce drooling.

Dental management

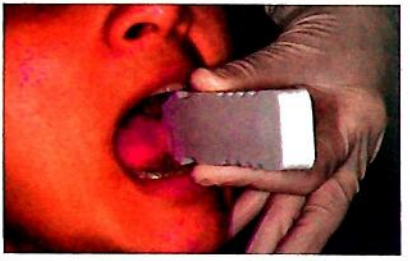
Reflex limb extension patterns may be triggered during dental visits if care is not taken. These contractions may occur during transfer of the patient from wheelchair to the dental chair. Discuss the transfer with the parent or caregivers before offering assistance. Use the hoist option for a safe transfer when available. This reflex may also be stimulated if the child's head is loose or unsupported. Ensure that the child is stabilized in the chair with blankets and pillows or restrained with a belt or webbing. If a reflex pattern occurs where the limbs are in extension:

- Raise the chair.
- Stabilize the head in the midline.
- Bring the arms forwards.
- Reassure the child.

Some patients are best treated in their own motorized wheelchairs. Remember to lock the wheels, recline the chair and use adequate head support (Figure 13.8).

Gag, cough, bite and swallowing reflexes may be impaired or abnormal in children with cerebral palsy. If the gag reflex is more exaggerated, treat the patient in a more upright position with the neck in slight flexion and the knees bent upwards, if possible. Mouth props may be used, however, for those patients with impaired swallowing, there is an increased risk of aspiration. Hand-held props and a floss ligature help to reduce this possibility. Rubber dam is especially useful in these cases as well. If the

Figure 13.8 Motorized wheelchair lift, allowing the patient to remain in the chair during dental treatment.



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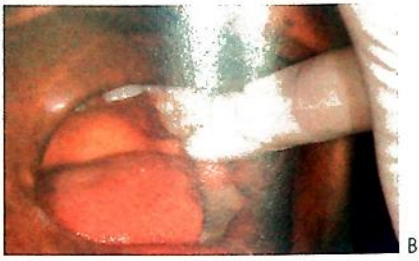

B

Figure 13.9 (A) A foam mouth-prop may also aid oral examination or tooth brushing. (B) It is very important to protect your fingers from being bitten, not least to protect against the risk of infection but also potential damage. By placing the index finger in the buccal sulcus and behind the last molar, the mouth can be opened and the operator's fingers safe.

patient's bite reflex to oral stimulation is still present, introduce instruments from the side rather than the front. To allow dental examination, apply gentle pressure with the forefinger on the anterior border of ascending ramus and in the retromolar triangle. This reduces the risk of a bitten finger. Alternatively in some cases, foam mouth props are available to assist in providing a safe oral examination in those patients with unpredictable behaviour who may unexpectedly bite down (Figure 13.9). Nitrous oxide sedation may help to reduce involuntary movements during dental treatment.

Hydrocephalus

Most cases of hydrocephalus result from obstruction to cerebrospinal fluid (CSF) flow, either within the cerebral ventricles or in the subarachnoid space. As the ventricles enlarge due to the accumulation of CSF, intracranial pressure increases, resulting in serious neurological impairment if not decompressed. The postnatal causes of hydrocephalus are varied including bacterial infection, haemorrhage and neoplastic obstruction, but prenatal causes are often undiagnosable. Treatment by insertion of a shunt is usually appropriate in infants with severe hydrocephalus. Many children with hydrocephalus have other developmental deficits such as learning disabilities or paraplegia.

Children with hydrocephalus undergoing dental treatment may require antibiotic prophylaxis if they have shunts that directly empty into the major blood vessels (ventriculoatrial) to prevent septicaemia and shunt infection. It is generally considered that children with ventriculoperitoneal and spinoperitoneal shunts do not require prophylactic antibiotic cover, unless specified by the neurologist. In all instances it is wise to seek advice and manage the patient in consultation with the their attending physician.

Spina bifida

In this condition, there is a herniation (meningomyelocele) of the spinal cord, nerve roots and meninges through a wide deficiency in the laminae and spinous process of one or more vertebrae, usually at the sacral or lumbosacral levels. The exposed cord is dysplastic and almost always non-functional, often resulting in paraplegia. Early operative closure is performed where possible to prevent infection and subsequent orthopaedic and urological management are necessary. Rehabilitation is best provided by coordinated multidisciplinary clinics.

Children with spina bifida have a higher prevalence of latex allergy (gloves, rubber dam) compared with the general paediatric population. The use of vinyl gloves is recommended. Many children are confined to a wheelchair and spinal comfort should be optimized in a similar way as for children with cerebral palsy. Otherwise, routine dental management can be undertaken in the clinic setting.

Muscular dystrophies

Muscular dystrophy is a progressive, genetically determined, primary degenerative myopathy. The clinical features include increasing muscle weakness, poor muscle tone, abnormal body movements, skin changes and progressive joint and skeletal deformity. Duchenne muscular dystrophy and myotonic dystrophy are the two most common forms and current treatment is to slow the effects of disuse atrophy. Ambulation is usually not possible after 12 years of age.

Oral manifestations include craniofacial deformity with protrusive spaced anterior teeth due to poor orofacial tone and associated mouth breathing, tongue thrust and open bite. Poor plaque control, gingivitis and anterior tooth trauma are common oral findings. Dental management strategies are similar to those used in children with cerebral palsy, using head and body supports and mouth props. Do not try and stop your patient's movements, instead work around them. Use check retractors in patients with loose or rigid oral musculature. Sedation and general anaesthesia are often necessary to manage children with muscular dystrophy due to their inability to tolerate routine procedures in the dental chair.

Sedation and general anaesthesia are often necessary to manage children with muscular dystrophy due to their inability to tolerate routine procedures in the dental chair. Anaesthetic techniques must be modified to minimize intra- and postoperative respiratory and cardiovascular depression and invasive monitoring, and access to intensive care may be warranted. Depolarizing muscle relaxants like succinylcholine should be avoided because of the risk of hyperkalaemia. This may result in the release of large amounts of potassium ions from the muscle cell into the bloodstream with subsequent,

rapid increase in potassium concentration in the blood resulting in life-threatening cardiac rhythm disturbances. Malignant hyperthermia occurs relatively frequently in patients with muscular dystrophy in the presence of succinylcholine or inhalation anaesthetics. This is characterized by an extremely elevated metabolism within the muscle cell. As a result, the temperature of the entire body rises to life-threatening levels. It is of tremendous importance that this is promptly diagnosed and appropriate measures are implemented.

Vision impairment

In addition to the obvious barriers to care that present for a child with vision impairment: the inability to see the dentist, the environment and what treatment is being done, is extremely threatening. Again, communication is the key to trust and success in treatment.

- The reception staff should introduce themselves and offer to lead the patient to the surgery and determine the level of assistance your patient needs. If the patient attends with a guide dog in harness, it indicates the dog is working.
- It is vital to assess the degree of visual impairment. Allow the child to make full use of their tactile sense and their sense of smell when familiarizing them with the dental environment and dental procedures.
- Always announce your entry and departure from the room. Offer verbal and physical reassurance to the child once a rapport has been established, as they cannot see non-verbal gestures.
- Paint a picture in the mind of your patients by describing the treatment and the environment throughout the procedure. A startle reflex may occur if patients are not warned before different instruments are introduced into the mouth without warning.
- Many visually impaired people are photophobic. It is important to ask parents and children about light sensitivity. Safety glasses should preferably be tinted.
- All written information, including appointments and oral hygiene instructions, should be provided in large text or Braille.

Hearing impairment

Children with hearing impairment present a unique challenge for dentists, as effective communication is the primary basis of successful child management. Hearing impairment may be sensorineural or conductive in origin, and range in degree of hearing loss from mild to profound. Recent advances such as the development of the cochlear implant, new surgical procedures and future stem cell therapy, have improved hearing outcomes significantly for many, but not all, hearing impaired children. It is useful to learn basic sign language or the appropriate manual finger-spelling alphabet (e.g. the two-handed alphabet in Britain, Australia and New Zealand; or the one-handed alphabet in the USA and Canada and, with some variation, many other countries) (see Figure 13.10). It should be noted that even within the English-speaking world, there are different sign languages which are mutually unintelligible (i.e. Auslan in Australia or American Sign Language in the USA and Canada). As with all behaviour management,

A

Figure 13.10 (A) Auslan two-handed alphabet.

B

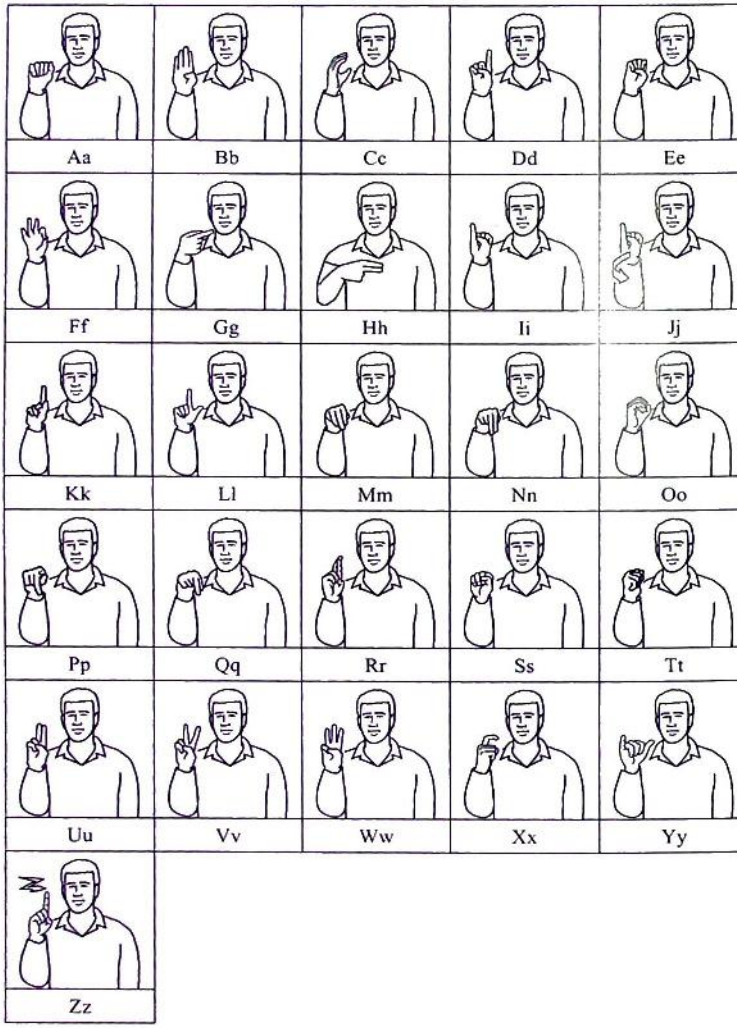

Figure 13.10 *Continued* (B) Auslan one-handed alphabet. (Reproduced with permission Johnston, T., Schembri, A., 2007. Australian Sign Language (Auslan): An introduction to sign language linguistics. Cambridge University Press, Cambridge.)

it is essential to win the trust of the child, be cognizant of their special needs and understand the unique difficulties they have in communication.

- Investigate how the child communicates. If the child uses a sign language as their first or preferred language, use basic signs and finger-spelling if you have previously learned this or, preferably, use the services of a sign language interpreter.
- If the child is hearing-impaired and is able to use their residual hearing with the help of hearing aids or a cochlear implant and lip-reading, use speech. A common fault is to talk loudly rather than slowly. Face the child and speak clearly and slowly. Remove your face mask and eliminate or reduce any background sounds (e.g. radio, etc.) before speaking with the child.
- Make it easy for patients to maintain visual contact, because these children may be startled if they are touched without visual contact.
- Children with hearing difficulties may be very sensitive to vibration, so introduce high-speed and low-speed drills carefully.
- If a hearing aid is worn, the volume may need adjustment. Try to avoid blocking the ears and the hearing aid with the forearms when operating, as this will create feedback.

Oro-motor dysfunction in patients with developmental disabilities

Children with cerebral palsy, trisomy 21 and global developmental delay often present with poor oral functions including:

- Hypertonicity.
- Hypotonicity.
- Dysphagia – difficulty in swallowing.
- Dysphasia – difficulty in speaking.
- Sialorrhoea – difficulty in swallowing, resulting in drooling.

Drooling

Parents will often present with their primary concern being excessive drooling. The paediatric dentist has a significant role in the management of sialorrhoea. Causes of drooling can range from poor competency of the lip and orofacial musculature, malocclusion, dysphagia, to oral habits.

The options for the management of drooling are:

Non-surgical

- Eliminate aggravating factors (dental caries, habits, malocclusions).
- Referral to a multidisciplinary team for oro-motor function therapy.
- Biofeedback using mouth mirrors for lip posture and use of tongue suck and swallow reflex.

Surgical management

- Severance of the parasympathetic supply.
- Re-routing the submandibular duct to the posterior tonsillar pillar; 70% cases described as good to excellent.

- Salivary gland duct ligation.
- Salivary gland excision.

Risks and side-effects
- Ranula formation.
- Loss of the gland.
- Increased caries risk.
- Aspiration of saliva due to dysphagia.

Pharmacological management
- Benztropine (Cogentin).
- Trihexyphenidyl hydrochloride (benzhexol hydrochloride; Artane).
- Scopolamine transdermal patches.
- Glycopyrrolate.
- Botulinum toxin A (Botox). It has a short duration of action (2–6 months) and necessitates the need for repeat general anaesthetics for some patients.

Side-effects of medications include:
- Xerostomia.
- Dental caries.
- Urinary retention.
- Flushing.
- Drying of all mucous membranes.
- Trihexyphenidyl can cause behavioural changes.

Oro-motor function therapy

Oro-motor function therapy is carried out by multidisciplinary teams that may include speech pathologists, occupational therapists, physiotherapists and dentists. The focus of oro-motor function therapy is to develop the oral motor skills required to manage saliva control. This multifaceted approach may include a number of elements such as:
- Behaviour modification.
- Proprioceptive neuromuscular facilitation.
- Postural adaptations.
- Oral screens and dental appliances designed to stimulate oral musculature.

Dental appliances

These are individually designed to produce the desired movement of the tongue, lips or jaw (Figure 13.11). Common goals include:
- Establishment of correct tongue position.
- Stimulation of lip closure.
- Stimulation of tongue elevation, lateralization.
- Stimulation of jaw stabilization.
- Reduction in mouthing behaviour.

Figure 13.11 A dental appliance with a movable bead in palate for use in oro-motor function therapy.

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14 Orthodontic diagnosis and treatment in the mixed dentition

John Fricker, Om P Kharbanda, Julia Dando

Introduction

The primary aim of orthodontic assessment in a growing child is to differentiate between a developing normal occlusion and a potential malocclusion, including any abnormal growth of the face and function of the stomatognathic system. It is essential to have a sound understanding of facial growth and dental development, and the ability to recognize the rate and direction of facial and dental growth. Many situations of apparent malocclusion in the mixed dentition are actually manifestation of the normal process of dental and facial development. Minor incisor irregularities, spacing and ectopic eruption of teeth, which may show up during the mixed dentition, could self-correct with growth and development.

Correction of dental arch irregularities, occlusal and jaw relation abnormalities and elimination of functional interferences may be classified as preventive or interceptive. The term 'preventive orthodontics' implies steps undertaken for elimination of factors that may lead to malocclusion in an otherwise normally developing dentition.

'Interceptive orthodontics' implies that corrective measures may be necessary to intercept a potential irregularity from progressing into a more severe malocclusion. Neither the appliances used nor the treatment itself should interfere with the often rapid changes in eruption of permanent teeth and the dynamic nature of occlusal adjustment. It is important to understand that even when such procedures are carried out, a majority of these children will go on to require some further treatment in the permanent dentition.

Orthodontic assessment of a child

An orthodontic assessment in common with other specialties, must include a good history, a thorough clinical examination and any relevant investigations. The information gathered leads to a diagnosis, which in turn allows treatment planning. This topic is covered in detail in Chapter 1. Additional points relevant to aid orthodontic diagnosis, however, will now be discussed.

The child should be assessed for skeletal and dental problems and abnormalities of functions of the stomatognathic system. Clinical assessment is performed in all the three dimensions of space, i.e. vertical, anteroposterior and transverse.

Skeletal classification

This describes the anteroposterior relationship between the maxilla and mandible relative to the cranial base:

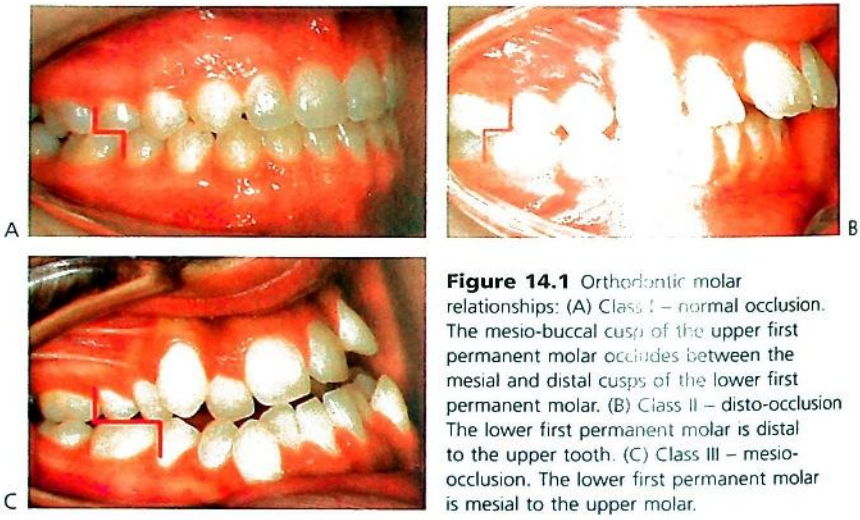


Figure 14.1 Orthodontic molar relationships: (A) Class I – normal occlusion. The mesio-buccal cusp of the upper first permanent molar occludes between the mesial and distal cusps of the lower first permanent molar. (B) Class II – disto-occlusion The lower first permanent molar is distal to the upper tooth. (C) Class III – mesio-occlusion. The lower first permanent molar is mesial to the upper molar.

- **Skeletal Class I** – the maxilla and mandible are in a normal relationship (orthognathic).
- **Skeletal Class II** – the mandible appears small relative to the maxilla (retrognathic). This could be due to: a small mandible, a large maxilla or a combination of both.
- **Skeletal Class III** – the mandible appears larger than the maxilla (prognathic). This could be due to: a large mandible, a small maxilla or a combination of both.
- Vertical assessment includes low or increased facial heights, while transverse assessment should evaluate a narrow or wide maxilla or mandible.

Dental relationships

Dental relationships are recorded with the teeth in occlusion. It describes the antero-posterior relationship of the upper and lower molars according to Angle's classification and the anteroposterior incisor relationship according to the British Standards Institute classification (1983). Angle's classification of malocclusion is based on the relationship of the upper and lower first permanent molars.

Molar relationship (Figure 14.1)

- Class I implies a normal anteroposterior relationship with the mesial cusp of the upper first permanent molar occluding with the fossa of the lower first permanent molar.
- Class II molar relationship implies disto-occlusion of the lower first permanent molar with the upper first permanent molar and is a reflection of a retrognathic skeletal pattern with increased over jet.

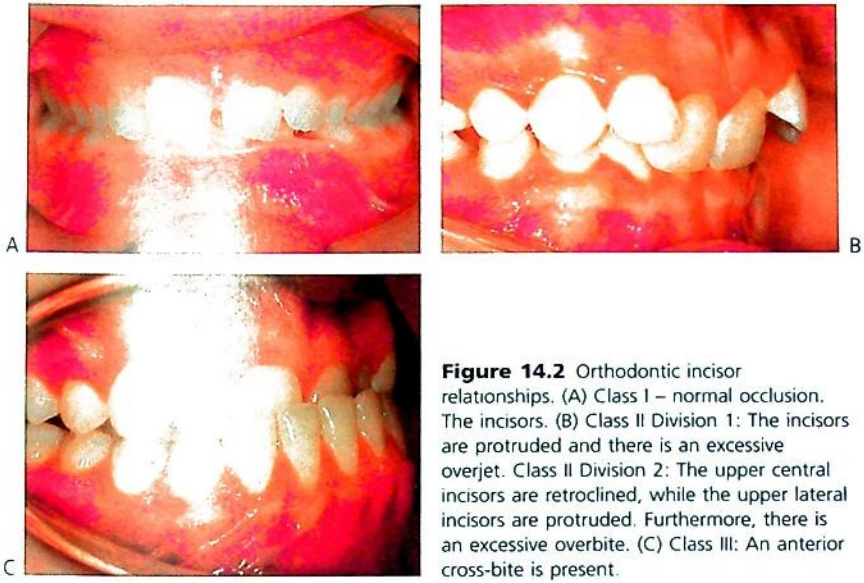


Figure 14.2 Orthodontic incisor relationships. (A) Class I – normal occlusion. The incisors. (B) Class II Division 1: The incisors are protruded and there is an excessive overjet. Class II Division 2: The upper central incisors are retroclined, while the upper lateral incisors are protruded. Furthermore, there is an excessive overbite. (C) Class III: An anterior cross-bite is present.

- Class III molar relationship implies a mesially positioned lower first permanent molar and is a reflection of a prognathic mandible and anterior cross-bite.

Incisor relationship (Figure 14.2)
- Class II Division 1 (proclined upper incisors).
- Class II Division 2 (retroclined upper incisors).
- Class III cross-bite of the anterior segment or negative over jet.

Vertical assessment includes normal or abnormal vertical overlap of the incisors, i.e. normal, deep or open bite. Transverse assessment should include any cross-bite or scissors-bite of the buccal segments of the dental arches.

Complicating factors in any malocclusion
These include:

Intra-arch problems (Figure 14.3)
- Crowding.
- Dentoalveolar disproportion (discrepancy between tooth and jaw size).
- Space loss (premature loss of primary teeth, delayed eruption of permanent teeth).
- Local tooth displacement.
- Spacing.
- Missing teeth (congenital absence, traumatic loss).

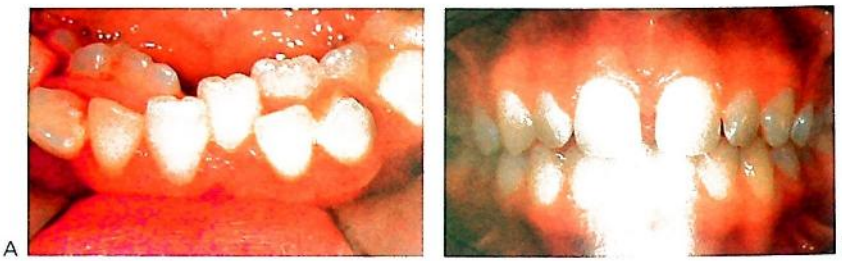


Figure 14.3 (A) Excessive crowding and a tooth to base bone discrepancy. (B) Excessive spacing with an anterior diastema.

- Supernumerary teeth.
- Eruption of teeth.
- Ectopic teeth.
- Impactions.
- Transpositions.
- Retained deciduous teeth.

Inter-arch problems
- Increased overjet.
- Increased overbite.
- Open-bites: Anterior (most common) or lateral.
- Cross-bites: Anterior, posterior (unilateral or bilateral).
- Tooth size discrepancies (Bolton's ratio).

Other factors
- Oral habits.
- Digit sucking.
- Pacifiers.
- Mouth-breathing.
- Abnormal swallowing.
- Trauma to dental tissues or facial trauma that may cause limited jaw opening or chin deviation during jaw opening.

Orthodontic examination

Extra-oral

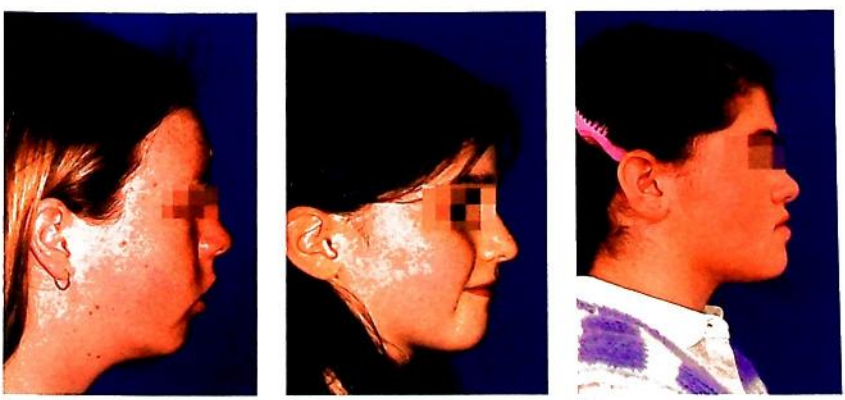
The physical status of the child should be included here, and if relevant, height and weight should be recorded on a standard growth chart. It is essential to determine if the face of the child is also growing normally. The face is examined with the child sitting upright. This is important because the mandibular rest position will change, if lying back.

Frontal view

- Shape of face – long and thin/normal/short and square.
- Symmetry – initial assessment from the front. Looking at the child from above and behind will confirm asymmetry. It is important to look at the position of the chin at rest and in occlusion. A deviation would suggest a functional shift is occurring rather than a true asymmetry.
- Facial proportions – viewed from the front, the face can be divided vertically into equal thirds. The height of the midface (supraorbital ridge to base of nose) should, therefore, equal that of the lower face (base of nose to chin). However, it may be increased or decreased.

Lateral view (Figure 14.4)

- Profile – convex/straight/concave.
- Skeletal pattern – Class I, II, III.
- Nose – small/normal/prominent.
- Chin – recessive/normal/prominent.
- Nasolabial angle – acute/normal/obtuse. If the angle is obtuse, orthodontic treatment involving the extraction of permanent teeth will have a detrimental effect on the profile.
- Lip position – competent (closed), incompetent (apart).
- Lower lip – normal/everted.
- Labiomental sulcus – normal/deep.
- FMPA – imagine a line connecting the ear to the eye (Frankfort horizontal) and construct an angle with the lower border of the mandible. This angle (FMPA) will help to assess growth direction.



A B C

Figure 14.4 Evaluation of facial profile. (A) Child with retrognathic chin and convex face suggestive of skeletal – Class II pattern. (B) Normally growing chin – Class I pattern. (C) Prominent chin with concave profile, suggestive of Class III skeletal pattern.

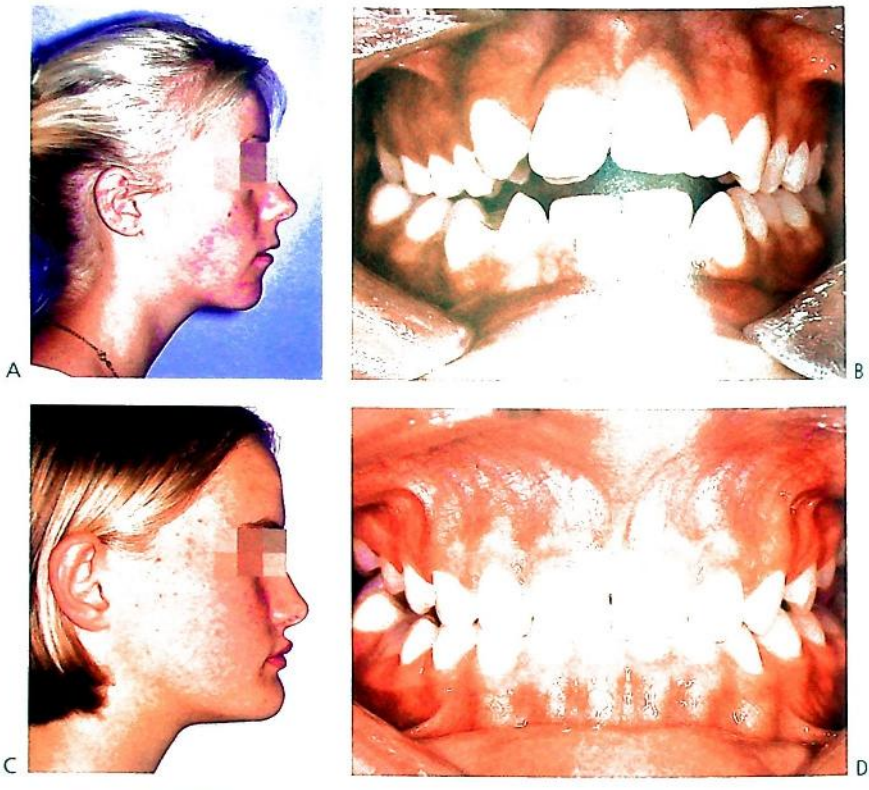


Figure 14.5 (A,B) Vertical grower with a long thin face, high FMPA. (B) Tendency for anterior open bite. Note the increase in lower face height. (C,D) Horizontal grower with square face and low FMPA. (D) Tendency for deep bite. Note the decrease in lower face height.

- Extreme types are vertical growers (with FMA angle >32°) and horizontal growers (with FMA angle of <20°). Growth direction is an important consideration in treatment planning (Figure 14.5).

Intra-oral

A record should be made of:

- Number of teeth present radiographically and intra-orally.
- Tooth quality and any existing restorations or active caries.

- Level of oral hygiene and assessment of gingival health.
- Assessment of crowding/spacing in both arches.
- Overjet, overbite, canine and molar relationships.
- Anterior or posterior cross-bites and any associated functional shift into occlusion.
- Centrelines
  - Dental centre lines in relation to each other (upper to lower).
  - In relation to the facial midline (upper to face and lower to face).
- Condition of the oral soft tissues
  - Abnormal frenal attachments. Thick upper labial frenum can be associated with median diastema. Tongue tie can be associated with articulation problems.
  - Any other pathology such as an epulis.
- Tongue position: at rest and during swallowing.
- Speech articulation problems (see Chapter 15).
- Opening and closing patterns.

It is important to identify any deviation of the mandible during opening and closing into full intercuspal occlusion. Often the underlying cause is interference from an erupting tooth leading to either functional forward or lateral shift that demands early treatment to restore the balance in the temporomandibular joint (TMJ).

Investigations

These are determined by the findings at examination.

- Panoramic radiograph – will give an overall picture of the developing dentition and jaws. It is the standard radiograph used in orthodontic assessment. Additional films may be needed to allow a more detailed analysis of suspected pathology.
- Bitewings.
- Periapical films.
- Lateral cephalogram – this is useful to assess skeletal discrepancy when treatment is to be started. Tracing of the film and subsequent cephalometric analysis will aid diagnosis and treatment planning. This film can also be used as a baseline to monitor future growth.
- Posteroanterior (PA) cephalogram – for transverse discrepancies or asymmetry.
- Anterior occlusal films – only for location of impacted canines, supernumeraries, or ectopic teeth.
- 3-dimensional CT may be required for evaluation of exact location of impacted or supernumerary teeth that are otherwise difficult to locate with standard 2D radiographs. Cone beam CT (CBCT) is becoming increasingly popular, since radiation doses are much less than CT (see Chapter 11).
- Study models are essential baseline records that are also used in treatment planning and space analyses.
- Any other relevant tests (i.e. pulp sensibility testing).

Evaluation of crowding

In the permanent dentition, it is easy to assess the amount of crowding by taking measurements directly from study models. Treatment will depend on the severity of the problem and may involve arch lengthening or extractions. In the mixed dentition, however, a prediction of future crowding is necessary.

Mixed dentition analysis

The purpose of a mixed dentition analysis is to determine the space available in the dental arch for the permanent successors to erupt. To complete this analysis, it is necessary to first record the arch length and the mesiodistal widths of the mandibular permanent incisors.

Measurement of arch length

The conventional way to determine arch length is to measure directly from a set of study casts. Soft brass wire can be adapted from the mesial of the first permanent molar to follow the arch form around to the mesial of the contralateral first molar. The wire should be shaped to the ideal arch form and not follow any teeth out of alignment. Once the arch length has been determined, it is then necessary to estimate the space required for the permanent successors. Mesiodistal dimensions of erupted teeth up to second premolars can be obtained directly from a study cast. Unerupted teeth can be measured by one of two methods:

- Using periapical radiographs with allowances for magnification (modified Hixon–Oldfather method).
- Using tooth size prediction formulas.

Both methods are based on the high correlation between the crown measurements of the permanent mandibular incisors and the combined sizes of two premolars and permanent canines. Thus, it is possible to forecast the amount of space required for the unerupted teeth and to plan interceptive and/or preventive space management requirements.

The difference in values between arch length and tooth size will indicate the amount of crowding or spacing present.

Summary

At the end of diagnosis, a clinician should have gathered the following information:

- Growth pattern – normal, Class I, Class II, Class III.
- Growth trend – horizontal, vertical.
- Sequence and stage of eruption of teeth.
- Number of teeth missing or supernumeraries.
- The dental arch relations – cross-bites.
- Overjet and overbite.
- Amount of crowding and spacing.
- Pattern of swallowing.
- Digit sucking/thumb sucking habit.
- Pattern of respiration, i.e. normal breathing or mouth breathing.
- Is the child likely to grow normally or would he/she benefit from orthodontic intervention?

Crowding and space management in the mixed dentition

Space management can minimize the development of crowding in the permanent dentition. It essentially involves:

- Space maintenance following the premature loss of primary molars.
- Utilization of the leeway space by placement of holding arches.

Space maintenance

The best space maintenance treatment is the preservation of the primary molars until natural exfoliation. Although dental health education and improved caries prevention have lowered the number of children who develop malocclusion because of premature loss of primary teeth, it is still one of the most common controllable causes of malocclusion.

When a primary second molar is lost prematurely due to caries or to the ectopic eruption of the first permanent molar, the first permanent molar will drift mesially. This is most pronounced in the maxilla with a more rapid shift of the molar and causing a Class II malocclusion. The earlier the loss of the second primary molar and the less the root development of the permanent molar, the greater will be the amount of bodily mesial shift of the permanent molar.

Factors to consider for placement of space maintainers

Placement of a space maintainer requires care of the appliance and oral hygiene maintenance. A child with poor oral hygiene and high caries risk is not the ideal case for such appliance therapy. Before a decision is made to provide a space maintainer, it is often essential to critically evaluate its merits, the need and the benefit it would provide to the development of normal occlusion.

Anterior teeth

- Loss of one or more primary incisors results in negligible space loss if canines and molars are present.
- If the eruption of a permanent incisor is delayed, space loss may occur because of migration of adjacent teeth.

Posterior teeth

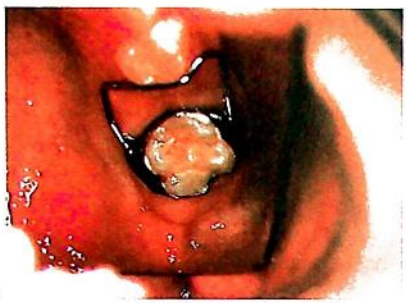
- Whenever a primary second molar is lost prematurely, whether before or after the eruption of the first permanent molar, there will be some loss of arch length caused by the mesial drift of the permanent molar.
- Space maintenance is critical in children who have a normal arch length and lose a primary molar. Any loss of space in these children will result in crowding of the permanent teeth.
- Where space has already been lost, it is necessary to regain space and then fit a space maintainer.

Types of space maintainer

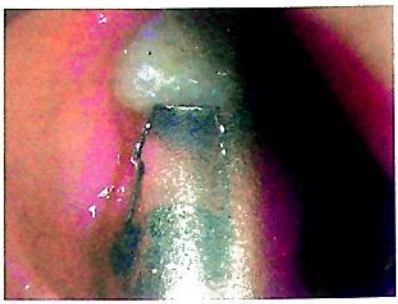
Removable

Removable space maintainers have shortcomings similar to all removable appliances:

- They may be worn at the whim of the patient.
- May be broken.
- Easily lost when removed by the patient.



A



B

Figure 14.6 (A) A band and loop space maintainer. The placement of a space maintainer must not compromise the permanent tooth. Bands should be cemented with a luting glass ionomer as a protection against caries and the appliance reviewed regularly. As the premolar erupts, the appliance is removed when there is interference with normal emergence. (B) A distal shoe space maintainer is placed following early loss of the second primary molar prior to the eruption of the first permanent molar. It prevents mesial migration of the permanent tooth.

A removable space maintainer that is only worn at night is often sufficient to hold space and prevent the mesial drift of permanent molars. Night-only wearing of the appliance also reduces the risk of loss or breakage by the patient. The appliance should be washed and inserted in place before going to bed, then removed, washed and placed in a safe place when not worn. Hawley's appliance is a typical example.

Fixed space maintainers (Figure 14.6)

- Fixed appliances have the advantage that they are worn continuously and do not require patient cooperation in wearing them.
- It should be noted that the placement of a fixed appliance in a child at high risk of caries may compromise those teeth which are banded as well as adjacent teeth.
- Band and loop appliance is typically used in cases of unilateral loss.
- Nance appliance or lingual arch can be used if the loss is bilateral.

Utilization of the leeway space

- The combined mesiodistal width of the deciduous molars is greater than that of the premolars. This residual space can be used to relieve mild crowding (1–2 mm) elsewhere in the arch.
- A transpalatal arch is used in the maxilla.
- A lingual arch is used in the mandible.

Regaining space

Within an arch, space may need to be regained when migration of permanent teeth has already occurred following the loss of adjacent deciduous teeth (Figure 14.7). Furthermore, space maintenance would then be needed until the permanent successor erupted. In the maxilla, this would intercept a developing Class II, dental relationship

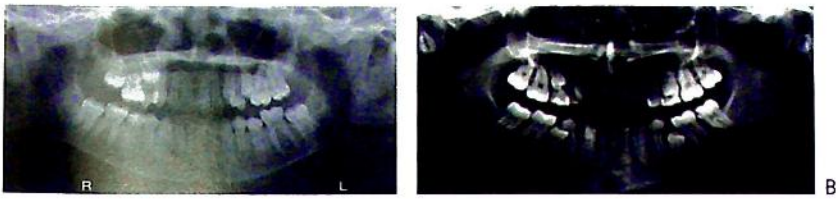


Figure 14.7 The failure to place space maintainers following bilateral loss of primary second molars has resulted in forward movement of the first permanent molars thereby reducing the available space for the second premolars. (A) More space tends to be lost in the upper arch than in the lower arch following loss of the second primary molars (B).

secondary to mesial migration and rotation of the first permanent molar. In the mandible, it could prevent a mild dental Class III relationship by uprighting tipped lower first permanent molars. In individuals with a developing skeletal discrepancy, the dental correction would have no effect on the underlying skeletal problem.

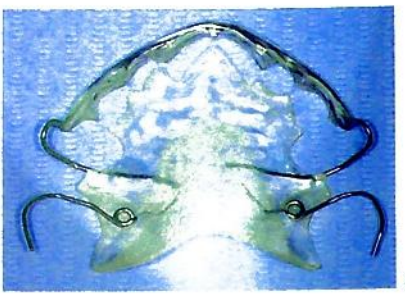
In general, tooth movement is slower in cases with severe horizontal growth pattern (low FMPA). Conversely, it is rapid in vertical growers, and space loss can occur very quickly. Early fitting of a space maintainer will prevent space loss. If space is to be regained, it is essential that the mechanics should not extrude the teeth at all.

Radiographs and study models are essential aids in assessing space needs. It is important to note whether teeth have moved bodily or have tipped into the space. Tipping can be easier to resolve than bodily tooth movement. Radiographic examination should also locate the permanent second molars and establish space available for distalization of the first permanent molars.

Appliances used to regain space

- Uprighting mechanics:
  - Sectional fixed appliance.
  - Removable appliances – Acrylic cervical occipital appliance (ACCO appliance). An ACCO appliance (Figure 14.8) is comprised of a palatal acrylic plate with an anterior bite platform to disclude the posterior teeth, allowing the first permanent molar to move freely. Retention is obtained via Adams clasps on the first premolars or deciduous molars and a labial bow across the permanent incisors. The bow should be supported with a band of acrylic across the labial surfaces of the incisors to increase the anchorage for the finger springs against the mesial surface of the molars to be distalized. These are most successful in the maxillary arch, where there is a dental and skeletal Class I pattern with normal vertical proportions and the regaining of space is by way of uprighting the first permanent molar.
  - Full arch fixed appliances.
- Distalizing appliances:
  - Distalizing springs or screws.
  - Open coil springs.
  - Extra-oral headgear.
- Lip bumpers – to upright and distalize lower molars.

A



B

C

Figure 14.8 (A–C) The ACCO appliance is used for uprighting maxillary permanent molars to regain space. To maximize anchorage, acrylic is flowed over the labial arch wire and this limits the proclination of incisors as the molars are distalized.

Timed extraction of teeth to resolve intra-arch crowding

The total amount of arch length deficiency is the key to planning of timed extractions. For this to be beneficial, a cephalometric analysis should show the child to be growing within a normal pattern and that all the permanent teeth are present radiographically and in the normal order of eruption.

Extraction of deciduous canines

- Premature loss of a primary canine as the permanent lateral incisor erupts will result in a midline shift to the same side. Extraction of the contralateral deciduous canine will help prevent a shift occurring.
- In cases with crowding, the loss of primary canines should be managed by placement of a fixed lingual arch to support the incisors and prevent lingual tipping as the midlines correct themselves.
- As the permanent canines erupt, it may be necessary to reduce some part of crown at the mesial of the primary first molars and then, as the first premolars erupt, reduce the mesial of the second primary molars.

Serial extraction

The purpose of serial extraction is to encourage the early eruption of the first premolars ahead of the permanent canines and should only be considered where there is an arch discrepancy of >4 mm. Serial extraction is usually limited to the upper arch as serial extractions in the lower arch usually results in lingual collapse of the lower anterior segment.

Contraindications

Serial extraction should not be performed in the following circumstances:

- Class I malocclusions where the lack of space is slight and the teeth show only mild crowding.
- Where there is a skeletal discrepancy in the dental arches.
- When there is a deep overbite or an open bite.
- When there are permanent teeth congenitally absent from the dental arch.

Treatment stages in serial extraction

- First, the primary canines are removed to allow spontaneous alignment of the permanent incisors.
- The primary first molars are removed to allow the eruption of the first premolars.
- Once the first premolars are erupted, they are removed and a space maintainer is issued to allow the permanent canines to erupt.
- Further orthodontic treatment is usually required to align teeth to achieve correct root angulation and incisor torque.

Thus, serial extraction is a planned procedure that demands a minimum of 5 years' supervision by the dentist of the developing occlusion. Without such a commitment, the objectives will not be fully achieved and at times, the child is then left with a more severe malocclusion.

Spacing

Spaces in the deciduous dentition are normal and such spacing indicates an increased chance of good alignment in the permanent dentition. During the early mixed dentition stage, physiological spacing is common in the anterior region with the incisors appearing splayed. As the permanent canines erupt, this will resolve spontaneously and early treatment should not be contemplated.

Dentoalveolar disproportion and tooth size discrepancies can also lead to spacing. Definitive treatment should be delayed until the permanent dentition, when options for space closure through orthodontics or tooth build-ups are available. If 'cosmetics' is a concern in the mixed dentition, then crown build-ups of the permanent incisors as a temporary solution, should be considered.

Unusually large spaces may be caused by an enlarged tongue. Isolated true macroglossia is not common and is usually only associated with Beckwith–Wiedemann syndrome. If tongue size is suspected to be enlarged, secondary causes such as increased secretion of growth hormone may also need to be investigated by a paediatrician.

Dentoalveolar disproportion and tooth size discrepancies can also lead to spacing. Definitive treatment is carried out in the permanent dentition, when space closure or tooth build-ups should be considered.

Hypodontia is the term used to describe the congenital absence of one or more teeth. These teeth have not developed from the initiation stage of tooth development (see Chapter 11).

Diagnosis

An understanding of the normal sequence and average age of eruption of permanent teeth will alert the practitioner to the possibility of congenital absence of a tooth or teeth. Any delay in the normal eruption time of permanent teeth or exfoliation of primary teeth should be investigated radiographically. The orthopantomogram (OPG) panoramic radiograph provides a good view for premolars and molars but is often unclear in the incisor region because of the narrow focal trough. It may be necessary to supplement this with either periapical films or, in the maxilla, an anterior occlusal film.

For most children, a radiographic survey at age 7 years will demonstrate the presence or absence of all permanent teeth, except for third molars. It should be noted, however, that there is a large variation, especially in the second premolar region. Third molars are generally not radiographically visible before the age of 9 years. A radiograph will show the tooth follicle before calcification begins, and there is a range in development time between the presentation of the follicle and calcification commencing, especially for second premolars.

Management

Where a permanent tooth is diagnosed as congenitally absent, there are two choices in management:

- Retain the space after loss of the primary tooth and insert a prosthetic replacement.
- Orthodontics to close the space.

The preferred treatment choice will depend on the severity of the condition (number of absent teeth), location of the missing teeth and the underlying skeletal pattern.

Class I patterns

The jaw relationship is normal. If the missing tooth is located in the posterior segments, space closure is often the treatment of choice. Occlusal relationships, however, will dictate the decision. On the other hand, if one or more lateral incisors are missing, the choices are to close spaces and substitute permanent canines for the absent laterals or open the spaces and fire the roots of the adjacent teeth in preparation for implant replacement. Planning such treatment is age-dependent as implants are not indicated until the patient is at least 22 years of age. Opening spaces in preparation for implants as a teenager will require long-term retention of the spaces and root angulations and often will require further orthodontics just prior to the placements of implants due to growth factors.

Class II patterns

This malocclusion is characterized by a smaller mandible with an increased overjet. The preferred option for missing teeth in the maxilla, is to close space and reduce the

overjet at the same time. The permanent canines can replace lateral incisors, but size, shape and colour must be considered. Restorative techniques using resin veneers and acid-etch can be used to reshape the canines as lateral incisors, restoring the anatomy of the substituted teeth and providing a balanced smile.

Class III patterns

The maxilla is proportionally smaller than the mandible and there can be a dental cross-bite either anteriorly or posteriorly. If teeth are missing in the lower arch, and the skeletal problem can be camouflaged with orthodontics only, it may be advantageous to close space. Conversely if teeth are missing in the maxilla, space opening and tooth replacement is the preferred option to avoid further constriction of the arch.

Tooth loss due to trauma

Traumatic loss of a maxillary incisor can be treated orthodontically within the same guidelines as those for congenital absence of teeth.

Orthodontic aspects of supernumerary teeth

Development and aetiology

Supernumerary teeth may be found in any part of the dental arch; however, the most frequent sites are in the regions of the maxillary midline and the third molars. Because the supernumerary teeth develop late, they are not often found in the primary dentition and when they do develop with the primary teeth, they usually erupt. Tubercular and inverted supernumerary teeth are most often unerupted, and they commonly delay or inhibit the eruption of the central incisors. Supernumerary teeth in the region of the lateral incisors, either in the primary or permanent dentition, usually erupt into the arch.

Orthodontic effects

Delay or failure of eruption

The failure of a permanent tooth to erupt leads to malocclusion as adjacent teeth shift into the area that should be occupied by the permanent tooth. Moreover, the supernumerary tooth can be a cause of ectopic eruption of other teeth, producing a malocclusion. Supernumeraries can result in:

- Displacement of permanent teeth (Figure 14.9).
- Rotations.
- Diastemas.
- Development of dentigerous cysts.
- Resorption of roots of adjoining teeth.

Treatment planning

- If a supernumerary tooth is obstructing the eruption path of an adjacent tooth, or has any associated pathology, it should be removed as soon as convenient. However, with all dentoalveolar surgery, there are risks that need to be considered, such as nerve injury or damage to adjacent teeth and anaesthetic risk. In some cases, the risks will outweigh the benefits and it may be better to leave the supernumerary tooth *in situ*.

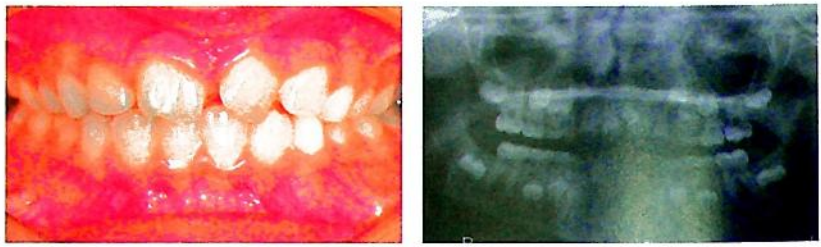


Figure 14.9 (A) Displacement of the upper left central incisor due to a midline tuberculate supernumerary lying in the palate (B).

- A supernumerary primary incisor may be retained if there is sufficient room for it. The tooth should be extracted when the permanent lateral incisor is ready to erupt.
- If there is an extra permanent lateral incisor present with the primary supernumerary tooth, it may be removed at the same time. Usually, the more distal of the two permanent teeth is the supernumerary tooth.
- Identification of a supernumerary tooth that is similar in form and size to the adjacent tooth can be made by comparing the teeth with those on the opposite side of the dental arch. The tooth that more closely resembles the size and shape the normal lateral incisor should be retained.

Extraction of over-retained primary teeth

The earlier that one can recognize and remove over-retained primary teeth that may be causing ectopic eruption of a succedaneous tooth, the better the chance that a permanent tooth will erupt in a satisfactory position (Figure 14.10). The greatest damage that may result from over-retained primary teeth comes in the wake of ankylosed primary molars.

Diagnosis

The ankylosed primary molar may not be recognized in the very early stage (Figure 14.11). The condition can be readily diagnosed a short time later because the vertical level of the occlusal surface of the ankylosed tooth becomes noticeably lower than the level of adjacent teeth, and as time progresses, this difference in vertical level becomes more extreme.

Because ankylosed teeth seem to be submerging, they have been called 'submerged' teeth, but the term cannot be applied accurately to ankylosed teeth. The continued vertical eruption of the uninvolved adjacent teeth and the vertical growth of the alveolar process and periodontium create the illusion that the ankylosed tooth is submerging.

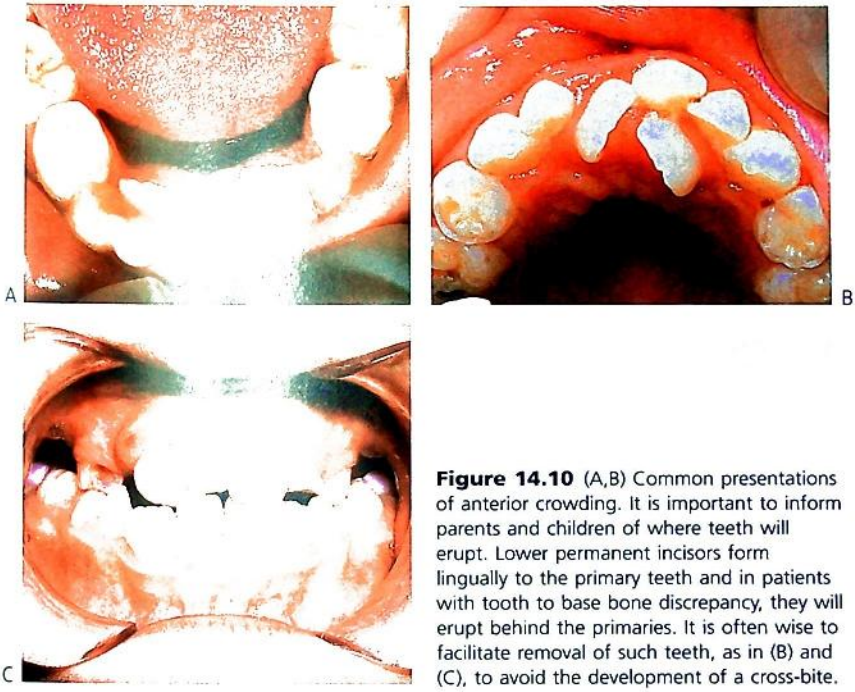


Figure 14.10 (A,B) Common presentations of anterior crowding. It is important to inform parents and children of where teeth will erupt. Lower permanent incisors form lingually to the primary teeth and in patients with tooth to base bone discrepancy, they will erupt behind the primaries. It is often wise to facilitate removal of such teeth, as in (B) and (C), to avoid the development of a cross-bite.

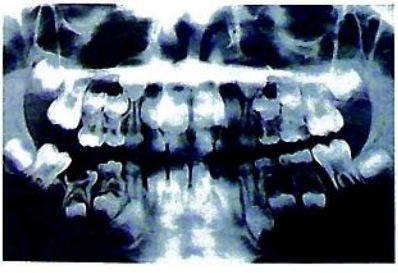


Figure 14.11 An ankylosed lower second primary molar in infraocclusion. The tooth is subgingival and grossly carious. Uprighting of the first permanent molar is required initially, followed by surgical removal, and placement of a space maintainer.

Management

- Ankylosed primary molars may be retained as long as they are maintaining arch length (i.e. preventing mesial shifting of the first permanent molars) or as long as they do not prevent the eruption of the succedaneous teeth.
- If there is evidence of root resorption, these teeth will eventually be lost normally and there is no indication for early removal.

- The union between the cementum and dentine of the tooth and the bone of the alveolar process is physically strong and removal may require a surgical procedure, depending on how far the tooth has been submerged.
- A space-maintaining appliance must be used if the primary tooth is removed before the imminent eruption of the succedaneous tooth.

An over-retained tooth often accounts for the ectopic eruption, or impaction, of the succedaneous tooth. Because the ankylosed tooth is ultimately unable to withstand the mesial shifting of the first molar and the loss of arch length, extraction of an ankylosed primary tooth is an effective means of interceptive-preventive orthodontics.

Ectopic eruption of permanent canines

The incidence of impacted canines in the maxilla is 2% and the majority lie in a palatal position. The anomaly can be associated with small or absent lateral incisors. In about 12% of cases with impacted canines, the lateral incisor root will undergo some resorption.

The normal age of eruption is 11±2 years and the crown should certainly be palpable in the labial sulcus at 9–10 years of age. If the canine is not palpable, further investigation is indicated to check for impaction or ectopic eruption. Intra-oral radiographs taken at right angles to each other and the technique of parallax can be used to localize their position or alternatively an OPG panoramic film. Radiographic scans with cone beam CT are the best for localizing ectopic or impacted permanent canines as these provide a 3D position of the tooth (see Chapter 11).

Interceptive extraction of the deciduous canines can improve the position of the permanent teeth and the maximum improvement will be seen within 12 months (Ericson & Kurol 1988). The success of this approach is reduced, however, if the arch is already crowded (Power & Short 1993).

Ectopic eruption of first permanent molars (Figure 14.12)

This can be an indication of an inadequate arch length, and a radiographic survey is required to confirm the presence of premolar teeth. The permanent teeth may resorb the distal margins of the second primary molars; this is more common in the maxilla.

Management

- Where there is impaction of the permanent molar against the distal of the second primary molar, slicing or discing of the distal surface of the primary molar will allow the spontaneous eruption of the permanent molar.
- Placement of orthodontic separators or brass ligature wire is usually difficult and uncomfortable, and has mixed success.
- Where the resorption of the primary molar is advanced, the loss of this tooth is indicated and space-regaining mechanics should be considered once the permanent molar has erupted (see above).

Figure 14.12 Ectopic eruption of the first permanent molars, causing resorption of the primary teeth. In this position, it is unlikely that the first permanent molars will erupt and space loss has already occurred. The primary molars were extracted and a space-regaining appliance constructed.

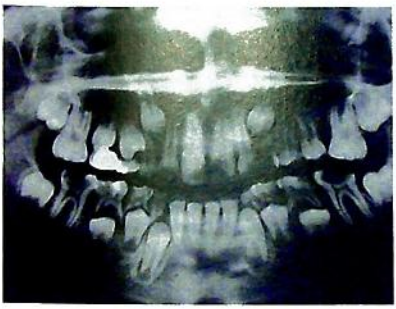


Figure 14.13 Gross caries affecting the lower first permanent molars. Both teeth are non-vital and should be removed. This is a perfect time for extraction as the second molars will migrate mesially. The upper molars should be retained with a night-time removable appliance to prevent overeruption.

- Parents should be warned that further orthodontic treatment is usually required because of arch length deficiencies.
- Primary failure of eruption (PFE) is a rare condition that is characterized by non-syndromic eruption failure of permanent teeth in the absence of mechanical obstruction. It typically affects first molars and/or second molars. The molars do not erupt and also do not respond to orthodontic traction rather efforts to pull them leads to ankylosis. This condition can be familial.

Extraction of first permanent molars (Figures 14.13, 14.14)

Gross caries involving the first permanent molars poses a difficult dilemma in treatment planning. The early presentation of the patient is essential in obtaining favourable results. The basic questions about whether these teeth should be removed or restored are:

- What is the long-term prognosis for the tooth?
- What is the status of the pulp?
- Are the root apices fully formed?
- Are the third molars present?

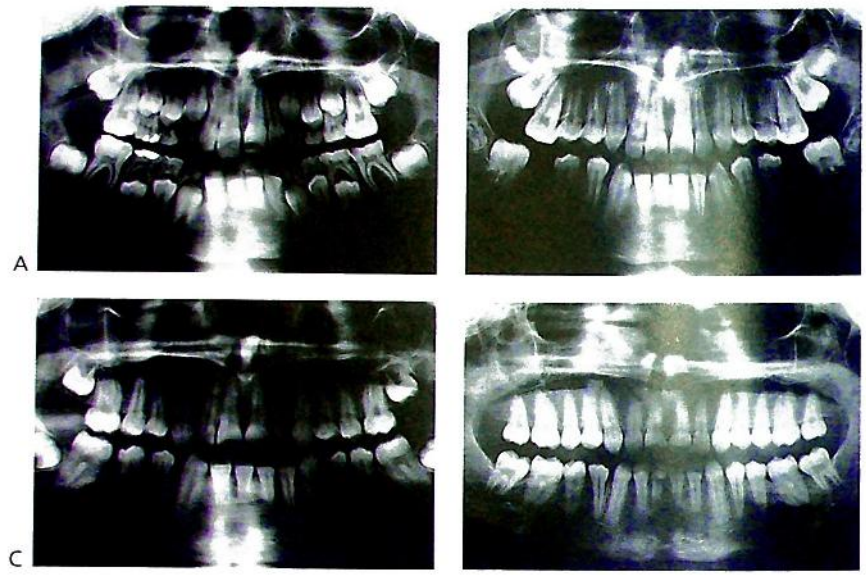


Figure 14.14 (A–D) Serial panoramic radiographs showing mesial migration of the second and third permanent molars following timed extraction of carious first permanent molars.

General considerations

- The decision to extract is often best made in conjunction with an orthodontist.
- If the tooth is not restorable no matter what the occlusion, then it should be removed. Even if successful root canal treatment can be completed, the status of the crown is most important. Commonly, these teeth have extensive loss of tooth structure, with only an enamel shell remaining.
- Non-vital immature teeth have a poor prognosis. Root canal treatment is usually not indicated in these teeth, especially as they would need an apexification procedure.
- If only the lower first permanent molars are removed, a removable appliance such as a Hawley should be used to prevent over eruption of the maxillary first permanent molars before the eruption of the lower second molars.
- The ideal time for lower first permanent molar extraction is before alveolar eruption of the second molar. These teeth will migrate mesially and assume the position of the first molar.
- If three molars are grossly carious and require removal, it is probably better to keep the extractions symmetrical and extract all four teeth.
- The presence or absence of third molars may influence a decision to extract the first molars, but ultimately, it will be the long-term prognosis of the first molars that

determines the final treatment plan. The goal is to have a long-term, functional occlusion with minimum maintenance.

Timing of extractions

Although the timing of extractions will be determined in individual cases, some general rules should be followed if possible.

Class I (with no crowding)

- Extract teeth that are not restorable.

Class I (crowding) or Class II

- Extract lower first permanent molars as early as practicable.
- Retain upper first permanent molars until the second molars begin to erupt.
- Extraction of the upper first permanent molars should coincide with ongoing treatment for crowding.

Class III

- Extract teeth that are not restorable.

Orthodontic appliance systems

Basic requirements of orthodontic appliances

- Permit control of the amount, distribution, duration and direction of the force they exert.
- Be atraumatic to the oral tissues and not be adversely affected by oral secretions.
- Allow teeth and soft oral tissues to function normally.
- Allow wearer to maintain oral hygiene.
- Exert sufficient force or offer sufficient anchorage resistance to induce histological bone changes necessary for desired orthodontic tooth movement.
- Respond to the control of the operator.
- Allow movement of individual teeth or of groups of teeth in desirable directions.

Safety measures in fixed appliance treatment

Appliances should be regularly examined. Loose molar bands can result in caries due to failure of the cement lute, or cause trauma to the soft tissues because of excessive movement from biting forces. Archwires should be carefully fitted with the distal ends either cut as they leave the molar tube, or turned in. Failure to do this will result in irritation of the buccal mucosa.

Removable appliances

Although removable orthodontic appliances cannot produce all types of tooth movement, they possess several advantages. They are laboratory fabricated and hence require less chair time; are easily removed by the child/patient for oral hygiene and are often low cost. The conventional removable appliance is the Hawley appliance.

Design

Removable appliances should include:

- Acrylic base plate or body.
- Retentive components
  - Adams' cribs.
  - C clasps.
  - Ball retainers.
  - Passive labial bow.
- Active components/tooth-moving components
  - Springs.
  - Screws.
  - Biteplanes.

Other design considerations

- For successful tooth/teeth movement to occur, the active components of the appliance should produce force in the desired direction whereas anchorage is derived from the acrylic plate that remains stationary.
- The acrylic plate should fit well against the palatal mucosa and must occupy the interdental spaces. The plate should be of even thickness, for strength and to house the retentive components and springs.
- The anchorage of the appliance is derived from palatal tissues, and from the teeth through the clasps.
- The posterior border of the maxillary appliance should be placed anterior to the junction of the hard and soft palate. It should be thin and gently merge with the palatal mucosa.
- The lower appliance should have smooth borders with sufficient relief to accommodate the functioning of the lingual frenum.

Springs

Spring design (finger, Z or retractors) should ensure adequate springiness and range of action while retaining strength.

For lingual/palatal movement:	Labial bow/long labial bow
For labial movement:	Z spring or T spring
For mesial movement:	Finger spring
For distal movement:	Finger spring
For canine retraction:	Canine retractor – labial/palatal

- The active arm of the spring should be in surface contact with the tooth to be moved.
- The point (area) of force application should be close to the cervical margin/gingival margin to minimize tipping.
- To increase the range of action, helices are incorporated into the spring design.
- Activate slowly, close to the gingival margins, without causing trauma to the oral tissues.
- There should be no hindrance/obstacle in the path of movement of teeth.

Labial bow

An active labial bow produces both horizontal and vertical force vectors resulting in palatal/lingual tooth movement and extrusion of tooth. It is used for retraction of

proclined incisors. Since the labial bow causes tipping of the crown, simultaneous labial root tipping occurs.

Prerequisites for activating a labial bow are:

- Bite opening – this should be achieved prior to incisor retraction.
- Ensure sufficient space is available for retraction.
- The activation should be gradual and gentle.
- Ensure sufficient relief of acrylic is provided palatal to the incisors before activation.

Anterior biteplanes

- The acrylic behind the upper incisors is thickened. In occlusion, there is contact of the lower incisors with the acrylic, with open bites in the buccal segments.
- In a growing child with a mild Class II tendency, bite opening may result in forward posturing of the mandible.
- Bite opening is a combination of supra-eruption of the mandibular molars and intrusion/stabilization of the vertical position of the mandibular incisors. Hence, a child with a normal or horizontal growth pattern is the most suitable case for bite opening.
- Children with vertical facial growth (that is, with a high FMPA) are not suitable cases for treatment with biteplanes. The biteplane will cause an open bite tendency to worsen, and have a detrimental effect on the profile.
- An active labial bow can be used to retract incisors after adequate bite opening, thus reducing an overjet.
- Biteplanes are also used to relieve interferences in the buccal segments, which may be hindering tooth movement.

Posterior biteplanes

- The acrylic is extended to include coverage of the occlusal surfaces of the posterior teeth.
- Allows rapid correction of an anterior cross-bite.
- Unlock the molars for maxillary expansion.

Treatment of anterior cross-bites

Up to 10% of children present with cross-bites. Three types of anterior cross-bite may present in the mixed dentition.

Ectopic incisors

An incisor may erupt ectopically either palatally in the maxilla or labially in the mandible to a cross-bite relationship in centric occlusion. This may occur in a child with a balanced skeletal relationship. Early treatment is necessary if there is a deviation on opening and/or closing or if there is a traumatic occlusion or periodontal concern.

Skeletal Class III malocclusion

Skeletal Class III malocclusions are associated with maxillary transverse and anteroposterior deficiency coupled with prognathic mandible. Patients who have a major component of maxillary deficiency and little vertical growing can be successfully treated

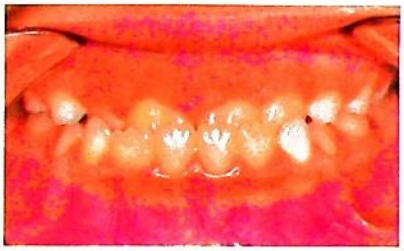


Figure 14.15 A pseudo Class III malocclusion, where the child protrudes into a cross-bite to avoid an anterior interference.

by early orthopaedic interventions. These include rapid maxillary expansion, maxillary protraction and mandibular chin cup therapy. Class III patients require follow-up and interventions to the age of 20 or beyond, until completion of mandibular growth.

Pseudo Class III malocclusion (Figure 14.15)
This pattern occurs where there is a habitual mandibular closure pattern such that the mandible goes into a protrusive bite and thus cross-bite of incisors avoiding traumatic occlusion with lingual position of one or more maxillary incisors. Thus, anterior shift of the mandible can affect the growth of both the maxilla and the mandible with undesirable muscle adaptation. These cases are managed more simply as the primary movement required is to procline the maxillary incisors with a removable appliance or inclined plane until the patient is able to occlude comfortably into a retruded position.

Management

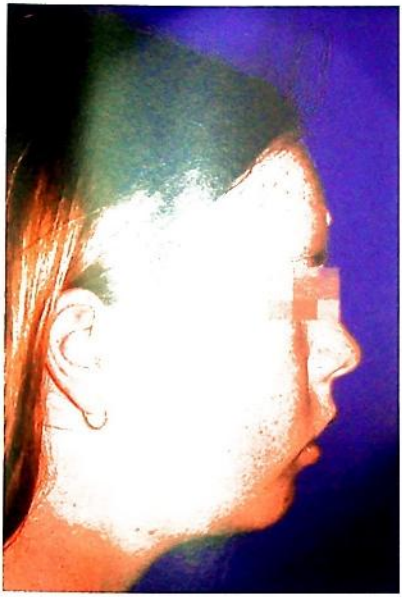
Tongue blade

If there is only one permanent incisor in cross-bite without an excessive overbite, a tongue blade, or paddle pop stick may be used to correct this. The stick is placed lingual to the upper tooth in cross-bite and the patient instructed to close firmly against the stick while it is held in position against the chin. The child should hold it there while biting against it and another person should count to 50 out loud, as in one apple, two apples, etc., for approximately 1 minute. Repeat this six times per day with an interval of at least half an hour. Correction is often complete within a few days.

Inclined planes

Where there is a functional shift of the mandible into an anterior cross-bite, an acrylic inclined plane can be fitted to the lower incisors to restrict the forward posturing and place pressure on the palatal of the maxillary incisors to push them labially. Alternatively, a composite build-up of the lower incisors will mimic the action of an incline plane. (It is preferable to choose a shade of composite resin that is easily distinguished from normal tooth structure to facilitate safe removal).

Treatment is usually complete within a few weeks. This appliance works best where there is a slight increase in overbite, which helps to retain the incisors in positive overjet once the appliance is removed.



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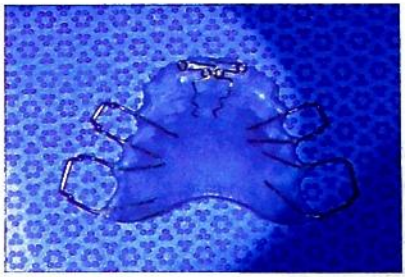

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Figure 14.16 (A) Anterior dental cross-bite involving tooth 11. (B) An upper removable appliance can be used to correct these malocclusions utilizing Adams' cribs on the first permanent and primary molars and two Z-springs. The Z-springs are activated approximately 2 mm to procline the incisor. Normally, the cross-bite should be corrected within 4 weeks and is often self-retaining dependent on the amount of overbite.

Removable appliances (Figure 14.16)

- These appliances should only be used to correct cross-bites of dental origin.
- A modified Hawley appliance can be used in the maxilla to correct one or two teeth in cross-bite.
- Ensure there is adequate space to move the teeth into the desired position and movement will occur rapidly.
- Occlusal surfaces of both the primary and permanent molars should be covered to open the bite and allow free labial movement of the teeth in cross-bite.
- Adams' clasps are placed on the first permanent molars.
- If the primary molars are present, ball-ended clasps can be fabricated to engage the interproximal areas of these teeth.

Where a single tooth is in cross-bite, a Z-spring placed palatally to the malposed tooth can be used, or if both central incisors are in cross-bite, two springs can be used to provide sweep arms on the palatal surface. Initially, the appliance should be fitted and checked for comfort with the springs passive. The springs are then activated 1–2 mm at a time. The patient is reviewed after 4 weeks to reactivate the springs as required and to check the retention of the appliance.

As with all removable appliances, the success of treatment is reliant on cooperation and compliance. If these qualities can be encouraged and the patient takes responsibility for the wearing of the appliance, treatment will progress satisfactorily. Occasionally,

the cross-bite may also be due to a labially placed lower incisor. This must also be corrected, but is dependent on available space. If this is not available, definitive treatment may need to be delayed.

Treatment of posterior cross-bites

A posterior cross-bite is an abnormal, buccolingual relationship of a tooth or teeth when the two dental arches are brought into centric occlusion. There are two types of posterior cross-bite:

Dentoalveolar
Insufficient arch length or prolonged retention of deciduous teeth can deflect teeth during eruption and produce a cross-bite. Prolonged digit sucking can also cause palatal tilting of teeth and narrowing of the maxillary arch.

Skeletal
A skeletal cross-bite is related to size discrepancy between the maxilla and mandible. This could be a narrow maxilla, a wide mandible, or a combination of both. It is possible that both dental and skeletal causes may contribute to cross-bites of variable severity.

Management

- In children with a normally growing mandible, posterior cross-bites should be treated as early as possible to allow normal growth and development of the dental arches and temporomandibular joints. When planning treatment it is important to determine whether the cross-bite is unilateral or bilateral.
- The majority of cross-bites are bilateral but often present as unilateral when the teeth are in full intercuspal position. In these cases, the dental midlines will not be coincident on closing and there will be a deviation of the mandible towards one side at the end on closing.
- When the teeth are closed with the dental midlines coinciding, the posterior segments will be in an edge-to-edge, buccolingual position, reflecting the overall constriction of the maxillary dental arch, and bilateral maxillary expansion is indicated.

Cross-elastics

- When only a single molar is in cross-bite, this can often be corrected with a bonded attachment, button or hook, to the palatal of the maxillary and buccal of the lower molar.
- An elastic is stretched between these teeth; it is worn 24 h per day and changed every time it breaks (which is often).
- Cross-bites will normally correct within 3–4 months with continuous wearing of the elastic. The major change will be reflected in the position of the maxillary molar because of the cancellous nature of the maxillary alveolar bone as against the denser bone around the mandibular molar.

Removable appliances

- Lateral maxillary expansion can be achieved with a parallel expansion screw that is housed in the upper acrylic plate (Figure 14.17).

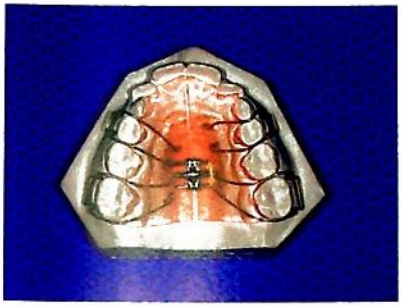


Figure 14.17 Maxillary appliance with a midpalatal expansion screw.

- To ensure delivery of sufficient force on the teeth and palate, the appliance should have excellent tissue contact and anchorage with clasps on teeth.
- Provide acrylic relief palatal to anterior teeth.
- The labial bow should be passive. When expansion occurs the bow becomes activated.
- The majority of the expansion appliances have a pitch of 1 mm. A full turn is achieved with four turns of the key.
- The conventional expansion schedule is one-quarter turn per week.
- Expansion appliances with posterior occlusal coverage work faster as they disclude the buccal occlusion.

It is extremely difficult to achieve unilateral expansion. A jackscrew offset in the palate will move one or two teeth, but there will usually be some expansion on the contralateral side. Always expand beyond the correction of the cross-bite and retain, because relapse potential is high. It is important to remember that correction is dental only, as the major component of tooth movement is tipping.

Fixed appliances

Slow maxillary expansion – quad helix/nickel titanium expanders

- A quad helix (Figure 14.18) is attached to the palatal surfaces of the cemented first molar bands. This may be hard soldered or removable via palatal tubes welded to the molar bands before cementation.
- The activation of this appliance is controlled by the dentist. This can be done intra-orally using pliers to open the individual helices or by removal of the appliance which is then expanded by hand. The quad helix is activated every 4 weeks and the appliance should be removed every 3 months for extra-oral activation and checking for loose bands or incipient caries.
- The expansion should continue until the molars are overcorrected, then retained with the same appliance for a further 3 months. The cross-bite is usually corrected within 4–6 months.
- The quad helix can be used simultaneously with full bonded appliance treatment.
- Thermal nickel titanium expanders require less adjustment than conventional stainless steel. Quad helix appliances provide a pre-determined amount of expansion. Cooling the expander softens the wire and allows it to be constricted and inserted into lingual tubes on the maxillary molars. As it warms to body temperature, it

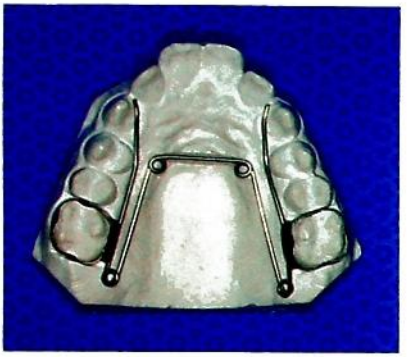
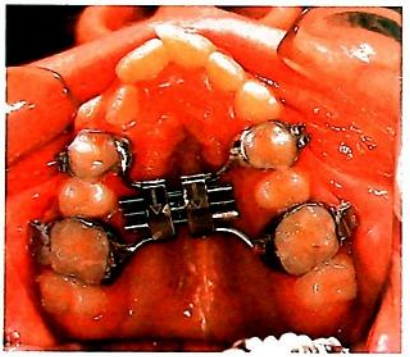


Figure 14.18 A quad helix appliance is used to correct posterior cross-bites in the mixed dentition. These appliances are simple to construct, are well tolerated by the patient and are efficient. They have the advantage that they are fixed and will also act as retainers once the malocclusion is corrected.



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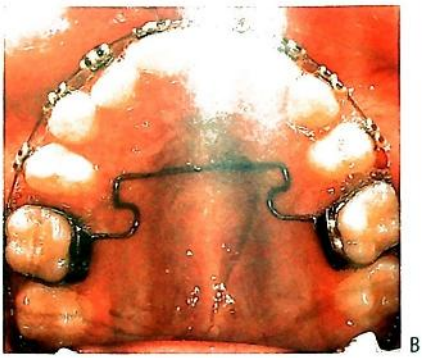

B

Figure 14.19 (A) Rapid maxillary expansion appliance. (B) Following expansion, a rigid retainer (e.g. transpalatal arch) should be used during the fixed appliance treatment phase to limit relapse.

becomes springy and exerts continuous force on the teeth, thereby causing arch expansion.

- The expanding forces also cause simultaneous derotation of the molars.

Rapid expansion – Hyrax screw (Figure 14.19)

- Rapid maxillary expansion (RME) is indicated for severe cases of bilateral cross-bite, in which correction requires skeletal expansion. It involves the splitting of the midpalatal suture producing an orthopaedic increase in maxillary width. This can occur in a growing child, preferably before the age of 9 years. The split is evident on an occlusal radiograph, being widest at the incisors.
- The appliance uses a midpalatal screw (Hyrax) soldered to bands on the first permanent molars and the primary molars or premolars. In contrast with the removable appliance, the screw is activated a quarter turn twice each day and the patient should be monitored once a week.

- As the expansion proceeds, a diastema will show between the central incisors. This will close as the overstretched supragingival trans-septal fibres relapse. The parents and patients should be warned of this. As with any expansion technique, the cross-bite should be overcorrected and retained in this position for at least 3 months with the same appliance.

Deleterious oral habits

Digit sucking

One of the most common oral activities of the infant and young child is thumb and finger sucking (Figure 14.20). Sucking habits are perfectly normal in infancy. The infant will suck on any object brought into contact with the lips. This reflex behaviour may last for several years. It is an adaptive reflex common to mammals. Because it is a normal activity, thumb and finger sucking may be ignored in infancy. Thumb or finger sucking that is discontinued by age 2–3 years produces no permanent malformation of the jaws or displacement of the teeth. Continued beyond the time that the permanent incisor teeth erupt, it is almost always a factor in producing malocclusion in the anterior portion of the mouth.

The majority of older children who continue thumb sucking have what is termed an 'empty' habit. It is just something they have always done. These children are usually receptive to reasons why they should stop and many actually want to give up. A minority, however (especially if the habit has restarted) may have underlying social or psychological problems and these should be investigated.

Malocclusion from digit sucking

- Proclination and protrusion of the upper incisor teeth.
- The lower incisors may or may not be displaced lingually by the abnormal sucking habit.
- Posterior cross-bite due to over activity of buccinator compressing the maxilla.
- Anterior open bite (Figure 14.20A).
- Tendency for the tongue to perpetuate open bite with anterior tongue thrust. Proclined maxillary incisors and an anterior open bite favour the forward positioning of the tongue.

Control of digit sucking

Chemical means

Chemical therapy employs either hot-tasting, bitter-flavoured preparations or distasteful agents that are applied to the fingers or thumbs. Such things as cayenne pepper, quinine and asafoetida have been used to make the thumb or fingers so distasteful that the child will keep them out of his or her mouth. These preparations are effective with a limited number of children, and only when the habit is not firmly entrenched.

Mechanical means

- A simple device for controlling thumb or finger sucking is the application of adhesive tape to the thumb or finger. In many instances, this changes the character of the finger sufficiently to call the child's attention to the fact that it is being placed in the mouth.

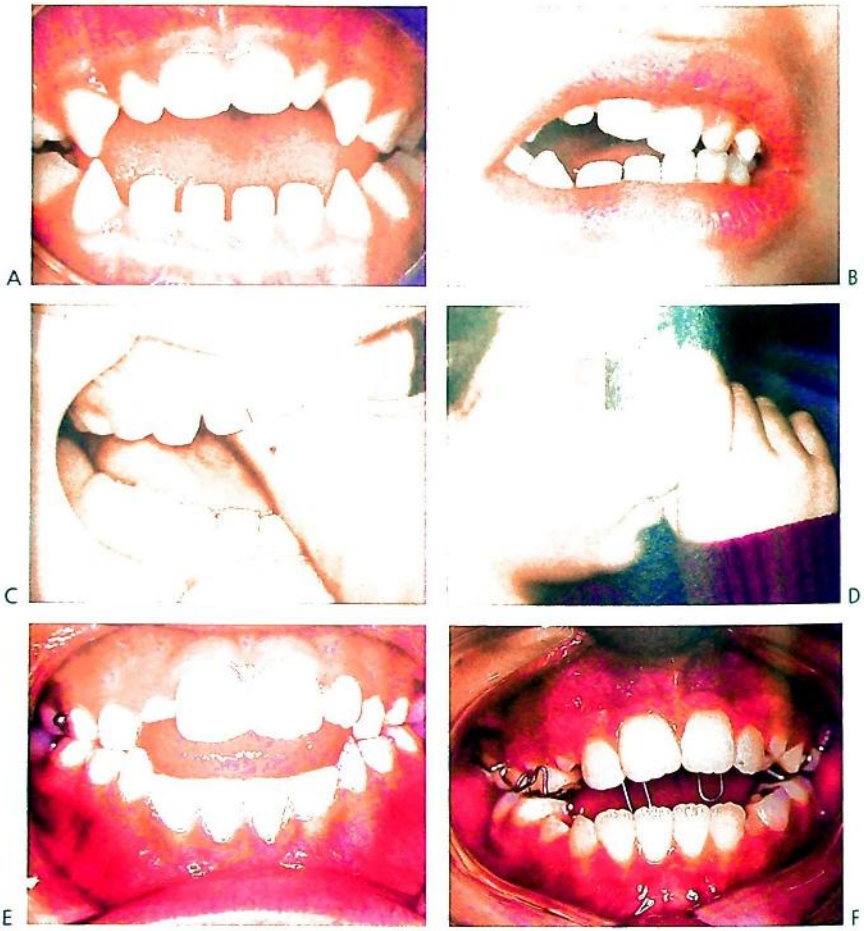

Figure 14.20 (A) An anterior open bite in the primary dentition caused by a dummy-sucking habit. Even a malocclusion of this size will improve once the habit has stopped and requires no active treatment. Note that the anterior teeth have been restored, due to early childhood caries associated with the dummy and a bottle at night. (B) Lateral open bite caused by use of a pacifier. (C) Position of a thumb exerting orthopaedic as well as orthodontic forces. (D) Note the abnormal activity of the perioral musculature due to thumb sucking. (E) Resultant proclination of the upper anterior teeth, anterior open bite and abnormal tongue position in the mixed dentition. (F) Tongue guard appliance incorporating a mid-palatal screw expander.

- A Hawley appliance with a palatal bar may be fitted as a habit reminder. This is important because in many instances thumb- and finger-sucking habits are at the subconscious level of the individual's attention. Even though there may be some desire on the part of the child to discontinue the act, they may find it difficult to do so unless made aware of when they are doing it.
- A fixed appliance consisting of bands on the first molars and an anterior tongue crib will ensure compliance, as the child cannot remove it (Figure 14.20F).
- Often the child will respond to simple encouragement and explanation of the effect of digit-sucking on the teeth. The child's own desire to break the habit means they react positively to such encouragement.
- The critical time for the elimination of digit-sucking is as the permanent incisors erupt. This generally coincides with entry into school, where peer pressure can be a powerful inducement to discontinue the habit.
- Psychological assessment is often beneficial in older children.

Mouth breathing

Mouth breathing is often associated with recurrent throat infections and nasal blockade. Obstructive mouth breathing can also be associated with severe deviated nasal septum or adenoids. Mouth breathing habit results in vertical growth pattern of the face, narrow maxillary arch, dryness of mouth causing gingivitis around maxillary incisors proclined maxillary incisors and inability to close lips. These facial features have been called 'adenoid facies'. Timely correction of cause can facilitate oral breathing, which can improve growth pattern of the face. Should the mouth-breathing patient be considered for orthodontics, it should be initiated only after appropriate consultation with an ENT surgeon.

Tongue thrusting

Infantile swallowing habits should change to mature swallowing patterns with the eruption of teeth and the establishment of the occlusion that helps to contain the tongue in the oral cavity lingual to the dental arches during swallowing. 'Teeth-apart' swallowing is called a tongue thrust that can lead to open bite. Tongue thrust habits can also be the outcome of clinical malocclusion such as skeletal open bite. Habit breaking appliances in the form of cribs/rakes and swallowing exercises can help restore normal swallowing. A persistent tongue thrust habit can also cause relapse of treated cases of malocclusion leading to spacing/open bite and may require orthognathic surgery to close the open bite.

Correction of developing Class II skeletal malocclusions

Developing Class II skeletal malocclusions may benefit by the use of functional appliances. Functional appliances are those that alter the abnormal functioning of orofacial musculature, thereby bringing about normalization of growth and occlusion (Figure 14.21).

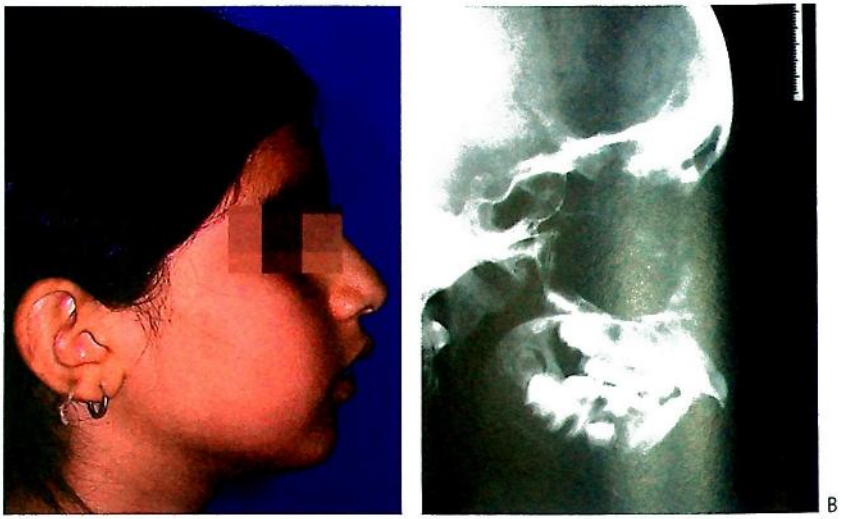

Figure 14.21 (A) An ideal case for treatment using a functional appliance: a child with a normal maxilla, a small mandible and a normal growth pattern. (B) Lateral cephalogram showing proclination of the upper incisors.

- By using functional appliances, there is an expectation of changes in the facial skeleton by growth modification.
- The simplest functional appliance is the anterior biteplane that can reposition the mandible more anteriorly in a growing child.
- Functional appliances can be fixed or removable.
- Functional appliances can be classified as tooth-borne (active or passive) and tissue-borne, depending upon the structures from which they derive anchorage. All removable functional appliances are tooth-borne, except the Frankel appliance, which is tissue-borne.
- Functional appliances help posture the mandible forward. The degree/amount of vertical and sagittal repositioning may cause variable tissue (muscle) responses.
- Those appliances that displace the mandible within the freeway space are intended to stimulate muscle activity and are called myodynamic appliances.
- Others can cause displacement of the mandible beyond freeway space and rely on passive muscle tension, and are called myotonic appliances.

Indications and case selection for functional appliances

- A growing child preferably during or prior to peak pubertal growth spurt.
- Retrognathic small mandible.
- Large overjet (>5 mm).
- Horizontal or normal growth trends.

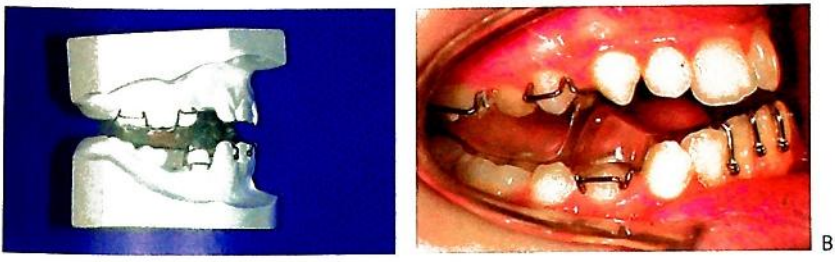

Figure 14.22 (A,B) Clark's twin block appliance.

- Normal or low incisor mandibular plane angle.
- Maxillary incisors not placed forward but can be proclined.
- No or minimal crowding in the upper and lower anterior teeth.
- A willing and cooperative child.

Effects of functional appliances

- Regulate the function of the oral/perioral musculature such as abnormal swallowing and thereby promote normal development.
- Mandible is repositioned forward.
- Remodelling of the glenoid fossa takes place.
- Retraction of maxillary anterior teeth.
- Bite opening and levelling of deep curve of Spee takes place with supra-eruption of lower posterior teeth.
- Mesial movement of lower buccal teeth and restraint or distalization of upper buccal teeth.
- Increase in lower posterior face height.
- Reduction in overjet.
- Proclination of the lower anterior teeth.
- Increase in maxillary arch width.
- Improvement in facial profile and lip seal.

Clark's twin block appliance

The twin block appliance is a two-piece functional appliance (Figure 14.22). The upper and lower blocks are made of acrylic and meet each other in the premolar region at an angle of about 70°. This is sufficient to maintain mandibular forward posturing. A child can speak, eat and live with the appliance in place. Being the only full-time functional appliance, it is expected to bring about rapid skeletal, dental and neuromuscular adaptations.

The upper twin block can house an expansion screw as well as springs for individual minor tooth/teeth movement. Hence, while mandibular repositioning is in progress, simultaneous expansion of the maxilla and alignment of minor tooth malpositions can take place, thereby eliminating the need for pre-functional phase treatment, resulting in an overall shorter treatment time.

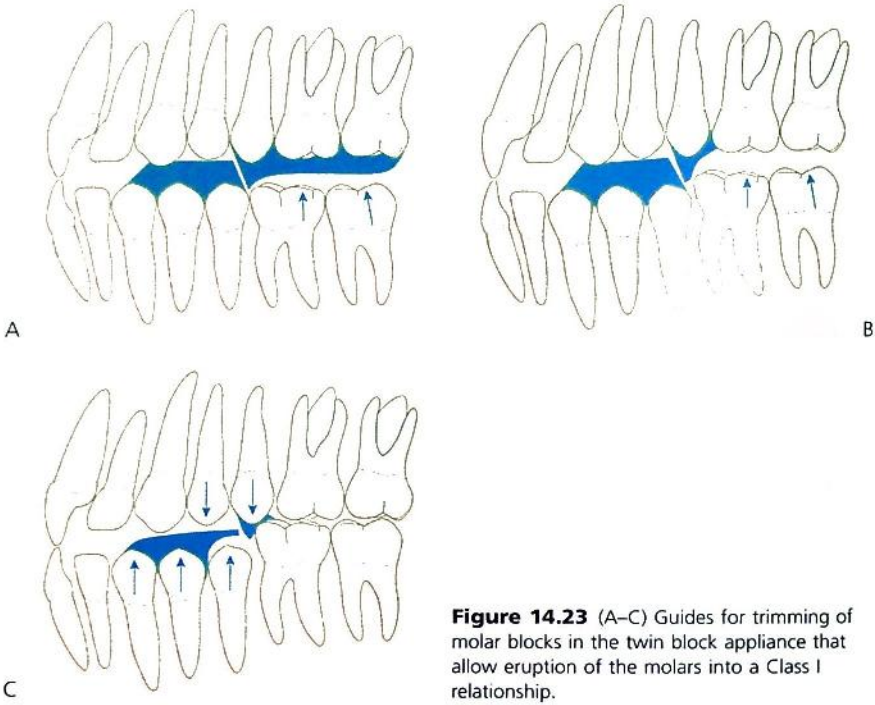

Figure 14.23 (A–C) Guides for trimming of molar blocks in the twin block appliance that allow eruption of the molars into a Class I relationship.

The twin block appliance offers flexibility of use with fixed appliances that may be required for finishing and detailing in the second phase of occlusal settlement. Hence, the total treatment time could be even shorter.

Treatment sequence (Figures 14.23, 14.24)

1. Case selection and treatment planning
   - Prepare complete records, i.e. study models, clinical photographs and X-rays.
   - Phase out the need for any major pre-functional orthodontic treatment or the possibility of simultaneous tooth movement with active phase.
   - Record bite – advance mandible to edge-to-edge position for bite registration.
2. Active phase
   - Appliance issue and follow-up.
   - Acrylic trimming for selective eruption of teeth for bite opening and sagittal molar correction.
3. Support phase
   - The modified acrylic biteplate holds the mandible forward.
4. Finishing and detailing with fixed appliances.

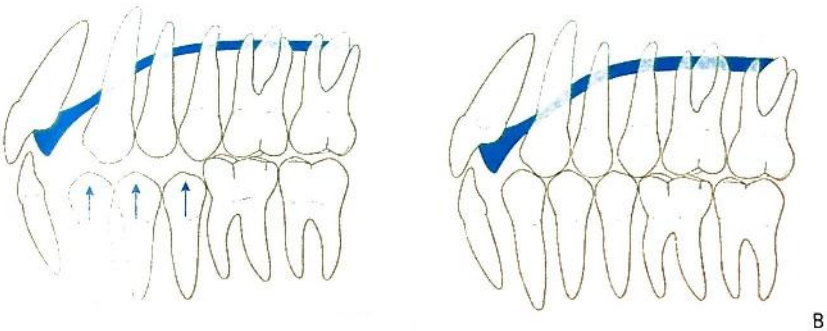

Figure 14.24 (A,B) The modified biteplate that maintains mandibular repositioning and allows settling of the buccal segments.

Instructions for patients wearing Clark's twin block appliance

- The appliance should be worn 24 h/day, except when cleaning and sports, including swimming.
- The key to success is eating with the appliance, as this enhances the adaptation of the muscles and ligaments of the temporomandibular joints as the mandible is protrusion.
- The usual time of treatment is 12 months, and this is usually followed by a second phase of treatment to detail the interdigitation of the upper and lower teeth.

Summary

Orthodontic assessment of a child requires a careful and detailed clinical examination. A child whose growth is yet to be completed and whose dentition is in a state of flux with several teeth shedding or erupting, needs to be diagnosed as having a state of normal/potentially abnormal or abnormal occlusion. This may require additional diagnostic records to be made, including input from a specialist orthodontist.

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15 Management of cleft lip and palate

Nicky Kilpatrick

Introduction

Epidemiology

Clefts of the lip and cleft palate are fusion disorders that affect the midfacial skeleton. They are one of the most common congenital anomalies with a worldwide incidence of between 0.8 and 2.7 cases per 1000 live births. The incidence of clefting varies by region, gender, ethnicity and maternal characteristics. Racial differences are apparent with Native Americans having the highest incidence (followed by Maoris, Chinese, Anglo Saxons) and African-Americans the lowest.

Aetiology

Despite being common, the aetiology of many clefts of the lip/palate remains obscure. In addition to chromosomal abnormalities and teratogenic effects, there are over 500 recognized Mendelian syndromes associated with a cleft of the lip/palate. In many instances, specific genetic mutations have been identified, e.g. van de Woude syndrome (*IRF6*) and Treacher Collins (*TCOF1*). However, over 70% of clefts of the lip and palate and up to 50% of clefts of the palate only are apparently isolated anomalies. The aetiology of these non-syndromic forms of cleft is multifactorial and the relative genetic and environmental contributions remain unclear. Consequently, while it is understood that offspring and siblings of individuals with a cleft have a higher risk of having a cleft themselves this lack of clarity makes it difficult to give families accurate information regarding the recurrence risks or to offer informed genetic counselling.

Embryology

Orofacial clefts result from a failure of complete fusion between any of the independently derived facial primordia that form the orofacial complex. Development of the human face begins around day 22 with the migration of cranial neural crest cells (ectomesenchyme) from the dorsal region of the anterior neural tube into the facial region. On either side of the foregut, ectodermal swellings form five pairs of branchial (pharyngeal) arches that are derived from primitive gill or visceral arches of fish. These facial primordial establish during the 4th week of gestation, consisting of five distinct prominences:

- A pair of mandibular prominences develop into the lower jaw.
- An unpaired frontonasal prominence that gives rise to a pair of lateral and medial nasal processes during the 5th week. During the following 2 weeks, the medial nasal processes enlarge and merge with each other and the flanking maxillary prominences, completing the formation of the upper lip.
- A pair of bilateral maxillary prominences that result in the upper jaw.

The secondary palate protrudes as a medial outgrowth from the maxillary prominences and consists of neural crest-derived mesenchymal cells. The shelves grow vertically (downwards) initially from the bilateral maxillary processes during the 6th week. Growth of the embryo causes rotation and extension of the head, dropping the tongue that was lying in the nasal cavity into the mouth (stomodeum). At the same time, the palatal shelves elevate to the horizontal position above the tongue during the 7th week of gestation. Following elevation, the palatal shelves continue to grow towards each other during the 8th week until the epithelial surfaces covering the opposing palatal shelves meet and fuse at the midline to form the secondary palate. The epithelium breaks down allowing mesenchymal consolidation.

Events that disrupt the fusion may result in clefting:

- Clefts of the primary palate (i.e. lip, alveolus and palate anterior to the incisive foramen) are caused by disruption of fusion of the medial nasal processes and maxillary prominences around Week 6
- Clefts of the secondary palate (i.e. hard and/or soft palate) are caused by disruption of the elevation and/or fusion of the palatal shelves around week 9.

Anatomy

Clefts of the lip and palate may be unilateral or bilateral, complete or incomplete. The presentation of clefting conditions can be categorized into the following groups:

Clefts of the lip and alveolus (primary palate)

The primary palate forms anterior to the incisive foramen. A cleft of the primary palate may vary from an incomplete cleft (forme fruste) to a minimal defect involving just the vermilion border, to a complete defect extending from the vermilion border to the floor of the nose with clefting of the alveolus. Even in the absence of a frank cleft of the bony alveolus there may be evidence of clefting in the form of a dental anomaly such as a missing or microdont lateral incisor (see Chapter 11). Clefts of the primary palate may be unilateral or bilateral.

Cleft lip and palate (CL/P)

Varying degrees of clefting of the lip and palate can also exist; complete to incomplete, in a wide range of combinations; uni- and bilateral, symmetrical and asymmetrical. In complete clefts, a direct communication exists between the oral and nasal cavities on the cleft side (Figure 15.1A). There can be substantial variation in the degree of palatal shelf separation and while the maxillary arch form generally appears normal at birth, medial collapse of the maxillary segments occurs soon after.

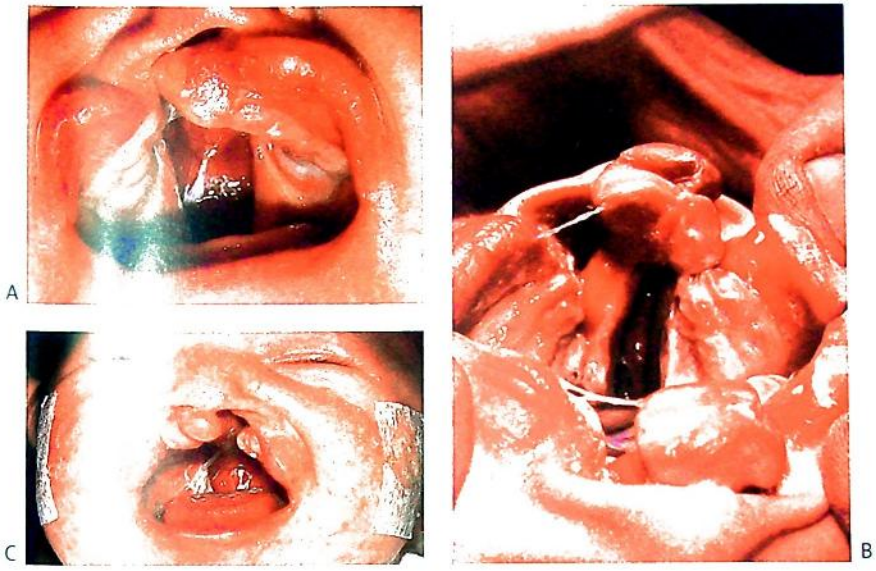


Figure 15.1 (A) Unilateral complete cleft lip and palate, showing the extent of the malformation in the palate. (B) Bilateral complete cleft lip and palate. The premaxillary segment is clearly visible as an extension of the nasal septum. The central incisors are contained within this process. (C) Anterior view of a child with a bilateral complete cleft of the lip and palate. The columella and philtrum are extremely short and there is a wide defect between the segments.

Bilateral clefts of the lip and palate can be either symmetrical or asymmetrical. In the bilateral complete cleft lip and palate, both nasal chambers are in direct communication with the oral cavity. The palatal processes are divided into two equal parts and the turbinates are clearly visible within both nasal cavities. The nasal septum forms a midline structure that is firmly attached to the base of the skull but is fairly mobile anteriorly, where it supports the premaxilla and columella (Figure 15.1B). In these clefts, the premaxilla protrudes considerably forward of the facial profile and is attached to a stalk-like vomer and the nasal septum. The 'lip' component of the medial segment contains only collagenous connective tissue. It is, therefore, grossly deficient in bulk and lacks the features normally produced by muscle (Figure 15.1C).

Cleft palate

A cleft of the palate may involve only the soft palate, both soft and hard palates, but almost never the hard palate alone. Deficiency of mucosa and bone are the main features of clefts of the hard palate (Figure 15.2). The cleft may extend forward from the uvula to varying degrees, from a bifid uvula (Figure 15.3) to a 'V'-shaped cleft extending through the hard palate to the incisive foramen. Some babies have a

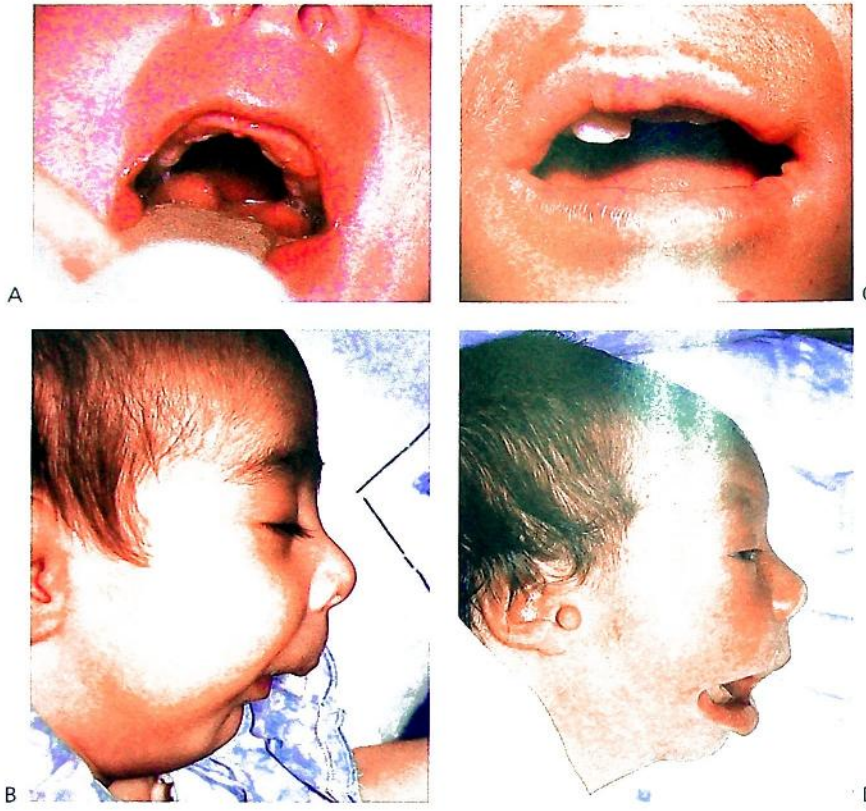


Figure 15.2 (A) Cleft of the palate with an intact lip and alveolus. This U-shaped cleft was associated with the Robin sequence and resulted from a failure of embryonic head rotation. This maintained the tongue in the oral cavity and the palatal shelves subsequently formed around the tongue, giving rise to the characteristic cleft shape. (B) Another manifestation of this condition is extreme micrognathia. The Robin sequence may also be associated with transverse clefts of the face. These transverse clefts may be rather subtle as demonstrated in (C), or severe as in (D). This latter patient has a first and second branchial arch deformity associated with Goldenhar's syndrome. Note the preauricular skin tag.

'U'-shaped cleft of the palate that is described in 'Robin sequence' (Figure 15.2). Robin sequence is characterized by upper airway problems associated with a very small mandible and glossoptosis and is thought to be secondary to mandibular hypoplasia in the 1st trimester, which causes the tongue to sit high in the mouth and prevent fusion of the palatal shelves.

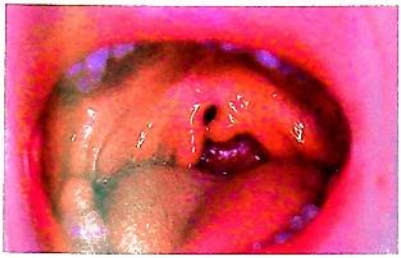


Figure 15.3 A bifid uvula associated with a submucous cleft palate. The fibres of tensor palati are not joined, although the epithelium is intact. These children may present with nasal air escape due to a shortened palate and velopharyngeal incompetence. There is often a notch felt at the posterior border of the hard palate and a bluish median line extends to the uvula.

Submucous cleft palate

Clefting of the velum or soft palate can be 'submucous' where the mucous membrane remains intact, despite clefting of the underlying musculature. Submucous clefts of the palate occur in around 1 in 1200 live births, with only half of the cases having clinically significant symptoms.

Diagnosis

Clefting disorders are now increasingly diagnosed prenatally as part of routine ultrasound screening between 18 and 20 weeks *in utero*. The reported diagnostic accuracy of routine 2D ultrasonography in a low risk population varies between 9–100% for complete clefts of lip and palate but is much lower (0–22%) for clefts of the palate only (Maarse et al 2010). Much higher accuracy is achieved using 3D ultrasound but again isolated clefts of the palate often remain undetected. Prenatal screening provides knowledge of the impending birth of a baby with a cleft, which allows both the family and appropriate clinicians to prepare for this event.

Management of individuals with clefts of the lip and/or palate

Although surgery can correct many of the structural defects, affected individuals can still face a lifetime of functional, social and aesthetic challenges. In addition, there is growing evidence that individuals with CL/P may also have subtle neuropsychological and developmental deficits, which are associated with learning difficulties and other educational challenges. The care of these infants often starts antenatally and continues from birth through to adulthood and involves a large multidisciplinary team of clinicians. The specialties involved in this team vary greatly but usually include:

- Plastic surgery.
- ENT (otolaryngology) and/or maxillofacial surgery.
- Speech pathology and audiology.
- Orthodontics.
- Paediatric dentistry.
- Paediatrics.
- Psychology.
- Specialist nursing and social services.

In addition, genetics, ophthalmology, neurosurgery, periodontics and implantology may be involved at different times. Such orofacial clefting places enormous burdens, both financial and psychosocial on, not just the individual themselves, but also their family and society more broadly.

While there is a lot of variation (and very little high-level evidence) in the sequencing and timing of treatment and the clinical techniques and interventions used, there are some commonly accepted aims and principles of treatment. The appropriate specialists (usually a specialist nurse and/or primary surgeon) within a cleft team aim to provide a rapid initial evaluation, often within hours of birth. Subsequent regular contact through either home visits or attendance at specialist cleft review clinics ensures that families are supported appropriately and social and psychological issues identified and resolved early. Treatment plans can then be formulated and implemented collaboratively by the multidisciplinary team in partnership with the families.

The goals of the treatment of a child with a cleft of the lip and/or palate are to restore both form (appearance) and function (especially speech and mastication), while optimizing an individual's general health and well-being.

Management in the neonatal period

Feeding

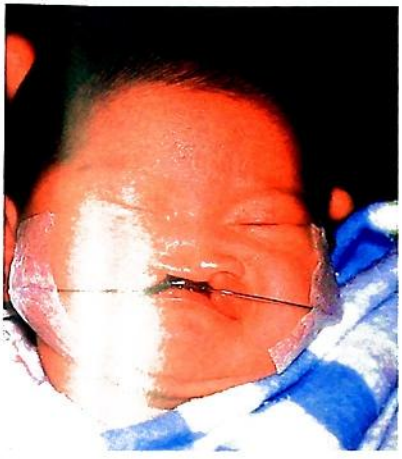
Efficient feeding is important for growth and development in infancy. A baby with a cleft of any kind may experience feeding difficulties. However, babies with cleft palate or combined cleft lip and palate usually have more problems than those with cleft lip. A cleft palate prevents the baby from sealing the oral cavity and generating the negative pressure necessary for efficient feeding. Babies with large clefts of the lip and palate may also have difficulty generating positive pressure (compression), which is also important for feeding. The early involvement of specialist nurses with experience of cleft care and in particular the feeding problems associated with clefting is essential. While there is no doubt that breast-feeding is best for babies and should be encouraged, it is usually unsuccessful for infants with a cleft of the lip and palate or palate alone, therefore bottle feeding (preferably using expressed breast milk) is often the most appropriate approach. Once a child is feeding well, and is progressively gaining weight, they will be able to cope with surgery.

Presurgical orthopaedics (Figure 15.4)

Presurgical orthopaedics (PSO) takes advantage of the plasticity of the nasal cartilage in the neonate by using orthodontic-like plates, with or without tapes and nasal stents to reposition the maxillary segments and the nasal structures. PSO needs to be started as soon after birth as possible to maximize the chances of an infant tolerating the appliances. It is technically complex and demanding, not least because taking a maxillary impression in a neonate is particularly challenging with the associated risks to the airway. PSO should only be done by appropriately trained and experienced clinicians. Its use remains controversial, not least because of the associated financial burden coupled with the lack of strong evidence of effectiveness.

Counselling and parent support

Careful counselling by an appropriately informed cleft specialist is often needed by parents. In many cleft teams, this is provided by the specialist cleft nurses who may



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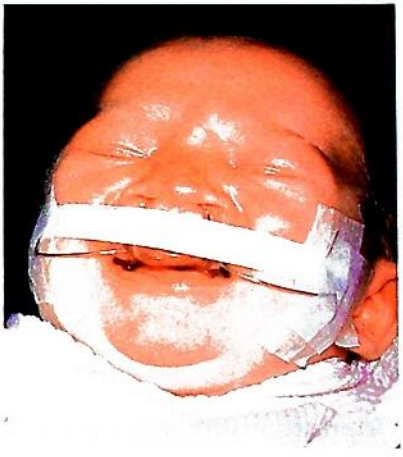


B

Figure 15.4 (A) Presurgical orthopaedic appliance. The plate is held by micropore tape to the cheeks. (B) Strapping aids in the positioning of the labial segments, especially in cases of bilateral complete clefts. Depending on the institution, these appliances are used in cases of bilateral cleft lip and those with very wide unilateral clefts as shown in (A).

also carry out home visits to provide additional support. Parents not only have many questions but are also often confused and anxious and may seek advice about the intermediate and long-term implications of the cleft defect and the necessary surgery. They also often appreciate, and benefit from, the assistance available from members of a parent support group such as CleftPALs (Australia), CLAPA (UK) and American Cleft Palate-Craniofacial Association.

Primary surgery

Many surgical techniques have been described for the primary closure of cleft lip and palate. However, there is still controversy regarding the timing of surgery and which is the most reliable technique consistent with ensuring optimal growth of the face and development of speech.

- **Lip repair** is less controversial than is palate repair and is generally undertaken around 10–12 weeks and almost certainly by 6 months of age, provided the infant is otherwise developing well. The aim of the lip repair is to restore the continuity of the orbicularis oris muscle of the lip, and with it, the appearance of the upper lip (Figure 15.5).
- **Palate repair** aims to reconstruct the abnormally inserted musculature of the soft palate to normalize movements of the soft palate and permit the development of normal speech. The extent and timing of palatal surgery is one of the major and continuing controversies in cleft management. This relates to the perceived balance between the benefits of good speech development (which is promoted by early closure) versus the deleterious effects on midfacial growth through surgical trauma and associated scarring (which can be minimized by delaying the surgery).

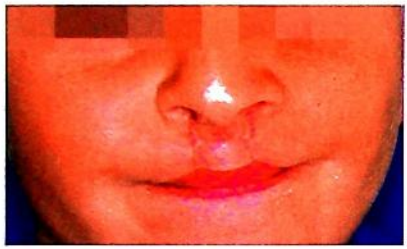


Figure 15.5 Surgical repair of a bilateral complete cleft lip with columella lengthening. The alar base is symmetrical, although there is an accentuation of the cupid's bow and eversion of the vermillion border.

To date, there is no strong evidence regarding the best approach to primary surgery. Attempts to address this gap in the evidence are being made through large multi-centre international clinical trials.

Management during childhood

Speech and language and ENT problems

Children with clefts and other craniofacial malformations are at an increased risk of speech and language difficulties. Regular assessments are required to monitor the speech and language acquisition process, to assist in making decisions about the need for either further surgery or speech therapy or both.

During childhood, care needs to be exercised to ensure hearing is optimal for speech and language development. Otitis media is common in infants and children with cleft palate and some may also have sensorineural loss. Suboptimal hearing can affect speech and language development so regular audiology assessments for all infants with clefts of the lip and palate is important.

Velopharyngeal inadequacy (VPI)

Children with clefts are at increased risk of VPI. Normal speech requires that the muscles that make up the velopharyngeal sphincter (predominantly the muscles of the soft palate and nasopharynx) work in a coordinated fashion. Defects in any aspect of the nasopharyngeal anatomy and/or physiology may lead to VPI. It is uncommon for otherwise healthy individuals to have VPI. However, for individuals with anomalies of their velopharyngeal sphincter such as a cleft of the palate (hard and/or soft, repaired or unrepaired) VPI can, if severe, compromise speech production rendering a child incomprehensible to others.

- The speech pathologist's assessment of speech production is critical in the identification and management of VPI and they work closely with the primary cleft surgeons in deciding what, if any, intervention is required to optimize speech.
- If significant VPI is identified, then surgery is usually needed. There are a number of different approaches to the surgical management of VPI, including narrowing the velopharyngeal opening (pharyngoplasty), lengthening the soft palate (intravelar veloplasty) and bulking up the posterior pharyngeal wall (fat augmentation). Although surgery can be expected to improve structural obstacles which hinder the development of normal speech, postoperative speech therapy may still be needed

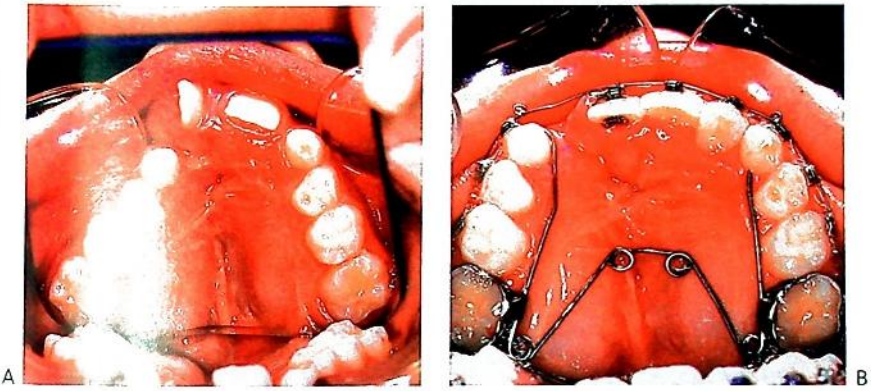


Figure 15.6 (A) One of the effects of early surgery and scarring is collapse of the palatal segments due to an inhibition of growth. Rotation of the central incisor adjacent the cleft can also be seen. (B) Palatal expansion with a quad helix appliance to correct the posterior cross-bite and open space for the bone graft.

(before and after surgery) to correct poor speech habits that have developed because of the structural malformation.

- Occasionally, surgical correction of VPI is not possible or does not bring resolution. In this case, a palatal obturator or a speech bulb may reduce the velopharyngeal space sufficiently for normal speech to develop.

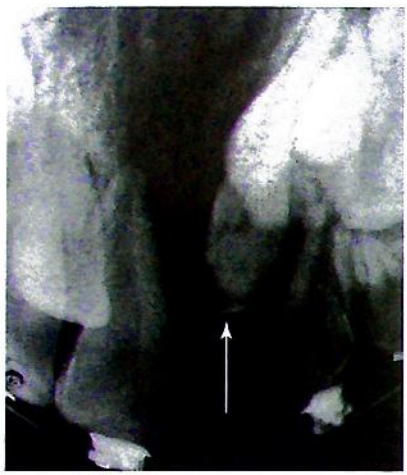
Orthodontics

Children with clefts of the lip and palate and those with cleft of the hard-palate are at risk of compromised growth of the maxilla as a result of post-surgical scarring. If severe, the growth restriction of the maxilla which affects all three dimensions can lead to the development of a skeletal class III relationship and an associated buccal crossbite. Individuals with complete clefts of the lip and palate may require extensive orthodontic treatment.

The first comprehensive orthodontic evaluation is generally made around the age of 8 years of age although occasionally, there may be a need for earlier review. Orthodontic management at this age can be subdivided into three main components:

Mixed dentition treatment (Figure 15.6)

- Interceptive treatment such as minor tooth alignment to facilitate anterior alignment or correction of an anterior incisal cross-bite.
- Palatal expansion prior to alveolar bone grafting comprises a relatively short period of fixed appliance therapy aimed at achieving an optimal arch shape to improve access for surgery. Bone grafting is usually undertaken when the cleft-side permanent canine (cuspid) tooth shows between half and two-thirds of root development (Figure 15.7). Where necessary, pre-bone graft expansion is commenced



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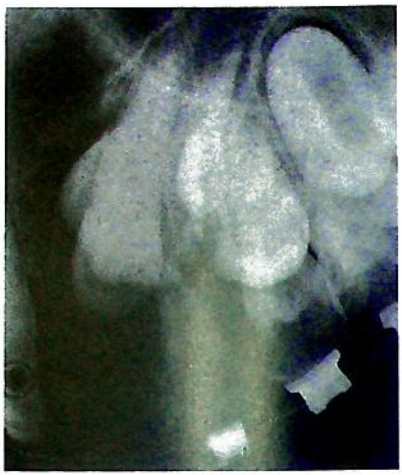

B

Figure 15.7 (A) Periapical radiograph of a cleft prior to bone grafting. The lateral incisor is present on the palatal aspect of the canine tooth. A small supernumerary tooth (arrowed) is also present, and was removed at the time of surgery. (B) The cleft has been filled with cancellous bone, harvested from the iliac crest. After 3 months, there is already movement of the lateral and canine and both these teeth erupted through the graft to the mouth, where they were aligned orthodontically.

approximately 12 months prior to surgery and is completed using an upper fixed appliance (Figure 15.6B). The appliance may then be maintained for 4–6 months post-graft, for stability.

Secondary alveolar bone grafting (Figure 15.7)
Alveolar bone grafting should be timed to precede the eruption of the permanent canine tooth on the cleft side, which usually occurs at approximately 11 years of age. The aim of bone grafting is to:

- restore the bony contour of the alveolus.
- stabilize the maxillary expansion.
- provide a bony matrix through which teeth (especially the canine) may erupt.
- allow the teeth to have a healthy supporting periodontium.

Surgery

- Presurgical palatal expansion (if required) with fixed appliances such as a quad helix (Figure 15.6B).
- Preparation of recipient area may be prepared by extraction of retained primary teeth several weeks prior to surgery.
- Bone harvest for the graft is usually autologous iliac crest cortico-cancellous bone, but may be obtained from the mandibular symphysis, tibia, rib, or calvarium.

Cleft management in adolescence and early adulthood

Orthodontics

Full permanent dentition correction

In the mixed dentition, very little active orthodontic treatment if any, is done, leaving the bulk of the orthodontic treatment to be carried out in the shortest possible time, when the full permanent dentition is present. Once the full permanent dentition is established (excluding the third molars), final correction of the malocclusion and tooth alignment is undertaken. The reasons for postponing the definitive orthodontic management to mid to late adolescence include:

- In the event of a significant skeletal discrepancy orthognathic surgery may be required in conjunction with orthodontic treatment the planning for which cannot occur until growth is complete.
- At 12–16 years dental suitability and motivation to cooperate with orthodontic treatment is assessed prior to the commencement of full fixed appliance treatment.
- Rapid palatal expansion and/or fixed appliance orthodontic treatment is undertaken at the optimum time – occasionally elective extraction of teeth for orthodontic reasons is indicated.

Orthognathic surgery

If a maxillary/mandibular skeletal discrepancy exists at the time of physical maturity, an osteotomy may be required along with any further soft-tissue revision which is also undertaken at the same time, if needed.

If necessary, further aesthetic surgery such as a rhinoplasty can be undertaken as a final surgical procedure. In some instances, functional lip and nose revisions are combined with alveolar bone grafting at an earlier stage to reduce the impact of the aesthetic deformity on the growing child but in general, such procedures are postponed until growth has ceased and orthodontic therapy completed.

The above procedures are only some of the many that may be needed by a patient with cleft lip and palate.

Importance of dental care in overall management

Children with cleft conditions require extensive interdisciplinary care throughout early life and the highest standard of oral health must be maintained because the presence of dental disease can severely compromise both surgical and orthodontic success. The paediatric dentist plays an essential role in coordinating the oral healthcare for these individuals, in order to ensure they reach adulthood with a healthy dentition and positive attitude to dental care. The paediatric dentist will generally coordinate all dental care aspects with those of other disciplines and as such, it is important for them, or their representative, to regularly review the child with a cleft lip and palate.

Figure 15.8 A tooth erupting in the cleft. Such teeth often become carious, but should be repaired and retained until bone grafting is performed. Where permanent teeth are congenitally absent, especially the lateral incisor, the supernumerary may be used if of adequate size.

The first appointment

Many specialist paediatric dental academies support the notion that all infants should be seen by a dentist within 6 months of eruption of the first tooth (i.e. before the age of 1 year). This is even more important in infants born with a cleft.

The purpose of this initial consultation is to explain:

- The dental aspects of the clefting process.
- The likely course of dental management. The involvement of different specialties including restorative, radiological, orthodontic and possible later oral surgical care.
- The probability of the absence of the normal tooth in the region of the cleft, and conversely, the possibility of one or more supernumerary teeth in the cleft region should be mentioned (Figure 15.8).
- The likelihood of the presence of crown and/or root morphological abnormalities and enamel hypoplasia of the incisor and canine teeth adjacent to the cleft should be indicated, with the positive reassurance that these can be treated relatively simply soon after they appear.
- The absolute importance of sound preventive care and regular dental visits should be emphasized.

It will almost certainly be necessary to reinforce the introductory information and advice given at this visit at many subsequent outpatient visits over the years of treatment. Many cleft palate clinics produce parent handbooks that are particularly useful.

Babies born with a cleft of the lip and palate not uncommonly have a tooth present at, or erupting soon after, birth – a natal tooth. This can worry parents and may contribute to the existing anxiety around feeding. Such teeth often sit in the premaxillary region and may be very mobile. In most instances, they can be extracted simply using topical anaesthetic cream. Care should be taken to protect the airway from the extracted tooth.

Preventive dental care

Children with clefts of the lip and/or palate may be at increased risk of developing dental caries for a number of reasons, however the evidence is fairly weak. Reasons include:

- PSO (if carried out) may predispose infant oral cavity to colonization by *mutans streptococci* and hence, to the early development of dental caries.
- Early infant feeding problems may lead to prolonged and more cariogenic feeding habits.
- Presence of developmental defects of enamel particularly around the cleft site.
- Presence of dental crowding, malocclusion and orthodontic appliances.
- Existence of other comorbidities, which may increase caries risk, e.g. reduced salivary flow associated with velocardiofacial syndrome.
- Risk of being 'spoilt' with sugary treats to compensate for hospitalizations and other healthcare exposures.

Preventive dental care is essential for these patients, using all known techniques including:

- Early and regular exposure to a fluoridated toothpaste.
- Oral hygiene technique instruction – for parents and later for the individuals themselves.
- Home application of topical fluoride agents.
- Fissure sealing of both primary and permanent teeth.
- Dietary advice to child and parents by paediatric dentist (or dietitian if necessary).
- Appropriate use of bitewing radiographs (in-line with national standards) to ensure early identification and treatment of carious lesions.

The prevention of dental caries and periodontal disease will help with cooperation for ultimate definitive orthodontic treatment, by reducing unpleasant visits for treatment in early childhood. Motivation is especially important for later orthodontic treatment in these patients. This should be assessed early, and enhanced during preventive visits during childhood, so as to ensure compliance.

In many countries, it is the role of the general dental practitioner (GDP) to provide routine dental care to all children. Despite being at increased risk of dental disease, there is no reason why children with craniofacial anomalies cannot receive such care from their local GDP. In many cases, this will be geographically more convenient. However, it is important that the GDP establishes early contact with the cleft palate team or surgeon and maintains a frequent dialogue.

Dental extractions and minor oral surgery

- Except in an emergency, dental extractions should not be performed for these children by the general dentist without first checking and clearing this with the supervising paediatric dentist or orthodontist.
- Primary molars should be retained by pulpotomy, or the space maintained after extraction as advised by the paediatric dentist.
- Erupted supernumerary teeth should be retained until 6–7 years of age, unless impossible to clean, resulting in progressive dental caries, gingival or mucosal inflammation.

Extraction of such teeth should then, in most cases, be carried out in the supervising hospital under either local or general anaesthetic. If a general anaesthetic is required for restorative treatment or other purpose, superficial or obstructing unerupted

supernumerary teeth may be removed at the same time but only after discussion with the coordinating paediatric dentist. If a bone graft is planned for the alveolar cleft, it may be necessary to retain primary teeth that are adjacent to the graft site. This, if done 3–4 weeks prior to the grafting procedure, ensures that the healing of the bone graft site is not compromised by leaving a deficiency where the tooth has been extracted at the time of grafting. If the child's behaviour does not permit the extraction of teeth prior to grafting, then they may be removed by the maxillofacial or plastic surgeon at the time of bone grafting.

Dental anomalies (see also Chapter 11)

Dental anomalies are extremely common in children with orofacial clefting. The most commonly affected tooth is the maxillary lateral incisor on the cleft side. This is due, in part, to disruption of the dental lamina. Anomalies may include:

- Agenesis of teeth.
- Supernumerary teeth.
- Concurrent agenesis and supernumerary teeth within or adjacent to the cleft.
- Disorders of morphogenesis (size and shape).
- Supernumerary teeth may occur in either the medial or distal segment and much less frequently in both segments (Figure 15.8).

Cosmetic restoration of malformed anterior teeth and alveolar cleft

The appearance of the teeth can be improved at any time, depending on the child's perceptions and wishes.

- Composite resin restorations may be placed for hypoplastic or morphologically abnormal permanent incisor teeth adjacent to the cleft(s) soon after eruption, however, enamel-bonded crowns or veneers should be reserved until after passive eruption and establishment of the gingival margin at the cementoenamel junction.
- As a temporary measure, and in suitable cases, an adhesive retained bridge may be placed to replace missing incisor teeth. This form of prosthesis is a superior alternative to a removable partial denture, however an osseointegrated implant remains the ultimate treatment solution when space cannot be closed orthodontically.

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16 Speech, language and swallowing

Sarah Starr

Introduction

The ability to communicate effectively is vital to a person's functioning in society. Speech and language acquisition is a developmental process occurring most dramatically in the first years of life but one that proceeds throughout a person's lifetime. Difficulties may be encountered at any point during the language acquisition process. Children may experience problems acquiring the sounds of the language, learning how to combine words meaningfully or comprehending others' questions and instructions. In all cases, a speech and language pathologist is the primary healthcare professional responsible for the identification and treatment of individuals with communication problems. Paediatric dentists should be aware of the symptoms and problems associated with communication impairment, particularly when it relates to orofacial or dentofacial anomalies. They should know how to refer children and their families to a speech pathologist.

Communication begins at birth and continues throughout a child's life through adolescence and into adult life. A child's communication development is influenced by many variables, including neurological status and motor development, oromotor status (anatomical and physiological), cognition, hearing, birth order, environment, communication modelling and experiences, as well as their personality.

This chapter will briefly describe some of the main communication disorders that can present with particular focus for paediatric dentists.

Communication disorders

There are six main areas to be considered when assessing a child's communication:

- Oral motor and feeding problems.
- Articulation.
- Language.
- Voice.
- Fluency.
- Pragmatics.

Oral motor and feeding problems

Problems in this area constitute the earliest at which children are referred to a speech pathologist. Significant problems can result when an infant does not develop control

of the oral mechanism sufficient for successful feeding. Early reflex development typically facilitates feeding behaviour, but neuromotor factors, prematurity, cleft lip and palate, long-term non-oral feeding and other reasons may interfere with a child's development of the movement patterns essential for sucking, swallowing and feeding. Since these patterns form the scaffolding of movement for early speech sound development, children with a history of feeding difficulties may have subsequent difficulties in producing sounds for speech.

Reasons for referral

- Sucking, swallowing or chewing difficulties.
- Gagging, coughing or choking with feeds.
- Moist vocal quality during or after feeds.
- Poor cough or gag reflex.
- Persistent drooling (not coincident with teething).
- Recurrent chest infections.
- Presence of a craniofacial malformation.
- Parental report of feeding difficulty or refusal.
- Poor oral intake and associated poor weight gain in infants and young children.

Articulation

Articulation refers to the production of speech sounds by modification of the breath stream using the various valves along the vocal tract: lips, tongue, teeth and palate. Problems in these areas can vary from a fairly mild distortion of sounds such as a lisp, where the child's speech is still easy to understand, through to a more severe speech production problem where all speech attempts are unintelligible or where the child makes very few speech attempts. Errors can be classified in the following ways:

- Speech sound omissions:
  - 'cu' for 'cup'.
  - 'te-y' for 'teddy'.
- Substitutions of sounds:
  - 'wed' for 'red'.
  - 'tun' for 'sun'.
- Distortions:
  - lateral lisps – 's' that sounds slushy.

Children learn to produce sounds in a developmental sequence, with adult-like sound systems expected by 8 years of age (Table 16.1). For example, it is quite acceptable for a 2-year-old to mispronounce an 's' sound, but it would be considered a problem if a similar error were made by a 7-year-old child.

Language

In contrast to the fairly straightforward examples listed above to illustrate speech sound learning, language development is much more complex. Skills emerge in two parallel levels.

Receptive language

This is the ability to understand language.

Table 16.1 Development of sounds with age

Age (years)	Sounds correctly produced	Comments
2 2½	m, n, h p, b, ng, w, d, g	Speech is sometimes difficult to understand, especially for unfamiliar people
3 3½	y, k, f, sh t, ch, dge	By 3 years of age, 80–90% of a child's speech should be easily understood
4 5	l, s, zh (measure) r	Blends of sounds (i.e. st, cl, dr) are acquired later than the individual sounds but are usually mastered by 5 years
5½ 7½ 8	z th	The ages quoted here are only a guideline as to when the average child acquires the sound, but by 8 years of age, all sounds should be mastered

Expressive language

This refers to the ability to produce verbal and non-verbal communication in the form of words and sentences and may include speech and written language.

A child with a language disorder may present with difficulties in both comprehension and expression of language or in only one area of language learning. Language learning proceeds in a predictable order but there is more variability in the emergence of these skills than the acquisition of speech sounds. Vocabulary grows as does a child's ability to progressively understand more complex language. Words are combined into phrases and eventually sentences, and comprehension becomes more adult-like over time. Eventually, language that is heard and said becomes the language of literacy, reading and writing. School success is highly correlated with language learning, especially in the early years.

Children may experience language learning problems at any stage of the acquisition process. There may be:

- Difficulties interpreting the meaning of words and gestures.
- Delays in the production of first words and phrases.
- A lack of understanding of questions and instructions.
- An inability to produce sentences that are grammatically correct.
- An inability to participate in conversations.

A delay at any single stage may not necessarily constitute a longstanding problem, although it should be investigated further. Problems with language acquisition are the most subtle indicators of difficulties with childhood development and therefore, should never be ignored.

Voice

Voice is produced when the vocal cords in the larynx are vibrated. Changes in air flow and the shape of the vocal folds can affect loudness, pitch and voice quality. Once voice is produced, its tone (resonance) and quality is modified by the throat, oral and nasal cavities. A child with a voice problem may present with the following:

Abnormal voice quality
Rough, breathy or hoarse voice in the absence of upper respiratory tract infection.

Abnormal resonance
Hypernasality (excessive nasal tone usually due to problems closing the velopharyngeal port during speech) or hyponasality (lack of nasal resonance usually due to some type of nasopharyngeal obstruction).

Inappropriate loudness levels
Voice too soft to be heard or so loud that it is distracting from the message of the speaker.

Problems with pitch
Pitch too high or low for age or sex.

Voice problems may be caused by:

- Poor vocal use, e.g. excessive yelling or screaming (in some cases producing vocal nodules).
- Neurological problems (e.g. cerebral palsy).
- Vocal pathology such as polyps or cysts.
- Muscular pathology.
- Vocal cord paralysis.
- Vocal irritants such as exposure to smoking, chemicals or aerosol sprays.
- Physical conditions including cleft palate, laryngectomy and hearing loss.

Fluency

Fluency refers to the smooth flow of speech. Where there are interruptions in the flow of speech, stuttering occurs. Many children experience brief periods of stuttering as they learn to speak in longer sentences and this early form of disfluency is not considered a disordered pattern, as it will usually pass. Early developmental disfluency is best resolved by reacting to the message the child is attempting to convey rather than the disfluency. When stuttering persists beyond the normal time period of approximately 3–6 months, and/or there is strong family history of stuttering or when it is becoming stressful for the child, referral to a speech pathologist is indicated.

A child or an adult with a stutter experiences involuntary repetition of words, prolongations of sounds in words and blocks where no sound is produced at all. Some speakers with a stutter will use words like 'um' to help them initiate speaking. Sometimes secondary features such as eye blinking and facial grimacing occur with the stutter.

Stutterers do not have more emotional or psychological problems when compared with the general population, nor is there evidence of decreased mental aptitude. Approximately 3% of the population stutter, with a predominance in males (3:1). The disorder usually has its onset in the early years of life and treatment is most successful in the preschool years. Hence, the importance of prompt and early referral.

Pragmatics

Pragmatics refers to *how* we communicate with others through our verbal and nonverbal language as well as our tone of voice, stress, pausing, pitch and loudness.

Pragmatics relates to our social use of language, including how we initiate, engage and maintain others in a communication dialogue through our eye contact, body language, physical space, choice and use of words, conversational turn taking and responses to others.

Pragmatic skills allow us to develop friendships and effectively communicate our messages in a social, educational and workplace setting. They are crucial to effective communication and develop from birth throughout our adult life. Pragmatic skills develop as part of normal cognitive development and are influenced by environmental and cultural modelling. Disorders in pragmatic skills can be isolated or part of a language, developmental, psychological or specific condition (e.g. autism).

Structural anomalies and their relationship to speech production and eating and drinking

The speech language pathologist is actively involved in the evaluation of orofacial, pharyngeal and laryngeal structures and functioning important for speech production and deglutition. Evaluation techniques may be perceptual or instrumental (e.g. videofluoroscopic X-ray studies of swallowing) and are often a combination of both, with input from other members of a multidisciplinary team. Results of diagnostic structural and/or functional testing direct which management strategies are available to the patient.

Speech sounds may be acoustically or visually distorted due to abnormal structure and/or function of the articulators (most commonly the lips, tongue, teeth and palate). There are three main ways in which speech sound production is influenced and these can coexist, thus differential diagnosis is important in decision-making for management.

1. Speech sounds may be omitted, substituted or added during early childhood, as mature speech patterns are mastered. For example, 3-year-old children often substitute alveolar sounds for velar sounds (cap → tap; go → do). Some children continue these substitutions for longer than expected and need speech therapy to learn mature speech patterns.
2. Speech sounds may be distorted due to underlying neurological impairment or oro-motor planning/coordination problems. Speech may be termed dysarthric (neurological) or dyspraxic (motor planning), depending on the aetiology and characteristics noted.
3. Structural problems may affect speech (and swallowing) in a number of ways. These are outlined below.

Dental anomalies

Malocclusion has the potential to greatly affect speech production although the ability of patients to compensate for abnormal dental relationships should never be underestimated. However, there is no definite proof that altering the position of a tooth may improve speech (Johnson & Sandy 1999).

Hypodontia/missing teeth causing interdental spacing

Chewing difficulties may result and a lateral or forward displacement of tongue during speech may occur, resulting in distortion of sounds. The presence or absence of teeth

and the position of these teeth in the dental arch is thought to be more significant for speech production than the condition, size or texture of the teeth (Shprintzen & Bardach 1995). In general, lingual-alveolar sounds (e.g. /s/, /z/) followed by lingual-palatal sounds ('j', 'sh', 'ch') are most affected by spaces in the dental arch. The tongue tends to move forwards into the interdental space causing a central or lateral 'lisp'. The speech sounds most resistant to changes in the dental arch are the velar consonants /k/ and /g/ (Bloomer 1971).

Class III malocclusion

A severe Class III malocclusion may be associated with distortion or interdentalization ('lisping') of sibilant and alveolar speech sounds (/s/, /z/, /t/, /d/, /n/, /l/) due to difficulty elevating the tongue tip to the alveolar ridge. The sound most likely to be affected is /s/. This is probably because production relies on precise placement of the tongue tip and blade in addition to sufficient space being available anterior to the tip in the anterior portion of the palate (Bloomer 1971). Forward tongue placement can also be associated with a tongue thrust swallow and more cumbersome oral preparation of food with poor lateral tongue transfers and imprecise tongue tip elevation during swallowing.

Class II malocclusion

A severe Class II malocclusion may interfere with lip closure during eating and drinking. Bilabial speech sounds (/p/, /b/, /m/) may be distorted or produced in a labiodental manner (with the upper incisors articulating with the lower lip). The speech sounds may be 'visually' distorted, that is they may look different but be acoustically acceptable (sound near normal).

Anterior open bite

An anterior open bite allows the tongue to move forward into the interdental space, causing interdentalization ('lisping') or distortion of speech sounds, particularly those which involve the tongue tip contacting the alveolar ridge (/t/, /d/, /n/, /l/) and palate (/s/, /z/).

Swallowing may also be different for children with an anterior open bite, compared with those with a normal occlusion. For example, genioglossus muscle activity is significantly higher in patients with anterior open bite than those without (Alexander & Sudha 1997). Difficulty biting with middle incisors is common when an anterior open bite is present with overfilling of the mouth, tearing of food or compensatory biting with more lateral teeth noted.

Maxillary collapse

This condition sometimes occurs after cleft palate surgery and leads to distortion of sounds requiring tongue and palatal contact ('s', 'z', 'sh', 'ch', 'j').

Speech and feeding problems are not always associated with these dental conditions. Each patient must be considered individually in light of their abilities to compensate for dental or occlusal anomaly. In cases where problems are identified, speech therapy is coordinated with dental and orthodontic management. Some children may not be able to improve their speech or feeding until their dental treatment is complete.

Lip anomalies

Cleft lip

Sometimes, tissue deficiency and excessive tightness or scarring of tissue affect speech. The speech sounds most likely to be affected are the bilabials (/p/, /b/, /m/). Problems with the facial nerve affecting speech have been reported in syndromes such as hemifacial microsomia and Moebius syndrome (Shprintzen & Bardach 1995).

Depending on the nature of the lip abnormality and range of lip movement, lip closure and protrusion during drinking (e.g. cup/straw drinking) or speech may be affected. Poor lip closure may relate to imprecise labial sounds (p, b, m) and poor lip protrusion associated with distortion of sounds such as 'w', 'oo', 'er'.

Palatal anomalies

The soft palate and pharyngeal walls work simultaneously to close the nasopharynx during speech production and swallowing (i.e. velopharyngeal closure). This action prevents excessive airflow into the nasal cavity during speech, maintains negative intraoral pressure during sucking and swallowing and prevents nasal regurgitation of food or fluid during the swallow. If there is a palatal abnormality, velopharyngeal closure cannot take place efficiently, and there is often associated poor sucking, slow feeding and possible escape of food and liquid into the nose. Speech is nasal and breathy, sounds are unclear and volume may be reduced.

Palatal anomalies may include:

- Cleft palate (with or without cleft lip).
- Submucous cleft palate, characterized by a bifid uvula, notching of the posterior margin of the hard palate, zona pellucida and abnormal insertion of the levator musculature into the free bony edge of the hard palate.
- Congenital palatal anomalies, including short palate, deep nasopharynx, uncoordinated or inefficient velopharyngeal movement.
- Acquired palatal abnormalities resulting from neurological damage, surgery or neoplasms.
- Neurological abnormalities influencing palatal movement (e.g. cerebral palsy, cranial nerve IX and X abnormalities, muscular dystrophy).

Children with palatal anomalies are best referred to a specialist cleft palate clinic where they can receive coordinated multidisciplinary assessment and management (see Chapter 12).

Lingual anomalies

Abnormalities of the tongue may affect the precision, range and speed of tongue movement, resulting in speech or feeding difficulties. The most common problem is ankyloglossia or tongue tie.

Ankyloglossia (Figure 16.1)

Tongue tie may occur with varying degrees of severity, which does not always correlate with severity of functional impairment. Some children have no problems with eating/drinking or speech and others do. Some of the possible are:

- Feeding difficulties such as difficulty sucking in infancy, poor tongue movement and chewing due to restricted tongue movement laterally and persistent messy eating (due to the child being unable to clear food from the buccal cavities and the lips).

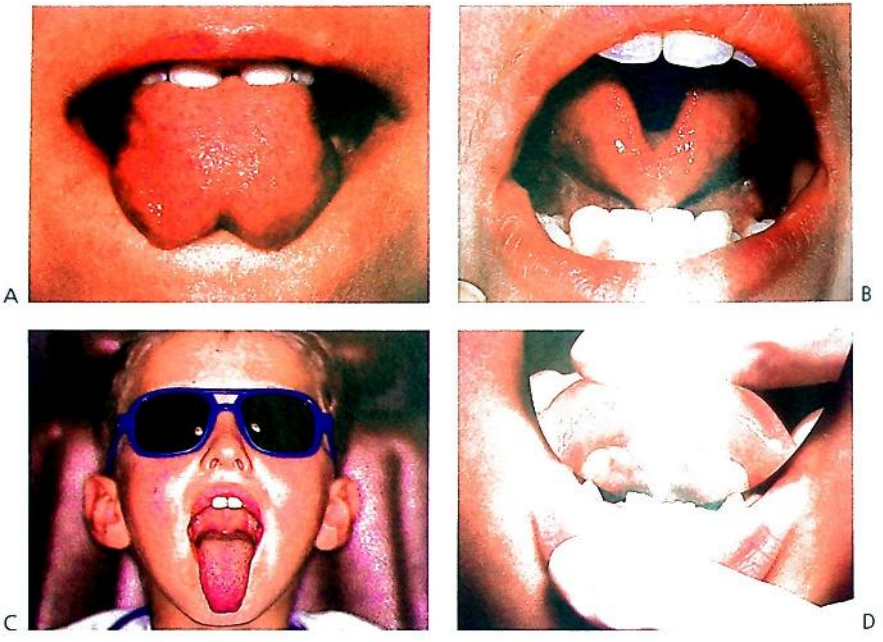

Figure 16.1 Ankyloglossia. (A,B) Notice the tethering of the frenum to the anterior tip of the tongue restricting elevation and protrusion. It is important to assess the position of the frenum into the body of the tongue as well as any attachment into the gingival margin that may cause periodontal complications. (C) Extent of protrusion after lingual frenectomy. (D) Another indication for frenectomy is where the attachment inserts into the free gingival margin, causing potential periodontal problems.

- Substitution and distortion of tongue tip (alveolar) sounds /l/, /t/, /d/, /n/, /s/, /z/ caused by restricted elevation of the tongue tip.
- Slower than normal speech rate or reduced speech intelligibility in conversational speech.
- Reduced speech precision during shouting. (Shouting requires the mouth to open more widely and thereby may result in the tongue tie having a more negative effect on speech precision.)
- Difficulty breast-feeding in infancy and persistent messy eating in later childhood (due to the child being unable to clear food from the buccal cavities and the lips).

If a child's speech or feeding appears to be affected by tongue tie, an assessment by a speech pathologist and a paediatric dentist is useful to determine whether lingual frenectomy is required.

Macroglossia

An abnormally large tongue may be associated with syndromes such as Beckwith–Wiedemann syndrome. Children with macroglossia may have difficulty correctly articulation dento–lingual sounds (e.g. 'th'), lingual–alveolar sounds /t/, /d/, /n/, /l/ and palatal–lingual sounds (e.g. 'ch', 'j', 'sh') In severe cases, the blade of the tongue may contact the upper lip affecting vowels and glides (e.g. /r/ and /w/).

Microglossia

An abnormally small tongue may be associated with syndromes such as hypoglossia-hypodactyly. In these cases, the tongue tip may not contact the teeth, palate or alveolus sufficiently for precise consonant production. Compensatory articulation of sounds may need to be developed.

Maxillofacial surgery and its relation to speech production

When orthognathic surgery is being considered, a consultation with a speech pathologist should be made to determine the possible consequences of the procedure on speech production.

Maxillary advancement procedures

When the maxilla is advanced anteriorly, the hard palate and soft palate are also displaced forwards, increasing the distance that the soft palate must move to achieve velopharyngeal closure. Most patients seem able to compensate for this alteration in nasopharyngeal relationships and their speech and swallowing are not affected. Some patients, however, are 'at risk' for deterioration in speech and swallowing characteristics (e.g. those with a repaired cleft palate). Forward displacement of the palatal structures may result in velopharyngeal insufficiency (VPI) and hypernasal speech production. Forward placement of the maxilla also means that the tongue contact on the palate may be altered for some sounds. Therefore, tongue placement and sound production may be improved in cases with severe Class III malocclusion.

Given the potential impact of surgical interventions on velopharyngeal function, the careful timing and selection of surgical techniques is recommended (Jacques et al 1997). Post-surgical speech review and possible speech therapy may also be warranted.

Referral to a speech pathologist

When the presence of a communication or feeding problem is suspected, referral should be made as soon as possible.

Dentists should refer any child who experiences the difficulties outlined below.

Feeding and swallowing

- Has difficulty sucking, swallowing and chewing.
- Is coughing, gagging or choking during feeds.
- Is drooling excessively.
- Has reported breast, bottle, drinking or eating difficulties.

Articulation
- Is not babbling a wide variety of sounds by 8–10 months.
- Is not easily understood by caregivers by 2 years.
- Is not easily understood by familiar adults by 3 years.
- Is having difficulty in producing sounds accurately by 5 years.

Language
- Is not understanding simple instructions and questions by 18 months.
- Is not using single words by 18 months.
- Is not combining two words by 2 years (i.e. 'more drink').
- Has difficulty following instructions or answering questions.
- Gives inappropriate answers or frequently ignores language spoken to them.
- Is constructing sentences that are incorrect or immature by 3–4 years (i.e. 'me go to him house').
- Cannot maintain a topic of conversation by 4 years.

Voice
- Has a hoarse or breathy voice or often loses their voice.
- Has a nasal voice.
- Often sounds as though they have a cold.
- Has a voice that seems too high or low for their sex or age.
- Continually speaks abnormally loudly or softly.
- Has a sudden onset of any of these problems.

Fluency
- Persistent stuttering at any age.

Pragmatics
- Does not initiate or engage others in communication at any age.
- Has poor eye contact.
- Constantly interrupts others.
- Displays inappropriate social use of language (e.g. inappropriate use of words for a situation).
- Talks excessively does to take conversational turns or goes off on a tangent when speaking.

Referral procedures

It will be necessary to locate the most appropriate service to meet your patient's needs. Most often, access to a speech pathologist through a local community health centre or hospital will be all that is required. Some patients may require a more specialized service such as a developmental disability service, cleft palate clinic, feeding specialist or feeding clinic. Some patients may prefer the services of a private practitioner.

Referral is most efficient when the dentist provides a written referral outlining the areas of concern. Following an assessment, a treatment plan will be devised according to the individual needs of the patient. Treatment can be provided in individual or group sessions and it may extend over a period of time, depending on the nature and severity of the condition. Most speech, language and feeding problems are best treated with parental participation. Preschool or school-based programmes are important for older children.

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Appendices

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Appendix A: Blood and serum testing and investigations

Laboratory examination may be divided into two general categories: screening and diagnostic. Screening studies are intended to identify individuals with disease in the early and asymptomatic stages. By definition, screening studies must be relatively simple and inexpensive, and are useful only when used to identify a disease which is relatively frequent (diabetes mellitus, anaemia, syphilis, blood disorders). Diagnostic examinations provide more specific information. The distinction between screening and diagnostic laboratory examination is not always rigid or absolute.

It must be remembered that laboratory examinations provide information that contributes to the diagnostic process. Seldom is this information of value by itself. The results must be interpreted in conjunction with other information that is available about the patient. It should also be noted that a laboratory value outside the normal range does not necessarily indicate disease. That value may represent normal for that specific patient. Usually, normal values are determined by testing supposedly healthy people, and these results are used to calculate the mean and normal range. Variables are not considered, and as a consequence, normal ranges are not always valid for all patients. Conversely, if a clinical diagnosis appears valid and is not substantiated by laboratory results, the tests should be repeated to rule out the possibility of laboratory error.

Haematology

Full blood count

A full blood count (FBC) usually includes a white blood cell count (WBC), red cell blood count (RBC), haemoglobin (Hb), haematocrit (Hct) and red blood cell indices (mean corpuscular haemoglobin, mean corpuscular volume, mean corpuscular haemoglobin concentration and platelet count).

Platelet function tests are rarely ordered, usually only in consultation with a haematologist. If one asks for FBC and 'differential', one will also receive a breakdown of the RBC and WBC as listed on the form. If an FBC detects anaemia, then one should request serum ferritin, red cell folate and serum vitamin B12. However, in interpretation of these results, one should seek counsel, as they are difficult.

Coagulation and bleeding tests

Problems relating to bleeding are relatively infrequent in dental practice. Most inherited defects will usually have been identified early in life, and so it is usually acquired bleeding problems about which the dentist must be aware. Screening studies will identify

Table A.1 Normal blood values

	Children		Adults	
White blood cells	$4.0–15.0\times10^9$/L		$4.0–11.0\times10^9$/L	
Neutrophils	$1.5–7.5\times10^9$/L	50%	$2.0–8.0\times10^9$/L	60%
Lymphocytes	$1.0–8.6\times10^9$/L	42%	$0.5–4.0\times10^9$/L	33%
Monocytes	$0.5–1.5\times10^9$/L	5%	$0.2–1.0\times10^9$/L	4%
Eosinophils	$0.3–0.8\times10^9$/L		$0.04–0.5\times10^9$/L	
Basophils	$<0.1–0.2\times10^9$/L		$<0.01–0.2\times10^9$/L	
Red blood cells	$4.0–5.5\times10^{12}$/L	(3–12 years)	$4.5–6.5\times10^{12}$/L	Male
			$3.8–5.8\times10^{12}$/L	Female
Haemoglobin	115–145 g/L	(3–12 years)	130–180 g/L	Male
			115–165 g/L	Female
Mean corpuscular volume	70–90 fL		80–96 fL	
Mean corpuscular haemoglobin	23–31 pg		27–32 pg	
Platelets	$150–450\times10^9$/L			
Erythrocyte sedimentation rate	0–10 mm/h		0–5 mm/h	Male
			0–20 mm/h	Female
Reticulocytes	2.0–6.0% or mean 150×10^9/L	Infants	0.2–2.0%	
	$10–100\times10^9$/L	Children		
Red cell folate	340–2500 nmol/L			
Serum folate	7–40 nmol/L			
Vitamin B^{12}	150–700 pmol/L			

Table A.2 Tests for bleeding problems

Activated partial thromboplastin time (APTT)	24–38 s
Prothrombin time	11–17 s
Factor VIII assay	50–200%
Mild haemophilia	20–25%
Moderate	2–5%
Severe	<1%
Skin bleeding time	<9 min

whether there is a bleeding problem, and in which of the three systems it is, namely, platelets, coagulation or vascular abnormalities.

Clinical chemistry

It is not appropriate to simply request a multi-blood analysis in the hope of finding a diagnosis or abnormality. The following abbreviations are often used when ordering tests, but will obviously differ from one institution or laboratory to another.

EUC Electrolytes, urea, creatinine
LFTs Liver profile, including serum proteins
CA Calcium
PHOS Inorganic phosphate, alkaline phosphatase

Blood chemistry

Table A.3 Normal blood chemistry

Sodium		136–146 mmol/L
Potassium		3.4–5.5 mmol/L
Chloride		94–107 mmol/L
Total CO_2		24–31 mmol/L
Urea		2.5–6.5 mmol/L
Creatinine		60–125 mmol/L
Glucose	Fasting 2 h postprandial	3.9–6.1 mmol/L <7.8 mmol/L
Maintenance range for IDDM		4–10 mmol/L
Calcium		2.13–2.63 mmol/L
Phosphate		0.18–1.45 mmol/L
Osmolality		275–295 mmol/L
Lactate		0.63–2.44 mmol/L
Alkaline phosphatase		60–391 U/L

Liver function tests

Table A.4 Liver function tests

Total bilirubin	2–21 mmol/L
Total protein	63–79 g/L
Albumin	35–53 g/L
Alkaline phosphatase (ALP)	30–115 U/L
γ-Glutamyl-transpeptidase (γGT)	
Male	8–43 U/L
Female	5–30 U/L
Alanine aminotransferase (ALT)	7–47 U/L

Iron studies

These tests are used when there is a suspicion of an underlying anaemia. The request for iron studies provides information regarding serum iron, total iron binding capacity and percentage iron saturation.

Table A.5 Iron studies

Ferritin	
Male	30–300 mg/L
Female: premenstrual	15–150 mg/L
Female: postmenstrual	25–200 mg/L
Iron	7.0–29.0 mmol/L
Transferrin	2.1–3.9 g/L
Saturation	0.09–0.52

Urine chemistry

Table A.6 Urine chemistry

Sodium	40–220 mmol/L
Potassium	25–120 mmol/L
Creatinine	8.0–18.0 mmol/L
Total protein	<0.15 g/dL

Arterial blood gas

Table A.7 Arterial blood gas

pH	7.35–7.45
pCO_2	35–45 mmHg
pO_2	75–100 mmHg
HCO_3	22–26 mmol/L
Base excess	–3 to +3 mmol/L
SaO_2	0.95–0.98

Appendix B: Fluid and electrolyte balance

Fluid and electrolyte replacement can be conveniently divided into:

- Maintenance replacement – The fluid and electrolyte losses occur during a normal day. These values are modified by other factors such as patient and environmental temperatures, age weight and metabolic rate.
- Deficit replacement – To replace any existing or ongoing abnormal losses such as dehydration from vomiting or diarrhoea and blood loss.

Maintenance replacement

The need for water and electrolytes is a function of the metabolic rate as they are substrates for metabolism. Thus, the younger the child, the higher the metabolic rate on a weight basis and the higher the turnover of water and electrolytes.

Water requirements

Table A.8 Water requirements

Infants	Day 1	60 mL/kg/day
	Day 2	80 mL/kg/day
	Day 3	100 mL/kg/day
	Day 4 to 1 year	120 mL/kg/day
Children	<10 kg	4 mL/kg/h
	10–20 kg	2 mL/kg/h+40 mL/h
	>20 kg	1 mL/kg/h+60 mL/h

Modifying factors

Increased maintenance requirement

- Fever – add 12% per degree above 37.5°C.
- Hyperventilation.
- Extreme activity.
- High environmental temperature.

Decreased maintenance requirement

- Cardiac failure.
- Inactivity (patient sedated in ICU) – decrease by 30%.
- Hypothermia – decrease 12% per degree <37.5°C.
- Head injury – decrease by 30%.
- Renal failure – decrease by 70%+urine output.

Electrolyte requirements

Normal electrolyte requirements are shown in Table A.9.

A total of 0.225% sodium chloride+3.75% dextrose at normal maintenance rates will supply adequate sodium and chloride; potassium should only be added to intravenous fluids if replacement is to continue for more than 24 h and adequate urine

Table A.9 Electrolyte requirements

	0–10 kg	11–20 kg	>20 kg
Fluids (mL/kg/day)	100	50	20
Energy (cal/kg/day)	100	50	20
Na (mmol/kg/day)	3.0	1.5	0.6
K (mmol/kg/day)	2.0	1.0	0.4

output is present. However, 0.45% saline+5% dextrose is now accepted as routine replacement fluid in many centres, as the risk of hyponatraemia is reduced in patients with stress related ADH release. Infants under 3 months of age will also require supplements of dextrose if they are to fast for longer than 4–6 h.

Fluid deficit

Fluid deficit is usually expressed as a percentage of body weight. This allows for easy calculation of replacement fluids. Fluid imbalance may be as a result of any of the following:

Water loss

- Decreased intake.
- Increased respiratory loss (especially seen in children with high respiratory rates).
- Renal concentration impairment.

Water and salt loss

- Vomiting.
- Diarrhoea.
- Increased sweating.

Blood volume loss

- Haemorrhage.
- Septic shock.
- Anaphylaxis.
- Burns.

Assessment of deficit

Deficit assessment is difficult even for experienced paediatricians. Some idea of the deficit can be gained from the history of the abnormal loss. For example:

- Has the vomiting persisted for more than 24 h?
- Has the child passed urine in the past 12 h?
- Is the child thirsty?

Dehydration is generally assessed by estimating weight loss.

Mild dehydration (2–3% acute weight loss)
- Thirst.
- Mild oliguria.
- No physical signs.

Moderate dehydration (5% acute weight loss)
- Slight decrease in skin tone.
- Sunken fontanelles in infants.
- Slight decrease in ocular tension.
- Tachycardia.

Severe dehydration (7–8% acute weight loss)
- Marked tachycardia.
- Loss of skin tone.
- Loss of ocular tension.
- Sunken eyes.
- Restlessness and apathy.

Profound dehydration (>10% acute weight loss)
- Circulatory collapse.
- Delirium and coma.
- Hyperpyrexia.
- Cyanosis.

Replacement of deficit
Usually the deficit is replaced with the fluid that most closely approximates the fluid that has been lost. Replacement therapy is aimed at restoring the fluid compartments in the following order. *If there is significant loss of blood volume, then this must be replaced as rapidly as is safely possible to preserve brain, heart and kidney perfusion.*

Blood volume loss
- Blood: packed red cells or whole blood.
- Colloid: 4% Albumex or Haemaccel.
- Hartmann's solution or normal saline.
- Inotropes if needed.

Salt and water loss
- Hartmann's solution or normal saline.
- 0.45% sodium chloride+2.5% dextrose (N/2 saline).

Water loss
- 0.255% sodium chloride+3.75% dextrose (N/4 saline).
- Maintenance fluids should be added to the deficit losses and given over the normal period.

Calculation of deficit

Deficit (mL) = % dehydration×weight (kg)×10.

Examples

MILD DEHYDRATION

Where there is no circulatory compromise, the loss will be water and electrolytes from all body compartments. This can be replaced with dextrose saline solution over many hours.

SEVERE DEHYDRATION

There is loss of intracellular, interstitial and most importantly, blood volume. The priority is to rapidly restore blood volume and with that, cardiac output with colloid or blood (10–20 mL/kg over 20–30 min) or saline solutions (20–40 mL/kg) to allow adequate vital organ perfusion. After this is achieved, electrolyte and water deficits should be replaced as calculated with dextrose saline solutions over a longer period of time (several hours to 24 h).

SEVERE BLOOD LOSS

In cases of trauma or bleeding, there will initially be loss of blood volume. This is treated in the same way as above, i.e. restoring circulating blood volume with colloids or blood and reassessment of losses. If the blood loss is unknown, then initial therapy is to start with 20 mL/kg over 10–20 min and then reassess. If the blood pressure has returned to normal and fallen again or has not responded to this initial bolus, then a repeat of the initial bolus of fluid is indicated, followed by reassessment. The signs of adequate fluid replacement without the aid of central venous pressure or urine output measurement are the return to normal values of blood pressure and heart rate without the need for further boluses of fluid.

Notes on rehydration

The above guidelines apply to previously healthy children. Those children with cardiac disease or significant systemic disease require intensive intravascular monitoring in a paediatric intensive care unit.

- Constant reassessment of fluid therapy is essential throughout replacement.
- Measurement of electrolytes is essential in the replacement of greater than moderate deficits and applies especially to potassium.
- Measurement of acid–base status with arterial blood gases is often necessary, as fluid deficit causes organ hypoperfusion and subsequent metabolic acidosis. This will usually correct itself with correction of blood volume and cardiac output over many hours.
- Fluid balance and acid–base disturbances are often very complex and life-threatening; if there is any doubt as to management, then specialist paediatric or anaesthetic advice should be sought.

Transfusion

Volume (mL) = weight (kg)×g% Hb rise required×3

Table A.10 Composition of intravenous crystalloid fluids

	Na^+ (mmol/L)	Cl^- (mmol/L)	Lactate (mmol/L)	Ca^{2+} (mmol/L)	Dextrose (g/L)
Normal saline	150	150			
0.45% NaCl+2.5% Dextrose	75	75			50
0.225% NaCl+3.75% Dextrose	37.5	37.5			37.5
0.18% NaCl+4% Dextrose	30	30			40
Hartmann's solution	130	110	5	3	

Table A.11 Composition of intravenous colloid fluids

	Na^+ (mmol/L)	Cl^- (mmol/L)	K^+ (mmol/L)	Ca^{2+} (mmol/L)	Colloid (g/L)
Haemaccel	145	145	5.1	6.25	Polygeline 35
Gelofusine	144	120			Gelatin 40
4% Albumex	140	128			Albumin 40

Appendix C: Management of anaphylaxis

Anaphylaxis
A symptom complex accompanying the acute reaction to a foreign substance to which the patient has been previously sensitized.

Anaphylactoid
Same symptoms but the reaction is non-immunological or unknown.

Incidence
- Anaesthesia – 1:5000 to 1:30000 (mortality rate of 4% with reactions).
- Radiographic contrast – 2%.
- Antibiotics– 1:5000.
- Latex allergy – 0.13%.
- Local anaesthetics – rare (usually to preservative).
- Foods, insects.

Latex sensitization in the general population is about 1%. Certain groups have a much higher incidence such as the healthcare workforce in which sensitization is estimated to be between 5% and 12% and children with spina bifida, who are repeatedly exposed to latex from birth.

Timing
Some 98% occur within 5 min of drug administration, but may occur up to hours later.

Clinical presentation
PRODROME
- Metallic taste.
- Apprehension.
- Coughing.
- Choking sensation.
- Paraesthesia.
- Arthralgia.

CUTANEOUS
- Blushing.
- Urticaria.
- Angio-oedema.
- Pallor and cyanosis.

CARDIOVASCULAR
- Tachycardia.
- Hypotension.
- Shock.

RESPIRATORY
- Bronchospasm.
- Laryngeal obstruction.
- Pulmonary oedema.

GASTROINTESTINAL TRACT
- Nausea, vomiting, diarrhoea.
- Abdominal cramps.

OTHERS
- Disseminated intravascular coagulation.
- Fitting.

Treatment
Adrenaline and colloid infusion are the mainstays of the treatment of anaphylaxis. Follow-up is essential. The patient must be transferred to an intensive care unit, as symptoms may return up to hours later. A letter must be sent with the patient describing the event and all the drugs used until skin testing can identify the offending drug. Skin testing of all drugs used is performed 3 months after the reaction. A MedicAlert bracelet should be worn by the child, identifying relevant drug reactions.

Notes on management (Figure A.1)
Adrenaline is the main drug used in the treatment of anaphylaxis and anaphylactoid reactions. Adrenaline *must* be used if anaphylaxis is suspected.

Table A.12 Treatment of anaphylaxis and anaphylactoid reactions with adrenaline

Children (<12 years)	Dilute 1 ampoule into 9 mL of saline 1:1000 becomes 1:10000 (1 mg/10 mL) Inject 0.25 mL per year of age intramuscularly; this approximates 5 µg/kg
Adults	Inject 1:1000 intramuscularly
Small adults (<50 kg)	0.25 mL
Average adults (50–100 kg)	0.50 mL
Large adults (>100 kg)	0.75 mL
	Intravenous access lines must be large gauge, preferably ≥16 gauge
	Colloid 10–20 mL/kg stat

- *The doses of adrenaline and colloid must be repeated if the patient's vital signs have not improved.*

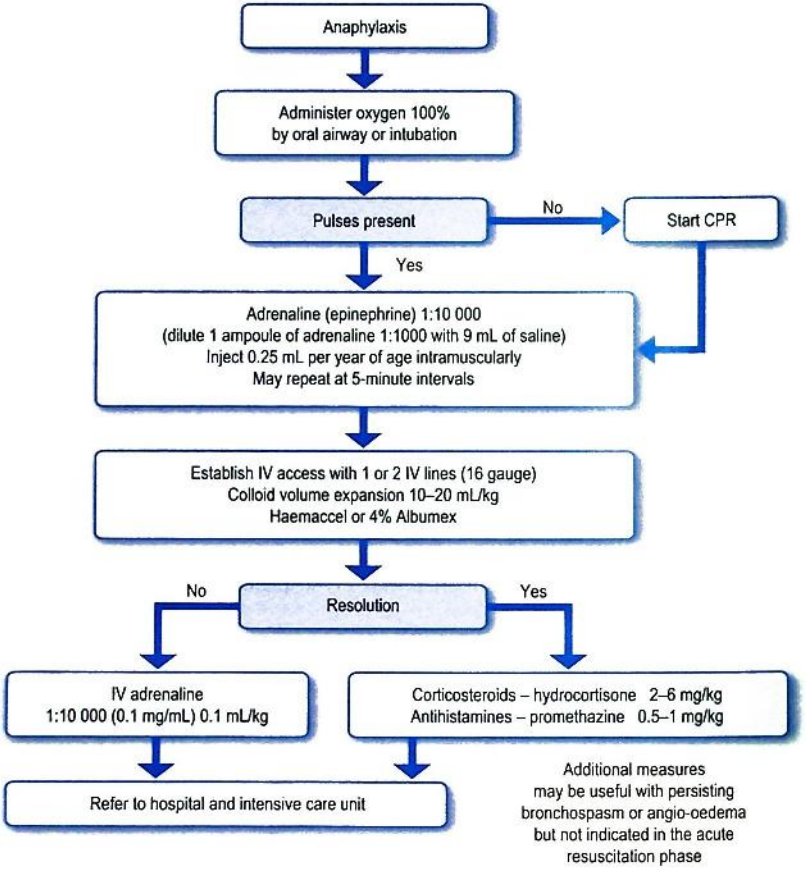


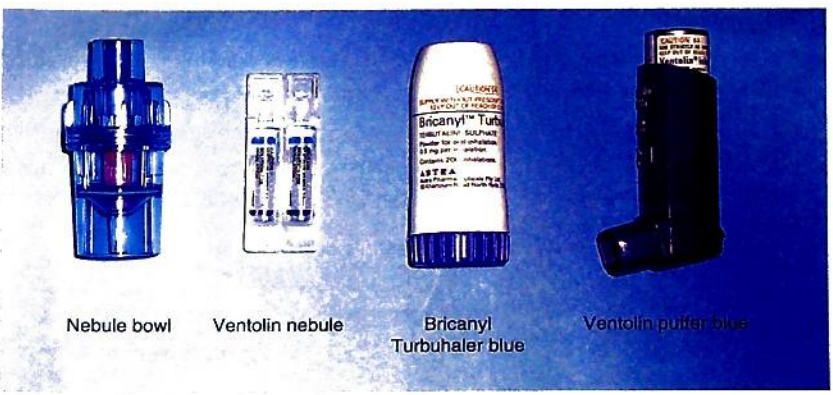
Figure A.1 Management of anaphylaxis.

Appendix D: Management of acute asthma

Asthma is one of the most common childhood diseases and accounts for significant mortality and morbidity. The emphasis of treatment today is on prophylaxis rather than merely treating attacks. Most children with known asthma will have treatment plans prescribed by their doctor for use in acute episodes. However, there are children who have undiagnosed asthma who are at risk of acute attacks.

Essential equipment for an asthma kit

- Ventolin puffer (Figure A.2A).
- Bricanyl turbuhaler.
- Ventolin nebules (2.5 mg).
- Ventolin nebules (5 mg).
- Nebulizer unit and tubing for wall oxygen.
- Child mask for nebulizer.
- Small volume spacer with face mask.
- Volumatic spacer or small volume spacer with mouthpiece.



A

B AeroChamber

Volumatic spacer with Ventolin puffer C

Figure A.2 A–C Drugs and devices used in the management of asthma.

Bronchodilators (Figure A.2A)

Ventolin

- Inhaler (blue): 100 µg/puff.
- Dose: 4–12 puffs (weight dependent) via spacer.
- Nebules: 2.5 mg or 5 mg given via nebulizer.

Bricanyl

- Turbuhaler: 500 µg/inhalation.
- In acute situations: preferable to use puffer and a spacer.

Apparatus for administration of bronchodilators

Small volume spacer: children 4 years and under (Figure A.2B)

- Position AeroChamber with mask over child's face.
- Four puffs from Ventolin puffer; patient inhales four to six times for each puff.

Volumatic spacer: children over 4 years (Figure A.2C)

- Position spacer between lips.
- Four puffs from Ventolin puffer, patient inhales four to six times for each puff.
- Encourage child to breath deeply for 6–10 s.

Nebulizer

- For very young children or in older children when condition is not improved by puffer with spacer after 10 min.
- <5 years: 2.5 mg nebule.
- >5 years: 5 mg nebule.
- Place contents of nebule in bottom of nebule bowl, fix to face mask and apply oxygen or air to mask at 6–8 L/min flow rate. Add normal saline to Ventolin nebule solution to make up to 4 mL total.
- A fine mist will form, which the child breathes deeply for about 10 min.

Management

See Figure A.3.

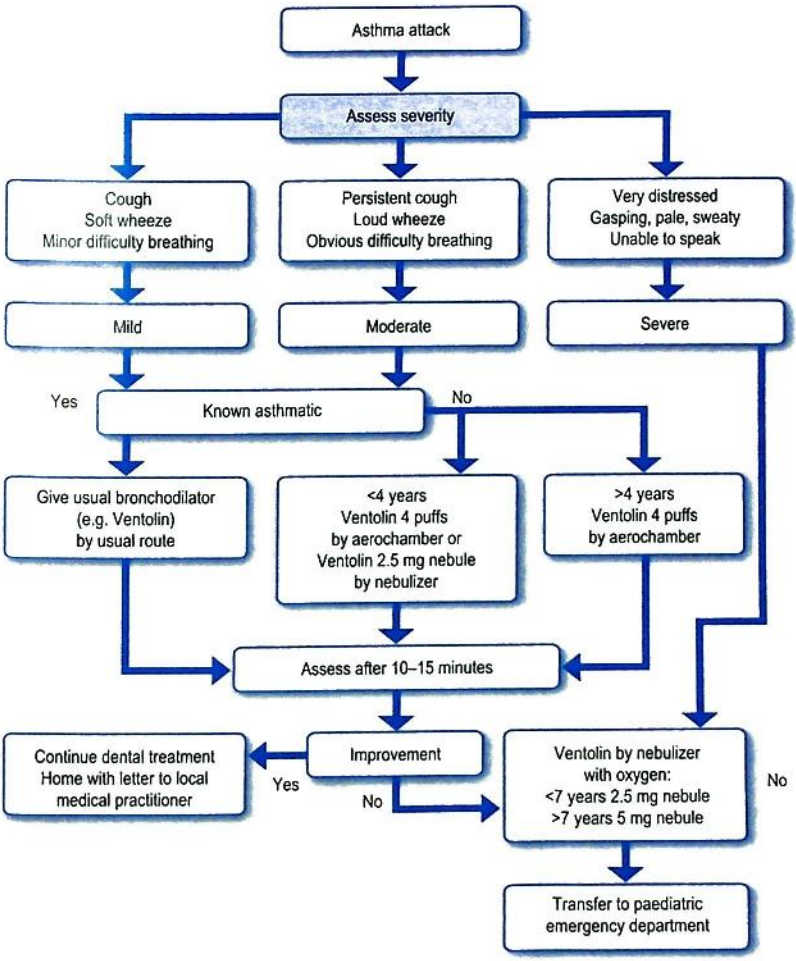


Figure A.3 Management of acute asthma.

Appendix E: Antibiotic prophylaxis protocols for the prevention of infective endocarditis

Infective endocarditis is a rare and potentially life-threatening disease with a reported annual incidence of 0.3 per 100 000 children in Western countries, which has remained unchanged in the past 40 years. Despite the advent of antibiotics, infective endocarditis still has a high rate of mortality, up to 25%, and is associated with significant morbidity.

Approved antibiotic prophylaxis regimens are still recommended for potentially at-risk patients receiving dental treatment, as there is evidence, predominantly based on animal models, that suggests infective endocarditis may follow dental treatment in susceptible patients. Recommendations differ from one institution to another and vary in different countries, and therefore it is the responsibility of clinicians to determine which guideline is the most suitable to their individual patient.

Pathogenesis

- Characterized by inflammation of the inner surface of the heart (endocardium).
- Generally due to bacterial infection.
- Most commonly affecting the heart valves.
- May also involve non-valvular areas.
- Implanted cardiac mechanical devices also affected, such as prosthetic heart valves.

Three conditions need to be met for infective endocarditis to occur:

- Pre-existing damage to the heart valve surface.
- Bacteraemia, that is the introduction and circulation of bacteria in the bloodstream.
- Presence of bacteria of sufficient virulence to evade the body's innate defences, to attach, colonize, invade and so cause infection of the damaged heart valve surface.

Epidemiology

The at-risk population principally consists of those with:

- Rheumatic fever. Previously, the commonest cause of heart valve damage was childhood rheumatic fever. Improved sanitation and living conditions and the availability of antibiotics has significantly reduced the incidence of rheumatic fever. In those areas with social and economic deprivation, rheumatic heart valve disease is still prevalent.
- Congenital heart disease.
- Prosthetic aorto-pulmonary shunts.
- Prosthetic heart valves.
- Patients with a previous history of endocarditis.
- Immunocompromised patients with long-term central venous lines.

Antibiotic prophylaxis

It has been long recognized that invasive dental procedures, typically extractions, cause an acute, substantive bacteraemia. However, there are increasing concerns regarding the cumulative bacteraemia associated with the activities of daily living, such as

chewing or tooth brushing, particularly in the presence of chronic dental disease. In spite of this, infective endocarditis is uncommon.

In an attempt to reduce the significant mortality and morbidity rates seen with infective endocarditis, numerous protocols recommending the prophylactic use of antibiotics have been published. Two of the most authoritative bodies, namely, the American Heart Association (AHA) and the Working Party of the British Society for Antimicrobial Chemotherapy (BSAC) have recently published significantly revised protocols for antibiotic prophylaxis for susceptible patients.

Cardiac conditions associated with the highest risk of adverse outcome from endocarditis for which prophylaxis with dental procedures is recommended:

- Previous history of infective endocarditis.
- Prosthetic cardiac valve replacement.
- Cardiac transplant recipients who develop valvulopathy.
- Specified congenital heart disease involving the presence or placement of shunts or conduits:
  - Unrepaired cyanotic shunts, including palliative shunts or conduits.
  - Completely repaired congenital heart defects with prosthetic material or device for at least 6 months after the procedure.
  - Repaired congenital heart disease with residual defects or adjacent to a site of prosthetic patch or material.

At-risk dental procedures

Antibiotic prophylaxis is now indicated for *any and all* dental procedures that involve manipulation of the gingival, mucosal or periapical tissues that is likely to cause bleeding (i.e. extractions, scaling, root canal instrumentation beyond the apex). The following procedures and events *do not* need prophylaxis:

- Routine anaesthetic injections through non-infected tissue.
- Taking dental radiographs.
- Placement of removable prosthodontic or orthodontic appliances.
- Adjustment of orthodontic appliances.
- Placement of orthodontic brackets.
- Shedding of deciduous teeth.
- Bleeding from trauma to the lips or oral mucosa.

Guidelines for clinicians

Past and current medical history

It is essential that the patient's medical status and history be assessed with respect to their cardiac problem. Consultation with the patient's doctor or cardiologist is essential. Dentists should be prepared to discuss with the treating doctor any issues surrounding the dental care of their patient.

Considerations in selecting appropriate antibiotics

- Anaphylaxis must be considered a risk in all patients taking any antibiotic, but particularly with any of the penicillins.
- Does the patient have a convincing history of allergy to any of the recommended antibiotics?

- Is the patient on long-term antibiotics? Or have they recently been taking an antibiotic? Alternative agents should be used.
- Does the patient have impaired renal function that will necessitate dose modification?
- Is the patient able to accept oral medications? Is there a history of vomiting with oral antibiotics? If so, consider parenteral medication.

Treatment planning

- 'Group' together a number of invasive dental procedures to be done in the minimal number of appointments to reduce the need for repeated courses of antibiotics.
- The same antibiotic should not be prescribed within 14 days.
- Is a general anaesthetic indicated? Consideration should be given to completing all possible treatment in one appointment.

Recommendations for use of protocols for antibiotic prophylaxis

The authors do not make a recommendation about the efficacy of one particular protocol over another. There are problems associated with prophylaxis regimens in that no two sets of guidelines are the same. Less than 10% of patients with endocarditis have had a recent invasive dental procedure and there is no direct evidence in humans that antibiotic prophylaxis is effective. Currently, there is disagreement over the efficacy of different protocols as to which patients and what dental procedures should be covered. Please note that the current Australian Guidelines make special reference for the need to provide antibiotic prophylaxis for indigenous Australians who have a history of rheumatic fever. The AHA Guidelines still recommend antibiotic prophylaxis for invasive dental procedures in select patients thought to be at higher risk of infective endocarditis. In contrast, the National Institute for Health and Clinical Excellence (NICE) published revised guidelines for the UK in 2008. The major recommendation of this group is that antibiotic prophylaxis is **NOT RECOMMENDED** for patients at-risk of infective endocarditis, undergoing any type of dental procedures, including invasive dental procedures. The NICE guidelines contend that the greatest risk of infective endocarditis is from the cumulative, incidental daily bacteremia. In light of this, the NICE guidelines place greater emphasis on the provision and maintenance of optimal oral hygiene and dental health. Of interest, since the introduction of the revised guidelines, there has not been an increase in the incidence of infective endocarditis in the UK.

Paediatric dosing

- The dose for any child should be calculated up to, but not exceeding the maximum adult dose.
- Dosage should always be prescribed according to weight (dose/kg).

Other considerations

- It is expected that some cases of endocarditis will occur, despite the use of optimal prophylaxis protocols.
- In circumstances where appropriate prophylaxis has not been given, antibiotics prescribed up to 6 h after a procedure may give effective cover.

Table A.13 Current protocols for susceptible patients

Situation	Agent	Regimen: Single dose 30–60 min before procedure	
		Adults	**Children**[c]
Oral	Amoxicillin	2 g	50 mg/kg to adult dose
Unable to take oral medications	Ampicillin *or* Cefazolin or ceftriaxone	2 g IV or IM 1 g IV or IM	50 mg/kg IV or IM 50 mg/kg IV or IM
Oral but allergic to penicillins or ampicillin	Cefalexin[a,b] *or* Clindamycin *or* Azithromycin *or* clarithromycin	2 g 600 mg 500 mg	50 mg/kg 20 mg/kg 15 mg/kg
Allergic to penicillins or ampicillin and unable to take oral medications	Cefazolin or ceftriaxone *or* Clindamycin	1 g IV or IM 600 mg IM or IV	50 mg/kg IV or IM 50 mg/kg IV or IM

IM, intramuscular; IV, intravenous.
[a]Or other first or second generation cephalosporin in equivalent adult or paediatric dose.
[b]Cephalosporins should not be used in an individual with a history of anaphylaxis, angio-oedema or urticaria with penicillins or ampicillin.
[c]Child dose must not exceed adult dose.
After Wilson et al. 2007.

- Good history-taking is essential.
- If in doubt, consult relevant medical authorities.

Further reading

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Appendix F: Somatic growth and maturity

As soon as the child enters the surgery, assessment should begin. At the outset, the dentist should always look at a child's size, development, appearance and behaviour in relation to their chronological age. Dental examination will initially include an assessment of dental age (based on time of exfoliation, eruption status and root development) in relation to the chronological age. Any marked discrepancies should then be investigated further.

Basic indicators of somatic growth and development

Height and weight

HEIGHT MEASUREMENT

- Measure the child with shoes off, standing straight, with the Frankfort plane horizontal to the floor.
- Measurement is taken on deep inspiration of the patient.
- Sequential measurements are ideally taken at the same time of day.

HEIGHT ABNORMALITIES

- Short stature <3rd percentile, tall stature >97th percentile over a 6-month period.
- Rate of growth <3–5 cm/year; consider both for referral to specialist growth unit at a paediatric hospital.
- Measurements must be considered in relation to height of parents and skeletal age.
- Height prediction possible using methods of Tanner and Whitehouse (1983) or Bayley and Pinneau (1952).
- Prediction of adolescent growth spurt is achieved by serial measurements, and may influence the subsequent timing of myofunctional orthodontic treatment.

WEIGHT MEASUREMENT

- Taken in light indoor clothing, with shoes off, ideally at the same time of day as height measurements.

WEIGHT ABNORMALITIES

- Children with an endomorphic appearance tend to mature early, while those who are ectomorphic (especially boys) tend to mature late.
- Underweight – consider anorexia/bulimia.
- Overweight – may indicate nutritional problems.
- When children are markedly outside norms for age, early referral to a paediatrician or dietitian is essential.
- Gross obesity may significantly alter drug metabolism and will affect the calculation of drug dosages.

SKELETAL ASSESSMENT

- It has been consistently shown that bone age as determined from hand-wrist radiographs using the Greulich and Pyle or Tanner and Whitehouse systems has a high correlation with stature and general body development.

- Other rarely used methods of bone-age calculation include the FELS method, use of knee joint or cervical vertebrae.
- Convention for anthropometric measurements uses the left hand.
- These methods assume bones of all patients consistently go through the same sequence of development, albeit at different rates. The Greulich and Pyle system is the most skeletally advanced for any age group, as it was derived in the USA from healthy children from a high socioeconomic group.

Greulich and Pyle method

Each bone is matched with a similar appearing bone in a series of standard radiographs of increasing age. Thus, each bone has a bone age assigned to it and the modal (or most frequent) of these bone ages is taken as the bone age of the hand and wrist. Frequently, the step of assigning bone ages to each separate bone is omitted and instead the patient's hand-wrist radiograph is matched to the nearest standard radiograph, thereby determining the patient's skeletal age. Radiographic standards are provided at 6- and 12-monthly intervals for both males and females.

Tanner and Whitehouse method

This method scores specified bones according to their stage of development, using a written description and a radiographic standard of each stage of development. The total score for all bones is used to derive a skeletal age from tables provided. Generally, the TW-2 (13 bone score) is used in preference to the Tanner and Whitehouse 20-bone method, as it is about as accurate but is quicker to use. A computerized image analysis system for estimating TW-2 bone age has been shown to be more reliable than the manual rating system. The Tanner and Whitehouse TW-2 method is easier and more accurate than the Greulich and Pyle method for the occasional user.

Significance

- Used to calculate potential for further increase in height.
- Used to predict adolescent growth spurt for timing of orthodontic treatment.
- Monitor growth abnormalities.

Dental development

Eruption times

- The emergence of teeth in the primary and permanent dentitions is unreliable because of environmental influences (i.e. early extraction of primary teeth will delay eruption of the succedaneous tooth, while late extraction of a primary tooth will hasten the eruption of the permanent successor).
- Eruption is not a continuous event.
- Racial variations – published data on eruption times are generally of northern European populations. Earlier eruption times may be the norm in Asian peoples, and later eruption times the norm in eastern and southern European groups.

Root development

- Use the scoring systems of Nolla, Moorees, Fanning, Demirijan and others. These quantify tooth development from initial calcification to final root closure, as seen on radiographs.

Sexual development: peak height velocity

- Hägg and Taranger (1982) observed that menarche occurred a mean 1.1 years after peak height velocity (PHV).
- Menarche is a highly reliable but not absolute indicator that PHV has been reached or passed.
- Menarche occurs at a bone age of 13.1 years.
- Boys attain a 'pubertal voice' (the pitch of the voice had changed noticeably but had not yet acquired adult characteristics) 0.2 years before PHV, and the 'male voice' (pitch of the voice had acquired adult characteristics) 0.9 years after PHV.
- Tanner (1978) found that in males, breaking of the voice happens relatively late in puberty and is due to the increased length of the vocal cords which follows the growth of the larynx. Voice breaking is often a gradual process and is not reliable as a criterion of puberty. Facial hair appears in boys usually somewhat later than the PHV.

Correlation between dental development and other maturity indicators

Evidence so far indicates that the skeletal system, as well as height and the onset of puberty, develop largely independently of the dental system. Teeth are partly of epithelial origin, whereas bone is derived from the mesoderm. Serious endocrinopathies, while severely retarding somatic growth and maturation, exert only a minor effect on the dentition. Demirjian (1978) found a very low correlation between dental age (root development) and skeletal age.

General observations on somatic growth

- Growth is nutrition dependent and a well-fed infant gains length before it gains weight.
- The pubertal growth spurt is governed by growth hormone and anabolic steroids (testosterone and oestrogens).
- Girls enter the growth spurt about 2 years earlier than boys; however, they complete the growth spurt only about a year earlier than boys.
- The growth spurt is of greater magnitude and of shorter duration in boys than girls, as testosterone has a greater anabolic effect than oestrogen.
- Growth ceases from the feet upwards, so limb growth stops before spine growth.
- The pubertal growth spurt adds 25–30 cm to final height over the childhood growth curve. Boys on average end up 12–13 cm taller than girls, as their growth spurt occurs after an additional 2 years of childhood growth.

Further reading

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Appendix G: Growth charts

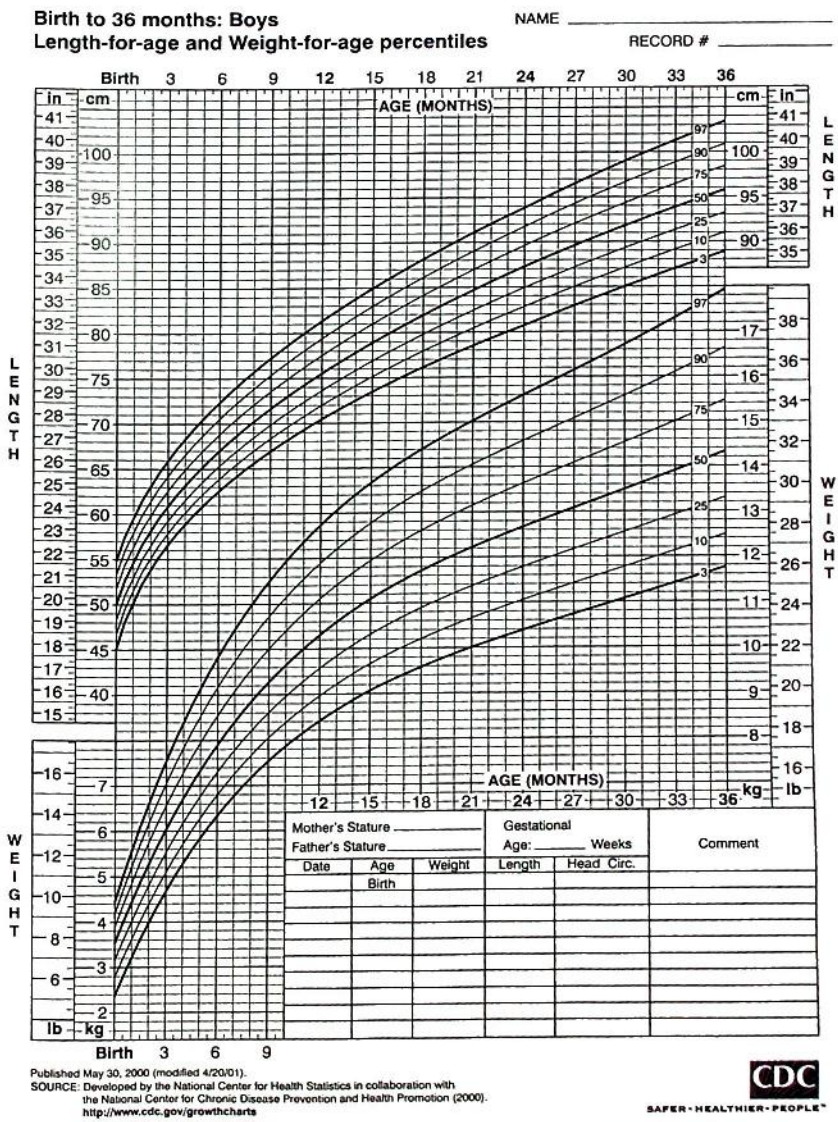


Figure A.4 Boys: Birth–36 months. Length-for-age and Weight-for-age percentiles. (Published 30 May 2000; modified 4/20/01.) *Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000), http://www.cdc.gov/growthcharts*

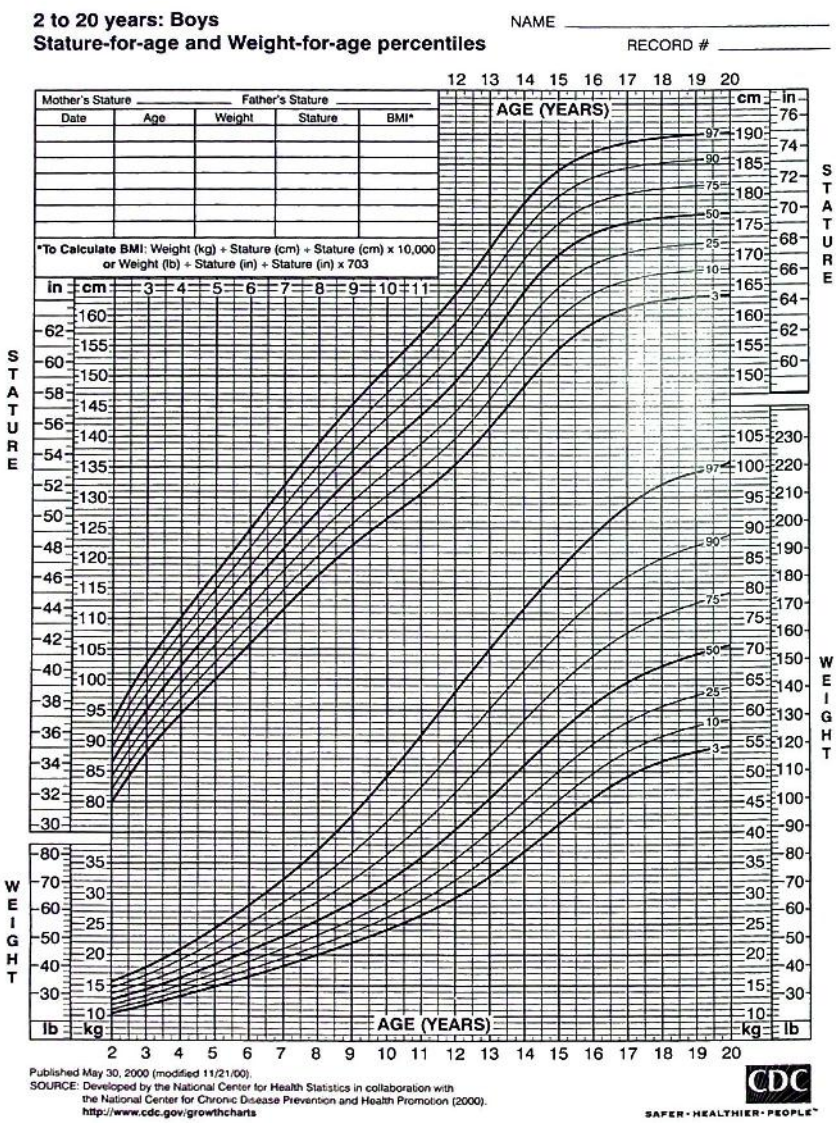


Figure A.5 Boys: 2–20 years. Stature-for-age and Weight-for-age percentiles. (Published 30 May 2000; modified 11/21/00.) *Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000), http://www.cdc.gov/growthcharts*

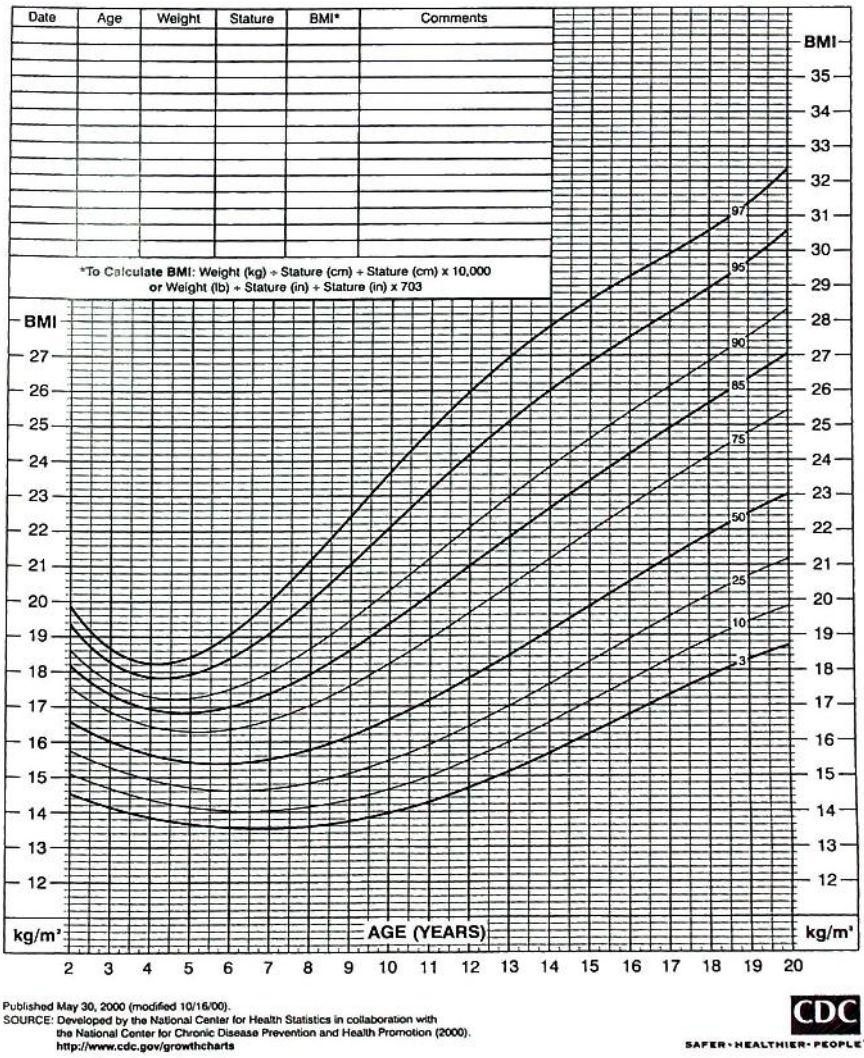


Figure A.6 Boys: 2–20 years. Body mass index-for-age percentiles. (Published 30 May 2000; modified 10/16/00.) *Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000), http://www.cdc.gov/growthcharts*

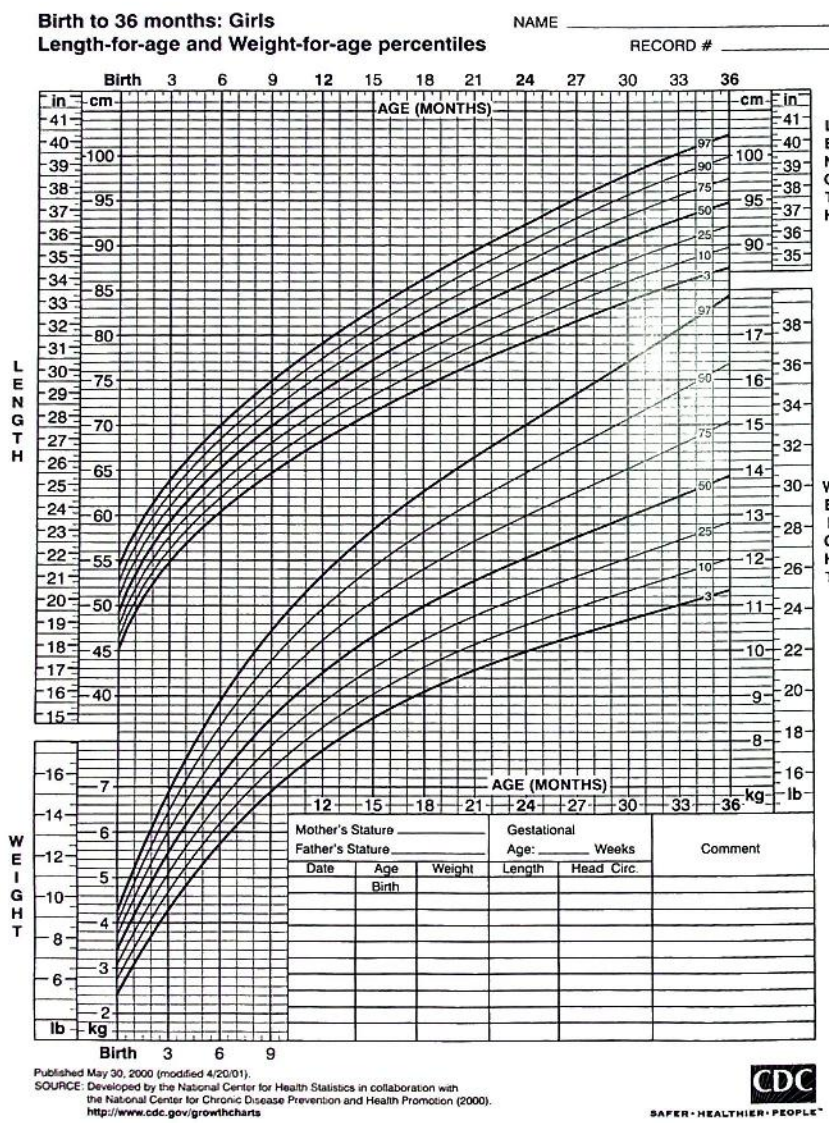


Figure A.7 Girls: Birth–36 months. Length-for-age and Weight-for-age percentiles. (Published 30 May 2000; modified 4/20/01.) *Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000), http://www.cdc.gov/growthcharts*

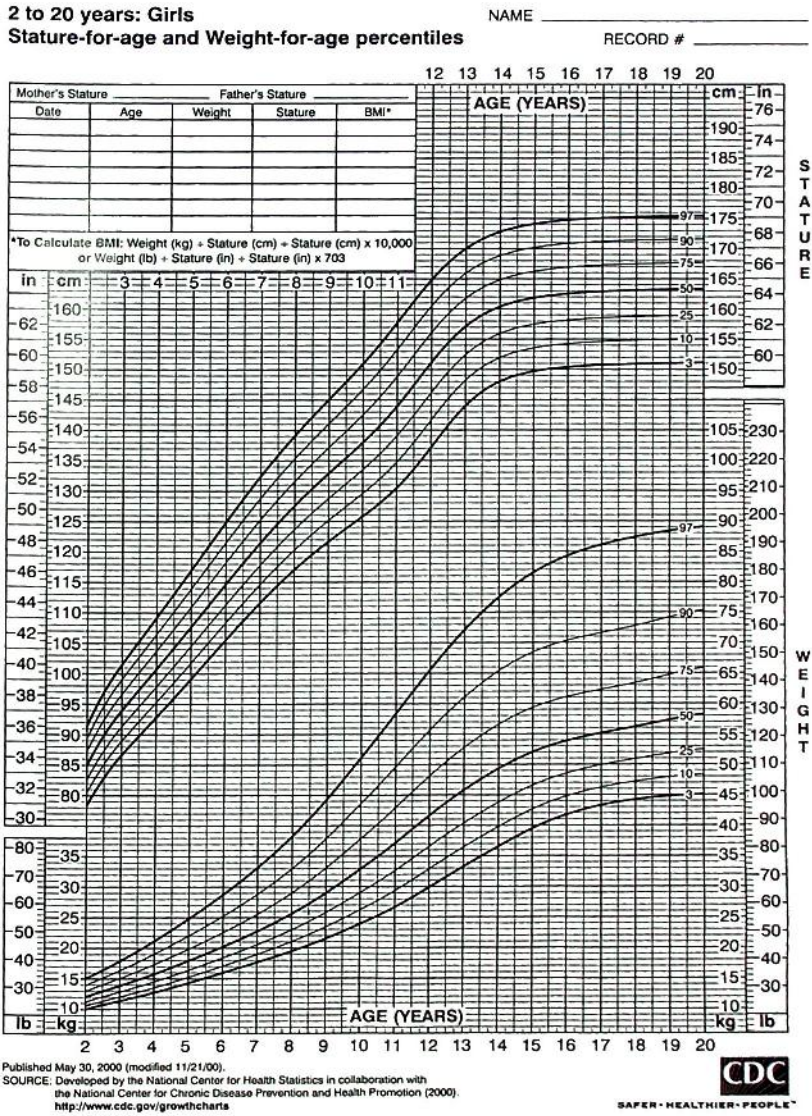
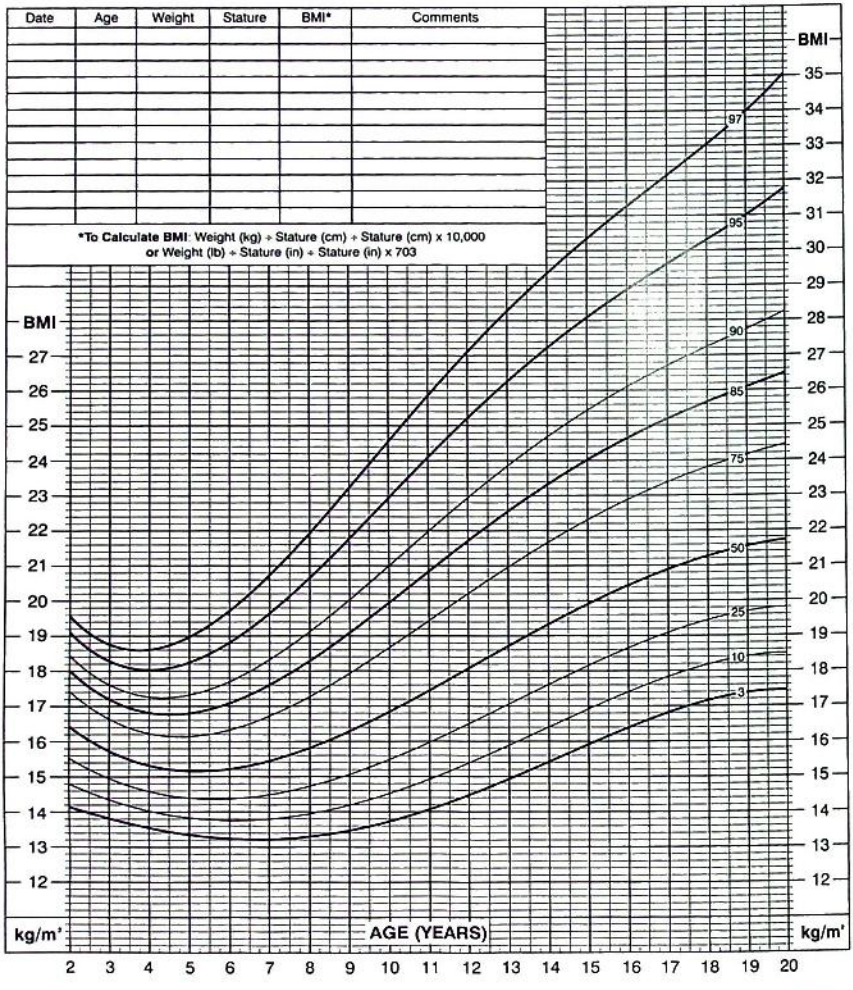


Figure A.8 Girls: 2–20 years. Stature-for-age and Weight-for-age percentiles. (Published 30 May 2000; modified 11/21/00.) *Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000), http://www.cdc.gov/growthcharts*

2 to 20 years: Girls
Body mass index-for-age percentiles

NAME _____

RECORD # _____



Published May 30, 2000 (modified 10/16/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000)
http://www.cdc.gov/growthcharts

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Figure A.9 Girls: 2–20 years. Body mass index-for-age percentiles. (Published 30 May 2000; modified 10/16/00.) *Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000), http://www.cdc.gov/growthcharts*

Appendix H: Glasgow Coma Scale

The Glasgow Coma Scale (GCS) is a rating score for head injury and the score gives an indication of degree of injury and level of consciousness. Table A.14 has been modified for children by the Adelaide Women's and Children's Hospital, as the response scores are usually lower in children. Children between 6 months and 2 years may localize pain but not obey commands, and before 6 months, the best score is withdrawal from pain or abnormal extension and flexion. There is no modification of the adult eye-opening scale. Verbal responses should be consistent with age.

Modified Glasgow Coma Scale Outcomes

In children with GCS scores of 3 or 4, there are significant mortality rates (between 20% and 70%), whereas in those with scores over 5, there is low mortality and morbidity (<30%). If a child does not die within the first 24 h, the risk of death falls to between 10% and 20%. Some 64% of children who do not open their eyes spontaneously within 24 h will die or survive in a vegetative state. It is important to note that over 90% of children who are comatose initially with a GCS score >3 will recover to an independent state, although 50% will have neurological impairment. If coma persists for longer than 3 months, there is almost always neurological and cognitive damage.

Table A.14 Modified Glasgow Coma Scale

	Response	Response for infants	Score
Eye opening	Spontaneously	Spontaneously	4
	To speech	To speech	3
	To pain	To pain	2
	None	None	1
Verbal	Orientated	Coos and babbles	5
	Words	Irritable cries	4
	Vocal sounds	Cries to pain	3
	Cries	Moans to pain	2
	None	None	1
Motor	Obeys commands	Normal spontaneous movements	6
	Localizes	Withdraws to touch	5
	Withdraws from pain	Withdraws from pain	4
	Abnormal flexion to pain	Abnormal flexion	3
	Extension to pain	Abnormal extension	2
	None	None	1
Best possible score			15

Appendix I: Common drugs usage in paediatric dentistry

Table A.15 Common drug usage in paediatric dentistry

Drug	Route	Dose	Frequency	Max dose	Indications	Contraindications/ notes
Antibiotics						
Amoxicillin	PO	15–25 mg/kg/ dose	t.d.s.	4 g/day	Antibiotic of first choice, except in allergic patients	Syrup or chewable tablets for young children
	IV	25 mg/kg/dose	t.d.s.	8 g/day	Antibiotic of first choice, except in allergic patients	
	PO IV	50 mg/kg up to adult dose 2 g	60 min prior stat	2 g adult dose	ENDOCARDITIS PROPHYLAXIS	
Amoxicillin plus clavulanic acid	PO	22.5 mg/kg/ dose	b.d.	1.5 g/day	Severe/persistent dental infections	For β-lactam resistant organisms only
Ampicillin	IV	25 mg/kg/dose	q.i.d.	12 g/day		
	IV	50 mg/kg up to adult dose 2 g	stat	2 g adult dose	ENDOCARDITIS PROPHYLAXIS	
Benzylpenicillin	IV	30 mg/kg/dose	q.i.d.	1.2 g/dose	First IV drug of choice for odontogenic infections	
Phenoxymethylpenicillin potassium	PO	10–12.5 mg/kg/ dose	q.i.d.	500 mg/dose		Must be given on an empty stomach
Cefalexin	PO	12.5–25 mg/kg/ dose	q.i.d.	1 g/dose		
	PO	50 mg/kg up to adult dose 2 g	60 min prior	2 g adult dose	ENDOCARDITIS PROPHYLAXIS	
Cefalotin	IV	25 mg/kg/dose	q.i.d.	2 g/dose		Not for use in pregnancy
Cefazolin	IV	50 mg/kg up to adult dose 1 g	stat	1 g adult dose	ENDOCARDITIS PROPHYLAXIS	

etronidazole	IV	12.5 mg/kg/dose	b.d.	500 mg/dose	Supplement to penicillins in cases of severe or protracted infection	Not for use in pregnancy
	PO	10 mg/kg/dose	t.d.s.	400 mg/dose		
Clindamycin	PO IV	10 mg/kg/dose	t.d.s.	450 mg/dose	Antibiotic of first choice in penicillin-allergic patients	Low risk of pseudomembranous colitis with protracted use
	PO IV	20 mg/kg up to adult dose 600 mg	60 min prior stat	600 mg adult dose	ENDOCARDITIS PROPHYLAXIS	Oral 1 h or IV stat before procedure
Azithromycin Clarithromycin	PO	15 mg/kg up to adult dose 500 mg	60 min prior	500 mg adult dose	Alternative for ENDOCARDITIS PROPHYLAXIS	Macrolide antibiotics – supersedes erythromycin
Antifungals						
Nystatin	PO	<2 years 50 000–100 000 U >2 years 100 000–500 000 U	q.i.d.		Topical antifungal	Drops – apply to affected area Tablets – chew slowly
Miconazole	PO	½ tbsp	b.d.–q.i.d.		Topical antifungal	Oral gel – apply to affected area
Fluconazole	PO IV	3–6 mg/kg/day	daily	400 mg/day	Systemic antifungal Candidiasis prophylaxis and treatment in immunosuppressed patients	Warfarinized and renal patients. Change to oral as soon as possible. Multiple drug interactions
Amphotericin B	PO	10 mg/dose (NOT kg)	6-hourly		Topical antifungal	Lozenge – patient to chew slowly
	PO	100 mg/mL	6-hourly		Topical antifungal	Suspension – apply to affected area
	PO	3%	6-hourly		Topical antifungal	Ointment – apply to affected area

Table A.15 *Continued*

Drug	Route	Dose	Frequency	Max dose	Indications	Contraindications/ notes
Antivirals						
Aciclovir	PO IV	20 mg/kg/dose 10 mg/kg/ dose	5-hourly tds		Early primary herpetic gingivostomatitis	Immunosuppressed patients, should be prescribed within 72 h of infection
Analgesics						
Xylocaine (viscous)	PO	2% 5 mL	3-hourly			Rinse mouth/gargle for 30 s No food or drink for 1 h after
Aspirin						Should not be used in children under 12 years of age due to the risk of Reye's syndrome
Paracetamol	PO PR	15 mg/kg/dose	q4–6-hourly	60 mg/kg/ day up to 4 g/day		Hepatotoxic if overdose. Be aware of different presentations of paracetamol
Ibuprofen	PO	5–10 mg/kg/ dose	q6–8-hourly	40 mg/kg/ day (2 g/ day)		
Naproxen	PO	10–20 mg/kg/ day	q8–12-hourly	1 g/day		
Diclofenac	PO PR	1 mg/kg/dose	q8–12-hourly	3 mg/kg/day (150 mg/ day)		Only in children >10 kg
Codeine phosphate	PO	0.5–1 mg/kg/ dose	q4–6-hourly	60 mg/kg/ dose		Similar side-effects of narcotics including nausea and constipation
Oxycodone	PO	0.1–0.25 mg/ kg/dose	q4–6-hourly			

Morphine	PO IV IM	0.2–0.5 mg/kg/dose 100–200 µg/kg/dose	q4–6-hourly q2–4-hourly	15 mg/dose		Should only be used in admitted patients
Tramadol	PO	1–2 mg/kg/dose	q6-hourly	6 mg/kg/day (400 mg/day)		
Naloxone	IM IV	5–10 µg/kg	Single dose	10 mg total	Narcotic overdose	May be repeated at 2–3 min intervals if necessary
Midazolam	PO PN IV	0.3 mg/kg 0.2 mg/kg 0.1–0.2 mg/kg	Single dose	10 mg 5 mg	Sedation	
Flumazenil	IV	5 µg/kg repeated every minute up to 40 µg/kg	Every minute	Total 2 mg/dose	Benzodiazepine reversal	Complex to administer – refer to product information sheet. Requires IV access in emergency resuscitation
Chloral hydrate	PO	10–20 mg/kg/dose 30–50 mg/kg	6-hourly Single dose	500 mg/dose 1 g	Sedation Hypnotic or premed	Avoid with renal or hepatic impairment
Temazepam	PO	0.3 mg/kg/dose	Single dose	20 mg/dose	Single dose for sedation	
Antiemetics						
Metoclopramide	PO IM IV	0.1–0.15 mg/kg	Single dose	0.5 mg/kg/day (30 mg/day)	Antinauseant/antiemetic	Single dose after narcotic if vomiting or nausea. Dystonic extrapyramidal reactions may occur
Ondansetron	IV	0.1–0.15 mg/kg	Single dose	4 mg/dose	Antinauseant/antiemetic Postoperative nausea and vomiting	Centrally acting Often used in chemotherapy recipients

Table A.15 *Continued*

Drug	Route	Dose	Frequency	Max dose	Indications	Contraindications/ notes
Corticosteroids						
Kenalog in Orabase	PO	Triamcinolone 0.1%	4–6-hourly	–	Mild–moderate oral ulceration	Ointment – apply to ulcers but do not rub in
Betamethasone in Orabase	PO	0.1% compound 1:1 w/w with Orabase	4–6-hourly		Moderate to severe ulceration	Ointment – severe ulceration Apply to ulcer but do not rub in
Dexamethasone	PO IV	0.1 mg/mL 0.1–0.2 mg/kg/ dose	4–6-hourly Single dose		Moderate to severe ulceration Reduction in postsurgical inflammation	Mouthwash for very severe mucosal ulceration 5 mL rinse for 5 min and spit out well
Prednisolone	PO	'Pulse' dose of up to 2 mg/ kg/day	6–12-hourly		Severe intractable immune-mediated oral ulceration	Week-long course, rapid taper
Antifibrinolytics						
ε aminocaproic acid (EACA)	IV	30 mg/kg	stat			Patients haemorrhagic diathesis loading dose of 100 mg/kg
Tranexamic acid	PO	15–20 mg/kg	q.i.d.			
Tranexamic acid	PO	10% compounded mouthwash	q.i.d.			Mouthwash – 5 mL rinse, then spit out well
DDAVP	IV	0.3 mg/kg	Slowly infuse over 60 min	20 µg/dose	Give before surgery	Infused over 1 h before surgery

Clinicians are warned to prescribe and administer any drug carefully. The dosages in the table above are provided as a guide to the usage of medications in paediatric dental practice and clinicians should also consult their relevant pharmacopoeia. Take care when determining maximal dose and frequency of administration.

Appendix J: Eruption dates of teeth

Table A.16 Development of primary teeth

Tooth	Initiation (weeks *in utero*)	Calcification begins (weeks *in utero*)	Crown formation at birth (38–42 weeks)	Crown complete (months)	Eruption (months)
Central incisor	7	13–16	5/6 maxilla 4/5 mandible	1–3	6–9
Lateral incisor	7	14–16	2/3 maxilla 3/5 mandible	2–3	7–10
Canine	7.5	15–18	1/3	9	16–20
First molar	8	14.5–17	Cusps united Occlusal surface complete 1/2 to 3/4 crown height	6	12–16
Second molar	10	16–23.5	Cusps united 1/4 crown height	10–12	23–30

From: Logan & Kronfeld (1933); Shour & Massler (1940).

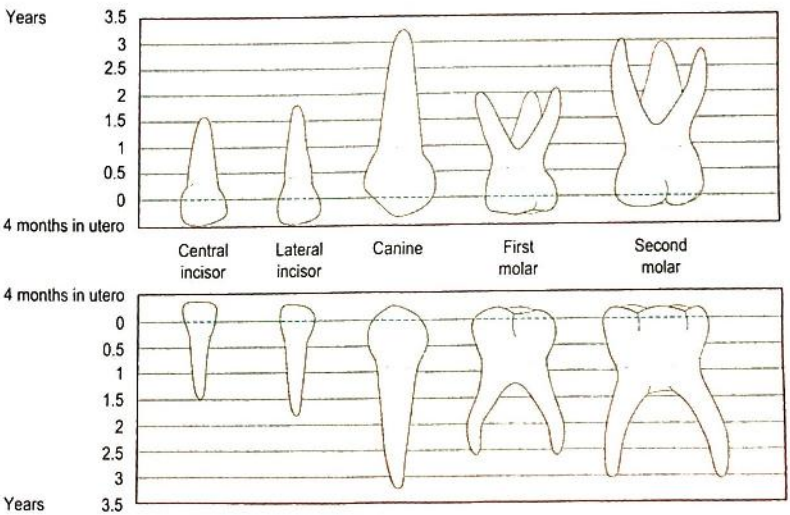

Figure A.10 Chronological development of primary dentition. (After Pindborg 1970 Pathology of the dental hard tissues. WB Saunders, Philadelphia, with permission.)

Table A.17 Development of permanent teeth

Tooth	Initiation	Calcification begins	Crown complete (years)	Eruption (years)
Mandible				
Central incisor	5–5.25 miu	3–4 months	4–5	6–7
Lateral incisor	5–5.25 miu	3–4 months	4–5	7–8
Canine	5.5–6 miu	4–5 months	6–7	9–11
First premolar	Birth	1.75–2 years	5–6	10–12
Second premolar	7.5–8 months	2.25–2.5 years	6–7	11–12
First molar	3.5–4 miu	Birth	2.5–3	6–7
Second molar	8.5–9 months	2.5–3 years	7–8	11–13
Third molar	3.5–4 years	8–10	12–16	17–21
Maxilla				
Central incisor	5–5.25 miu	3–4 months	4–5	7–8
Lateral incisor	5–5.25 miu	11 months	4–5	8–9
Canine	5.5–6 miu	4–5 months	6–7	11–12
First premolar	Birth	1.25–1.75 years	5–6	10–11
Second	7.25–8 months	2–2.5 years	6–7	10–12
First molar	3.5–4 miu	Birth	2.5–3	6–7
Second molar	8.5–9 months	2.5–3 years	7–8	12–13
Third molar	3.5–4 years	7–9 years	12–16	17–25

miu, months *in utero*.

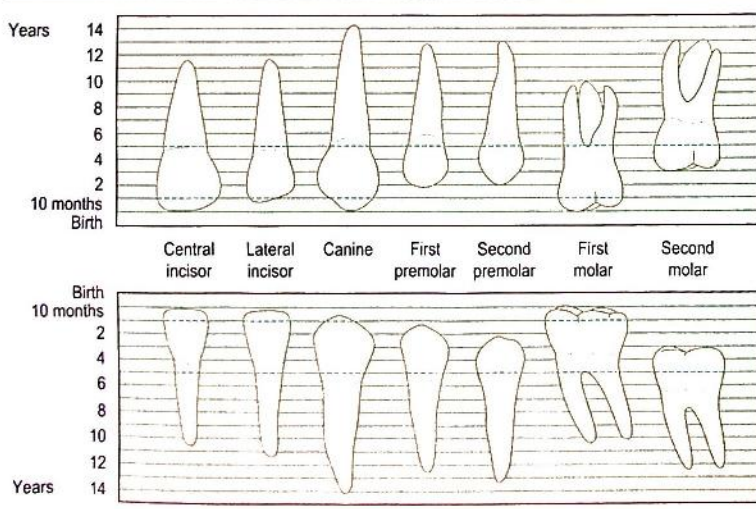

Figure A.11 Chronological development of permanent dentition. (After Pindborg 1970 Pathology of the dental hard tissues. WB Saunders, Philadelphia, with permission.)

Notes on eruption of teeth

All these values are based on work that was published over 50 years ago. At time of writing, there has been very little up-to-date work on the eruption of teeth. It should be noted that there is extreme variability within normal populations and it is of more value to compare the eruption pattern of the whole dentition rather than one particular tooth. Eruption sequence is of particular importance and may be indicative of pathology, e.g. a supernumerary tooth blocking the eruption of a central incisor.

Further reading

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Shour, I., Massler, M., 1940. Studies in tooth development. The growth pattern of human teeth. Journal of the American Dental Association 27, 1918–1931.

Appendix K: Construction of family pedigree

Pedigree is a useful presentation of families in clinical notes. It displays information about past generations and the transmission of genetic traits through families. The symbols used in constructing pedigrees are shown in Figure A.12.

The affected individual at examination is termed the proband and an arrow is placed indicating this patient. Generations are numbered with roman numerals, and arabic numbers are used to indicate individuals within each generation. An example of a family pedigree displaying a sex-linked transmission is shown in Figure A.13.

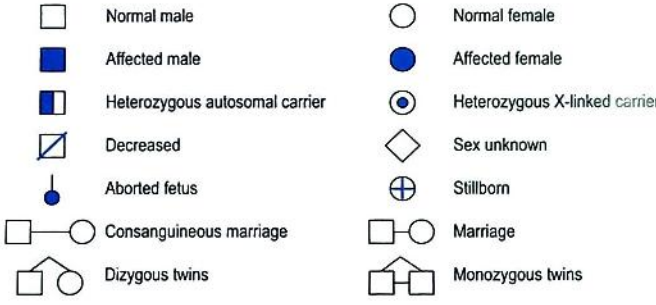

Figure A.12 Symbols used in the construction of pedigrees. (After Pindborg 1970 Pathology of the dental hard tissues. WB Saunders, Philadelphia, with permission.)

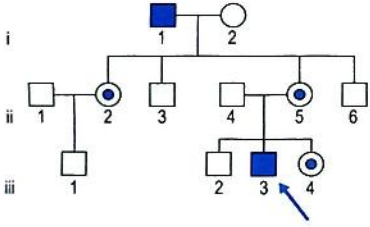

Figure A.13 A pedigree of a family with ectodermal dysplasia, demonstrating sex-linked inheritance. In the first generation, the grandfather (i1) of the proband (iii3) (arrow) had no hair, was hyperthermic and only had three permanent teeth. Of his offspring, all the females were heterozygotes (ii2 and ii5). The younger had sparse hair and eczema and was missing seven teeth, including the lower canines. It is important to note that there is no male-to-male transmission. The daughter then passed the mutation to one of her sons (iii3), who fully expresses the gene, and one of her daughters, who is a carrier. (After Pindborg 1970 Pathology of the dental hard tissues. WB Saunders, Philadelphia, with permission.)

Appendix L: Calculating fluoride values for dental products

The basic unit used for measuring and comparing fluoride products is 'parts per million' (ppm). This is a water engineering term that has been adopted by the dental profession. One ppm is equal to 1 mg in 10^6 mg (or 1 kg). One ppm is 1 mg dissolved in 1 L of water; 1 L of water weighs 1 kg.

Some useful analogies for thinking about 1 ppm are as follows:

- 1 inch in 16 miles.
- 1 minute in 2 years.
- 1 cent in $10000.
- 1 drop in 10 gallons.
- 1 mL in 1000 L.

The molecular weight ratio (MWR) is used to calculate the fluoride content of fluoride products. Most fluoride products are labelled with the concentration of the compound (e.g. 2% NaF), rather than the fluoride content. An exception to this labelling occurs with the 1.23% APF gels that are unusual in being labelled with the fluoride concentration (i.e. 1.23% F or 12300 ppm F).

Sample calculations

Sodium fluoride

A NaF compound contains:

- Na (atomic weight: 23) and F (atomic weight: 19).
- NaF then contains 19+23 = 42 (combined atomic weight). In this compound.
- F then represents 19/42 = 0.45 (MWR for F).
- Na represents 23/42 = 1.8 (MWR for Na).

Consider a 2%NaF product:

In order to calculate the %F in this product, the numerical % concentration is multiplied by the MWR for F: $2 \times 0.45 = 0.91\%$F.

To convert 0.91%F to ppm, multiply by 10^4 (since 1%F = 10000 ppmF):

- $0.91 \times 10^4 = 9100$ ppmF.

Sodium monofluorophosphate (MFP)

Na_2FPO_3:

- 2Na (atomic weight: 46).
- F (atomic weight: 19).
- PO_3 (atomic weight: 79). In this compound.
- F represents 19/144 = 0.132% F.
- 0.76% MFP toothpaste contains: $0.76 \times 0.132 = 0.1\%$ F or 1000 ppm F.

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