



ABC of oral health: Oral health and disease

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BMJ 2000;320:1652-1655
doi:10.1136/bmj.320.7250.1652

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ABC of oral health

Oral health and disease

Ruth Holt, Graham Roberts, Crispian Scully

A healthy dentition and mouth is important to both quality of life and nutrition, and oral disease may affect systemic health, as discussed in later articles in this series.

Development of the dentition

Teeth form mainly from neuroectoderm and comprise a crown of insensitive enamel surrounding sensitive dentine and a root that has no enamel covering. Teeth contain a vital pulp (nerve) and are supported by the periodontal ligament, through which roots are attached into sockets in the alveolar bone of the jaws (maxilla and mandible). The fibres of the periodontal ligament attach through cementum to the dentine surface. The alveolus is covered by the gingivae, or gums, which, when healthy, are pink, stippled, and tightly bound down and form a close fitting cuff with a small sulcus (gingival crevice) round the neck (cervical margin) of each tooth.

The primary (deciduous or milk) dentition comprises four incisors, two canines, and four molars in each jaw (total of 20 teeth). The normal permanent (adult) dentition comprises four incisors, two canines, four premolars, and six molars in each jaw (32 teeth).

Tooth development begins in the fetus, at about 28 days in utero. Indeed, all the primary and some of the permanent dentition start to develop in the fetus. Mineralisation of the primary dentition begins at about 14 weeks in utero, and all primary teeth are mineralising by birth. The permanent incisors and first molars begin to mineralise at or close to the time of birth, while the other permanent teeth start to mineralise later. Tooth eruption occurs after formation and mineralisation of the crown are largely complete but before the roots are fully formed.

Neonatal teeth are uncommon and may be loose. They may damage the mother's nipple during suckling, in which case they might need to be removed.

- Tooth development begins in utero
- Root formation finalises after eruption
- Full primary dentition has 20 teeth
- Full permanent dentition has 32 teeth

Teething

Eruption of primary teeth may be preceded by a bluish gingival swelling, usually a result of a transient haematoma and, rarely, an eruption cyst, which usually ruptures spontaneously. Tooth eruption may be associated with irritability, disturbed sleep, cheek flushing, drooling, and sometimes a small rise in temperature or a circumoral rash, but it does not cause diarrhoea or bronchitis (although these may occur coincidentally).

Delays in tooth eruption

A delay in eruption of up to 12 months may be of little or no importance in an otherwise healthy child. Localised delays often result from local factors such as a tooth in the path of eruption,

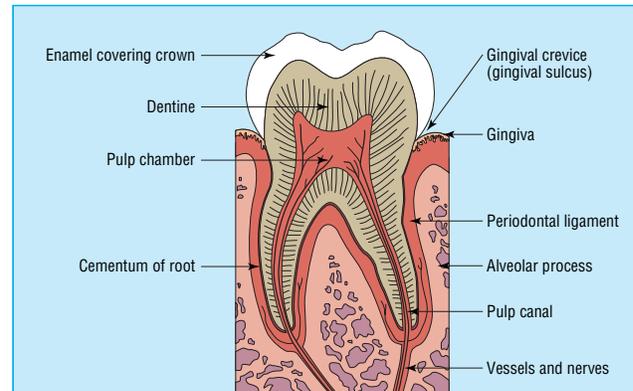
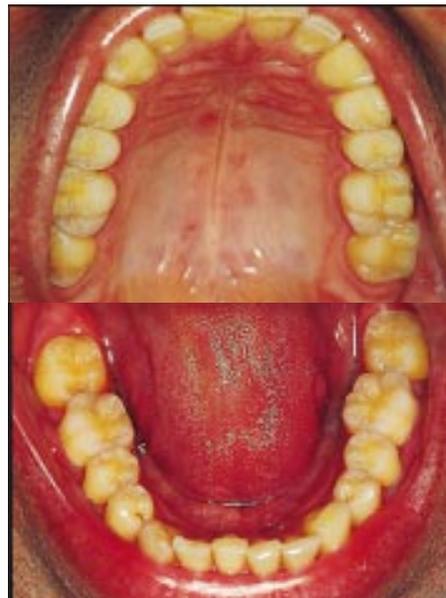


Diagram of a tooth and supporting structures



Healthy dentition, increasingly seen as caries declines

Average times of tooth eruption

	Upper teeth	Lower teeth
Primary teeth		
Central incisors	8-13 months	6-10 months
Lateral incisors	8-13 months	10-16 months
Canines (cuspids)	16-23 months	16-23 months
First molars	13-19 months	13-19 months
Second molars	25-33 months	23-31 months
Permanent teeth		
Central incisors	7-8 years	6-7 years
Lateral incisors	8-9 years	7-8 years
Canines (cuspids)	11-12 years	9-10 years
First premolars (bicuspid)	10-11 years	10-12 years
Second premolars (bicuspid)	10-12 years	11-12 years
First molars	6-7 years	6-7 years
Second molars	12-13 years	11-13 years
Third molars	17-21 years	17-21 years

insufficient space in the dental arch, or dental infection. Ectopic positioning and impaction most often affect the third molars, second premolars, and canines, possibly because these are the last teeth to erupt.

More generalised failure of eruption is rare but may be associated with a variety of systemic causes.

- Teething may cause irritability, drooling, and a small rise in body temperature
- Failed eruption of single teeth is often caused by impaction

Early loss of teeth

Early tooth loss is usually because of extraction as a result of dental caries or, in adults, periodontal disease. Teeth, particularly incisors, may also be lost through trauma, such as from sports, assaults, or other injuries.

Unexplained early tooth loss in children or adults may be a feature of Down's syndrome, diabetes, immune defects, or non-accidental injury, or of rare conditions such as eosinophilic granuloma, hypophosphatasia, or Papillon-Lefèvre syndrome (palmoplantar hyperkeratosis).

- Most tooth loss is due to caries, periodontal disease, or trauma
- Early tooth loss may have a systemic cause

Variations in tooth number

Teeth missing from the normal series may have failed to develop (hypodontia) or to erupt or have been lost prematurely.

Hypodontia is not uncommon and is probably of genetic origin. The teeth most often missing are the third molars, second premolars, and maxillary lateral incisors, and other teeth may be reduced in size. Several teeth may be absent in disorders such as Down's syndrome and ectodermal dysplasia.

Mixed dentition—It is not uncommon to see what seem to be two rows of teeth in the lower incisor region, when permanent teeth erupt before the primary incisors have exfoliated. This is particularly likely when there is inadequate space to accommodate the larger permanent teeth. The situation usually resolves as primary incisors are lost and the mandible grows.

Supplemental teeth—Extra teeth are uncommon. Of unknown cause, they are most often seen in the regions of the maxillary lateral incisors, premolars, and third molars. Additional teeth of abnormal form (supernumerary teeth) are also rare. They are usually small and conical in shape and are seen particularly in the maxillary midline, where they may remain unerupted and may cause a permanent incisor to impact. Additional teeth often occur alone in otherwise healthy individuals but occasionally occur in association with rare disorders such as cleidocranial dysplasia and Gardner's syndrome.

- Missing teeth may be due to failed eruption, tooth loss, or hypodontia
- Hypodontia is genetic and is seen in Down's syndrome and ectodermal dysplasia
- Supplemental or supernumerary teeth are genetic and occasionally occur with systemic disorders

Causes of delayed tooth eruption

Local

- Impacted teeth

Iatrogenic

- Cytotoxic therapy
- Radiotherapy

Uncommon or rare systemic causes

- Down's syndrome
- Cleidocranial dysplasia
- Congenital hypopituitarism
- Congenital hypothyroidism
- Gaucher's disease
- Osteopetrosis

Main causes of early loss of teeth

Local causes

- Caries
- Periodontal disease
- Trauma

Systemic causes

Main systemic features

Systemic causes	Main systemic features
<i>Genetic defects</i>	
Down's syndrome	Learning disability, short stature
Papillon-Lefèvre syndrome	Palmar-plantar hyperkeratosis
Juvenile periodontitis and related disorders	Sometimes neutrophil defects
Ehlers-Danlos syndrome type VIII	Hypermobility
Chédiak-Higashi syndrome	Recurrent infections
Eosinophilic granuloma	Bone lesions
<i>Immune defects</i>	
Neutropenia	Recurrent infections
Neutrophil defects	Recurrent infections
Monocyte defects	Recurrent infections
Interleukin 1 abnormalities	Recurrent infections
HIV infection and AIDS	Recurrent infections
<i>Enzyme defects</i>	
Acatalsia (absent catalase)	Recurrent infections
Hypophosphatasia (low alkaline phosphatase)	Recurrent infections



Hypodontia with many missing permanent teeth, including upper lateral incisors and lower central incisors. This appearance may be seen in ectodermal dysplasia



Supernumerary teeth erupting palatal to upper central incisors. Such teeth often remain unerupted and may impede eruption of permanent incisors

Tooth size, shape, structure, and colour

A variety of local and generalised factors may act during tooth formation or mineralisation. Although tooth development in utero is generally well protected, it may be affected by maternal disease and intrauterine infection and by systemic disturbance during early life. Intrauterine infections that may affect tooth structure include rubella and cytomegalovirus. The classic hutchinsonian incisors and Moon's (or mulberry) molars of congenital syphilis are extremely uncommon in developed countries.

Between birth and 6 years of age, the permanent teeth, particularly those of cosmetic importance, may be damaged. Upper permanent incisors may show defects as a consequence of trauma to the primary predecessor. Local infection or trauma may cause a defect in a single tooth or group of teeth. Malformed lower premolars secondary to periapical infection of their primary predecessors are not uncommon and are termed Turner's teeth. More generalised defects may be seen in a range of systemic disorders (prematurity, infections, jaundice, malabsorption, and cytotoxic therapy) during tooth formation and mineralisation, the defect relating to the timing, severity, and duration of the disorder.

Teeth, especially the third molars, may vary in size, form, and structure because of genetic factors. Microdontia (teeth smaller than usual) is largely of genetic origin and usually affects the lateral incisors, which are conical or peg shaped. Teeth that are larger than normal (megadont) are uncommon. Double teeth may be seen occasionally. These seem to be the result of fusion of two teeth and occur most often in the primary dentition, when they are likely to be followed by extra tooth elements in the succeeding permanent dentition.

Superficial tooth discoloration is usually caused by poor oral hygiene or habits such as smoking, consuming certain foods and beverages (such as tea), or taking drugs such as iron, chlorhexidine, or long term oral antimicrobials. In some cultures chewing betel causes staining. Discoloration of a single tooth is usually because the tooth is non-vital, heavily filled, or carious.

Intrinsic staining of a brown or grey colour may be caused by tetracyclines given to pregnant or lactating women or to children under the age of 8 years. Excessive fluoride ingestion during early life may also result in enamel opacities, but, except in those parts of the world where water supplies contain very high levels of fluoride, these are usually extremely mild.

Enamel and dentine defects of genetic origin are rare but are occasionally severe and may take a variety of forms and vary in their inheritance. They can occur in isolation—as amelogenesis imperfecta (defective enamel) or dentinogenesis imperfecta (defective dentine)—or as part of a disorder such as epidermolysis bullosa dystrophica or osteogenesis imperfecta. In some genetic defects of dentine, for example, newly erupted teeth may seem brownish and translucent, an appearance seen in some patients with osteogenesis imperfecta.

- Developing teeth can be damaged by infection, jaundice, metabolic disorders, drugs, and irradiation
- Tetracyclines given to pregnant or lactating mothers, or to children, can discolour teeth
- Inherited disorders of enamel or dentine may cause malformation or discoloration
- Most tooth discoloration is due to poor oral hygiene, diet, or habits



Dilacerated upper central incisor resulting from injury to primary predecessor during permanent tooth development. Severely malformed teeth such as this may require removal, but less severely affected teeth may be treated conservatively



Microdont upper left lateral incisor. The tooth is small in size and conical in form

Causes of tooth discoloration

Extrinsic discolorations (typically brown or black)

- Poor oral hygiene
- Smoking
- Food and drink (such as tea, coffee, red wine)
- Drugs (such as iron, chlorhexidine, antimicrobials)
- Chewing betel

Intrinsic discolorations

Localised

- Trauma (yellow to brown)
- Caries (white, brown, or black)
- Restorative materials (such as black of amalgam)
- Internal resorption (pink spot)

Generalised

- Tetracyclines (brown)
- Excessive fluoride (white or brown)
- Rare causes
 - Amelogenesis imperfecta (brown)
 - Dentinogenesis imperfecta (brown or purple)
 - Kernicterus or biliary atresia (green)
 - Porphyria (red)



Characteristic appearance of teeth of patient with osteogenesis imperfecta who also shows dentinogenesis imperfecta

Anatomical variants

Patients sometimes become concerned after noticing various anatomical variants in the mouth.

Tori and exostoses are bony lumps that appear during tooth development and are especially common in Mongoloid and Negroid races. Torus mandibularis consists of bilateral, asymptomatic, benign bony lumps lingual to the lower premolars. Also common is torus palatinus, a slow growing, asymptomatic, benign bony lump in the midline of the palate. These lumps are usually left alone but are occasionally excised or reduced if they cause severe difficulties with dentures.

Sebaceous glands may be seen as creamy-yellow dots (Fordyce spots) along the border between the lip vermilion and the oral mucosa. Probably 50-80% of the population have them, but they are not usually clinically evident until after the age of 3 years, and they increase during puberty and then again in later adult life. They are totally benign, although occasional patients or physicians become concerned about them or misdiagnose them as, for example, thrush or lichen planus. No treatment is indicated other than reassurance.

Foliate papillae—The size and shape of the foliate papillae on the posterolateral margins of the tongue are variable. These papillae occasionally swell if irritated mechanically or if there is an upper respiratory infection. Located at a site with a high predilection for lingual cancer, they may give rise to anxiety about cancer.

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The ABC of oral health is edited by Crispian Scully and will be published as a book in autumn 2000.

Crispian Scully is grateful for the advice of Rosemary Toy, general practitioner, Rickmansworth, Hertfordshire.

BMJ 2000;320:1652-5



Torus palatinus, with large central torus on palate



Fordyce's spots—sebaceous glands close to vermilion border between lip and buccal mucosa

Further reading

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A message from Holland

Eleven years ago a lively 85 year old man came to my surgery. He had been a famous violinist and concert leader for most of his professional life and still enjoyed playing music. He asked me if I would "help" him should he find himself the victim of a serious illness. In the Netherlands this really means, "What is your view on euthanasia, Doc?" I replied that given such circumstances I would be prepared to help him.

During the next few years I visited him several times, and then at the age of 95 and still in possession of his mental capacities he was diagnosed with prostate cancer and metastases in the lumbar spine causing a spastic paraparesis. My patient was treated with radiation, but this failed to prevent the growth of the tumour. Doubly incontinent—he was catheterised and wore a continence pad as precaution—this elderly and proud man had to be assisted in most activities of daily living. The pain was hardly relieved by treatment with morphine.

He found his total dependence on others intolerable and requested euthanasia. After a wonderful life, he wished to die with dignity. Fully supported by his wife, he repeated his request several times and also set out his wishes and reasoning in a letter. In his eyes he was experiencing intolerable suffering with no hope of relief and the sole prospect of losing his dignity. His request was consistent, voluntary, and documented. Thus all Dutch criteria for euthanasia were met and I had no fear of prosecution.^{1,2} All that I needed was a second opinion from an uninvolved colleague. Yet, I had grave doubts. Had I really tried all aspects of palliative care? In my opinion his life was more than worth living: he still composed music and received visitors every

day. With the utmost difficulty I was able to postpone the decision by convincing him of the importance of a family discussion at which I would be present.

On the dreaded day I approached with lead in my boots. I knew that I was not ready to perform euthanasia; this man was not in a terminal phase and was still involved in many aspects of life. However, I was nervous of his reaction. I had the nagging thought that I was going back on a promise made 10 years earlier.

His daughter opened the door and with a smile on her face exclaimed, "Dad has decided to live." I was offered a glass of champagne (a few bottles had already been cracked) and we all toasted life. I asked the maestro what had made him have such a sudden change of heart. He told me that his 93 year old sister had visited him the day before and had had a few words. "John," she said, "I hear you are considering euthanasia. Mother would never have approved." As if by magic, he changed his mind. I realised that despite my best moral efforts I had not been able to sound the right chord. A few words from his mother, already 50 years in eternity, sufficed to reach his soul. Half a year later he died peacefully, a natural death, with dignity.

H A Thiadens *general practitioner, Amersfoort, the Netherlands*

- 1 Van der Maas PJ, vander Wal G, Haverkate I, de Graaf CLM, Kester JGC, Onwuteaka-Philipsen BD, et al. Euthanasia, physician-assisted suicide, and other medical practices involving the end of life in the Netherlands, 1990-1995. *New Engl J Med* 1996;335:1699-705.
- 2 Sheldon T. Dutch doctors tighten euthanasia guidelines. *BMJ* 1995;311:708.