

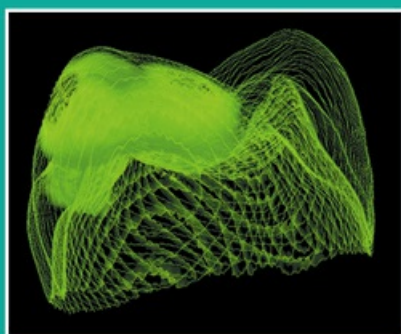
THIRD EDITION

Pediatric Dentistry

A Clinical Approach

EDITED BY

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WILEY Blackwell

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[Figure 8.5 "Tell-show-do" technique might be useful to obtain good child cooperation. Before exposure of the child the radiographic procedure can be demonstrated on a teddy bear while the child is watching from a "safe" place.](#)

[Figure 8.6 Image receptor holders for bitewing radiographs. The three to the right have an extraoral beam aiming device.](#)

[Figure 8.7 Radiographic principle for three-dimensional object localization in the horizontal plan. A metal ball positioned on the facial side of the tooth crown and a metal paper collage positioned on the oral side—nearest the image receptor as shown in \(a\) will appear as superimposed objects on a radiograph \(c\) exposed with the X-ray beam orientated perpendicular at the surface of the image receptor \(b\). On a radiograph exposed with the receptor in the same position as in \(b\) but with the X-ray beam orientated left-sided eccentric at the surface of the receptor \(d\), the metal objects appear separated from each other \(e\). The paper collage—placed nearest to the receptor—has moved in the same direction as the X-ray beam \(to the left\) whereas the metal ball—placed nearest to the X-ray focus—has moved in the opposite direction \(to the right\).](#)

[Figure 8.8 Horizontal, three-dimensional object localization of an impacted 13. \(a\) A periapical radiograph exposed with the X-ray beam orientated perpendicular at the region for 13. \(b\) A periapical radiograph exposed with the X-ray beam orientated mesioeccentric at the region for 13. Since 13 moves in the same direction as the X-ray beam \(mesially\) in relation to the root of 12, it is placed nearest the receptor, which means palatally to the root of 12.](#)

[Figure 8.9 Different panoramic segments. \(a\) A dental panoramic radiograph, \(b\) a right-sided panoramic radiograph, \(c\) a lower jaw panoramic radiograph, and \(d\) a bilateral premolar panoramic radiograph.](#)

[Figure 8.10 Stereo scanogram for depth localization of an impacted 15. The X-ray beam orientation at 15 is "disto-eccentric" for the scanogram to the left and "mesio-eccentric" for the scanogram to the right. As 15 moves mesially in relation to the root of 14 on the scanogram to the right compared with the scanogram to the left it is located palatally to the root of 14.](#)

[Figure 8.11 \(a\) Frontal part of a panoramic image showing impaction of 13 and 23. \(b\) CBCT scanning for detailed examination of 23. From 2D-reconstructed images in the coronal \(upper left corner\), sagittal \(upper right corner\) and axial \(lower left corner\) plane transposition of the impacted 23 with 22 is seen. Palatally to](#)

[the crown of 23 a compound odontoma \(blue arrow\) is present. From the 3D model \(lower right corner\) the odontoma is also visible, and it seems to interfere with the eruption of 23.](#)

[Figure 8.12 \(a\) Conventional intraoral radiograph showing an impacted, transversally located 11. \(b\) On a three-dimensional image model from a CBCT examination it is clear that 11 is lacerated and placed with the crown palatally and the root facially to the neighboring teeth.](#)

[Figure 8.13 A neck shield.](#)

Chapter 09

[Figure 9.1 Pain perception based on classical conditioning: a painful procedure based on an unconditioned stimulus \(e.g., tissue damage when drilling into dentin\), combined with other stimuli such as the sound and the water spray of the drill \(conditioned stimuli\), may result in pain perception when the conditioned stimuli are presented alone.](#)

[Figure 9.2 The perception of procedural pain due to tissue damage \(nociceptive pain\) depends on a variety of factors, owing to the fact that the stimuli are modulated in the central part of the brain before reaching the sensory cortex. Previous pain experiences \(psychological pain memory\) and fear are the most important factors contributing to the affective component of pain perception.](#)

[Figure 9.3 \(a\) Topical application of local analgesia ointment on a cotton bud at the injection spot. \(b\) Infiltration analgesia followed by \(c\) a transpapillary injection started from the buccal and \(d\) continuing to the palatal mucosa. Note blanching of the palatal papilla and mucosa in \(c\).](#)

[Figure 9.4 Mandibular block analgesia in a preschool child.](#)

[Figure 9.5 The position of the mandibular foramen changes during growth. However, the mandibular foramen is below the occlusive plane in children. The foramen is always situated on the line, where the ramus is narrowest, two-thirds of the way back from the anterior concavity.](#)

[Figure 9.6 Pictures of skulls. The mental foramen is located closer to the primary mandibular first molar in children and the permanent second premolar in adults.](#)

[Figure 9.7 \(a\) The STA™ System and \(b\) Calaject™ are examples of computerized systems that delivers the analgesic solution with a constant and very slow speed which minimizes the pressure to the tissue and thereby the pain from the injection. \(c\) The AMSA injection technique.](#)

[Figure 9.8 During an injection, the assistant can support the child by holding a hand and also keep the other hand on the child's head to be sure the child does not make a sudden movement.](#)

[Figure 9.9 Side effect of mandibular block analgesia. Bite wound in lower lip.](#)

[Figure 9.10 Blanching of the cheek \(sympathetic reaction\) after local analgesia injection in a child.](#)

[Figure 9.11 Nitrous oxide–oxygen sedation.](#)

[Figure 9.12 \(a\) Oral administration with midazolam mixed in a juice. \(b\) Applicator for rectal administration of midazolam. \(c\) Oral sedation with needleless syringe in a 2-year-old child.](#)

[Figure 9.13 Dental treatment under general anesthesia in a hospital setting. Notice the comprehensive equipment and the large amount of personnel.](#)

Chapter 10

[Figure 10.1 Contribution of initial and manifest lesions, filled and missing \(due to caries\) surfaces of the total caries index in Norwegian children.](#)

[Figure 10.2 Mean number of decayed and filled tooth surfaces according to age in 1973, 1983, 1993, 2003, and 2013, Jönköping, Sweden. For 3- and 5-year-olds only caries in primary teeth were recorded, while only caries in permanent teeth were recorded in subjects 10 years and older. Initial, noncavitated carious lesions were included.](#)

[Figure 10.3 Distribution of 15-year-old Danes according to DMFS \(initial, noncavitated lesions not included\) in 1988, 2006, and 2013.](#)

Chapter 11

[Figure 11.1 The triad forming the base for evidence-based practice.](#)

[Figure 11.2 Basic design of the randomized controlled caries clinical trial. Study subjects should be assigned randomly to one or more experimental or intervention groups and a control or no intervention group. The outcome of the trial is measured by comparing caries increment in the group\(s\) from the beginning to the end of the trial \(\$M_1\$ to \$M_2\$ \).](#)

[Figure 11.3 A conceptual model, where disease is explained as occurring when a number of component causes \(sections of the circle\) act together to form a sufficient cause \(closed circle\). \(a\) High level of plaque, high sugar intake, and high susceptibility to caries will result in a caries lesion. \(b\) If the sugar intake is reduced, \(c\) if the plaque level is reduced, or \(d\) if the susceptibility is decreased, caries lesions will not occur.](#)

[Figure 11.4 Caries balance as influenced by social, behavioral, and biological factors.](#)

[Figure 11.5 Caries risk assessment with Cariogram. Ten variables are computed and the program indicates a 44% chance of avoiding new caries lesions in the near future \(green sector\). The blue sector indicates that high sugar amounts and frequent intakes are the main contributing factor in this case. Targeted preventive measures linked to the individual caries risk profile are suggested. The program is available in several languages at](#)

www.mah.se.

[Figure 11.6 Caries progression. \(a\) An 11 month old girl exposed to frequent intakes of stewed fruits. The parents could not change the diet. \(b\) One year later the incisors had to be extracted. \(c\) A 4 year old boy with developing initial caries lesions. Good parental cooperation. \(d\) Status after 1 year shows no progression of the caries lesions. \(e\) A 6 year old boy with active caries. Intense prophylaxis. \(f\) Status 1 year later shows complete control of caries progression.](#)

[Figure 11.7 The amount of fluoride toothpaste \(little fingernail\) for a 2 year old child.](#)

Chapter 12

[Figure 12.1 How is caries defined? Caries disease is assessed by its signs and symptoms that depend on the severity of the disease. The figure shows the time dependent development of a lesion from a subclinical level to increasing destruction of dental hard tissues.](#)

[Figure 12.2 \(a\) ICDAS based criteria for severity grading of caries on free smooth and occlusal tooth surfaces. \(b\) Alternative index using a five graded scale for severity grading of caries on free smooth, occlusal, and approximal tooth surfaces.](#)

[Figure 12.3 Active and inactive/arrested caries lesions. Upper row shows initial \(noncavitated lesions\) and the lower row shows cavitated lesions. \(a\) Active noncavitated lesions close to the gingival line on the buccal surfaces of primary upper incisors in a 2 year old. There is loss of luster and the lesions are rough on probing. \(b\) Arrested noncavitated lesions on the buccal surfaces of primary upper incisors in a 4 year old. The lesions are situated at a distance from the gingival line, and are shiny and hard on probing. \(c\) Active cavitated lesion in a primary lower second molar in a 5 year old. The dentin is soft on probing and the cavity borders are blunt and irregular. \(d\) Inactive/arrested cavitated lesion in a primary lower first molar in a 7 year old. The dentin is brownish black, hard on probing, and the cavity borders are sharp and regular.](#)

[Figure 12.4 Sectioned premolar with an enamel caries lesion in the fissure before probing \(left\). Intense probing \(right\) destroys the surface zone of the lesion.](#)

[Figure 12.5 A small but obvious occlusal cavity in the central fossa of a permanent first molar \(arrow\). The borders around the cavity are whitish and rough in texture suggesting an active caries process. There is a shadow from underlying dentin caries. The radiograph reveals a substantial radiolucency in the dentin \(arrow\).](#)

[Figure 12.6 \(a\) Light brown discolored fissures in a permanent first molar of an 8 year old. The enamel around the central fossa is whitish and there is softened enamel at the entrance of the fissure indicating an active lesion \(arrow\). \(b\) The radiograph reveals radiolucency in the dentin \(arrow\). \(c\) Dark brown/black discolored fissures in a permanent first molar of a 19 year old with a low caries activity. The fissures are hard on probing indicating an arrested \(inactive\) lesion.](#)

[Figure 12.7 Caries lesions on distal surfaces of two mandibular second premolars: both radiographs \(a and c\) showed radiolucency in outer dentin, but during cavity preparation, a clinical cavity was observed only in one of them \(b\).](#)

[Figure 12.8 Hidden caries under a seemingly sound occlusal surface of a permanent lower second molar in a 14 year old. \(a\) Visual-tactile examination of the surface did not reveal any clear signs of caries. \(b\) The bitewing radiograph shows, however, an obvious radiolucency in the dentin. The presence of soft carious dentin was confirmed at drilling.](#)

[Figure 12.9 \(a\) to \(j\) A 14 year old boy who was treated with bone marrow transplantation \(BMT\). Three months later he developed graft versus host disease, which prolonged his hospitalization to a total of 4 months. His condition and the treatment he received resulted in hyposalivation, and during the treatment period he experienced frequent supply of sugary drinks, inadequate dental hygiene, and little fluoride exposure.](#)

[Figure 12.10 \(a\) A 3 year old boy with high caries activity due to frequent intake of high sucrose containing meals. \(b\) After gross excavation of the caries lesions and application of a temporary zinc oxide-eugenol cement the child is ready for nonoperative treatment.](#)

[Figure 12.11 \(a-c\) A 13 year old girl with active caries and low motivation, treated by the use of general and local caries arresting interventions. \(d\) Five years later: adequate caries control. \(e\) Another 3 years later: still adequate caries control. Observe the glossy surfaces of the previously active initial caries lesions.](#)

[Figure 12.12 Rubber dam for isolating the operation field before restorative therapy of primary molars.](#)

[Figure 12.13 Rubber dam for isolating and drying the operation field before restorative therapy of maxillary incisors.](#)

[Figure 12.14 Severe early childhood caries in a 4 year old child. The caries process started in the upper incisors during the eruption of the teeth, due to sugary drinks at night. Note that first primary molars are also heavily affected. No oral hygiene habits had been introduced.](#)

[Figure 12.15 Use of a thin layer of GIC for the arrest of approximal dentin caries in primary incisors of a 2 year old child. A hand instrument was used to remove soft carious tissue before application of the cement.](#)

[Figure 12.16 Small Class II cavity preparation for GIC in primary molars. Basically, the outline of the cavity is determined by the extent of the caries lesion, but some mechanical retention is advocated in the directions indicated by the arrows.](#)

[Figure 12.17 Large Class II cavity preparation for compomer or composite materials in a primary molar. Retention of the filling is based on mechanical as well as adhesive techniques.](#)

[Figure 12.18 \(a\) Lower right second molar with severe multi surface caries suitable for a stainless steel crown.](#)

(b) Removal of carious tissue reveals pulp exposure. (c) After pulpotomy the cavity is restored with GIC and the crown is prepared for a stainless steel crown. (d) The tooth is restored with a stainless steel crown.

Figure 12.19 (a) Only just enough tooth tissue is removed to adjust the crown to the occluding and neighboring teeth. The gingival contour should be kept intact to retain the crown. (b) Extensive carious defects are initially restored with GIC and the crown is subsequently adapted to the restoration.

Figure 12.20 Fissure sealing covering all parts of the fissure without overfilling and overextension.

Figure 12.21 Enamel surface seen in scanning electron microscope after etching with 30% phosphoric acid for 45 seconds. As an effect of lack of cleaning before etching, black areas with organic material remain (white arrows; magnification $\times 300$).

Figure 12.22 Preventive resin/GIC restoration; R = restorative material (resin or GIC), S = sealant.

Figure 12.23 Class III cavity in maxillary incisor.

Figure 12.24 The relative percentage distribution of approximal (appr) and occlusal (occl) DFS (decayed and filled surfaces) at ages of 13, 19, and 27 years evaluated from radiographic examinations. For approximal surfaces decayed (D) equals a radiolucency at the enamel–dentin border or deeper and for occlusal surfaces D equals an obvious radiolucency in dentin. The same individuals ($n = 250$) were followed from 13 to 27 years of age.

Figure 12.25 Cumulative survival curves of occlusal surfaces (first and second molars) from radiographically sound to obvious radiolucency in dentin. The slopes of the curves show that most new dentin lesions occurred between 12 and 15 years of age and particularly concerned the second molar. It should be noted that, for the first molar, the data show only those who were radiographically sound at age 12 years.

Figure 12.26 Caries rates (number of new lesions/100 tooth surface years) of approximal surfaces from 11 to 22 years of age. Median values of all surfaces.

Figure 12.27 Survival times of approximal caries lesions from 11 to 22 years of age. The ninetieth percentiles of three progression states: from 0 to 2, from 2 to 4, and from 3 to 4.

Figure 12.28 Median values of survival times (years) from caries state 3 to state 4 (progression within dentin) at different approximal surfaces.

Figure 12.29 Cumulative survival curves of approximal surfaces from radiographically sound to inner enamel caries, from inner enamel caries to caries in outer dentin, and from caries at the enamel–dentin border to caries in outer dentin from 12 to 27 years of age.

Figure 12.30 Two 12 year olds; one has almost fully erupted premolars and second molars while the other is still in the process of shedding primary molars.

Figure 12.31 (a) Bitewing radiographs from the same individual from 15 to 21 years of age showing slow progression of lesions on the distal surface of upper left first premolar and mesial surface of the second premolar. The enamel lesions on these surfaces have not progressed into the dentin during these 6 years. (b) A significant example of the rapid progression in newly erupted first molars (tooth 26) in an 11 year old girl. At the follow up after 1 year no bitewing radiographs was taken. At the 2 year follow up the tooth was extracted due to severe caries with pulp involvement.

Figure 12.32 The saucer shaped cavity design: (a) before filling and (b) after filling with a composite resin.

Figure 12.33 (a) The design of the saucer shaped cavity is based on adhesion of composite resins to enamel and dentin. (b) The saucer shaped cavity design (dotted line) saves more tooth tissue than the traditional Class II preparation.

Figure 12.34 Conventional Class II amalgam preparation with minimal buccal–lingual extension.

Chapter 13

Figure 13.1 Distribution of dental erosion by tooth and by gender. Percentages of boys and girls with dental erosion by teeth at (a) 12 years of age and (b) at 15 years of age.

Figure 13.2 Severe erosion on the palatal surface of the upper incisors. The enamel close to the gingival margin is intact and a shoulder is shown.

Figure 13.3 (a) Cuppings on a permanent first lower molar (tooth 36) in a young teenager. (b) Study cast from the same case.

Figure 13.4 Dental erosion as a result of lemon sucking in a 6 year old child. Dental erosion has caused severe endodontic problem especially in region 52 and 51.

Figure 13.5 Near to pulp exposure on the palatal surfaces of anterior primary teeth. Secondary and tertiary dentin are shown.

Figure 13.6 pH decrease (mean values) for three methods of drinking and nipping from a baby's bottle using the microtouch method and Cola Light. Holding = holding the drink in the mouth for 2 min. Long sipping = sipping from a glass for 15 min. Gulping = swallowing quickly three times over 5 min intervals. Baby bottle = nipping from a baby's feeding bottle for 15 min.

Figure 13.7 Dose response relationship between frequency of soft drink intake and number of teeth affected by dental erosion in 7th grade (12 year old) and 10th grade (15 year old) children.

Figure 13.8 Clinical signs of erosion: cuppings on primary molars (teeth 83 and 85) and permanent molar (tooth 46).

[Figure 13.9 Illustrations of different severities of dental erosion graded according to the scale in Box 13.6: \(f\) is a study model of the patient in \(e\) illustrating that intraoral photographs could be combined with grading of study models as the two complement each other in assessment of dental erosion.](#)

[Figure 13.10 \(a\) Fifteen year old girl with palatal erosion and sensitivity on maxillary anterior teeth. \(b\) Composite restoration took place on tooth 12, establishing a new vertical dimension sufficient for restoring 11, 21, and 22 \(c\). \(d\) As a result, posterior disocclusion is present which, however, is normalized after approximately 4 weeks due to \(e\) compensatory eruption and alveolar growth.](#)

Chapter 14

[Figure 14.1 Clinically healthy primary tooth gingiva.](#)

[Figure 14.2 Clinically healthy permanent tooth gingiva.](#)

[Figure 14.3 Chronic gingivitis.](#)

[Figure 14.4 Supragingival calculus.](#)

[Figure 14.5 Bitewing radiographs showing proximal calculus on primary and permanent teeth. Arrow heads indicate subgingival calculus.](#)

[Figure 14.6 Radiographs of a 14 year old boy showing proximal subgingival calculus and minor bone loss at mandibular permanent first molars. Arrow indicates bone loss and arrow heads indicate subgingival calculus.](#)

[Figure 14.7 Black stains can be observed in children.](#)

[Figure 14.8 Gingivitis in relation to dens geminatus \(tooth 41\) in the incisal region of the lower jaw. \(a\) Frontal view of tooth 41. \(b\) Distofacial view of tooth 41.](#)

[Figure 14.9 Poor oral hygiene and gingivitis in a patient undergoing orthodontic treatment.](#)

[Figure 14.10 Chronic gingivitis associated with mouth breathing.](#)

[Figure 14.11 Edematous gingival inflammatory reaction during puberty.](#)

[Figure 14.12 A 14 year old boy with localized aggressive periodontitis \(c, d\). Previously obtained and filed radiographs from the age of 8 years show loss of bone support \(a, b\) \(arrows\)](#)

[Figure 14.13 A 3 year old boy with a generalized form of aggressive periodontitis. The primary teeth in all quadrants are involved.](#)

[Figure 14.14 \(a\) A 13 year old girl with a localized form of aggressive periodontitis, clinically identified with a diastema between permanent maxillary incisors. \(b, c and d\) The radiographs show bone destruction in the same area as well as in the permanent molar regions \(arrows\).](#)

[Figure 14.15 Severe periodontal involvement in a young child suffering from diabetes mellitus.](#)

[Figure 14.16 Aggressive periodontitis in a 19 year old patient with diabetes mellitus with poor metabolic control. \(a\) Clinical picture \(b\) Panoramic radiograph.](#)

[Figure 14.17 Alveolar bone loss in a child with hypophosphatasia.](#)

[Figure 14.18 \(a, b c\) Chronic periodontitis at permanent first molars of a 13 year old girl. Scaling and root planning were performed. \(d, e\) Radiographs taken 6 months later show healing of bone defects.](#)

[Figure 14.19 \(a\) Aggressive periodontitis at permanent molars of an 18 year old adolescent. Thorough scaling and root planing were performed. \(b\) Radiographs taken 1 year later and \(c\) 3 years later show substantial healing of the bone defects.](#)

[Figure 14.20 Periodontal surgery of a 14 year old girl with localized marginal periodontitis in the lower left molar region. \(a\) A bitewing radiograph showing bone loss on tooth 36. \(b\) Intracrevicular incision. \(c\) The gingiva is retracted. \(d\) The exposed root surfaces are subjected to mechanical debridement. \(e\) The flaps are replaced and sutured.](#)

[Figure 14.21 Gingival recession at permanent lower central incisor; \(a\) in a mixed dentition and \(b\) in a permanent dentition.](#)

[Figure 14.22 Phenytoin induced gingival overgrowth.](#)

[Figure 14.23 Gingival fibromatosis \(a\) in a newborn child and \(b\) at 5 years of age after surgical correction.](#)

[Figure 14.24 \(a\) Traumatic ulcerative gingival lesion and \(b\) after treatment.](#)

[Figure 14.25 Foreign body \(a plastic ring or rubber band from children's toys\) in the oral cavity of a 6 month old baby. \(a\) Blue plastic ring/rubber band around tooth 72; \(b\) Blue plastic ring around the column of tooth 72 after extraction.](#)

Chapter 15

[Figure 15.1 Impetigo contagiosa in a 7 year old girl.](#)

[Figure 15.2 Forchheimer's spots on the soft palate in a child with rubella.](#)

[Figure 15.3 Koplik's spots in the cheek mucosa in a child with measles.](#)

[Figure 15.4 Vesicular lesions on the skin in a child with varicella \(chickenpox\).](#)

[Figure 15.5 Vesicles on the mucosa of the tongue in a child with varicella.](#)

[Figure 15.6 \(a\) Herpes simplex lesions spread over the alveolar mucosa and \(b\) tongue.](#)

[Figure 15.7 Herpes labialis.](#)

[Figure 15.8 Wart.](#)

[Figure 15.9 Candidiasis lesion in cheek mucosa.](#)

[Figure 15.10 *Candida albicans* infection of the palatal mucosa in child with a partial denture.](#)

[Figure 15.11 Aphthous ulcer.](#)

[Figure 15.12 Erythema multiforme: \(a\) oral lesions showing vesicles and bullae which rapidly burst; \(b\) lesions after 2–3 days; \(c\) crust formation \(healing\) 2 days later.](#)

[Figure 15.13 Tongue mutilation after local anesthesia.](#)

[Figure 15.14 Crohn's disease, gingival characteristics: \(a\) incisal region; \(b\) molar region.](#)

[Figure 15.15 Geographic tongue is a relatively common condition found in children.](#)

[Figure 15.16 Bohn's nodules in a newborn baby.](#)

[Figure 15.17 \(a\) Mucous retention cyst in the lower lip. \(b\) Surgical removal of the cyst. \(c\) Healing after 1 week.](#)

[Figure 15.18 Ranula located in the floor of the mouth.](#)

[Figure 15.19 Pyogenic granuloma.](#)

[Figure 15.20 Peripheral calcifying granuloma.](#)

[Figure 15.21 Peripheral giant cell granuloma.](#)

[Figure 15.22 Capillary hemangioma.](#)

[Figure 15.23 Cavernous hemangioma in the palate of a newborn. Note also the capillary hemangioma in the cheek.](#)

[Figure 15.24 Extraction of primary teeth: \(a, b\) molars in upper jaw and \(c, d\) lower jaw – loosen the tooth carefully with an elevator, place the forceps around the tooth and apply apical pressure and buccal–lingual movements before the tooth is lifted out. \(e–g\) Incisors are extracted by slight apical pressure and rotary movement.](#)

[Figure 15.25 Primary first molar with pulpitis. Roots of the tooth encircle the permanent tooth bud.](#)

[Figure 15.26 Primary molar cut into two halves before extraction.](#)

[Figure 15.27 \(a\) Radiographs of upper right canine in palatal ectopic position with risk for resorption of the permanent incisors. \(b\) After a palatal muco,periosteal flap is raised and bone is removed the canine is exposed. \(c\) Brackets and gold chain are etched to the lingual surface of the canine. \(d\) The flap is sutured and the free end of the chain is temporarily fastened to a premolar. The contralateral canine was treated at the same session. The patient is now ready for the orthodontic treatment.](#)

[Figure 15.28 \(a\) Delayed eruption of upper central incisor. \(b\) Denudation of gingival mucosa to enhance eruption.](#)

[Figure 15.29 \(a\) Extensive dentigerous \(follicular\) cyst in left maxilla emerging from a supernumerary tooth and displacing tooth germs and disturbing normal eruption in a 5-year-old girl. \(b\) Radiographic examination after 5 months revealed that the cyst had been reduced considerably and was now available for surgical removal without risk of disturbing the involved teeth. \(c\) After 4 months and surgical removal of supernumerary teeth and residual cyst tissues. \(d\) Obturator inserted to facilitate rinsing of the cyst. The obturator was removed after 6 weeks.](#)

[Figure 15.30 Fibrous frenulum causing bony defects in a 5-year-old girl.](#)

[Figure 15.31 Lingual frenuloplasty. \(a\) Lingual frenulum restricting the movements of the tongue. \(b\) Local anesthesia. \(c\) Curved hemostat is placed close to the tongue. \(d, e\) Frenulum is cut. \(f\) Healing after 10 days.](#)

Chapter 16

[Figure 16.1 Histologic view of partial chronic pulpitis of a primary molar. Note the restricted area of chronic inflammation at the site of carious exposure.](#)

[Figure 16.2 Radiograph showing stepwise excavation performed in two primary second molars in order to keep them in situ and without subjective symptoms at least until the permanent first molars are in occlusion.](#)

[Figure 16.3 Radiograph of primary second molar after partial pulpotomy. The hard tissue barrier indicates wound healing; note absence of periradicular pathology.](#)

[Figure 16.4 Pulpotomy performed in lower left second primary molar with pulp exposure and pulpitis \(a\). The tooth was maintained in the arch with healthy bone in the bifurcation \(b\), after it was restored with a stainless steel crown \(c\).](#)

Chapter 17

[Figure 17.1 Stepwise excavation of a permanent molar, \(a\) at the time of treatment and \(b\) 9 months later. The formation of dentin between the temporary filling and the pulp has proceeded and the risk of pulp exposure at the time of final restoration of the tooth is minimal.](#)

[Figure 17.2 Partial pulpotomy of permanent molar. \(a\) Radiograph taken at the time of treatment and \(b\) 2 years after treatment.](#)

[Figure 17.3 Partial pulpotomy of a permanent incisor with complicated crown fracture. \(a\) At the time of](#)

treatment; (b) application of calcium hydroxide; (c) radiograph at the time of treatment; and (d) several years later.

Figure 17.4 Diagrammatic representation of calcium hydroxide apexification technique. (a,b) Access pulp chamber, (c) light filing or no filing and irrigation with 0.5% sodium hypochlorite, (d) drying the canal using paper points, (e) placement of calcium hydroxide to the apical foramen (spiral filler could be used), (f) placement of cotton pledget (white) and glass ionomer filling (brown) for a minimum of 3 months until clinical or radiographic evidence of a calcific barrier is seen (g).

Figure 17.5 Radiograph showing cervical root fracture of a tooth treated with calcium hydroxide apexification.

Figure 17.6 Periapical radiographs showing calcium hydroxide apexification of the upper left lateral incisor over a period of 9 months. (a) 15 May 2004, (b) 21 September 2004, (c) 15 January 2005, and (d) 11 February 2005.

Figure 17.7 Diagrammatic representation of MTA plug and thermal guttapercha obturation of immature teeth. (a,b) Access pulp chamber, (c) light filing or no filing of canal and irrigation with 0.5% sodium hypochlorite, (d) drying of the canal using paper points, (e) placement of 4–5 mm white MTA (light green) in the apical third of the canal, (f) incremental obturation using Obtura (orange), (g) placement of a glass ionomer based (gray) base and composite filling (brown).

Figure 17.8 Radiographs showing a case treated with MTA plug and Obtura.

Figure 17.9 Radiographs showing continuation of root development in a patient treated with RET using blood clot as a scaffold. (a) 23 October 2013, (b) 23 October 2014.

Figure 17.10 Diagrammatic representation of RET of immature teeth. (a,b) Access pulp chamber, (c) light filing or no filing of canal and irrigation with 2.5% sodium hypochlorite, followed by 5 mL sterile saline, (d) drying of the canal using paper points. (e) A mixture of metronidazole (100 mg) and ciprofloxacin (100 mg) is mixed with distilled water and delivered into the root canal system, (f) placement of cotton pledget and a temporary glass ionomer filling until symptoms and signs of infection resolve. (g) At the second stage and after accessing the tooth again, the canal is irrigated by copious amounts of normal saline followed by 10 mL 17% EDTA and thoroughly dried with paper points. (h) A sterile sharp instrument (needle or a finger spreader) with a length of 2 mm beyond the working length is pushed past the confines of the root canal, into the periapical tissues to intentionally induce bleeding into the root canal. (i) The bleeding is then allowed to fill the root canal followed by placement of a cotton pledget for 5 min until a clot has formed. (j) Once the clot has formed, the access cavity is then hermetically sealed with three layers of material to prevent coronal leakage and contamination: Portland cement, followed by glass ionomer and then composite resin.

Chapter 18

Figure 18.1 A 4-year-old boy with lateral luxation of three primary incisors and extensive gingival laceration.

Figure 18.2 The patient has had an impact where the force has been transmitted through the upper lip to the teeth and the alveolar process. Note the lip laceration and abrasion and the displacement of the right central and lateral incisors.

Figure 18.3 Percentage distribution of 1275 children with traumatic dental injuries related to age at the time of injury.

Figure 18.4 Distribution of injuries of the most frequently injured permanent teeth: 97% of all injuries affected the incisors.

Figure 18.5 (a) Crown fracture of mandibular lateral incisor and mandibular lip lesion. (b) A radiograph reveals the fractured tooth fragment hidden in the lip lesion.

Figure 18.6 (a) Clinical condition immediately after severe intrusive luxation of the primary right central incisor. (b) The occlusal exposure shows foreshortening of the intruded tooth, indicating buccal displacement away from the permanent follicle. (c) This is evident in the lateral radiograph, since the apex of the intruded incisor is forced through the buccal bone plate.

Figure 18.7 (a–c) Clinical appearance after lateral luxation of the right central incisor. (d–g) One occlusal and three periapical radiographs. Note that the occlusal exposure is optimal for showing the buccal displacement of the root. (h) The lateral radiograph illustrates where the fracture of the buccal bone plate has occurred (arrow).

Figure 18.8 Percentage distribution of diagnoses for traumatized primary teeth.

Figure 18.9 Distribution of 2019 traumatized permanent teeth according to diagnosis in 1275 children aged 7–18 years.

Figure 18.10 Schematic drawing illustrating developmental disturbance of permanent tooth bud at the age of 2 years. The crown of the primary incisor is displaced buccally, forcing the root into the crown of the developing permanent incisor.

Figure 18.11 Procedure for examination of a young child's mouth (see text).

Figure 18.12 (a) Fractured roots of both central incisors with dislocation of the coronal fragments. (b) Normal resorption of the apical fragments after removal of the coronal fragments.

Figure 18.13 Severe soft tissue damage with extensive hemorrhage. Both central incisors and the right lateral incisor are extruded and extremely mobile.

Figure 18.14 (a) Severe palatal luxation of the right central incisor. No treatment other than observation was performed. (b) Two months later, the tooth is back in normal position due to tongue pressure.

[Figure 18.15 Clinical condition immediately after buccal displacement of the left central incisor in an 8-month-old girl.](#)

[Figure 18.16 \(a\) Clinical examination after trauma of an 18-month-old child. The parents assumed that the right central incisor was lost. \(b\) The radiograph reveals severe intrusive luxation. Additional radiographs should be taken to disclose the exact direction of the intrusion \(see Figure 18.6\).](#)

[Figure 18.17 \(a\) Condition immediately after intrusive luxation of both central incisors. \(b\) Re-eruption is evident 6 months later.](#)

[Figure 18.18 Uncomplicated crown fracture involving either mesial corners or entire incisal edge. The gingival bleeding indicates that intrusive luxation has also occurred in the right central incisor.](#)

[Figure 18.19 \(a\) Both subluxation and uncomplicated crown fracture have occurred in the left central incisor. \(b\) The tooth is stabilized with a splint, and a temporary crown restoration is applied.](#)

[Figure 18.20 \(a\) Enamel–dentin fracture of the left central incisor in an 8-year-old boy. \(b\) The fractured crown fragment. \(c\) Condition immediately after reattachment of the fragment.](#)

[Figure 18.21 \(a\) A 12-year-old girl with enamel–dentin fracture of the left central incisor. \(b\) Condition shortly after the composite crown build-up.](#)

[Figure 18.22 Right central incisor with a small pulpal exposure, but with loosening and marked tenderness to percussion. Partial pulpotomy was decided to be the treatment of choice.](#)

[Figure 18.23 Crown–root fracture of the left lateral incisor. \(a\) Buccally, the fracture line is located close to the gingival margin. \(b\) The radiograph only demonstrates the position of the buccal part of the fracture, whereas the palatal part cannot be seen.](#)

[Figure 18.24 \(a\) Root fracture in the right central incisor with severe dislocation of the coronal fragment. \(b\) Optimal repositioning performed within 1 hour. \(c\) Condition 1 year later, with normal findings in the fracture area and partial pulp canal obliteration.](#)

[Figure 18.25 \(a\) Subluxation of both central incisors with mobility in both the horizontal and vertical directions. \(b\) The teeth are stabilized with an orthodontic twisted wire, resin material, and the acid-etch technique \(see Box 18.7\).](#)

[Figure 18.26 \(a\) Extrusive luxation of the right central incisor. The tooth appears elongated and is also very mobile. \(b\) The radiograph shows increased periodontal width apically. \(c\) A radiograph taken after repositioning illustrates optimal position of the tooth in its socket.](#)

[Figure 18.27 Palatal luxation of the crown. The apex is forced through the buccal bone. Repositioning requires disengagement of the tooth from its bony lock. Apply firm digital pressure in an incisal direction, and move the tooth back through the fenestration into the socket. Thereafter, axial pressure will bring the tooth back to its original position.](#)

[Figure 18.28 Resin splint \(Protemp[®]\) applied after surgical repositioning of severely intruded left central incisor.](#)

[Figure 18.29 Complete intrusion of the left central incisor in a 9-year-old boy. \(a\) The incisal edge is barely visible 5 days after the accident. It was decided to await re-eruption. \(b\) Partial re-eruption is evident a month later. \(c\) Complete re-eruption 10 months after trauma. \(Delayed eruption of the right incisor is due to a supernumerary tooth.\)](#)

[Figure 18.30 \(a\) A 10-year-old girl with avulsion of the left central incisor after a skiing accident. The tooth was found in the snow 4 hours later. \(b, c\) The avulsed tooth is replaced in the socket with gentle finger pressure and a splint is applied.](#)

[Figure 18.31 Successful replantation of the left central incisor in a 7-year-old boy. The avulsed tooth was immediately pushed back in place by the boy's father. \(a, b\) Six days and 1 year after replantation, respectively. \(c\) Four years after the accident with completed root formation and almost total pulp canal obliteration. Note also obliteration in the right central incisor after a subluxation injury.](#)

[Figure 18.32 Frequency of dental and other sports injuries per 10,000 people from 1981 to 1983 in Norway.](#)

[Figure 18.33 \[www.dentaltraumaguide.org\]\(http://www.dentaltraumaguide.org\).](#)

Chapter 19

[Figure 19.1 Later wound healing events: macrophages \(m\) form the healing front, followed by endothelial cells \(e\) and fibroblasts \(f\).](#)

[Figure 19.2 Nature of trauma in the case of separation injury.](#)

[Figure 19.3 Nature of trauma in the case of crushing injury.](#)

[Figure 19.4 Severe malformation of permanent lateral incisor following intrusive luxation of predecessor at the age of 2 years. \(a\) Condition 1 year after trauma. \(b,c\) Further development of deformed incisor and uncomplicated eruption, respectively.](#)

[Figure 19.5 \(a\) Radiograph taken 1 week after slight intrusive luxation of right central incisor. \(b\) Three months after the trauma there is marked periapical inflammation \(arrows\).](#)

[Figure 19.6 Subluxation of both central incisors leading to pulp canal obliteration. \(a\) At time of injury. \(b\) Two years later, there is almost total obliteration of the pulps.](#)

[Figure 19.7 Disturbance of development of permanent tooth bud due to intrusion of primary incisor. Due to laceration of the follicle, disturbances in enamel formation will develop.](#)

[Figure 19.8 Enamel defects in three mandibular incisors \(arrows\) resulting from avulsion of corresponding predecessors at the age of 2 years.](#)

[Figure 19.9 External enamel hypoplasia of right central incisor caused by intrusion of predecessor at the age of 18 months. The hypoplastic area is covered with composite.](#)

[Figure 19.10 A simplified model of the different stages of amelogenesis. The secretory stage where the ameloblasts are secreting the enamel matrix and the early and late maturation phases where the enamel crystals are growing and the mineral content reaches 96% prior to tooth eruption. The reduced enamel epithelium is fusing with the oral epithelium when the tooth is erupting.](#)

[Figure 19.11 \(a\) Intrusive luxation of immature right central incisor. \(b\) Spontaneous reeruption, closure of apical foramen, and pulp canal obliteration have occurred.](#)

[Figure 19.12 Partial pulp canal obliteration in left central incisor. \(a\) At time of injury. \(b\) Condition 15 years later. Pulp chamber completely obliterated and root canal slightly reduced in size \(arrow\).](#)

[Figure 19.13 Obliteration after successful replantation of right central incisor. The tooth was replanted within a few minutes. \(a\) Normal findings 3 weeks later. \(b\) A radiograph taken 6 months later shows apical closure. \(c\) Seven years after replantation, there is total pulp canal obliteration and no sign of root resorption.](#)

[Figure 19.14 \(a\) Left central incisor in 10-year-old boy discolored within 1 week after subluxation injury. \(b\) Three months later. Discoloration has disappeared and the tooth responds normally to electrometric pulp testing.](#)

[Figure 19.15 Pulp necrosis of right central incisor following intrusion. \(a\) Reeruption took place 3 months after injury. \(b\) No further root development. Hard tissue formation \(arrow\) is found together with continued vitality. \(c\) Pulp necrosis is diagnosed from a periapical radiolucency \(arrow\), which developed 1 year after injury.](#)

[Figure 19.16 Spontaneous root fracture of nonvital immature central incisor. \(a, b\) During long term treatment with calcium hydroxide. \(c\) The fracture was observed 1 year after completed endodontic treatment.](#)

[Figure 19.17 External repair related resorption along the root surface and the apex of an extruded central incisor. \(a, c\) Clinical and radiographic appearance at the time of injury. \(d, f\) Repair related resorption is diagnosed after one year \(arrows\).](#)

[Figure 19.18 External infection related root resorption along root surface of an intruded lateral incisor. \(a\) Six weeks after injury. \(b\) During endodontic treatment, the pulp cavity was temporarily filled with calcium hydroxide. Persistent defects are seen on the root surface \(arrows\), but no further progression has taken place.](#)

[Figure 19.19 External infection related root resorption following intrusive luxation of a right central incisor. \(a\) An area of resorption \(arrow\) was seen 8 weeks after injury. The pulp canal was temporarily filled with calcium hydroxide. \(b\) Two years later, there is a persistent defect \(arrow\), but no further progression of resorption.](#)

[Figure 19.20 Progression of replacement resorption after avulsion and subsequent replantation of left lateral incisor. \(a–c\) Radiographs taken 6 months, 2 and 4 years after injury. \(d\) Condition at time of removal of lateral incisor 7 years after replantation.](#)

[Figure 19.21 Infraposition of a left central incisor due to replacement resorption \(ankylosis\).](#)

[Figure 19.22 Radiographs and diagrams illustrating various modalities of healing after root fractures. \(a\) Healing with calcified tissue. \(b\) Interposition of connective tissue. \(c\) Interposition of bone and connective tissue. \(d\) Interposition of granulation tissue.](#)

[Figure 19.23 Root fractures of both central incisors. \(a\) Retained pulpal vitality and calcified tissue repair in the right incisor, whereas radiolucency corresponding to fracture line \(arrow\) indicates necrosis in the left incisor. \(b\) Two years after completed root filling of left incisor PDL union between segments is evident.](#)

[Figure 19.24 Successful replantation of a left central incisor \(arrows\). The tooth was stored in the mouth of the child's mother for 45 min. \(a–c\) Radiographs taken before, 12 days and 6 months after replantation, respectively. \(d\) One year after injury, with continued root development and narrowing of pulp canal.](#)

Chapter 20

[Figure 20.1 Perikymata shown as horizontal lines on buccal surfaces of central incisors. White, opaque patches and lines represent enamel disturbances \(mild dental fluorosis\). Seven-year-old child.](#)

[Figure 20.2 Schematic representation of hemisectioned tooth with incremental lines in enamel. SEM images show perikymata on the enamel surface \(a\) and striae of Retzius \(arrows\) and cross-striations of enamel prisms \(arrowheads\) in an acid-etched longitudinal section \(b\). Prism cross-striations indicate a daily rhythm while striae of Retzius indicate a longer \(about 7–10 days\) rhythm in enamel formation. Perikymata are the surface representations of the striae of Retzius and run horizontally around the tooth. Prism-free enamel \(PFE\) is shown close to enamel surface. P = prism.](#)

[Figure 20.3 Schematic overview showing a systematic approach to the collection of data and classification of findings related to developmental disturbances of the dental hard tissues.](#)

[Figure 20.4 Classification and examples of types of enamel defects due to disturbances in the tooth formation. The natural appearance of the incisor 21 \(a\) is manipulated to illustrate \(b\) demarcated opacity \(white\), \(c\) diffuse opacities \(white\) with no well-defined margins, \(d\) discolored enamel which has some translucency, \(e\) single hypoplasia \(single deep pit\), \(f\) multiple hypoplastic defects, and \(g\) posteruptive breakdown due to](#)

[hypomineralized enamel. Note that very often in cases with enamel defects, several teeth in the dentition will be affected.](#)

[Figure 20.5 Hypoplasia and opacity of permanent maxillary left central incisor due to intrusion trauma of primary incisor at the age of 30 months \(the child is now 10 years old\).](#)

[Figure 20.6 Dental mutilation resulting in \(a\) hypoplasia with exposure of dentin of the right lower permanent canine and \(b\) absence of the left lower permanent canine due to removal of the tooth germ \(germectomy\). \(c\) Panoramic radiograph shows absence of the permanent left canine. \(d\) Radiograph of the right lower permanent canine shows apparently normal development of the root despite the malformed tooth crown due to the damage previously performed towards the tooth germ. \(e\) Intraoral radiograph of a malformed and hypoplastic permanent mandibular canine in a 14-year-old Ethiopian girl as a result of dental mutilation. An odontoma on the root surface and periapical destruction are present.](#)

[Figure 20.7 Erupting defective 24 in a 6-year-old girl who had 64 extracted 6 months earlier. The extracted tooth had a longstanding, chronic periapical periodontitis.](#)

[Figure 20.8 Arrested root development in a 13-year-old girl who was treated by irradiation \(50.4 Gy/28 fractions\) at age 6 because of rhabdomyosarcoma in the pharynx.](#)

[Figure 20.9 Neonatal lines of dentin \(left\) and enamel \(right\) demarcate the respective hard tissues formed/mineralized before and after birth.](#)

[Figure 20.10 Hypoplasia of enamel of a preterm child.](#)

[Figure 20.11 A 7-year-old child with pigmentation in both primary and permanent teeth due to bilirubine deposits in teeth caused by biliary atresia in early life.](#)

[Figure 20.12 \(a\) A 14-year-old girl with cystic fibrosis who was treated with repeated courses of tetracyclines for recurrent chest infection. \(b\) Histologic sections of a tooth showing the bands of tetracycline staining.](#)

[Figure 20.13 Mild fluorosis in a 15-year-old girl.](#)

[Figure 20.14 Dental fluorosis with posteruptive breakdown of the enamel and tooth wear in a 12-year-old boy with natural high \(\$\approx 5\$ ppm\) fluoride concentration in the drinking water.](#)

[Figure 20.15 \(a\) Mild dental fluorosis in a 14-year-old girl. \(b\) The surface is rubbed using a wooden pin with 18% hydrochloric acid in pumice. \(c\) The surfaces are covered by 2% sodium fluoride gel. \(d\) Treatment result.](#)

[Figure 20.16 Porcelain veneers in a 13-year-old girl with moderate dental fluorosis.](#)

[Figure 20.17 Molar–incisor hypomineralization in an 8-year-old boy showing different manifestations in similar teeth: \(a\) 16 with a defective restoration; \(b\) 26 with seriously disintegrated enamel and caries; the tooth is very sensitive and toothbrushing is impossible; \(c\) 46 healthy; \(d\) 36 disintegrated enamel, but no frank cavitation; \(e\) demarcated white opacities in the upper front teeth.](#)

[Figure 20.18 X-ray micro-computed tomography image of a MIH tooth. The more opaque areas represent hypomineralized enamel.](#)

[Figure 20.19 \(a\) A severely hypomineralized lower first molar is erupting. Normal morphology. \(b\) Heavy disintegration 6 months later.](#)

[Figure 20.20 Scanning electron microscopy. The cut surface has been etched with 30% phosphoric acid for 30 s. \(a\) Normally mineralized enamel. \(b\) Hypomineralized enamel. \(c\) The border between normal and hypomineralized enamel.](#)

[Figure 20.21 \(a\) An 8-year-old girl with an MIH-affected molar to be restored. Porous, soft enamel was removed by a bur until hard, sound enamel appeared. \(b\) After acid etch of the cavity walls, dentin–enamel bonding agent was applied and the tooth was restored with composite.](#)

[Figure 20.22 \(a\) A 13-year-old boy with MIH. \(b\) Enamel disintegration in 11 was restored with a porcelain veneer.](#)

[Figure 20.23 \(a\) Hypomineralized permanent first molars with opacities, posteruptive breakdown of enamel and insufficient conservative treatment, i.e., posteruptive enamel breakdown along the margins of composite fillings. \(b\) Treatment with cast gold copings after minimal preparation due to the scarce thickness required to be able to make a cast gold coping. \(c\) A cast gold coping on the cast. \(d\) The cast gold coping can be made with surface roughening by use of sugar crystal impression method with the purpose of improving the retention.](#)

[Figure 20.24 Clinical manifestation of amelogenesis imperfecta. \(a\) Hypomaturation type \(generalized white opacities with brownish discolorations on upper central incisors\). \(b\) Hypomaturation type \(generalized yellowish opacities\). \(c\) Hypomaturation type, mixed dentition. Chipping of enamel. \(d\) Hypocalcified type. \(e\) Hypoplastic type \(rough, pitted\). \(f\) Hypoplastic type \(rough, vertical grooves\). \(g\) Hypoplastic type \(rough, thin enamel\) \(h\) Combination of hypomaturation and hypoplastic type \(g\) and \(h\)](#)

[Figure 20.25 Hypocalcified amelogenesis imperfecta \(AI\) in the primary dentition of a 3-year-old girl.](#)

[Figure 20.26 \(a1\) A 9-year-old girl with AI \(hypoplastic type\). \(a2\) Same girl at the age of 14 years. Maxillary incisors restored with ceramic veneers. \(b1\) Dentition of a 12-year-old boy with AI \(hypocalcified type\) before orthodontic treatment and placement of crowns. \(b2\) Same boy at the age of 20 years with single metaloceramic crowns in the mandible and single conventional gold crowns with composite facades in the maxillary teeth](#)

[Figure 20.27 A boy with teeth affected by hypocalcified AI: \(a\) 3.5 years, problems with sensitivity and chipping of enamel; \(b\) 3.5 years, the molars are restored by stainless steel crowns; \(c, d\) 9 years, time for dressing the](#)

permanent molars with steel crowns due to chipping and attrition of enamel.

[Figure 20.28 Treatment of hypoplastic, rough pitted AI. \(a\) Prior to treatment, \(b\) pits are cleaned by the bur, \(c\) etching, and \(d\) the teeth are dressed with flowable composite.](#)

[Figure 20.29 A 5-year-old child with DI showing extreme tooth wear in the primary molars. No operative treatment has been offered so far, although the child would have benefited from stainless steel crowns in the molar regions to keep the occlusion. Permanent lower central incisors are in eruption.](#)

[Figure 20.30 Panoramic radiograph of a 10-year-old boy with DI inherited as a single trait \(type II\). Note the obliterated pulp chambers and the cervical constrictions of the crowns.](#)

[Figure 20.31 Osteogenesis imperfecta. \(a\) Blue sclerae in a 4-year-old boy. \(b\) Characteristic dentin defect \(DI type I\) in the same boy.](#)

[Figure 20.32 A boy with DI type II. \(a\) Mandibular teeth at the age of 3 years 5 months. The primary left second molar was infected due to attrition and had to be extracted. Stainless steel crowns have been made to prevent further attrition. \(b\) The permanent molars have been fitted with stainless steel crowns and the incisors with laboratory made composite crowns \(c\) Same patient at the age of 10 years 4 months](#)

[Figure 20.33 Dentin dysplasia type II. \(a\) A 3-year-old boy with DD type II. \(b\) Panoramic radiograph showing pulpal obliteration in the primary molars. \(c\) Radiograph of the mother showing characteristic thistle tube shaped pulp chambers and narrowed root canals.](#)

[Figure 20.34 Vitamin D deficiency rickets \(nutritional rickets\). \(a–c\) Chronological and symmetrical hypoplasia in permanent incisors and first molars in a breastfed 8-year-old boy who was not given a vitamin D supplement in the first year of life. \(d\) Defects can often be seen on a panoramic radiograph. \(e\) An 11-year-old boy with severe hypoplasia on upper permanent incisors due to vitamin D deficiency rickets. Often cusp tips of permanent canines are affected.](#)

[Figure 20.35 Familial hypophosphatemia. \(a\) A 6-year-old boy with several infected primary teeth. \(b\) Radiograph of mandibular primary teeth in a 4-year-old boy. Note the enlarged pulp chambers. \(c\) Microradiograph of a primary molar. Note the enlarged pulp chamber and pulp horns extending to dentino–enamel junction. Defective circumpulpal dentin, mantle dentin seemingly normal. \(d\) Panoramic radiograph showing periapical radiolucency in the lower incisor region and two lower incisors that have already been endodontically treated in a 14-year-old boy. Note also the enlarged pulp chambers and the extended pulp horns in the permanent dentition. \(e\) Clinical picture of the lower jaw of the same 14-year-old boy.](#)

[Figure 20.36 A 7-year-old boy with damage of permanent maxillary teeth after localized, high dose radiation \(56 Gy\) at 3.5 years of age due to a tumor in the right maxilla. Very short roots in permanent teeth are seen in the affected area.](#)

[Figure 20.37 Hypophosphatasia. \(a\) A 3-year-old boy with a benign form of hypophosphatasia. Mandibular right central incisor is poorly attached and mobile. \(b\) Radiograph of incisor teeth showing severe alveolar bone loss in the lower incisor region. \(c\) Histologic section showing thin cementum on the root surface.](#)

[Figure 20.38 Regional odontodysplasia. \(a\) Malformed teeth in the lower right quadrant in a 3-year-old boy. An abscessed incisor is seen. \(b\) Dental treatment under general anesthesia consisted of extraction of all teeth in the lower right quadrant except a more robust lower right second primary molar which would contribute in maintenance of the functional height of the primary dentition and in continuation of some alveolar growth in the region. \(c\) First primary molar after tooth extraction showing enamel hypoplasia of the tooth crown. \(d\) The complete tooth is fragile, and the amount of tooth substance is scarce. The root complex has an open apex, and the pulp chamber is abnormally large. \(e\) A denture was inserted when the boy was 4 years old. As seen on the picture where the child is 6 years old, the denture may need adjustment when primary teeth are gradually lost due to shedding. \(f\) Panoramic radiograph shows that the successors are also affected. The denture was in place when the radiograph was taken as the retentive elements are visible on the photograph.](#)

[Figure 20.39 Regional odontodysplasia. \(a\) Abscessed incisors in the maxillary left quadrant. \(b\) Panoramic radiograph shows involvement of all the teeth in the quadrant. \(c\) The child was treated by extraction of the affected teeth except the permanent first molar, which was thought to maintain some alveolar growth in the region. The maxillary partial denture was placed after extractions at the age of 3 years.](#)

Chapter 21

[Figure 21.1 Changes in body proportions during development and growth.](#)

[Figure 21.2 Changes in proportions of the head.](#)

[Figure 21.3 Facial growth in a normal girl from 2 months to 5 years 2 months of age. Superimposition was made on the nasion–sella line registered at sella. Note the magnitude of mandibular growth from a marked retruded position.](#)

[Figure 21.4 With a mesial step in the terminal plane of the primary dentition, the permanent molars may erupt directly into normal occlusion \(left\). If the primary dental arches end in the same vertical plane, the permanent molars will erupt into a cusp to cusp relation \(right\).](#)

[Figure 21.5 The permanent maxillary incisors erupt at a more labial inclination than their primary predecessors. Consequently, the dental arch becomes wider and longer.](#)

[Figure 21.6 The primary canines and molars occupy more space than is necessary for the corresponding group of permanent teeth. The difference is called the “leeway space” and is greater in the mandible \(2.5 mm\) than in the maxilla \(1.5 mm\).](#)

[Figure 21.7 Posterior cross bite \(patient's left side\), midline deviation and mandibular lateral shift, and frontal open bite in a dummy sucker.](#)

[Figure 21.8 Results of finger sucking. Asymmetrical left side open bite and overjet.](#)

[Figure 21.9 Bilateral scissors bite in combination with forced distal occlusion.](#)

[Figure 21.10 Skeletal class III malocclusion without compensation. Note mesial relations between canines.](#)

[Figure 21.11 Dentoalveolar anterior cross bite.](#)

[Figure 21.12 Unilateral scissors bite in the primary dentition.](#)

[Figure 21.13 Forced posterior cross bite. The jaws are symmetrical, but the maxillary jaw is narrow. In the retruded position, the molars occlude cusp to cusp \(a\). Full occlusion needs forcing of the bite to the left \(b\).](#)

[Figure 21.14 "Classical" unilateral posterior cross bite. Narrow maxilla with the mandibular midline forced to the cross bite side. Note lack of space in the maxilla.](#)

[Figure 21.15 \(a\) Incompetent lip closure, \(b\) maxillary overjet, incisor proclination, distal occlusion, and \(c\) deep bite is a common combination.](#)

[Figure 21.16 Dentoalveolar frontal open bite.](#)

[Figure 21.17 \(a\) Unilateral cross bite in the early mixed dentition. \(b\) Note the narrow maxilla in retruded position which needs active transversal expansion.](#)

[Figure 21.18 Radiograph showing two mesiodens as the cause of large median diastema.](#)

[Figure 21.19 Boy at 18 years of age with persisting 85 and agenesis of 45. The tooth is in good condition and may continue functioning for many years.](#)

[Figure 21.20 Early extraction \(a, b\) of the primary canine may spontaneously change the path of eruption \(c\) of palatal ectopic maxillary canines.](#)

[Figure 21.21 Ankylosis of primary molars resulting in infraposition of 75 \(secondary retention\) and tipping of 36.](#)

[Figure 21.22 Persisting 65 due to a slight ectopic position of 25. The buccal roots are resorbed, while the palatal root is still intact.](#)

[Figure 21.23 \(a, b\) Ectopic eruption of 26.](#)

[Figure 21.24 Ectopic eruption of 13,23 has almost completely destroyed the roots of 12,22 and also resorbed the roots of 11,21. Extraction of 12,22 was needed.](#)

[Figure 21.25 Same case as in Figure 21.24 after extraction of 12,22 and orthodontic treatment and grinding of canine cusps.](#)

[Figure 21.26 Removable appliance for frontal expansion of the maxillary front.](#)

Chapter 22

[Figure 22.1 Diagrams showing prevalence of one or more \(a\) subjective symptoms and \(b\) clinical signs of TMD presented in different epidemiologic studies of children and adolescents. The single dots represent the prevalence figure found in different studies. Note the wide variations within the same age groups, ranging from single percent to more than 70%.](#)

[Figure 22.2 Measurement of maximal jaw opening capacity.](#)

[Figure 22.3 Bilateral palpation of the lateral aspects of the TMJs.](#)

[Figure 22.4 Palpation of the anterior part of the superficial portion of the right masseter muscle.](#)

[Figure 22.5 Schematic illustration of the apparent paradoxical peripheral delineation of the condylar heads of normal and pathologic joints in the child and the adult. A normal delineation of the condylar head of a child \(1\) has characteristics in common with that of a pathologic adult TMJ \(4\) and the pathologic condylar head of a child \(2\) with that of a normal adult \(3\).](#)

[Figure 22.6 Examples of therapeutic jaw exercises. \(a\) Active jaw opening against a slight resistance. \(b\) Active lateral excursion to the right against a slight resistance.](#)

[Figure 22.7 Soft interocclusal appliance placed in the mandible.](#)

[Figure 22.8 \(a\) A 12-year-old girl in whom the canines in the maxilla are not completely erupted. \(b\) She is provided with a hard acrylic appliance \(Shore plate\), where the acrylic is removed in the region of the canines to allow further eruption. \(c\) A 7-year-old boy with a mixed dentition and a deep bite. Severe bruxism and frequent headaches. \(d\) He is provided with a hard acrylic bite plate to prevent the teeth from further wear, to unload the jaw muscles, and eventually to decrease the deep bite.](#)

Chapter 23

[Figure 23.1 A 3-year-old boy with cerebral palsy and showing severe gingival overgrowth.](#)

[Figure 23.2 \(a\) A 10-year-old girl on phenytoin medication exhibiting severe gingival overgrowth. \(b\) At 13 years of age, exhibiting a normal gingival 1 year after gingivectomy and discontinuation of phenytoin medication.](#)

[Figure 23.3 \(a\) A 15-year-old boy with aplastic anemia prescribed cyclosporine medication. \(b\) At 18 years of age, deteriorating gingival health coincidental with a hematological crisis.](#)

[Figure 23.4 A 14-year-old girl with restricted and asymmetric growth of the mandible due to unilateral TMJ arthritis in her right side.](#)

[Figure 23.5 A 9-year-old boy with a congenital HIV infection exhibiting periodontal disease.](#)

[Figure 23.6 A 3-year-old boy with a malabsorption syndrome with daily vomiting and regurgitation. Note erosive change on anterior teeth.](#)

[Figure 23.7 A 16-year-old boy with severe obesity exhibiting high caries activity and extensive gingivitis.](#)

[Figure 23.8 Discoloration and enamel hypomineralization in a 14-year-old boy with cystic fibrosis.](#)

[Figure 23.9 A 12-year-old boy with immunoglobulin A and G deficiency exhibiting ulceration of the marginal gingiva.](#)

[Figure 23.10 Panoramic radiograph showing severe periodontal breakdown in a 6-year-old boy with cyclic neutropenia.](#)

[Figure 23.11 \(a\) Blistering of hand and fingers in a 10-year-old boy with dystrophic epidermolysis bullosa. \(b\) Oral blistering and scarring in a 1-month-old boy with dystrophic epidermolysis bullosa.](#)

[Figure 23.12 Severe oral ulceration in a 5-year-old boy during induction chemotherapy for acute lymphoblastic leukemia.](#)

[Figure 23.13 \(a\) Panoramic radiograph showing premature apical closure of permanent first molars in an 8-year-old boy treated with 10 Gy total body irradiation at 5 years of age. \(b\) At 17 years of age, all permanent teeth exhibit short V-shaped roots.](#)

Chapter 24

[Figure 24.1 The ICF theoretical, biopsychosocial model defines functioning and disabilities in relation to outcomes between the health conditions of a person and the context in which they operate. This context is defined at three levels of functioning, at the level of a body part, at the level of the whole individual, and the functioning that occurs at the level of society.](#)

[Figure 24.2 Examples of photographs that can be used as pedagogic tools in patients with neuropsychiatric disorders. Based on individual needs: pictures showing the entrance, the dental chair, the operatory light, a toothbrush, a mirror and an open mouth \(symbolizing “open your mouth”\) were chosen in this case. The photos can be put in sequence in a photo album.](#)

[Figure 24.3 Large, fissured tongue of a boy with Down syndrome.](#)

[Figure 24.4 Drooling in a child with cerebral palsy.](#)

[Figure 24.5 A vacuum-moulded splint in place to prevent further self-mutilation in a child with cerebral palsy \(note calculus as a consequence of nil-by-mouth PEG feeding\).](#)

[Figure 24.6 \(a\) A prop, with and without an occlusal “guard”, and \(b\) in use.](#)

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[Figure 24.8 A Superbrush[®] on the left and two Collis Curve[®] brushes on the right.](#)

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To Anna, Lena Hallonsten

Pediatric Dentistry

A Clinical Approach

Third Edition

Edited by

**Göran Koch
Sven Poulsen
Ivar Espelid
Dorte Haubek**

WILEY Blackwell

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Preface to the Second Edition

Pediatric Dentistry: A Clinical Approach was first published in 2001 with the aim of providing a comprehensive review of pediatric dentistry with special emphasis on evidence-based oral health care for the child and adolescent. In this second edition of this textbook, we have built upon the earlier volume by thoroughly updating the text, as well as replacing chapters and adding new ones as necessary.

The sciences behind pediatric dentistry, as well as strategies of clinical approaches, have developed rapidly over the past few decades. These advances have been reflected in the considerable work undertaken by the many clinicians and scientists who have contributed to this textbook.

Pediatric dentistry aims to improve the oral health of children and adolescents through health promotion, prevention and systematic and comprehensive oral care. It is concerned with the expression of, and interventions against, the major dental diseases as well as with a number of dental and oral conditions specific to childhood and adolescence. These comprise all aspects of dental and occlusal developmental disturbances, traumatic injuries to the teeth, periodontal conditions, oral pathological conditions, pain control, dental need and treatment of handicapped and medically compromised children. Pediatric dentistry applies principles from other clinical disciplines, medical and behavioral sciences and adapts them to the special needs of the growing and developing individual from birth through infancy and childhood to adolescent late teens.

Our objective is that this book will serve not only as a basis for undergraduate training in pediatric dentistry but will also be of relevance to postgraduate students and dental practitioners who want to increase their knowledge and skills in order to deliver up-to-date pediatric dental care.

Göran Koch and Sven Poulsen
Editors

Preface to the Third Edition

This third edition of *Pediatric Dentistry: A Clinical Approach* follows up on the two previous editions published in 2001 and 2009. It represents a considerable effort from a large group of highly experienced colleagues within the field of pediatric dentistry and related disciplines.

The present edition is expanded to include new chapters on recently developed essential subjects within the field of pediatric dentistry, i.e., genetics in pediatric dentistry, child abuse and neglected children, and ethics in pediatric dentistry. The previous chapter on pedodontic endodontics has been split into two chapters – one on primary teeth and one on young permanent teeth. Most of the chapters have been thoroughly revised and several new illustrations are included. The concept of evidence-based care has been given more attention.

It is our hope that this edition will also serve the objective stated for the previous editions.

Göran Koch, Sven Poulsen, Ivar Espelid, Dorte Haubek
Editors

About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/koch/pediatric_dentistry

The website includes:

- Interactive multiple choice questions

CHAPTER 1

Pediatric Oral Health and Pediatric Dentistry: The Perspectives

Sven Poulsen, Göran Koch, Ivar Espelid, and Dorte Haubek

Children are special

Pediatric dentistry is defined as “the practice, and teaching of and research in comprehensive preventative and therapeutic oral health care of children from birth through adolescence” [1]. The central element in this definition—and that which distinguish it from other clinical fields in dentistry—is *children*, further qualified as individuals *from birth through adolescence*.

In this book, we adopt the United Nations (UN) Convention definition of a child as “every human being below the age of 18 years unless, under the law applicable to the child, majority is attained earlier” [2]. That children are different from adults has not always been recognized. Previously, children were depicted as “small adults” (Figure 1.1), but recent research reflect that health services for children need to consider that children are growing and developing individuals who are dependent on an adult caregiver. This requires oral health professionals with special competencies, so called *child competency* (Box 1.1).



Figure 1.1 Until the eighteenth century, children were considered to be small adults (sort of “miniature grown-ups”) as shown in this painting from a medieval church. Note the similarity of the facial features of the adults and the children.

Source: Epitaph in Norra Sandsjö parish church, Sweden, of Johan Printzensköld and Anna Hård af Segerstad and their five children.

Box 1.1 Professionals should recognize that children are not “small adults” and that special competency (child competency) is needed, when meeting children

Children are different from adults in a number of ways:

- children are individuals in growth and development
 - physical
 - psychological
 - social
 - cognitive
 - emotional
- oral health, including attitudes and behavior relating to oral health, is formed during childhood and adolescence
- children’s situation is different from the situation of adults:
 - they are in the care of and dependent on adults
 - they are not able to foresee consequences of their own decisions and behavior.

Child competency is:

- a specific insight into the dental and oral health for the child and adolescent
- an ability to communicate effectively with children, adolescents, and their parents
- a positive professional attitude towards children, adolescents, and their parents.

Today, a satisfactory definition of health needs to include somatic as well as non-somatic dimensions. Consequently, oral health should include not only sound teeth and surrounding oral structures, but also absence of dental fear and anxiety as a prerequisite for good oral health during later periods of life. This is consistent with recent concepts of oral health as a determinant factor for quality of life [3].

Community responsibility: the population perspective

By the end of the nineteenth century, a number of large epidemiologic studies on caries in children carried out in the Nordic countries showed that more than 80% of the children had carious teeth and that only a few per thousand had received any dental treatment. These studies were the major reason why children’s dental health was conceived as a problem, requiring public intervention in terms of organized public dental health services for children.

It is interesting to note that the arguments for better oral services for children in the Nordic countries were based on epidemiologic data. Using epidemiologic information to document a health problem is to adopt a population approach rather than an individual clinical approach. This illustrates that in the Nordic countries, organized child dental care has for more than a century been considered a collective responsibility rather than the responsibility of the individual on their own. Formal legislation and regulations concerning child dental care were passed by the parliaments of all Nordic countries several decades ago and dental services, including outreach preventive services, have been developed to serve the whole child population. The epidemiologic starting point of child dental care in the Nordic countries also explains why the child dental services in these countries have collected valuable epidemiologic information to continually monitor the level of disease in the target groups.

The clinical perspective

Pediatric dentistry encompasses all aspects of oral health care for children and adolescents. It is based on basic knowledge from various odontological, medical, and behavioral sciences that are applied to the unique situation of the developing child and young person. Prevention is still the cornerstone of pediatric dentistry. Starting prevention in early childhood makes it possible to maintain sound erupting teeth and keep oral structures healthy. Pediatric dentistry also implies early diagnosis and treatment of the multitude of oral diseases and conditions found in the child’s and the adolescent’s oral cavity, including caries, periodontal diseases, mineralization disturbances, dental erosion, disturbances in tooth development and tooth eruption, and traumatic injuries in otherwise healthy individuals as well as oral health care of sick and disabled children. The realm of pediatric dentistry is constantly expanding, and now includes such areas as early identification of children suspected to suffer from syndromes, and of children suspected to suffer from child maltreatment. Ethical considerations superimpose all these areas.

The quest for evidence-based interventions—preventive, diagnostic or rehabilitative—is urgent in pediatric dentistry as well as in all other fields of dentistry, and recent research has identified the need for more high-quality research in a number of the domains comprising pediatric dentistry [4]. It is important that diagnosis, risk assessment, prevention, treatment, and follow-up of children are based on scientific evidence, when available. Translation of evidence into clinical guidelines will thus help to secure quality of dental care for all children. The burden of dental disease is not equally distributed and it is a goal to diminish the inequality. Health technology assessment (HTA) bodies in many countries have provided useful guidelines about important topics in pediatric dentistry. In Scandinavia, the Swedish

Council on Health Technology Assessment (SBU) has produced relevant guidelines for pediatric dentistry.

Education in pediatric dentistry: the perspectives

The undergraduate education and training in pediatric dentistry in the Nordic countries today is well balanced and aims to give the student sufficient knowledge and competence to deliver basic dental care to preschool children, school children, and adolescents. During the last decades the undergraduate curriculum has increased the emphasis on prevention and behavioral and social sciences.

The need for postgraduate courses and training was recognized early. To provide dental care to complicated cases, often with special needs and in a multidisciplinary team, requires specialized knowledge and child competence as obtained in a specialist education in pediatric dentistry. The European Academy of Paediatric Dentistry presented guidelines for a specialist education in pediatric dentistry in 1995 [5]. The education is a 3-year full-time course given at universities and institutes preceded by at least 2 years' practice as a general practitioner. This program has been adopted by most educational centers in Europe during the last decades.

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CHAPTER 2

Growth and Pubertal Development

Anders Juul, Sven Kreiborg, and Katharina M. Main

The evaluation of growth charts and pubertal development in children and adolescents is an important tool for any clinician in the assessment of health status. Optimal thriving and height attainment in accordance with family potential can only be achieved in an environment providing optimal socioeconomic conditions, health care, and psychosocial support. Thus, failure to thrive or to grow may be the first indication of an underlying problem that may need attention. In turn, treatment of children may need to consider the specific growth and developmental windows in order not to disturb this delicate balance.

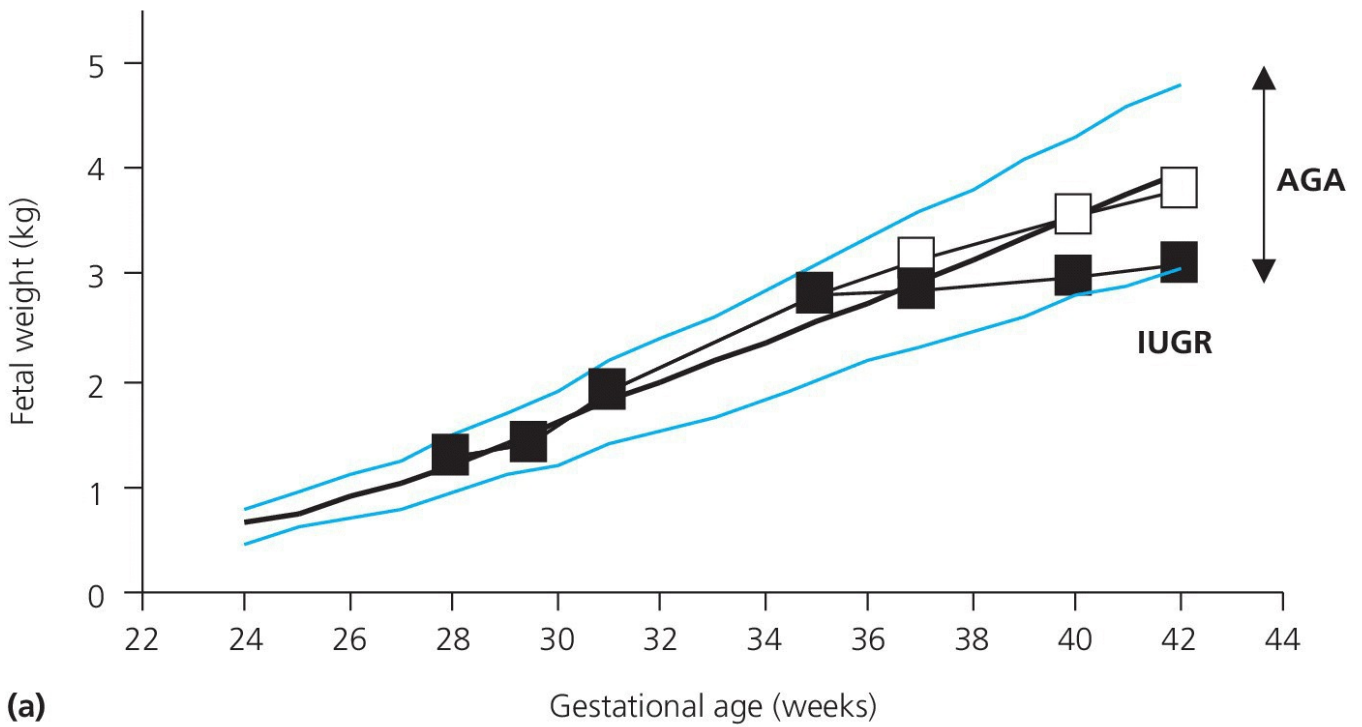
Measurement of growth in different phases of life

The current concept of prenatal and postnatal growth suggests that there are distinct growth phases, which should be considered separately.

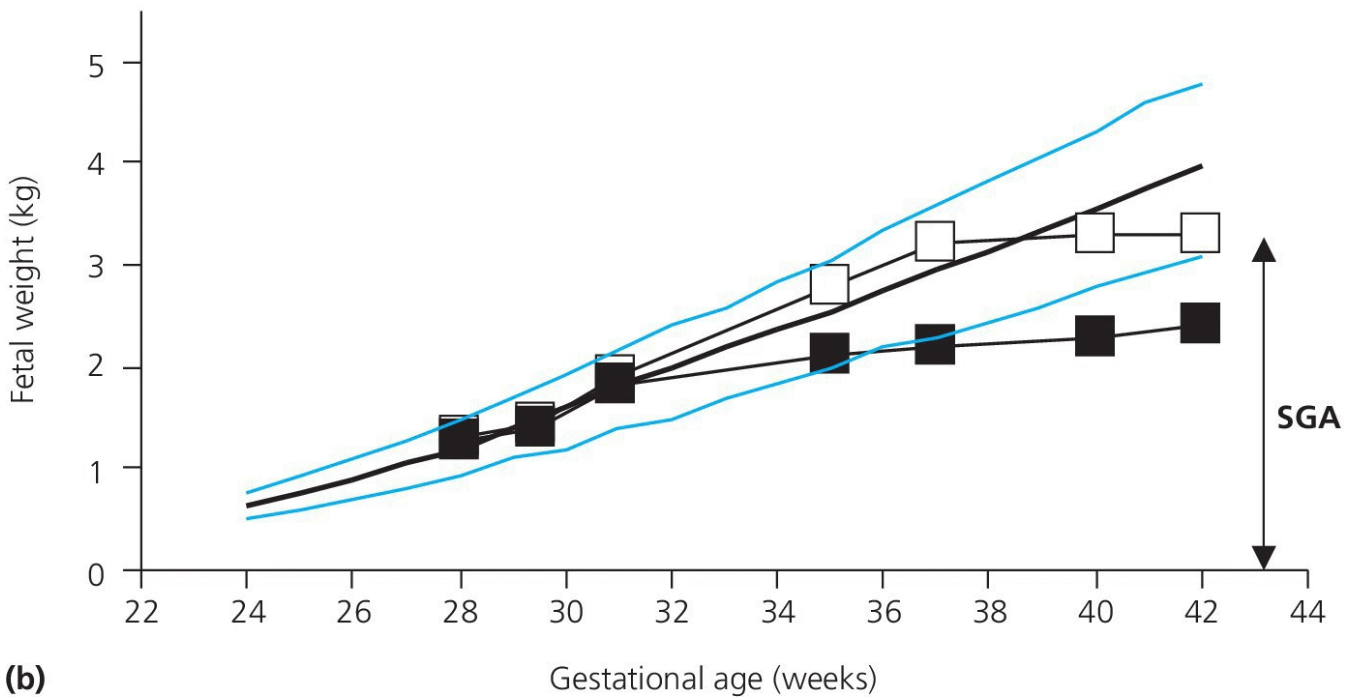
Prenatal growth

Prenatal growth is divided into three trimesters (by convention). The first trimester is characterized by organogenesis and tissue differentiation, whereas the second and third trimesters are characterized by rapid growth and maturation of the fetus. Fetal growth can be assessed by serial ultrasonography in the second and third trimesters. Abdominal circumference, head circumference, and femoral length of the fetus can be determined, and from these parameters fetal weight can be estimated using different algorithms [1]. The fetal weight estimate should be related to normative data. Some reference curves for fetal growth are based on children born prematurely [2], and hence such curves tend to underestimate normal fetal weights from healthy pregnancies. Alternatively, reference curves based on ultrasound studies of normal healthy infants exist [1] and should preferably be used. Based on the changes in fetal weight estimates over time, the fetus can be considered as having a normal fetal growth rate, or alternatively as experiencing intrauterine growth restriction (IUGR) [3]. Children born at term (gestational age 37–42 weeks) are considered mature. Children born before 37 weeks of gestation are premature, and children born after 42 weeks of gestation are postmature. At birth, weight and length can be measured and compared to normative data correcting for gestational age at birth. Based on these comparisons, a newborn child can be classified as either appropriate for gestational age (AGA), small for gestational age (SGA), or large for gestational age (LGA).

IUGR fetuses will often end up being SGA at birth, but not necessarily so. Thus, IUGR infants may end up lighter than their genetic potential but remain within normal ranges (i.e., AGA). Therefore, IUGR and SGA are not synonymous entities, although they are often referred to as such in the literature ([Figure 2.1](#)). Height velocity *in utero* is higher than at any time later in life, leading to an average birth length of 50–52 cm and birth weight of 3.5–3.6 kg after 37–42 weeks of gestation. It is therefore not surprising that growth disturbances during this phase may have long-lasting effects on growth and health later in life. Whereas the first trimester is dominated by tissue differentiation and organ formation, the second and especially third trimesters show a rapid gain first in length and then in weight. Fetal and placental endocrinology is highly complex and hormones such as insulin, leptin, placental growth hormone, insulin-like growth factor (IGF)₂, and thyroid hormone are only some of the many growth factors involved in the regulation of fetal growth.



(a)



(b)

Figure 2.1 Reference ranges for fetal weight according to gestational age during pregnancy denoted by the blue lines (10th, 50th, and 90th percentiles) (8). Panel (a) shows examples of children with normal birth weights at term; a normally growing fetus ending with a birth weight which is appropriate for gestational age (AGA) and (•) a fetus with third trimester intrauterine growth restriction (IUGR) ending with a birth weight below the genetic potential but within normal limits (AGA). Panel (b) shows examples of fetuses with intrauterine growth retardation (IUGR) ending up AGA (□) or SGA (•).

Postnatal growth

Postnatally, height can be determined by measuring length in the supine position in the first 2–3 years of life. After 2–3 years of age standing height can be measured, preferably using a wall-mounted stadiometer. Height is determined without shoes, shoulders towards the wall, arms hanging down, and the face straight forward (Figure 2.2). The eyes should be horizontally aligned with the external ear opening. The means of three measurements are recorded. The stadiometer should be calibrated on a daily basis.