Pediatric Dentistry
Pediatric Dentistry
A clinical approach
Second edition

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To Anna-Lena Hallonsten
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.

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Pediatric oral health care: the perspectives

Sven Poulsen and Göran Koch

How it began: the historical perspective

The history of pediatric dentistry as we know it today is usually considered to have started in the latter part of the nineteenth century. However, in 1743, Robert Bunon published his book *Essay sur les Maladies des Dentes*, where he for the first time discussed at length dental problems during childhood. Bunon emphasized the connection between the diet and health of the pregnant mother and the mineralization of her child’s teeth. He also studied the influence of infectious diseases on dental development and described the principles of serial extraction. The importance of good dietary habits for prevention of dental disease was repeatedly stressed in his book. Robert Bunon rightly deserves the title “the father of children’s dentistry”.

One of the first dentists who dedicated himself to providing regular dental care for children was John Greenwood, who practiced in New York in the 1780s. He advertised a reduced fee for children who signed up for regular dental treatment. It is also known that around 1800, C.F. Delabarre undertook dental treatment in overfilled Parisian orphanages in wartime France. The first known proposal for a regular dental care program for children was put forward in 1851 by A.-F. Talma, dentist to King Leopold I of Belgium, and was based on regular examination of all children between the ages of 5 and 12 years. Similar programs were proposed in many other countries.

In this early era, enthusiastic pioneers – often on their own initiative – organized dental care for children (Fig. 1-1).

Community responsibility: the population perspective

The growing interest in dental care for children at the end of the nineteenth century was partly caused by large epidemiological studies on caries in children published in 1893–1895. These studies showed that more than 80% of the children had carious teeth and that only a few per thousand had received any dental treatment. These results were thoroughly discussed at world dental congresses and led dentists in many countries to urge their societies to organize public dental health services for children. The first municipal dental clinic for children was opened in Strasbourg in 1902 under the direction of the Danish-born Ernst Jessen. This very first school dental clinic became a model for the development of children’s dental clinics in a number of countries.

It is interesting to note that the arguments for better dental services for children were based on epidemiological data. Using epidemiological information to document a health problem is to adopt a population approach rather than an individual clinical approach. This indicates that in the Nordic countries, organized child dental care has for more than a century been considered a collective responsibility rather than the problem of the individual. 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 epidemiological starting point of child dental care in Nordic countries may also explain why the child dental services in these countries have collected valuable epidemiological information to monitor continually the level of disease in their target groups.

From birth to adolescence: the oral health perspective

As already mentioned, child dental services originally focused on school-age children, often being limited to extraction of diseased teeth and restorative care of the permanent teeth. This focus had no rational foundation,
but was merely due to the fact that children could easily be reached in schools. This philosophy is still prevalent in many countries in spite of the pain untreated dental disease and emergency care may inflict on the pre-school child with subsequent dental fear and anxiety. In this book we adopt the United Nations (UN) Convention definition of a child (every human being below the age of 18 years unless, under the law applicable to the child, majority is attained earlier). Thus, we see pediatric oral care as a continuous effort to secure good oral health as defined in Box 1-1 from birth to 18 years of age.

The World Health Organization (WHO) definition of health emphasizes that health cannot be isolated from the subjective experience of the individual (Box 1-1). In other words, a satisfactory definition of health needs to include somatic as well as nonsomatic dimensions. As oral health should be considered an integrated part of general health, a definition of oral health should also include not only sound teeth and oral structures, but also absence of dental fear and anxiety. This is consistent with recent research on patients’ assessment of how their dental and oral conditions influence the quality of their daily life. Methods to measure oral health-related quality of life in children are now available, and it has been shown that dental conditions such as early childhood caries reduce children’s oral health-related quality of life. It has also been shown that the oral health-related quality of life of children with a severe caries treatment need is improved after rehabilitation under general anesthesia (see Chapter 6).

**Box 1-1** WHO definition of general health and an analogous definition of oral health

**General health**
A state of complete physical, mental and social well-being and not only the absence of disease or infirmity (1).

**Oral health**
A state of sound and well-functioning dental and oral structures as well as absence of dental fear and anxiety.

**UN Convention on the Rights of the Child: the ethical perspective**

Today, concern for the child’s specific needs and rights is attracting increasing interest as reflected in the UN Convention on the Rights of the Child (2). This document is important to all professionals who relate to children – including pediatric dentists.
One of the key items in this document is that children have rights as human beings and also need special care and protection, as often expressed by the saying that “children are not small adults” (Fig. 1-2). In order to secure children and adolescents high-quality oral health care, emphasis is now being placed on the concept of child competency (Box 1-2).

The UN Convention on the Rights of the Child was adopted by the UN General Assembly on November 20, 1989, and has had a marked impact on the lives of children in a number of countries. The overriding point in the Convention is that children have rights. They have a right to be protected against bad or unfair treatment and they also have a right to be respected as individuals. Another principle is the “best interest of the child”. Whenever we take a decision affecting children, their best interest should be a primary consideration. Further, the views of the child should be respected. The child should be free to express opinions and these should be given due weight “in accordance with the age and maturity of the child”. In other words, children should have a say in matters of concern to them.

A number of the articles in the convention have obvious implications for pediatric dentists and the way we organize and deliver oral health care to children (Box 1-3).

**Pediatric dentistry: 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. Starting prevention in early childhood makes it possible...
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 mouth, including caries, periodontal diseases, mineralization disturbances, disturbances in tooth development and tooth eruption, and traumatic injuries in otherwise healthy as well as sick and handicapped children (Fig. 1-3).

The quest for evidence-based interventions – preventive, diagnostic or rehabilitative – is urgent in pediatric dentistry as well as in all other fields of dentistry.

References
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 (9). The fetal weight estimate should be related to normative data. Some reference curves for fetal growth are based on children born prematurely (11), and hence such curves therefore tend to underestimate normal fetal weights from healthy pregnancies. Alternatively, reference curves based on ultrasound studies of normal healthy infants exist (9) 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) (8). 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 (Fig. 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.

Postnatal growth

Postnatally, height can be determined by measuring length in the supine position 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 (Fig. 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.

Importantly, the body proportions (such as head circumference, facial appearance, sitting height, and arm span) may be helpful in the differential diagnosis of growth disorders (Fig. 2-1). This can simply be done by assessing the sitting height with subsequent calculation of the sitting height to standing height ratio. This enables quantification of whether or not a growth failure is proportional or disproportional (such as in hypochondroplasia). Reference ranges for this ratio exist (5).

Changes in height can be separated into infant, childhood, and pubertal growth phases according to the infancy–childhood–puberty (ICP) model described by Karlberg (7). The majority of children will follow the distinct growth patterns of these phases.

**Infancy**

After a brief initial weight loss of up to 10% of the birth weight, growth during the first months postnatally follows to a large extent fetal growth rate during the third trimester with 30 g/day and 3.5 cm/month. After that a rapid decline in growth rate occurs, in both weight and height. However, this period still represents a major growth phase during the lifetime with a three-fold increase in weight over 6 months. Very little is known about the regulatory factors of growth during this period.

![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 (■).](image-url)
of life, but nutrition and living conditions play a major role. Recently, the World Health Organization (WHO) published a new growth chart reference for infancy based on breastfed infants from different countries and ethnic origins living under optimal socioeconomic conditions. This chart did not find significant differences in growth patterns between these children, which indicates that genetic differences may first become evident later in life (14).

**Childhood**

In this phase growth is relatively constant, with a gradual decline in growth velocity over time. From 2 to 4 years children grow approximately 7 cm and 2 kg/year. Beyond 5–6 years of age this rate has decreased to approximately 5 cm/year. This growth phase is highly dependent on growth and thyroid hormones.

**Puberty**

During the pubertal growth spurt, which typically stretches over 4–5 years, total height gain is on average 20–25 cm for girls and 25–30 cm for boys with large interindividual variations. There is some tendency that early maturers obtain a higher peak height velocity compared to late maturers (Fig. 2-3). Sex steroids increase the pulsatile growth hormone secretion, which in turn increases IGF-1. Weight gain is highly individual and may occur both before and after peak height velocity.

In girls, the onset of the growth spurt is early and may even precede the development of secondary sexual characteristics in some. Typically, breast buds appear before pubic hair at 10–11 years of age, but occasionally this succession may be reversed (9). Both breast development and pubic hair attainment are graded into five stages (B1–B5 and PH1–PH5) according to Tanner and Whitehouse (13). The first menstruation, menarche, is a sign of adult level estradiol production and follicle maturation and occurs late during the growth spurt at approximately 13 years of age. Height attainment after menarche is small, with 4–8 cm over 1.5–2 years.

In boys, the pubertal growth spurt occurs relatively late during development. Puberty commences with enlargement of testis size from 3 to 4 ml at 11–12 years of age, and this very first sign of pubertal onset is usually

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**Figure 2-2** Standing height determined by a wall-mounted stadiometer (a). Height is recorded as the mean of three measurements. Sitting height is determined by a specifically designed chair (b). Head circumference is determined using a measuring tape (c). Armspan length is determined by measuring the distance from fingertips to fingertips (d).
not noticed by the boy or even less so by the parents. Pubertal development in boys is graded into five genital stages (G1–G5) according to Tanner and Whitehouse (13). Testis growth continues and within 6–12 months pubic hair can be seen. Testicular volume can be determined by the use of an orchidometer to which the size of the testes is compared. Maximum height velocity often occurs at a testis size of 10–12 ml at around 14 years of age, at the time when the voice breaks and facial hair appearance occurs. Thus, boys are already relatively virilized at the time of the adolescent growth spurt (10). In midpuberty, many boys develop physiological gynecomastia, which usually disappears within 6–12 months.

The onset of puberty is approximately a year earlier in girls than boys, which consequently results in earlier growth arrest in girls than boys (14–15 versus 16–17 years of age). The timing of puberty may also differ by 1–2 years according ethnicity and nationality.

**Growing pains**

A significant number of children and adolescents experiences intermittently pains, localized to the shins or legs when going to bed after a physically active day. The etiology of this phenomenon is unknown, but local warmth, gentle massage, and mild pain medication, if the child is in real discomfort, can normally ameliorate the problem, which resolves spontaneously.

**Evaluation of growth charts**

Growth evaluation should be based on observations over time by applying longitudinal measurements of height and weight on an age- and gender-specific growth chart. These charts are available for many populations and also for a variety of growth disorders and syndromes. Due to the secular trend in height, country-specific reference ranges should be constructed at regular intervals (6). Repetitive measures of growth will result in a trajectory of growth, which then can be evaluated against family potential (parental stature, growth of siblings). As some children show considerable seasonal variation in growth, follow-up periods of 6–12 months may be necessary. In children approaching puberty, pubertal staging (13) will additionally be necessary for adequate assessment.

Growth charts are usually based on cross-sectional data from children and adolescents, covering 95% of the population (±2 standard deviations). Charts may depict percentiles or standard deviation lines. Per definition, 2.5% of the population will be below or above the outer limits (Fig. 2-4). In contrast, height velocity curves are based on longitudinal follow-up studies of healthy children (Fig. 2-5).

In the evaluation, both the position within the growth chart in relation to the parental potential and the trend of the individual growth curve are important. Deviations from the expected may represent two separate pathological conditions. In populations with a significant secular trend in height attainment due to recently improved socioeconomic conditions, the growth of siblings in comparison to the patient may be helpful as well. The simplest method to determine the family growth potential is based on calculation of midparental height (Box 2-1).

During childhood, most children will follow their trajectory of growth, which ideally should follow the
family potential. There are, however, two phases in life where this trajectory may not be followed without necessarily representing pathology: (a) during the first 2 years of life, children may “catch up” or “slow down” depending on their intrauterine growth and size at birth, a phenomenon also called regression towards the mean, and (b) during puberty, early maturation will lead to a growth spurt above average (vice versa for late maturation) and the individual child will therefore almost always deviate upwards (or downwards) compared to the mean on the growth chart (due to the cross-sectional design of the growth charts). In general, tall children have a tendency to enter puberty early, short children to enter puberty later.

Acute diseases during childhood and adolescence will often only result in a temporary weight loss with rapid catch-up after recovery. In contrast, height attainment will often get compromised in long-term or serious illness. These children may show considerable catch up growth after recovery, if their bone age allows further

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**Figure 2-4** Normal (Gaussian) distribution of heights illustrating the 95% reference interval by percentiles or standard deviations (SDs).

**Figure 2-5** Normal height curve (a) based on healthy children. Lines denote mean ±1 standard deviation (SD) and ±2SD. One individual patient is depicted on the curve (●) before and after operation for a pituitary tumor (craniopharyngeoma) resulting in growth hormone deficiency. A typical deceleration is seen prior to diagnosis. Horizontal lines (red arrow) denote bone age. Following operation the child suffers from pituitary insufficiency and is substituted with L-thyroxine, hydrocortisone growth hormone, (GH) (arrow), and testosterone (Te) (arrow). This results in a final height well within target height. T = target height range, F = father’s, and M = mother’s height expressed as SDs. (b) Normal height velocity curve based on Tanner’s longitudinal study of healthy children. The same child (●) is depicted on this curve illustrating the marked growth acceleration following GH therapy, as well as the acceleration when puberty is initiated.

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**Box 2-1** Calculation of family growth potential (equal to target height or genetic height potential)

**Girls**

\[
\frac{\text{Maternal height (cm)} + \text{paternal height (cm)}}{2} - 6.5 \text{ cm}
\]

**Boys**

\[
\frac{\text{Maternal height (cm)} + \text{paternal height (cm)}}{2} + 6.5 \text{ cm}
\]

To allow for growth variation within a family, the target height range is calculated as midparental height of ± 6.5 cm for both genders.

Potential pitfalls of this approach are: (a) the parents differ considerably in height centiles and (b) one of the parents is not of normal stature.
growth potential. Thus, growth deceleration is seen commonly in the year(s) prior to diagnosis of severe chronic disease (e.g., brain tumors or malignancies) which is often first noticed in retrospect.

Detailed evaluation of growth includes bone age determination and final height predictions.

**Bone age determination**

Linear growth continues until the fusion of the ossification centers. Thus, determination of bone maturation may help to assess the growth potential in an individual, as many disorders of growth are associated with either delayed or accelerated bone age. Bone age is mostly measured with a radiograph of the left hand and wrist and a comparison of the epiphyseal growth plates with age- and gender-specific references (Fig. 2-6). Two main systems are used clinically: (a) the Greulich–Pyle method (3) and (b) the TW method (12). Computer-based automated bone-age assessments are currently being developed and will help to reduce interobserver variation and time expense.

**Final height prediction**

Both methods of bone-age determinations (Greulich–Pyle and TW methods) can be employed in prediction models (Bayley–Pinneau and TW method, respectively) for final height (1,12), with a broad margin of error. Both methods are based on studies of healthy children who were followed up until final height, in whom bone ages were determined at various ages. Heights at each bone age were assigned a certain percentage of the final height, e.g., a 13-year-old boy with a bone age of 14 years is assumed to have reached approximately 90% of his final height according to the Bayley–Pinneau tables, and his current height can then be transformed into a final height estimate. Pitfalls in this approach are (a) the normal biological variation of bone age in comparison to chronological age which is ±1 year and (b) the fact that prediction models are based on normally growing children and may therefore both underestimate and overestimate final height in pathological conditions.

**Dental age determination**

Dental age or dental maturity may be assessed in different ways. The simplest method is to record the teeth erupted and compare to normative data. A more precise method is to judge the development of the teeth from radiographs. Haavikko (4) has given normative data for individual permanent teeth, while Demirjian (2) has developed a scoring system based on assessment of all lower left permanent teeth (except the third molar) from an orthopantomogram. Demirjian’s method has

![Figure 2-6](image-url) Two radiographs of the left hands of two healthy children. Note that the mineralization of the small bones has not yet occurred in the younger child (left).
In general, the correlation between dental age and bone age is, however, relatively low (Fig. 2-7).

**Disorders of growth and puberty**

**Intrauterine growth restriction**

Many adverse conditions can lead to impairment of intrauterine growth and development. Infections, medications, environmental chemicals, exposure to tobacco, maternal diseases, and uteroplacental insufficiency may cause early or late growth restriction. A fetus may follow a growth trajectory below normal throughout pregnancy and be born SGA, or growth restriction may have its onset during the third trimester and lead to IUGR.

Over the past few decades research has revealed that antenatal and early postnatal growth patterns may have health consequences in adult life, which may be caused by fetal programming to accommodate adverse conditions. Links have been established to cardiovascular disorders, dyslipidemia, diabetes mellitus, pubertal timing, and reproductive function. The majority of children born SGA or IUGR (80–85%) will show spontaneous catch-up growth after birth, typically within the first 2–3 years of life. Thus, the remaining 10–15% of IUGR/SGA children do not show catch-up growth and remain short in childhood and end up as short adults. These children respond well to treatment with biosynthetic growth hormone. Silver–Russell syndrome is associated with prenatal as well as postnatal growth failure, and children typically respond with significant improvement of final height despite the fact that they generally have no evidence of growth hormone deficiency.

**Postnatal growth failure**

Today, being of short stature is less well accepted by many societies than being tall. Therefore, many children are presented in the clinic (Fig. 2-8). In the majority of cases a growth curve evaluation will reveal that the child is within its family potential. A typical growth curve of a child with familial short stature is shown in Fig. 2-8(a). These children are typically growing at a normal growth rate and thus following their growth trajectory. These families need reassurance, as there is today no convincing treatment schedule available that will reliably and significantly increase final height. If, however, the child’s position on the growth curve does not correspond to the familial potential or the growth trajectory deviates downwards due to low growth velocity, the child should be investigated further. Many chronic and systemic diseases (e.g., asthma, sleep apnea, malabsorption, and metabolic diseases) and systemic steroid treatment may lead to growth disorders. In rare cases even large doses

![Figure 2-7](image-url) Illustrative examples showing low correlation between bone age and dental age. (a) A healthy girl, aged 10 years 9 months, with advanced dental maturity (nearly complete permanent dentition: DS4, M1) compared to the skeletal maturity (prepubertal hand–wrist radiograph). (b) A healthy girl, aged 11 years 6 months, with delayed dental maturity (early mixed dentition: DS2, M1) compared to the skeletal maturity (postpubertal hand–wrist radiograph).
Figure 2-8