

# **Paediatric Dentistry**



Edited by Richard Welbury Monty S. Duggal Marie Thérèse Hosey paediatric dentistry

## paediatric dentistry

#### **FIFTH EDITION**

#### Edited by

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## Preface to the first edition

The child and adolescent deserves the best of care in all the different disciplines of dentistry. I was delighted to be given the opportunity of trying to draw together all the different aspects of paediatric dentistry and am most grateful to my colleagues for agreeing to contribute the various chapters which make up the book. We have tried to cover as much ground as possible within the obvious publishing restrictions and the finished product will inevitably reflect the editor's perceptions of where current needs and deficiencies exist. The sudden increase in both erosive tooth surface loss and cosmetic awareness in our younger patients made their inclusion in Chapter 8 important, where previously they have not achieved such prominence in paediatric texts. Similarly, periodontal disease (Chapter 10), oral pathology and oral surgery (Chapter 14), and disability (Chapter 16) are as deserving of detailed inclusion in a 'paediatric' as much as in any 'general' text.

The book was written with undergraduate dental students in mind, but we hope it will also be useful to those engaged in postgraduate studies and to general dental practitioners. Exhaustive references are deliberately not given but suggested 'Further reading' lists are included to help expedite further enquiry and learning.

I hope we have shown in *Paediatric Dentistry* that the early years of life are the time to get it right for the child and adolescent and there is no reason why our young patients should be denied correct and appropriate care.

R.R.W. Newcastle upon Tyne *April 1996* 

## Preface to the second edition

I am delighted to be given the opportunity to edit the second edition of this popular textbook and am grateful to my colleagues for their continuing contribution. The reviews of the first edition identified the need for a chapter on the treatment of caries in the preschool child and I am grateful to Stephen Fayle for undertaking this task.

There have been small modifications and updates to most chapters, which should keep the reader abreast of current theory and practice. Greater use has also been made of 'Key points' for revision purposes.

I hope the second edition will continue to help both undergraduates, postgraduates, and general dental practitioners in their practice of paediatric dentistry.

> R.R.W. Newcastle upon Tyne January 2001

## Preface to the third edition

I was very pleased when my younger colleagues Marie Thérèse Hosey and Monty Duggal accepted my offer to join me in editing this third edition. Our book has now sold four and a half thousand copies since its launch in 1997 and it is essential that we maintain a contemporary outlook and publish changes in techniques and philosophies as soon as they have an evidence base.

Since 2001 and the second edition, there have been a significant number of changes of authorship, as well as a change of chapters for some existing authors.

Gerry Winter died in December 2002. He was a wise colleague and friend who was a mentor to many of us. I continue to miss his expertise and availability for consultation, by post or telephone, which he freely gave even after his retirement.

John Murray, Andrew Rugg-Gunn, and Linda Shaw have now retired from clinical practice. I am indebted to them all for their support, both in my own personal career and in the production of out textbook. I am grateful to them for allowing the new chapter authors to use their texts and figures.

The restorative section of the book has been remodelled. The endodontics chapter in the previous editions has now been incorporated into either Chapter 8 or Chapter 12, and there are separate chapters relating to the operative care of the primary and the permanent dentitions. Without the help and friendship of Jim Page the original 'Operative care of dental caries' chapter would not have been possible. I am grateful to Jim for allowing us to continue to use his original illustrations from that chapter.

Although designed for the undergraduate we hope the new edition will continue to be used by undergraduate, postgraduate, and general dental practitioner alike, and that their practice of paediatric dentistry will be both fulfilling and enjoyable.

R.R.W. Glasgow January 2005

## Preface to the fourth edition

It is difficult to believe that 17 years have passed since work began on the first edition. Our Portuguese edition was marketed in 2007 and this has broadened our market significantly in Brazil.

This edition contains some new contributors, Liege Lourenço-Matharu, Lucy Burbridge, Jenny Harris, and Toby Gillgrass, and their enthusiasm and insight have been invaluable. We are grateful to the comments from reviewers and OUP staff which have resulted in revision of a number of chapters, and so we hope that the end product will be as well received as previous editions.

The excitement of a new edition is tinged with sadness at the loss of Nigel Carter at such a young age. Nigel always gave very generously of his time to paediatric colleagues. On a happier note, Peter Gordon is now enjoying a well-earned retirement and we would all like to thank him for his contribution to the three previous editions.

In looking at the author list for the fourth edition we are struck by the number of contributors who have gained promotion to professorial positions since they originally contributed to *Paediatric Dentistry*. We must be doing something right!

Richard Welbury Marie Thérèse Hosey Monty Duggal *April 2012* 

## Preface to the fifth edition

It is now 21 years since the first edition of *Paediatric Dentistry* was published. We are privileged to be a recommended text in all UK and Ireland Dental Schools and also in many Schools across the world, especially in Brazil where *Paediatric Dentistry* is now published in Portuguese.

We welcome many new contributors to the fifth edition: Barbara Chadwick, Fiona Gilchrist, Guy Jackson, Anjali Kandiah, Sondos Albadri, Simon Stone, Susan Parekh, Kathryn Harley, Agnes Bloch-Zupan, Alex Keightley, Alexander Crighton, and Graeme Wright. It has been a pleasure to have you working with us.

We would like to thank colleagues who contributed previously but who have now retired from clinical practice: Lindsay Hunter, Peter Crawford, and Michael Aldred. Thank you for your time and dedication and best wishes for the future.

As always, as clinical teachers we are especially proud of our former students who are now colleagues and contributors to *Paediatric Dentistry*.

Finally, thank you to our patients and their parents who have put their trust in us.

Richard Welbury Marie Thérèse Hosey Monty Duggal *August 2017* 

## Contents

Abl Cor	previations htributors to the fifth edition	xiii xvi
1	Craniofacial growth and development T.J. Gillgrass and R. Welbury	1
2	Introduction to the dental surgery A.S. Blinkhorn and B.L. Chadwick	16
3	History, examination, risk assessment, and treatment planning <i>F. Gilchrist and H.D. Rodd</i>	31
4	Safeguarding children J.C. Harris and R. Welbury	49
5	Management of pain and anxiety M.T. Hosey, L. Lourenço-Matharu, and G.J. Roberts	67
6	Local anaesthesia for children J.G. Meechan and G. Jackson	84
7	Diagnosis and prevention of dental caries C. Deery and K.J. Toumba	97
8	Treatment of dental caries in the preschool child S.A. Fayle and P. Kandiah	117
9	Operative treatment of dental caries in the primary dentition <i>M.S. Duggal and P.F. Day</i>	129
10	Operative treatment of dental caries in the young permanent dentition J.A. Smallridge and S. Albadri	155
11	Advanced restorative dentistry N.M. Kilpatrick and L.A.L. Burbridge	183
12	Periodontal diseases in children P.A. Heasman and P.J. Waterhouse	208
13	Traumatic injuries to the teeth R. Welbury, J.M. Whitworth, S.J. Stone, and M.S. Duggal	227
14	Anomalies of tooth formation and eruption S. Parekh, K. Harley, and A. Bloch-Zupan	257
15	The paedodontic–orthodontic interface T J. Gillgrass and A.J. Keightley	277

### XII Contents

16	Oral medicine and oral surgery in children A. Crighton and J.G. Meechan	310
17	Medical disability M.T. Hosey and R. Welbury	341
18	Childhood impairment and disability J.H. Nunn and G. Wright	359
Ind	lex	377

## Abbreviations

AAGBI	Association of Anaesthetists of Great Britain and Ireland
ABH	angina bullosa haemorrhagica
AC	alveolar crest
AD	autosomal dominant
ADHD	attention-deficit hyperactivity disorder
ADJ	amelodentinal junction
AI	amelogenesis imperfecta
AIDS	acquired immunodeficiency syndrome
ALL	acute lymphocytic leukaemia
ALOSS	attachment loss
ALP	alkaline phosphatase
AML	acute myeloid leukaemia
AMSA	anterior middle superior alveolar nerve block
AP	anteroposterior
APAGBI	Association of Paediatric Anaesthetists of Great Britain and Ireland
APECED	autoimmune polyendocrinopathy-candidosis-ectodermal dystrophy
APF	acidulated phosphate fluoride
ASA	American Society of Anaesthesiologists
ASD	atrial septal defect
BASCD	British Association for the Study of Community Dentistry
BDA	British Dental Association
BNF	British National Formulary
BoNT-A	botulinum neurotoxin type A
BPA	bis-phenol A
BPE	Basic Periodontal Examination
BRIT	bi-rotational injection technique
BSPD	British Society of Paediatric Dentistry
BW	bitewing
CAT	computed axial tomography
СВСТ	cone beam computed tomography
CCLAD	computer-controlled local anaesthetic device
CDC	US Centers for Disease Control and Prevention
CEJ	cemento-enamel junction
CJD	Creutzfeldt–Jakob disease
CLD	certainly lethal dose
CLP	cleft lip and palate
СМС	chronic mucocutaneous candidiasis
CNS	central nervous system
COP-DEND	Committee of Postgraduate Dental Deans and Directors
COSHH	Control of Substances Hazardous to Health
СР	cleft palate
CPP-ACP	casein phosphopeptide-amorphous calcium phosphate
СТ	computed tomography
DDAVP*	1-desamino-8-D-arginine vasopressin
DI	dentinogenesis imperfecta
DIC	disseminated intravascular coagulation
DMFS	decayed, filled, and missing tooth surfaces
DMFT	decayed, missing, and filled teeth
DO	distal–occlusal
DPC	direct pulp capping
EACA	epsilon-aminocaproic acid

extra-alveolar dry time
European Academy of Paediatric Dentistry
extra-alveolar time
ethyl chloride
early childhood caries
electrocardiogram
ectodermal dysplasia
external inflammatory resorption
ear, nose, and throat
electric pulp tester
erbium: vttrium aluminium garnet
fluoride
fibre-optic transillumination
glucose-6-phosphate dehydrogenase
general anaesthetic
gamma-aminobutyric acid
general dental practitioner
glass ionomer cement
gastrointectinal tract
general medical practitioner
general medical practitioner
ganaral practitionar
buman borness irus
human herpesvirus
human initiationente entigen
human leucocyte anugen
hand over mouth exercise
noimium: yttrium aluminium garnet
numan papilioma virus
Intercollegiate Advisory Committee for Sedation in Dentistry
Inferior alveolar nerve block
International Carles Diagnosis and Assessment System
Index of Complexity, Outcome, and Need
intellectual disability
insulin-dependent diabetes mellitus
intramuscular
international normalized ratio
Index of Orthodontic Treatment Need
indirect pulp capping
intermediate restorative material
intra-ligamentary anaesthesia
idiopathic thrombocytopenic purpura
intravenous
keratinized gingiva
iodoform paste
local anaesthetic
leucocyte adhesion deficiency syndrome
Langerhans cell histiocytosis
low-level light therapy
Modified Dental Anxiety Scale
Modified Child Dental Anxiety Scale (faces)
metered dose inhaler
multidisciplinary team
sodium monofluorophosphate
major histocompatibility complex
molar incisor hypomineralization, molar incisor hyperplasia
molar incisor hypomineralization, molar incisor hyperplasia measles, mumps, and rubella

MTA	mineral trioxide aggregate
NAI	non-accidental injury
Nd:YAG	neodymuim: yttrium aluminium garnet
NECAT	New England Children's Amalgam Trial
NF	neurofibromatosis
NICE	National Institute for Health and Clinical Excellence
NRL	natural rubber latex
NSAID	non-steroidal anti-inflammatory drug
NUG	necrotizing ulcerative gingivitis
OCA	operative care advised
OFD	oral–facial–digital
OFG	orofacial granulomatosis
ОНІ	oral hygiene instruction
OME	otitis media
ΟΡΤ	orthopantomogram (also abbreviated to OPG)
PAR	Peer Assessment Rating
PASA	palatal approach superior alveolar nerve block
PCA	preventive care advised
PCS	patient-controlled sedation
PDL	periodontal ligament
PEG	percutaneous endoscopic gastrostomy
PGE.	prostaglandin E.
PILS	Paediatric Immediate Life Support
PJC	porcelain jacket crown
PL	periodontal ligament
PLD	potentially lethal dose
PLS	Papillon–Lefèvre syndrome
РМС	preformed metal crown
ppm	parts per million
PRR	preventive resin restoration
psi	pounds per square inch
PVAC-PE	polyvinylacetate-polyethylene
RCOA	Royal College of Anaesthetists
RCT	root canal treatment
RET	regenerative endodontic technique
RMGIC	resin-modified glass ionomer cement
SBE	subacute bacterial endocarditis
SCAP	stem cells of the apical papilla
SCC	squamous cell carcinoma
SDCEP	Scottish Dental Clinical Effectiveness Programme
SIGN	Scottish Intercollegiate Guidelines Network
SLS	sodium lauryl sulphate
SMMCI	solitary median maxillary central incisor
STA	single-tooth anaesthesia
STD	safely tolerated dose
TAB	transient apical breakdown
TENS	transcutaneous electrical nerve stimulation
TMD	temporomandibular joint disorder
TSL	tooth surface loss
ТТР	tender to percussion
TTS	titanium trauma splint
TWA	time-weighted average
TWI	tooth wear index
VSD	ventral septal defect
WHO	World Health Organization
WSN	white sponge naevus

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# Craniofacial growth and development

#### T.J. Gillgrass and R. Welbury

#### **1.1** Introduction

This chapter describes, in general terms, the prenatal development and postnatal growth of the craniofacial skeleton, and the occlusal development of the primary and permanent dentitions.

#### **1.2** Prenatal development

Understanding of embryological development is essential for the dental practitioner who may frequently face patients with common craniofacial anomalies such as cleft lip and/or palate. For routine care, an understanding of their development and aetiology will bring insight to their likely presenting signs and symptoms.

This section will include a brief summary of the development of the face, including the neural crest and pharyngeal arches. It is not the intention of this summary to be in any way a complete or thorough description but simply to describe some of the key cells/interactions and structures.

#### 1.2.1 Neural crest

Neural crest cells are derived from the neural fold, and are highly migratory and specialized cells capable of predetermined differentiation. The differentiation occurs after their migration and is essential for the normal development of face and teeth (Fig. 1.1).

#### **1.2.2** Branchial arches

By week 4 the primitive mouth or stomatodeum is bordered laterally and from the developing heart inferiorly by the pharyngeal or branchial arches (Fig. 1.2). These are six bilateral cylindrical thickenings (although the fifth and sixth are small) which form in the pharyngeal wall and into which the neural crest cells migrate. They are separated externally by the branchial grooves and internally by the pharyngeal pouches. The first groove and pouches are involved in the formation of the auditory apparatus and the Eustachian tube.

Each arch has a derived cartilage rod, muscular, nervous, and vascular component. The first two arches and their associated components are central to the development of the facial structures.

This period is also characterized by the development of the organs for hearing, sight, and smell, namely the otic, optic, and nasal placodes.



Fig. 1.1 A child with the hemifacial microsomia part of the oculoauricular-vertebral spectrum. The unilateral inhibition of neural crest migration and bronchial arch development results in (a) marked asymmetry and (b) ear defects.



**Fig. 1.2** Week 6–7 embryo showing branchial arches and migration of neural crest cells into the branchial arch system. Reprinted from Ten *Cate's Oral Histology*, 6th edition, Antonio Nanci, copyright (2003) with permission from Elsevier.

#### 1.2.3 Facial development

By the end of week 4, thickenings start to develop in the frontal process. The medial and lateral frontonasal processes develop from these, together with the nasal placodes.

The maxillary process develops from the first pharyngeal arch and grows forward to meet the medial and nasal processes, from which it is separated by distinct grooves at week 7 (Fig. 1.3). Its eventual fusion with them creates the upper lip and, from the two medial nasal processes, the incisor teeth and the primary palate. Where this fusion is disturbed a cleft of the lip may form (Fig. 1.4).

The lower lip is formed by fusion of the mandibular process from the first arch.

By week 8 the odontogenic epithelium, which will differentiate into tooth-forming cells, can be determined on the inferior border of the maxillary process, the lateral aspect of the medial process, and the superior border of the mandibular processes.

#### 1.2.4 Secondary palate

Development of the secondary palate starts around week 7. It is formed from three processes: the nasal septum develops in the midline from the frontonasal process and the two palatine shelves develop from

#### **1.3** Postnatal craniofacial growth

There is a great deal of individual variation in the process of postnatal growth and the final form of the craniofacial structures. This section presents a simplified and rather idealized account of bone growth in general and as part of craniofacial growth. Occlusal development is then the maxillary processes. At this stage the palatal shelves are directed downwards on either side of the tongue. Between weeks 7 and 8 they elevate to meet the primary palate and nasal septum, to which they fuse (Fig. 1.5).

The trigger for this elevation is still unclear, although high concentrations of glycosaminoglycans which attract water and increase turgidity in the shelves, contractile fibroblasts, and the position of the tongue have all been implicated. Once in contact, the epithelial covering of the shelves must disappear to allow the fusion. Various methods including cell death ('apoptosis') and cell transformation have been suggested as methods by which this epithelial covering is lost.

If the shelve fusion fails, this is likely to result in clefts of the palate. The extent of these clefts varies clinically from submucous clefts, affecting the bony structure of the palate and underlying muscular attachment and clefts of the soft palate which may or may not have significant effects on speech, to those including the hard palate producing communication between the nasal and oral cavities (Fig. 1.6). There appear to be distinctive differences between clefts of the palate and those of the lip and palate within different geographical and sexual distributions. This also suggests different disruptive mechanisms and timings, as lip closure occurs earlier in development than palatal fusion. However, as clefts of the palate alone and lip with palate can occur in certain families, it suggests that the distinction may not be complete.

described, before going on to discuss the effect of individual variation in producing departures from this idealized pattern.

An individual's stature can be charted on standard growth charts during growth. This will present an overall view of the process of growth and



Fig. 1.3 Diagrammatic representation of early facial development from 4 to 10 weeks i.u.: (a) 4th week *in utero* (i.u.); (b) 28 days i.u.; (c) 32 days i.u.; (d) 35 days i.u.; (e) 48 days i.u.; (f) 10 weeks i.u. Reproduced from Mitchell L., *Introduction to Orthodontics*, 4th edition, fig 4.2, page 37, 2013 with permission from Mitchell L, Oxford University Press.



**Fig. 1.4** Failure of fusion resulting in cleft lip and primary palate.

help to detect instances where growth is not proceeding in the usual manner. However, it disguises the fact that the various tissues of the body grow at different rates at different ages (Fig. 1.7).

In order to maintain harmonious facial growth, bone growth must synchronize with that of other tissues. For example, growth of the calvarium is linked to growth of the brain. The cranial vault initially grows much more rapidly than the facial bones in order to keep pace with the developing brain, 90% of which is complete by 5 years of age.

## **1.3.1** Assessment of postnatal craniofacial growth

One way of assessing the changes that take place during craniofacial growth is to superimpose tracings of two lateral skull radiographs taken of the same person at different ages. The two radiographs can be compared, as shown in Fig. 1.8, and the changes that have taken place during growth can be examined. A potential difficulty with this approach is that the various bones of the skull grow at different rates at different ages, and there is no single central point about which growth occurs in a radial fashion, i.e. there is no valid fixed radiographic landmark on which to superimpose the films. One convention is to superimpose the tracings of the sella to the frontonasal suture to orientate the films. If this method of superimposition is used, it appears that the cranium expands in a more or less radial fashion to accommodate the brain and the facial skeleton then grows downwards and forwards, away from the cranial base.

Another difficulty is that radiographs only produce a two-dimensional representation of what is a three-dimensional structure. Newer



**Fig. 1.5** Diagrammatic representation of palatal shelf elevation and subsequent fusion. (a) During week 7 i.u. the palatal shelves begin to develop and lie on either side of the tongue. (b) During week 8 i.u. the palatine shelves elevate rapidly owing to the internal shelf-elevating force and developmental changes in the face. (c) During week 9 i.u. the shelves fuse with each other, the primary palate, and the nasal septum. MC, Meckel's cartilage; asterisks, palatal shelves. Reproduced from Mitchell L., *Introduction to Orthodontics*, 4th edition, fig 4.3, page 37, 2013 with permission from Mitchell L, Oxford University Press.



Fig. 1.6 Diagrammatic representations of some of the different types of clefts of the lip and palate: (a) normal; (b) unilateral cleft lip; (c) unilateral cleft lip and anterior palate; (d) bilateral cleft lip and anterior alveolus; (e) cleft of posterior palate (hard and soft); (f) unilateral cleft of the lip and anterior and posterior palate. Reproduced from Johnson D.R and Moore W.J, *Anatomy for Dental Students*, third edition, fig 2.25, page 52, 1997 with permission from Oxford University Press.



**Fig. 1.7** Postnatal growth patterns for neural, lymphoid, somatic, and genital tissues shown as percentages of the total increase. The patterns for the maxilla and mandible are shown in blue. Reproduced from Scammon R.D., *The Measurement of the Body in Childhood*. In Harris J.A. (Ed.) *The Measurement of Man*. 1930. With permission the University of Minnesota Press.

methods of radiological assessment using computed tomography or lower-radiation-dose cone-beam computed tomography (Fig. 1.9) are capable of producing three-dimensional volumetric images of the facial skeleton (Benington et al. 2010).

Soft tissue growth and facial changes are also important for understanding the effects of underlying bony changes. Although computed tomography will capture the soft tissue, it does not accurately depict the colour and texture, and, particularly in the case of conventional computed tomography, produces a significantly higher radiation dose than plain radiographs.

Non-invasive laser scanning or stereophotogrammetry is capable of producing photo-realistic topography of the facial soft tissues without exposure to radiation. Sequential capture is being used for longitudinal growth studies where colour mapping can help illustrate areas of maximum growth (Fig. 1.10) over a substantial time period.

#### **1.3.2** Bone growth

Mineralized bone is formed through a process known as 'ossification'. This occurs in two ways, **intramembranous ossification** and **endochondral ossification**. Intramembranous ossification occurs



**Fig. 1.8** Superimpositions on the cranial base showing overall downward and forward direction of facial growth: solid line, 8 years of age; broken line, 18 years of age. Reproduced from Mitchell L., *Introduction to Orthodontics*, 4th edition, fig 4.7, page 39, 2013 with permission from Mitchell L, Oxford University Press.



Fig. 1.9 Cone-beam computed tomography of a child with a cleft of the alveolus, manipulated to produce a three-dimensional image.

by membrane activity and is seen in the bones of the calvarium, the facial bones, and the mandible. **Endochondral ossification** occurs by replacement of a cartilage framework. Classically, endochondral ossification is described in long bones but it also occurs in the craniofacial region, most notably in the cranial base.

Growth in bones formed by endochondral ossification occurs at growth centres known as epiphyseal plates in long bones and synchondroses in the cranial base. These are primary growth centres within which the chondroblasts are aligned and clear zones of cell division, hypertrophy, and calcification occur. The most notable of the three synchondroses within the cranial base is the sphenooccipital synchondrosis. The condylar cartilage has a different histological appearance to that of the epiphyseal plates and synchondroses. Although capable of producing bone, its stimuli appear more reactionary to growth around it rather than the primary growth sites which react to both internal and external stimuli.



Fig. 1.10 Colour mapping after sequential laser scanning showing facial growth in a forward direction (red) and a negative direction (blue) in the AP plane. Courtesy of Professor Steve Richmond.

The apparent growth of the facial bones is a function of **remodelling** and **displacement** or **translation**. Remodelling results in an alteration in the size and shape of bones by deposition and resorption of material on the external and internal surfaces of the bone and suture systems. It is a function of the 'periosteum' or 'osteogenic membrane'. Deposition and resorption go hand in hand; one seldom occurs without the other. Deposition of bone on one aspect of a cortical plate of bone is accompanied by resorption on the other aspect. Displacement or translation occurs when one bone is moved relative to another, primarily due to another area of growth; for example, the maxilla is translated downwards and forwards by growth of the spheno-occipital synchondroses and nasal septum. Such translation will be accompanied by a degree of remodelling.

The suture systems form bone when subjected to traction. In the case of the calvarial bones, the suture systems form new bone and enable the bones to stay in contact with each other when the expansion of the growing brain would otherwise move them apart.

The suture systems allow the bones to respond to growth in neighbouring soft tissue. The suture systems lying between the maxilla and the cranial base allow the downward and forward translation of the maxilla in response to the growth of the soft tissues of the face. It is not proliferation of the vascular connective tissue in the sutures that pushes the bones apart; the whole arrangement of the connective tissue in a suture seems to be designed to enable the suture to respond to a tensile force.

#### 1.3.3 Soft tissue growth

The effects of bony growth can be masked or accentuated by the overlying soft tissues. Notably, this is shown intra-orally in the positions of the dental arches in the so-called neutral zone between the effects of the tongue, lips, and cheeks. The soft tissues are also responsible for dento-alveolar compensation where the position of the teeth attempts to compensate for skeletal jaw discrepancies.

The growth of the soft tissues, particularly the nose and the length and thickness of the lips, has a profound effect on the appearance of the face. Soft tissue growth shows sexual dimorphism, with changes occurring later and for longer in boys. Changes in the the nose continue into adulthood.

#### 1.3.4 Mechanisms of growth

The mechanisms controlling the process of facial growth are not completely understood. In the post-genomic era it is becoming apparent that genetically encoded factors have a major effect on craniofacial growth; after all, children tend to resemble their parents in facial appearance (Carlson 2005). This may be particularly noted in class III patients and those with a class II division 2 malocclusion. The alternative school of thought is that growth is only loosely under genetic control; rather, the final shape is under the control of its soft tissue environment. This is known as the 'functional matrix theory'. This is best shown in the cranial vault where the bone growth is reactionary to neural expansion. However, it fails to explain mid-facial growth through the synchondroses.

Therefore it is likely that both mechanisms come into play, The genetically encoded factors can be affected by factors outwith the DNA 'epigenetic' that are able to switch them off or on. If this is the case, it should be theoretically possible to influence them (e.g. the use of functional appliances to encourage mandibular growth). At present, however, although a positive response is possible, it appears extremely variable and unpredictable between individuals.

#### 1.3.5 Cranial growth

At birth, the cranium is some 60–65% of its adult longitudinal dimensions, and this increases to about 90% by the age of 5 years. The calvarial bones are carried away from each other by the expanding brain and respond by forming new bone in the sutures that separate the bones of the vault of the skull (Fig. 1.11). The six fontanelles that are present at birth reduce in size. The largest (the anterior fontanelle) closes at about 1 year of age and the last to close (the posterolateral fontanelle) closes at about 18 months. The calvarial bones undergo a process of remodelling, with areas of bone deposition and resorption altering the contour of the bones as the volume of the brain cavity increases. Early fusion of the cranial sutures, or 'craniostenosis', results in compensatory growth from the other sutures. This can result in unusual head shapes, and may produce detrimental effects on brain growth and development as it may be accompanied by increased intracranial pressure. The most common craniostenosis involves the sagittal suture. Compensatory growth results in a head shape that is increased in the anteroposterior direction and narrow laterally—'dolichocephaly'. If the suture fusion is asymmetric, the deformation is also asymmetric— 'plagiocephaly' (Fig. 1.12).

The cranial base also grows to accommodate the changes in the size and shape of the brain, but the process is different to that seen in the calvarial bones. There is considerable lateral growth of the cranial base



Fig. 1.11 The skull at birth showing the sagittal, coronal, frontal, and lamboid sutures. Reproduced from Johnson D.R and Moore W.J, *Anatomy for Dental Students*, third edition, fig 2.46, page 71, 1997 with permission from Oxford University Press.







(b) DOLICHOCEPHALY

(c) ANTERIOR PLAGIOCEPHALY

Fig. 1.12 Skull morphology: (a) normal; (b), (c) abnormal due to early fusion of cranial sutures.





as the cerebral hemispheres expand, but less increase in the anteroposterior dimension. No sutures are present to allow for expansion of the deeper compartments of the cranial base, a process that takes place by surface deposition and extensive remodelling. In addition, the three synchondroses (spheno-occipital, intersphenoid, and spheno-ethmoidal) in the mid-ventral floor of the cranial base allow for increases in the anteroposterior dimension by endochondral ossification. Growth in the spheno-occipital synchondrosis does not cease until about the age of 15 years in boys and rather earlier in girls, and it closes fully at about the age of 20. The spheno-occipital synchondrosis has a significant influence on the growth of the facial region as the condylar fossa is posterior to it, but the anterior cranial base, and therefore the nasomaxillary complex to which it is attached by a suture system, sits anteriorly. As a consequence, as it grows it has an effect on how the maxilla and mandible relate to each other (Fig. 1.13).

The pattern and the timing of growth in the cranial base is intermediate between the neural type of growth that characterizes the growth of the calvarial bones and the musculoskeletal pattern of growth exhibited by the facial skeleton.

The shape of the cranial fossae is much more complex than the relatively smooth form of the bones of the vault of the skull. Surface deposition and subsequent remodelling occurs, with the final size and shape of the compartments being determined by the size of the lobes of the brain forming the partitions which separate the cranial fossae.

#### **1.3.6** Nasomaxillary growth

The nasomaxillary region, which makes up the middle third of the face, is a complex area comprising a number of bones joined to each other and to the anterior cranial base by a suture system. The nasomaxillary complex grows downwards and forwards relative to the cranial base. This is accomplished through cranial base growth and deposition within the suture system as it is carried forward; there is also deposition in the region of the tuberosity, lengthening the alveolus in this region. The anterior surface is remodelled not by apposition, as might be expected in a bone that is growing forward, but by resorption. Failure of the cranial base to lengthen, as seen in achondroplasia and a number of other syndromes (Fig. 1.13), results in characteristic faces with lack of mid-face prominence.

Unlike the growth of the cranium, which occurs in conjunction with the growth of the brain, the nasomaxillary complex grows fastest at about the time of the pubertal growth spurt, in conjunction with the general growth of the musculoskeletal system.

As bone is deposited on the external aspect of the maxilla in the region of the tuberosity, and vertically with the development of the alveolus through tooth eruption, it is also resorbed from the internal aspect of the bone in this area, thereby enlarging the maxillary sinus. As the bone is translated downwards, the nasal cavities and the maxillary sinus expand by a process of bone resorption at the floor of the nose and the sinus, together with bone deposition on the palatal aspect of the maxilla.

#### **1.3.7** Mandibular growth

Growth of the mandible, like that of the maxilla, is coordinated with the pattern of general musculoskeletal growth, growing at its fastest rate at about the time of the pubertal growth spurt. Growth of the mandible has to be coordinated with the downward and forward growth of the maxilla. This task is made more complicated by the fact that the mandibular condyles articulate in the glenoid fossa, which lies behind the spheno-occipital synchondrosis, while the maxilla lies in front of it; therefore growth of the mandible has to keep pace not just with the translation of the maxilla, but also with growth in the cranial base (Fig. 1.14).

Taking the anterior cranial fossa as a stable reference area, it appears that the mandible, like the maxilla, grows downwards and forwards. As is the case with the maxilla, this downward and forward growth is not achieved by deposition of bone on the anterior aspect of the mandible, but by translation of the bone accompanied by growth in the region of the ramus and the mandibular condyle. Bone is deposited on the posterior aspects of the ramus and the coronoid processes and resorbed from the anterior aspect of the ramus. At the same time the condylar cartilage contributes to growth of the mandibular condyle, although its growth appears more reactionary than a primary growth cartilage.



**Fig. 1.14** Anteroposterior growth at the spheno-occipital synchondrosis affects the anteroposterior relationship of the jaws. Reproduced from Mitchell L., *Introduction to Orthodontics*, 4th edition, fig 4.10, page 40, 2013 with permission from Mitchell L, Oxford University Press.

That is, it is not proliferation of the condylar cartilage that pushes the mandible downwards and forwards, but the condyle essentially 'fills in' as the mandible is translated.

As the ramus of the mandible grows upwards and backwards, its anterior aspect undergoes resorption and becomes remodelled into the body of the mandible. This process involves resorption on the lateral aspect of the bone and deposition on the lingual aspect, which forms new bone in correct alignment with the body of the mandible and helps maintain an appropriate intercondylar width. Growth of the mandibular ramus and condyle has to keep pace with changes in the position of the maxilla, in both vertical and horizontal directions, and with growth in the middle cranial fossa. Until puberty the mandible will grow at approximately 1–2mm per year, but after puberty this may double. It is easy for a small discrepancy to arise, for example in the amount of vertical growth of the mandibular ramus, resulting in a rotation of the body of the mandible and a corresponding tilt of the occlusal plane (Houston 1988). These rotations have been demonstrated using implant studies (Bjork 1955). The rotations may be partially masked by resorption, but are capable of a significant effect on the vertical dimension of the face and on skeletal relationships.

#### **1.3.8** Normal variation

There is always variation between individuals. Variation in the pattern of facial growth is only to be expected, and there are a number of compensatory mechanisms which operate to minimize the impact of such variation (Solow 1980). Variation in the position or size of one structure is often compensated by corresponding change in another. The process of growth is constantly creating imbalances, as related structures grow and develop at different rates, but the overall direction of growth is towards some position of overall balance or harmony.

Anteroposterior discrepancies can arise during facial growth because of the position of a bone, or an imbalance in the sizes of bones, or a mixture of both. A class II skeletal pattern can be caused by insufficient growth of the ramus of the mandible in a backward direction; alternatively, a class II skeletal pattern can be the result of a backward tilt of the middle cranial fossa. This change in angulation results in the maxilla having a more anterior position, relative to the glenoid fossa, than would otherwise have been the case. The normal-sized mandible, occluding in the glenoid fossa, now has a class II relationship with the normal-sized maxilla. In a similar way, a converse alteration in the pattern of growth—excessive backward growth of the ramus of the mandible or a more vertical tilt of the middle cranial fossa—can produce a class III skeletal relationship, with the accompanying dental malocclusion.

Vertical growth in the nasomaxillary region has to be combined with vertical growth of the mandibular ramus. Maxillary growth that is not matched by mandibular growth will result in mandibular rotation. If there is an excess of vertical maxillary growth that is not matched by vertical growth of the ramus, the effect will be to produce a downward and backward rotation of the mandible. This downward and backward rotation will, in turn, produce an anteroposterior discrepancy, with a tendency towards a class II relationship.

Horizontal and vertical discrepancies tend to be accompanied by **dento-alveolar compensations**. In the case of a class III skeletal pattern, the upper incisor teeth are frequently proclined and the lower incisor teeth retroclined, as illustrated in Fig. 1.15.



(a)

(b)

Fig. 1.15 Dento-alveolar compensation: (a) cephalometric radiograph; (b) clinical photograph.





These compensations are almost certainly brought about by muscular activity in the soft tissue integument affecting the position of the teeth, and they minimize what might otherwise have been a large reversed incisor overjet. In the case of class II skeletal patterns, the dentoalveolar compensation can take two forms. If the lips function in front of the upper incisor teeth, these teeth are generally retroclined, with the effect that the incisor overjet is virtually normal. However, if the lower lip functions behind the upper incisors, these teeth are usually proclined and the lower incisors are retroclined to make way for the lip. This results in an increased incisor overjet (Fig. 1.16).

Downward and backward mandibular rotations tend to be accompanied by a vertical drifting of the premolar, canine, and incisor teeth to compensate for an arrangement that would otherwise have produced an anterior open bite. This vertical drifting should be distinguished from over-eruption, which would produce a lengthening of the clinical crowns of the teeth.

## **1.3.9** Modification of the pattern of facial growth

Attempts to modify the pattern of facial growth have met with a certain amount of success. Orthodontic appliances have been designed which hold the mandible postured downwards and forwards. This moves the condyle out of the glenoid fossa and encourages upward and backward growth of the mandibular ramus. At the same time, the stretched

#### **1.4** Tooth development

Teeth start to form during week 5 of embryonic life, and the process of tooth formation continues until the roots of the third permanent molars are completed at about 20 years of age. The stages of tooth formation are the same

muscles of mastication exert an upward and backward force (through the appliance) to the maxilla, which tends to inhibit its downward and forward growth. The effect on the growing face is to help correct a developing class II skeletal pattern. If this pattern is being caused not so much by underdevelopment of the mandible (which seems to be the most common cause) but by excessive forward growth of the maxilla, an appliance may be used simply to exert an upward and backward force on the maxilla without involving the mandible, thus helping to influence the course of facial growth.

These appliances apply forces to the developing maxilla and mandible via the teeth-the appliances are attached to the teeth-and work partly by inducing a dento-alveolar compensation for the underlying skeletal discrepancy. This process is sometimes referred to as 'orthodontic camouflage'. There is likely to be some restraint of the downward and forward growth of the maxilla, but the appliances seem to have only a minimal effect on the eventual size of the mandible. The so-called myofunctional appliances, which derive their impetus from the muscles of mastication, are used most often to help correct class II malocclusions. The appliances work by maintaining a forward and downward posturing of the mandible. However, while it is possible to use myofunctionalfunctional appliances to correct a developing class III malocclusion, they are less often used in this context as it is difficult to obtain the necessary backward posturing of the mandible. In addition, the dento-alveolar compensations that these appliances tend to produce are often already present in untreated class III occlusions.

whether the tooth is of the primary or the permanent dentition, although obviously the teeth develop at different times. The tooth-germs develop from the dental lamina, a sheet of epithelial cells which itself develops from the primary epithelial band. This is a layer of thickened epithelium which forms around the mouth in the area soon to be occupied by the upper and lower jaws. The primary epithelial band quickly organizes into two discrete epithelial ingrowths—the vestibular lamina and the dental lamina. The vestibular lamina grows down into the underlying ectomesenchyme, and the epithelial cells enlarge and then break down, thereby forming the cleft which becomes the sulcus between the cheeks and the alveolar processes.

The dental lamina forms a series of epithelial buds that grow outwards into the surrounding connective tissue. These buds represent the first stage in the development of the tooth-germs of the primary dentition. The epithelial bud continues to grow and becomes associated with a condensation of mesenchymal cells to form a tooth-germ at the cap stage of development. The epithelial bud develops into the enamel organ, and the condensation of mesenchymal cells constitutes the dental papilla and extends around the enamel organ to form the dental follicle. The cells at the margin of the epithelial bud continue to proliferate and grow to enfold the mesenchymal cells of the dental follicle, producing a toothgerm at the bell stage of development. Around this time—the transition from cap stage to bell stage—a process of histodifferentiation produces the recognizable structures of the enamel organ, with its external and internal enamel epithelia, stratum intermedium, and stellate reticulum.

Further proliferation of the cells of the dental lamina at a point adjacent to each primary tooth-germ, but on its lingual aspect, produces the tooth-germ of the permanent successor. The tooth-germs of the permanent molar teeth which have no primary precursors are formed by distal extension of the dental lamina which tunnels backwards as the jaws lengthen posteriorly.

The cells of the inner enamel epithelium lengthen to a columnar shape, with the cell nuclei occupying the portion of the cell beside the

#### **1.5** Tooth eruption

For tooth eruption to occur, a force must be generated to propel the tooth through the bone and gingival tissue. In the case of the secondary dentition the primary tooth roots must also be removed.

stratum intermedium, away from the dental papilla. The cells of the dental papilla adjacent to the internal enamel epithelium also elongate to a columnar form, with their nuclei aligned away from the enamel organ and towards the centre of the dental papilla. These columnar cells of the dental papilla differentiate into odontoblasts, the cells which form dentine. Dentine formation, which is induced by the cells of the internal enamel epithelium, always precedes enamel formation. Although dentine is formed by the odontoblasts of the dental papilla, the process is initiated by the epithelial cells of the enamel organ. Once dentine formation begins, the cells of the internal enamel epithelium differentiate into ameloblasts and commence the formation of enamel. Dentine and enamel formation initially occurs in the region of the cusp tips and incisal edges of the teeth, and then continues towards the cervical margin of their crowns.

Differentiation of odontoblasts and the formation of dentine is induced by the cells of the internal enamel epithelium. A similar process is involved in the production of dentine to form the roots of teeth. The epithelial cells at the cervical loop of the enamel organ proliferate and migrate in an apical direction to form a tubular epithelial sheath around the dental papilla. These epithelial cells—the root sheath of Hertwig—induce the formation of odontoblasts from the cells of the dental papilla and the production of dentine to form the roots of the teeth, a process which is not complete until 3–5 years after the eruption of the crown of the tooth.

The stages of tooth development are the same for both primary and permanent teeth, although progression through the stages occurs at different times and varying rates for the different teeth. Tables 1.1 and 1.2 summarize the chronology of tooth development for the two dentitions.

The method by which the force is created is open to debate. Some have suggested cellular proliferation at the apex of the tooth; alternatively, a localized change in blood pressure has been implicated. It

					-						
Stage of	Central incisor		Lateral incisor		Canine		First molar		Second molar		Time
development	Мах	Mand	Max	Mand	Мах	Mand	Max	Mand	Max	Mand	
Hard tissue formation begins	13–16	13–16	14.7–16.5	14.7–16.5	15–18	16–18	14.5–17	14.5–17	16–23.5	17–19.5	Weeks after ovulation
Crown formation complete	1.5	2.5	2.5	3	9	8–9	6	5–6	11	8–11	Months after birth
Beginning of eruption	8–12	6–10	9–13	10–16	16–22	17–23	13–19	14–18	25–33	23–31	Months after birth
Completion of root formation	33	33	33	30	43	43	37	34	47	42	Months after birth

#### Table 1.1 The chronology of the development of the primary dentition

Max, maxilla; Mand, mandible.

Adapted from Oral Structural Biology: Embryology, Structure, and Function of Normal Hard and Soft Tissues of the Oral Cavity and Temporomandibular Joints, Schroeder, H.E., Copyright (1991) with permission from Georg Thieme Verlag, Thieme Medical Publishers, Inc.

Stage of	Central incisor		Lateral incisor		Canine		First pre	First premolar		Second premolar	
development	Мах	Mand	Max	Mand	Max	Mand	Мах	Mand	Max	Mand	
Hard tissue formation begins (histology)	3–4	3–4	10–12	3–4	4–5	4–5	18–24	18–24	24–30	24–30	Months after birth
Hard tissue formation begins (radiology)	-	-	-	-	6	6	19	19	36	36	Months after birth
Crown formation complete	3.3–4.1	3.4–5.4	4.4–4.9	3.1–5.9	4.5–5.8	4.0–4.7	6.3–7.0	5–6	6.6–7.2	6.1–7.1	Years of age (decimal)
Beginning of eruption	6.7–8.1	6.0–6.9	7.0–8.8	6.8–8.1	10.0–12.2	9.2–11.4	9.6–10.9	9.6–11.5	10.2–11.4	10.1–12.1	Years of age (decimal)
Completion of root formation	8.6–9.8	7.7–8.6	9.6–10.8	8.5–9.6	11.2–13.3	10.8–13.	0 11.2–13.6	11.0–13.4	11.6–14.0	11.7–14.3	Years of age (decimal)
Stage of develop	ment First n		olar	Secon	d molar		Third molar		Time		
		Max	Mand	Мах	Man	d	Max	Mand			
Hard tissue formatio (histology)	on begins	7–8ao	7–8ao	30–36r	no 30–3	6mo	7–9yr	8–10yr	Months after ovulation (ao) or months (mo) and years (yr) after birth		n (ao) or rs (yr) after
Hard tissue formatio (radiology)	on begins	2mo	2mo	36–48r	no 36–4	8mo	9–10yr	9–10yr	Months (r birth	no) or years	(yr) after
Crown formation co	mplete	2.1–3.5	2.1–3.6	6.9–7.4	6.2–7	'.4	12.8–13.2	12.0–13.7	Years of a	ge with decir	mal fractions
Beginning of eruption	on	6.1–6.7	5.9–6.9	11.9–1	2.8 11.2-	-12.2	17.0–19.0	17.0–19.0	Years of a	ge with decir	mal fractions
Completion of root	formation	9.3–10.8	7.8–9.8	12.9–1	6.2 11.0-	-15.7	19.5–19.6	20.0–20.8	Years of a	ge with decir	mal fractions

 Table 1.2
 The chronology of the development of the permanent dentition

Max, maxilla; Mand, mandible.

Adapted from Oral Structural Biology: Embryology, Structure, and Function of Normal Hard and Soft Tissues of the Oral Cavity and Temporomandibular Joints, Schroeder, H.E., Copyright (1991) with permission from Georg Thieme Verlag, Thieme Medical Publishers, Inc.

has also been suggested that the force of eruption causes resorption of bone in the tooth's path, although this has also been questioned. Animal experiments have shown that the resorption process can be uncoupled from the eruption process, i.e. it is not necessary for the tooth to erupt to cause resorption of bone.

The follicle has been shown to play an essential role in active tooth eruption, but it is not fully understood how the coronal part of the follicle is activated to initiate osteoclastic activity in the alveolar bone ahead of the tooth and clear a path for tooth eruption. Once the tooth has

#### **1.6** Occlusal development

#### **1.6.1** The primary dentition

The first tooth to erupt is usually the lower central incisor. Occasionally, this tooth is present at birth, but the average age for its eruption is about 7 or 8 months, although inevitably there is some individual variation.

broken through the alveolar crestal bone it reaches the supra-alveolar phase of eruption where it is likely that the follicle plays a lesser role.

What causes the tooth to erupt through the opening in the crypt created by the resorption process is also open to question. Suggested theories including root elongation, periodontal ligament, and local changes in vascular pressure have been discounted as major factors, and although bone growth at the base of the crypt is essential for eruption it is possible that this is simply reactive to tooth movement. It is fair to say that this source of eruption remains elusive.

The other incisor teeth follow soon after, with the upper central incisors erupting at about 10 months followed by the upper lateral incisors at about 11 months and the lower lateral incisors at about 13 months. The first primary molars put in an appearance at about the age of 16 months, followed by the primary canine teeth at about 19 months. The



Fig. 1.17 Spaced upper arch in the primary dentition of a patient.



Fig. 1.18 Anthropoid spaces and flush terminal plane.

second primary molars erupt at about 27–29 months, with the lower teeth usually erupting before the upper ones. While the eruption sequence—the order in which the teeth erupt—is usually as described in this section, there is considerable variation in the actual age at which the teeth erupt. In any event, there is an almost continuous process of tooth eruption between the ages of 7 and 29 months.

Some occlusal features occur relatively frequently in the established primary dentition (Foster and Hamilton 1969).

- 1. The incisor teeth tend to be spaced (Fig. 1.17). If the primary teeth are not spaced, the permanent teeth will be crowded because the larger permanent incisor teeth have to fit into the same space as their smaller primary predecessors. Crowding of the permanent incisor teeth is a relatively common occurrence, but it is seldom so severe that there is no spacing of the primary incisors.
- So-called anthropoid spaces between the upper lateral incisor and the canine and between the lower canine and the first primary molar are particularly common (Fig. 1.18).
- 3. In the case of a class I occlusion, the mesiobuccal cusp of the primary upper second molar occludes in the mesiobuccal groove of the primary lower second molar, a situation analogous to the class I occlusion of first permanent molars. However, the primary lower second molar is much longer mesiodistally than the upper second molar. Therefore the class I occlusion of the mesiobuccal cusp of the upper molar means that the distal surfaces of the teeth are in the same vertical plane, which is known as the flush terminal plane (Fig. 1.18).

The incisor relationship tends more towards edge-to-edge than is the case with permanent teeth (although with increasing wear of the primary teeth there may be a postural element in this) and the upper incisors tend to be more upright. There is sometimes an anterior open



**Fig. 1.19** The primary dentition with an anterior open bite and unilateral cross-bite consistent with habit.

bite associated with a sucking habit, and there may be cross-bites of the buccal segment teeth (Fig. 1.19). However, in general the teeth in the primary dentition tend to be well aligned. While there may be some anteroposterior, lateral, or vertical discrepancy, these deviations are seldom so marked that they give rise to comment.

#### **1.6.2** The mixed dentition

The primary dentition erupts more or less continuously over a two-year period. However, the permanent dentition erupts in two stages: first, the incisor teeth and the first permanent molars erupt, and then the other teeth in the buccal segments. The lower central incisor and the first permanent molars erupt at about the age of 6 years. The upper central incisor and the lower lateral incisor erupt at about the age of 7 years and the upper lateral incisor at about the age of 8 years. As with the primary teeth, while some variation in the timing of tooth eruption is only to be expected, this eruption sequence should not vary. In particular, the upper central incisor should erupt before the upper lateral incisor. The upper incisors are usually spaced, and the lateral incisors in particular have a distal inclination. During this period of physiological spacing the canine is closely associated with the distal aspect of the lateral incisor (Fig. 1.20) and should be palpated clinically high in the buccal sulcus from the age of 10 years. The resultant spacing will usually close significantly as the canine erupts.

If the upper lateral incisor erupts before the upper central incisor, almost certainly there is something impeding the eruption of the central incisor, for example a supernumerary tooth or a dilaceration of the root of the central incisor (Fig. 1.21).

The lower canine and the first premolar teeth are the next to erupt at about 10 years of age, followed by the upper canine and the second premolar teeth at about the age of 11 and the second molar teeth at about the age of 12. Third molar teeth start to erupt from about the age of 16 onwards, but the eruption of third molars is very variable; not uncommonly, these teeth are impacted against their neighbours and fail to erupt at all.

The upper central incisors are more proclined than their primary counterparts. This allows some forward repositioning of the mandible when the first permanent molars erupt in a cusp-to-cusp relationship with their opponents. The distal surfaces of the second primary molars tend to be in the same vertical plane, so the first permanent molars erupting behind them tend to adopt a class II occlusal relationship. The forward repositioning of the mandible allows the establishment of a class I intercuspal position. The primary teeth in the buccal segments



#### (a)



(b)

Fig. 1.20 (a) The mixed dentition with spacing between the upper central and lateral incisors. The lateral incisors have a distal inclination. (b) The orthopantomogram (OPT) radiograph of the same patient showing the unerupted canine teeth closely associated with the apex of the lateral incisors.



**Fig. 1.21** Early mixed dentition where the upper left central incisor and both lateral incisors have erupted out of sequence with the upper right central incisor.

have a larger combined mesiodistal width than the permanent teeth which replace them. Thus, provided that the primary teeth are exfoliated in the ordinary way, there should be no problem with a lack of space for the permanent teeth in the buccal segments of the dental arch. The 'leeway space' as it is sometimes called—the amount by which the combined size of the primary canine and primary molar teeth exceeds the combined mesiodistal widths of the permanent canine and permanent premolar teeth—amounts to 1.5mm in the upper arch and 2.5mm in the lower with its large second primary molar (Fig. 1.22).

The size and shape of the anterior segments of the dento-alveolar arches do not change much following the eruption of the permanent incisor teeth. There is not much growth by deposition of bone on the labial aspect of the maxilla or mandible—these bones grow by forward



**Fig. 1.22** Early permanent dentition where a lower left second primary molar has recently been exfoliated and the lower left second premolar is erupting. Note the space mesial and distal to the erupting tooth which is due to the discrepancy in size between the primary molar and premolar.

and downward translation, with deposition of new bone on their posterior surfaces. If there is insufficient space to accommodate the teeth in the dental arches when the teeth erupt, it is unlikely that the situation will improve as growth proceeds (Leighton 1969). There is a small expansion in the width of the dental arch, with deposition of bone on the lateral aspect of the maxilla and the mandible, most of which occurs as the lateral incisors are erupting. While most of the 'leeway space' is taken up by mesial movement of the posterior teeth, the mesial molar movement may be restrained using orthodontic appliances to allow some improvement in the alignment of crowded incisors.

#### **1.6.3** The permanent dentition

As with the primary dentition, it is possible to identify occlusal features of the established permanent dentition that occur consistently in ideal dental arches (Sinclair and Little 1983).

- 1. The mesiobuccal cusp of the upper first permanent molar occludes in the mesiobuccal groove of the lower first permanent molar, with the distal aspect of the upper first molar contacting the mesial aspect of the lower second molar.
- 2. The teeth should have a normal labiolingual inclination. In the case of the anterior teeth, the crown inclination must be sufficient to prevent over-eruption of the teeth and an increased incisor overbite. In the case of the posterior teeth, the upper teeth should have a lingual inclination which remains constant in the premolar and molar regions, while the lower teeth have a lingual inclination which becomes more pronounced towards the back of the arch.
- **3.** The teeth should have a normal mesiodistal angulation, with the crowns of the teeth more mesially positioned than their roots.
- 4. There should be no rotated teeth.
- 5. There should be no spacing or crowding of the teeth.
- 6. The occlusal plane should be flat or have only a mild curve of Spee.

Not all these features are necessary for dental health, but taken together they provide a definition of the ideal class I occlusion.

#### 1.7 Summary

The aim of this chapter has been to outline the pattern of normal craniofacial growth.

- 1. While normal variation of this pattern will produce the differences that occur between individuals, the underlying patterns of growth should not be radically different from those described here, i.e. if development is to proceed in a normal fashion.
- 2. The growth of the brain and the cranium is almost complete by the age of 5 years.
- 3. The facial skeleton grows downwards and forwards relative to the cranial base, starting to grow rapidly at about the time of the pubertal

growth spurt and with facial growth virtually complete by the age of about 15.5 years in girls, and slightly later in boys.

- 4. With regard to occlusal development, while the exact age at which structures form will vary from person to person, the overall pattern of tooth development and the process by which the dental occlusion becomes established is largely as described in this chapter.
- 5. The eruption sequence—the order in which the teeth erupt—is more important than the age at which the teeth erupt. Therefore if there is a local problem with regard to the establishment of a normal occlusion, it is likely to become apparent in the first instance as a disturbance of the eruption sequence.



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# 2 Introduction to the dental surgery

#### A.S. Blinkhorn and B.L. Chadwick

#### 2.1 Introduction

It is a common belief among many individuals that being 'good with people' is something you are born with and cannot be taught. It is true that some individuals have a more open disposition and can relate well to others (Fig. 2.1). However, everyone can adopt approaches that put children and parents at ease.

It is particularly important for dentists to learn how to help people relax, as failure to empathize and communicate will result in disappointed patients and an unsuccessful practising career. Communicating effectively with children is of great value, as 'being good with younger patients' is a practice-builder and can reduce the stress involved when offering clinical care. All dental training should include a thorough understanding of how children relate to an adult world, how the dental visit should be structured, and what strategies are available to help children cope with their apprehension about dental procedures. This chapter will consider these items, beginning with a discussion on the theories of psychological development and following this up with sections on parents and their influence on dental treatment, dentist–patient relationships, anxious and uncooperative children, and helping anxious patients to cope with dental care.

#### 2.2 Psychology of child development

The psychological development of children was originally viewed as a series of well-defined phases, but is now seen as a continuum. The phases of development may well differ from child to child, so cannot be rigidly applied, but for clarity are described as a series of psychological developmental milestones from infancy to adulthood.

The most important theoretical perspective now influencing thinking about child development is John Bowlby's attachment theory. Bowlby suggested that child development could best be understood within the framework of patterns of interaction between the infant and the primary caregiver. If there were problems in this interaction, the child was likely to develop insecure and/or anxious patterns that would affect the ability to form stable relationships with others, to develop a sense of self-worth, and to move towards independence. The other important concept to note is that development is a lifelong process—we do not switch off at 18—nor is it an even process. Development is uneven, influenced by periods of rapid bodily change.

The psychological literature contains many accounts of the changes accompanying development. Therefore a general outline of the major 'psychological signposts' of which the dental team should be aware is presented in this section. As the newborn child is not a 'common' visitor



Fig. 2.1 Being good with patients is not necessarily an inborn art! With thanks to David Myers and kind permission of Eden Bianchi Press.

to the dental surgery, no specific description of newborn behaviour will be offered; instead, general accounts of motor, cognitive, perceptual, and social development from birth to adolescence will be included. It is important to understand that the thinking about child development has become less certain and simplistic in its approach; hence dentists who make hard and fast rules about the way they offer care to children will cause stress to both their patients and themselves.

#### 2.2.1 Motor development

A newborn child does not have an extensive range of movements, but these develop rapidly and by the age of 2 years the majority of children can walk on their own. The 'motor milestones' occur in a predictable order, and many of the tests used by paediatricians assess normal development in infancy in terms of motor skills. The predictability of early motor development suggests that it must be genetically programmed. Although this is true to some extent, there is evidence that the environment can influence motor development.

Motor development is completed in infancy; the changes which follow the walking milestone are refinements rather than the development of new skills. Eye-hand coordination gradually becomes more precise and elaborate with increasing experience. The dominance of one hand emerges at an early age and is usually linked to hemisphere dominance for language processing. The left hemisphere controls the right hand and the right hemisphere controls the left. However, whereas the majority of right-handed people appear to be strongly left-hemisphere dominant for language processing, only 20% of lefthanded people have right-hemisphere dominance for language processing. Some children with motor retardation may fail to show specific right or left manual dominance and will lack good coordination between the hands.

Children aged 6–7 years usually have sufficient coordination to brush their teeth reasonably well. Below that age many areas of the mouth will be missed and there is a tendency to swallow relatively large amounts of toothpaste; therefore preventive guidelines recommend parental involvement in brushing prior to age 7 years and supervision of brushing thereafter.

#### 2.2.2 Cognitive development

The cognitive capability of children changes radically from birth through to adulthood, and the process is divided into a number of stages for ease of description. The Swiss psychologist Piaget formulated the 'stages view' of cognitive development on the basis of detailed observations of his own children, and suggested that children pass through four broad stages of cognitive development (Table 2.1).

#### Table 2.1 Piaget's stages

Sensorimotor	This stage lasts until about 2 years of age. The prime achievement is 'object permanence'. The infant can think of things as permanent—which continue to exist when out of sight—and can think of objects without having to see them directly.
Preoperational thought	This runs from 2 to 7 years of age. The sensorimotor stage is further developed, allowing the child to predict outcomes of behaviour. Language development facilitates these changes. Thought patterns are not well developed, being egocentric, unable to encompass another person's point of view, single-tracked, and inflexible (sums up most politicians, some dental professors, and hospital administrators). Typically, children in this age band are unable to understand that areas and volumes remain the same despite changes in position or shape.
Concrete operations	This is the stage of thinking that occurs from about 7 to 11 years of age. Children are able to apply logical reasoning, consider another person's point of view, and assess more than one aspect of a particular situation (Fig. 2.2). Thinking is rooted in concrete objects; abstract thought is not well developed.
Formal operations	This is the last stage in the transition to adult thinking ability. It begins at about 11 years of age and results in the development of logical abstract thinking so that different possibilities for action can be considered.



Fig. 2.2 Children aged 7–11 years are able to consider another person's point of view. With thanks to David Myers and kind permission of Eden Bianchi Press.

#### Dogma bites the dust

These stages have been highlighted because of the importance of Piaget's early work on cognitive development. However, an over-reliance on 'dogma' may well limit the development of a subject, and this was the case with cognitive development. Few scientists challenged Piaget's findings and the field of infant perception became a rather sterile area for a number of years, but this changed with the work of Bowlby. Enormous developments in research since then have led to many doubts being raised about Piaget's original interpretation of his data. He underestimated the thinking abilities of younger children, and there is evidence to show that not all preschool thinking is totally egocentric. (See Key Point 2.1.)

#### Key Point 2.1

Babies and children are not unfinished adults. They are able to create, learn, and explore.

#### 2.2.3 Are adults sensible?

Of just as much interest is the modern view that not all adult thinking is logical; most of us are biased and illogical some of the time. How else, given the overwhelming evidence for the benefits of fluoride-containing toothpaste, can the use of non-fluoridated toothpaste be explained?

However, there is a serious point to this observation on adult illogicality. We must be prepared for parents who do not agree with our perceived wisdom (Fig. 2.3) or do not understand the basic tenets of specific programmes. Dentists will lead less stressful practising lives if they remember that not all their patients will always agree with or follow oral health advice.

So Piaget should be seen as a pioneer who really set in motion work on cognitive development, but it is now recognized that the developmental stages are not as clear-cut and many kids are smarter than we think!

#### 2.2.4 Perceptual development

Clearly, it is very difficult to discover what babies and infants are experiencing perceptually, so much research has concentrated on eye movements. These studies have shown that, with increasing age, scanning becomes broader and larger amounts of information are sought. Compared with adults, 6-year-old children cover less of the object, fixate on details, and gain less information. However, children do develop their selective attention, and by the age of 7 years can determine which messages merit attention and which can be ignored. Concentration skills also improve. Children must be involved in their treatment and some dental advice can be offered to children of this age but, given the importance of the home environment, parents should be the main focus of any information given on oral healthcare.

With increasing age children become more efficient at discriminating between different visual patterns and reach adult proficiency by about 9 years of age.

The majority of perceptual development is a function of the growth of knowledge about the environment in which a child lives, hence the



Fig. 2.3 Be prepared for parents who don't agree. With thanks to David Myers and kind permission of Eden Bianchi Press.



**Fig. 2.4** Spend time explaining the facts about dental care. With thanks to David Myers and kind permission of Eden Bianchi Press.

necessity to spend time explaining aspects of dental care to new child patients (Fig. 2.4).

#### 2.2.5 Language development

A lack of appropriate stimulation will retard a child's learning, particularly language. A child of 5 who can only speak in monosyllables and has no sensible sentence structure will not only be unable to communicate with others but will also be unable to think about the things he/she sees and hears. Stimulation is important, as language development is such a rapid process in childhood that any delay can seriously handicap a child. Newborn children show a remarkable ability to distinguish speech sounds, and by the age of 5 years most children can use 2000 or more words. Language and thought are tied together and are important in cognitive development, but the complexities of the relationship between the two are not well understood.

Dentistry has a highly specialized vocabulary and it is unlikely that many children, or even adolescents and parents, will understand our meaning if we rely on jargon. The key to successful communication is to pitch your advice and instructions at just the right level for different age groups of children. Clearly while a small child may happily accept that you are going to 'tickle the brown spot on their tooth', a streetwise 10-year-old who is a computer games afficionado will be less than impressed. There is no universal approach to patients, so careful treatment planning and assessment are required before children or their parents are given specific written or verbal advice. Communication is more than the words we use, and for children with limited vocabularies the tone and body language are as important as the words. (See Key Point 2.2.)

#### Key Point 2.2

Members of the dental team must assess children's linguistic ability before offering advice. Voice tone and body language matter. Tailor advice to individual families.

#### 2.2.6 Social development

Until fairly recently it was believed that newborn infants were individuals who spent most of their time sleeping. However, recent research reveals that babies interact quite markedly with their environment, often initiating interactions with other humans by movement of their eyes or limbs.

#### Separation anxiety

Babies tend to form specific attachments to people and are prone to separation anxiety. At about 8 months infants show a definite fear of strangers. This potential for anxiety separation remains high until about 5 years of age, when separation anxiety declines quite markedly. This is consistent with studies of children in hospital, which show that after the age of 5 there is less distress on entering hospital. Separation anxiety should also be considered by dentists who insist that all young children must enter the dental surgery alone. Clearly, this will cause severe anxiety to patients under 5 years of age. (See Key Point 2.3.)

#### Key Point 2.3

Parents in the dental surgery cannot be governed by a rule. Children under 5 benefit from parental presence. Each child and parent has different needs. A caring health professional should adopt some flexibility in his/her approach to offering clinical care with or without parents present.

It has been reported that a loving early parental attachment is associated with better social adjustment in later childhood and is a good basis for engendering trust and friendship with peers. This is important, as a successful transition from home to school depends on the ability to interact with other individuals apart from parents. The home environment will play a major part in social development, but the effects of community expectations should not be underestimated. We are all products of our broad social environment, mediated to some extent by parental influences.

#### 2.2.7 Adolescence

The waning of parental influence can be seen in the final stage of child development—adolescence. This is the end of childhood and the beginning of adulthood. It is conceptualized as a period of emotional turmoil and a time of identity formation. This view is a Western creation and is culturally biased. In many societies 'terrible teenagers' do not exist; childhood ends and adult responsibilities are offered at a relatively early age.

It is interesting to note that even in Western industrialized societies there is little real evidence to support the idea that the majority of adolescents are rebellious and nonconformist. The main change is the evolution of a different sort of parental relationship. There is increasing independence and selfsufficiency. The research does show that young people tend to be moody, are oversensitive to criticism, and feel miserable for no apparent reason, but on the whole they do not rebel against their parental role models.

There are some clear messages to dentists who wish to retain their adolescent patients. Treat them as independent from their parents and recognize them as individuals in their own right. Don't criticize them excessively as this may compromise their future oral health. These patients are looking for support and reassurance. Remember that their personal behaviour patterns are usually not related to health issues. Until there are acute problems 'health per se' is of little relevance to adolescents, being a rather abstract concept. Their major issues of

concern are finding employment, exploring their sexuality, and having the friendship and support of their peers.

#### 2.3 Parents and their influence on dental treatment

Children learn the basic aspects of everyday life from their parents. This process is termed socialization, and is ongoing and gradual. By the age of 4 years children know many of the conventions current in their culture, such as male and female roles. The process of transmitting cultural information early in life is called primary socialization. In industrialized countries, obtaining information on many aspects of life is gained formally in schools and colleges rather than from the family. This is termed secondary socialization.

#### 2.3.1 Socialization

Interestingly, primary socialization can have a profound and lasting effect. For example, fear of dental treatment and when we first begin to clean our teeth can often be traced back to family influence, so parents can shape a child's expectations and attitudes about oral health. Thus every attempt should be made to involve them when attempting to offer dental care or change a child's health habits. (See Key Point 2.4.)

#### Key Point 2.4

Maintaining a healthy mouth begins in early childhood as the teeth erupt. It is far simpler to encourage healthy habits than to change ingrained behaviour.

#### 2.3.2 Avoid victim blaming

Involving parents means that the dentist must look to positive reinforcement rather than 'victim blaming'. Parents accused of oral neglect may well feel aggrieved or threatened. Often children's oral health is compromised by a lack of parental knowledge, so programmes should be designed to reduce parental guilt and guide families to improved



**Fig. 2.5** Positive reinforcement is important. With thanks to David Myers and kind permission of Eden Bianchi Press.

preventive behaviours. Guilt may result in parents spending more time seeking excuses for problems rather than trying to implement solutions.

Parents who believe their child has an oral health problem which can be solved react more positively both to their dental advisor and to the preventive programme itself. It is especially helpful if the preventive strategy can include a system of positive reinforcement for the child (Fig. 2.5). Features that encourage desired behaviour such as brushing charts, diet sheets, gold stars for brushing well, and extra pocket money for curtailing thumb-sucking are all useful tips to help parents maintain a child's enthusiasm for a particular dental project.

It should be emphasized that preventive programmes must be planned to include only one major goal at a time. Parents cannot cope if too much is expected of them at any one time. Programmes that involve families have higher success rates than those which concentrate solely on the patient. Interestingly, families also have a profound influence on levels of dental anxiety among their children. Dentally anxious mothers have children who exhibit negative behaviour at the dentist. Hence dentists need to look 'beyond' the child when assessing the reasons for dental anxiety.

## **2.3.3** Should parents join children in the surgery?

One of the great debates in paediatric dentistry centres on whether parents should be allowed in the dental surgery while their child is receiving treatment. A child's family, it could be argued, can offer emotional support during treatment. There is no doubt that within the medical field there is great support for the concept of a parent actually 'living in' while a child is hospitalized. However, the issue is not so clear cut in dentistry (Fig. 2.6).



**Fig. 2.6** Should we allow parents into the surgery? With thanks to David Myers and kind permission of Eden Bianchi Press.



Fig. 2.7 Some parents can be very irritating by repeating all your requests. With thanks to David Myers and kind permission of Eden Bianchi Press.

The first issue that must be raised is whether dentists have the ethical/moral right to bar parents from sitting in with their children when dental care is being undertaken. Clearly, parents have views and anxiety levels may be raised if parents feel that their familial rights are being threatened, and a child may be stressed by tension between his/her parents and the operator.

In their comprehensive book on child management, Wright et al. (1987) summarize the advantages of keeping parents out of the surgery as follows.

- 1. The parent often repeats orders, annoying both the dentist and the child patient (Fig. 2.7).
- 2. The parent intercepts orders, becoming a barrier to the development of rapport between the dentist and the child.
- The dentist is unable to use voice intonation in the presence of the parent because he/she is offended.
- 4. The child divides his/her attention between the parent and the dentist.
- The dentist divides his/her attention between the parent and the child.
- Dentists are probably more relaxed and comfortable when parents remain in the reception area.

These suggestions have merit but feel authoritarian, stressing the ordering and voice intonation rather than sympathetic communication.

#### 2.4 Dentist–patient relationship

The way a dentist interacts with patients will have a major influence on the success of any clinical or preventive care. Clearly, only broad guidelines can be presented on how to maintain an effective relationship with a patient, as all of us are unique individuals with different needs and aspirations. This is especially the case in paediatric With the exception of the importance of parental separation there is little research to inform whether parents should be in or out of the surgery, so it is usually a personal decision taken by the dentist in the light of parental concerns and clinical experience. But parental involvement is key to other aspects of care, and the negative impacts can be removed by working with parents and outlining how they can successfully support their child with a supportive but silent presence. As in any branch of medicine there can be no hard and fast rules for dealing with the general public; an adherence to any type of dogma, come what may, is a recipe for confrontation and stress. Allowing parents to sit in with children should be a decision taken for each individual rather than implementing a 'keep parents out' policy.

#### 2.3.4 Each patient is a unique individual

Patients with special needs require a high degree of parental involvement in oral healthcare, particularly those children with educational, behavioural, and physical difficulties. For example, toothbrushing is a complex cognitive and motor task which will tax the skills of many disabled children. A parent will have to be taught how to monitor the efficiency of the plaque removal and intervene when necessary to ensure that the mouth is cleaned adequately. Diet is also important, so clear advice must be offered and reinforcement planned at regular intervals.

dentistry where a clinician may have to treat a frightened 3-year-old child at one appointment followed by a recalcitrant 15-year-old at the next. However, there are common research findings which highlight the key issues that will cause a dentist–patient consultation to founder or progress satisfactorily.

#### 2.4.1 People like friendly dentists

The first question that must be considered is 'Why me—what factors did the parents take into account before making an appointment at my practice?'

The obvious answers are that your practice is closest to the bus stop, has good parking, and is the only one open after 6.00p.m. Surprisingly, the choice is not so simple. Most people try to find out details about different dental practices from friends and colleagues. While the technical skill of the dentist is of some concern, the most important features people look for are that he/she has a gentle friendly manner, explains treatment procedures, and tries to keep any pain to a minimum. (See Key Point 2.5.)

#### Key Point 2.5

Patients seek out friendly, kind dentists. Taking time to talk and interact with individuals can build a practice.

As with any health issue the social class background of the respondents influences attitudes and beliefs. For example, parents of high socioeconomic status are more interested in professional competence and gaining information, whereas parents from poorer areas want a dentist to reassure and be friendly to their child.

So which dentist parents choose to offer care to their child will depend to some extent on reports about technical skill from family and friends, but the major driving force is well-developed interpersonal skills. A major point to emphasize is that technical skill is usually judged in terms of caring and sympathy, a finding which adds further weight to the importance of dentists developing a good 'chair-side manner'. The ability to communicate empathy with tone of voice and body language may enhance or undermine the words used. A calm warm tone coupled with open body language and when appropriate a smile is more reassuring to an anxious parent than an impersonal businesslike approach delivering the same message.

Explanation, 'taking the time to talk us through what our child's treatment will entail', is another factor which rates highly, and may actually influence the rate of attendance for follow-up appointments.

#### 2.4.2 Structure of the dental consultation

To help students and new graduates improve their dentist-patient interaction skills it is possible to give an outline structure for a successful dental consultation. The proposed model consists of six stages, and is based on the work of Wanless and Holloway (1994). (See Key Point 2.6.)

#### Key Point 2.6

Most consultation visits should have a set plan. Focus on communication before intervention.

- 1. *Greeting.* The dentist greets the child by name. Avoid using generalized terms such 'Hello, sunshine', which are general rather than specific to the patient (Fig. 2.8). If parents are present include them in the conversation, but the child should be central to the developing relationship. A greeting can be spoilt by proceeding too quickly to an instruction rather than an invitation. For example, 'Hello, Sarah, jump in the chair' is rather abrupt and may prejudice an interactive relationship. The greeting should be used to put the child and parents at ease before proceeding to the next stage.
- 2. Preliminary chat. This phase has three objectives: to assess whether the patient or parents have any particular worries or concerns, to settle the patient into the clinical environment, and to assess the patient's emotional state. The following sequence represents one way of maximizing the effect of the 'preliminary chat'.



Fig. 2.8 Always greet your patient by name. With thanks to David Myers and kind permission of Eden Bianchi Press.