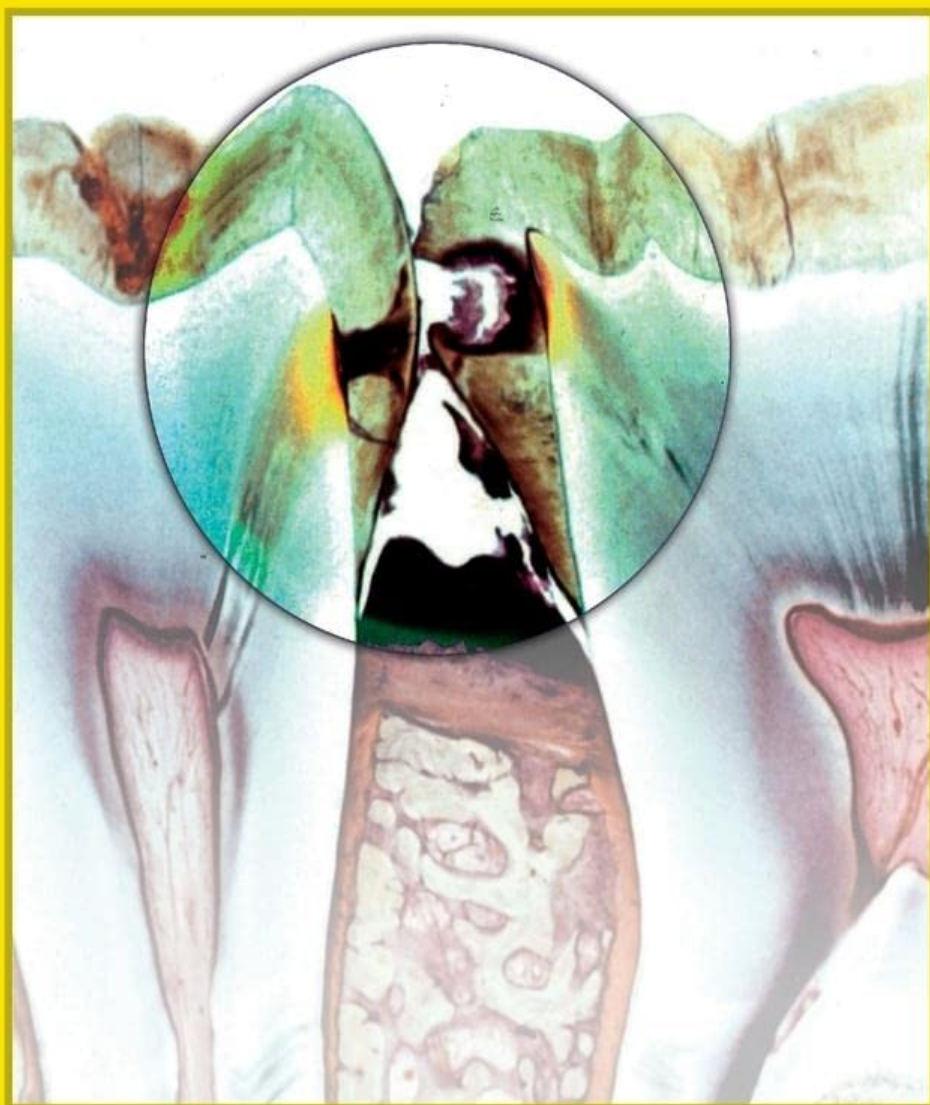


Dental Caries

The Disease and Its Clinical Management

Third Edition



Edited by
Ole Fejerskov, Bente Nyvad and Edwina Kidd

WILEY Blackwell

Dental Caries

Dental Caries

The Disease and Its Clinical Management

Third Edition

Edited by

**Ole Fejerskov, Bente Nyvad,
and Edwina Kidd**

WILEY Blackwell

This edition first published 2015
© 2015 by John Wiley & Sons, Ltd
© 2003, 2008 by Blackwell Munksgaard Ltd

Registered Office

John Wiley & Sons, Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial Offices

9600 Garsington Road, Oxford, OX4 2DQ, UK
The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK
1606 Golden Aspen Drive, Suites 103 and 104, Ames, Iowa 50010, USA

For details of our global editorial offices, for customer services and for information about how to apply for permission to reuse the copyright material in this book please see our website at www.wiley.com/wiley-blackwell

The right of the author to be identified as the author of this work has been asserted in accordance with the UK Copyright, Designs and Patents Act 1988.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by the UK Copyright, Designs and Patents Act 1988, without the prior permission of the publisher.

Designations used by companies to distinguish their products are often claimed as trademarks. All brand names and product names used in this book are trade names, service marks, trademarks or registered trademarks of their respective owners. The publisher is not associated with any product or vendor mentioned in this book. It is sold on the understanding that the publisher is not engaged in rendering professional services. If professional advice or other expert assistance is required, the services of a competent professional should be sought.

The contents of this work are intended to further general scientific research, understanding, and discussion only and are not intended and should not be relied upon as recommending or promoting a specific method, diagnosis, or treatment by health science practitioners for any particular patient. The publisher and the author make no representations or warranties with respect to the accuracy or completeness of the contents of this work and specifically disclaim all warranties, including without limitation any implied warranties of fitness for a particular purpose. In view of ongoing research, equipment modifications, changes in governmental regulations, and the constant flow of information relating to the use of medicines, equipment, and devices, the reader is urged to review and evaluate the information provided in the package insert or instructions for each medicine, equipment, or device for, among other things, any changes in the instructions or indication of usage and for added warnings and precautions. Readers should consult with a specialist where appropriate. The fact that an organization or Website is referred to in this work as a citation and/or a potential source of further information does not mean that the author or the publisher endorses the information the organization or Website may provide or recommendations it may make. Further, readers should be aware that Internet Websites listed in this work may have changed or disappeared between when this work was written and when it is read. No warranty may be created or extended by any promotional statements for this work. Neither the publisher nor the author shall be liable for any damages arising herefrom.

Library of Congress Cataloging-in-Publication Data

Dental caries (Fejerskov)

Dental caries : the disease and its clinical management / edited by Ole Fejerskov, Bente Nyvad, Edwina Kidd. – Third edition.
p. ; cm.

Includes bibliographical references and index.

ISBN 978-1-118-93582-8 (cloth)

I. Fejerskov, Ole, editor. II. Nyvad, Bente, editor. III. Kidd, Edwina A. M., editor. IV. Title.

[DNLM: 1. Dental Caries—diagnosis. 2. Dental Caries—therapy. 3. Dental Restoration, Permanent. WU 270]

RK331

617.6'7—dc23

2014046642

A catalogue record for this book is available from the British Library.

Wiley also publishes its books in a variety of electronic formats. Some content that appears in print may not be available in electronic books.

Set in 10/12 pt Minion Pro by SPi Publisher Services, Pondicherry, India
Printed and bound in Singapore by Markono Print Media Pte Ltd

1 2015

Correct citation of this text:

Fejerskov, O., Nyvad, B. & Kidd, E. (eds) (2015) *Dental Caries: The Disease and Its Clinical Management*, 3rd Edn. Oxford, Wiley Blackwell.

Contents

Contributors

xi

Part I	Dental caries: what is it and how widespread is it globally?	
Chapter 1	Prologue	3
	O. Fejerskov, B. Nyvad, and E.A.M. Kidd	
	<i>Introduction</i>	3
	<i>The role of cariology in restorative dentistry</i>	3
	<i>The content of this textbook</i>	4
Chapter 2	Dental caries: what is it?	7
	O. Fejerskov, B. Nyvad, and E.A.M. Kidd	
	<i>The disease</i>	7
	<i>Terminology</i>	9
	<i>Background literature</i>	9
	<i>References</i>	10
Chapter 3	Clinical features of caries lesions	11
	O. Fejerskov and B. Nyvad	
	<i>What do caries lesions look like clinically?</i>	11
	<i>The deciduous dentition</i>	12
	<i>The permanent dentition</i>	13
Chapter 4	How big is the problem? Epidemiological features of dental caries	21
	V. Baelum and O. Fejerskov	
	<i>Introduction</i>	21
	<i>What? Defining the health issue at hand</i>	21
	<i>Who? The distribution of caries in populations</i>	28
	<i>Where? The geography of caries</i>	35
	<i>When? Trends in caries</i>	37
	<i>Why? The causes of caries</i>	37
	<i>References</i>	41
Part II	The caries lesion and its biological determinants	
Chapter 5	Pathology of dental caries	49
	O. Fejerskov	
	<i>Introduction</i>	49
	<i>Human dental enamel at time of eruption</i>	51
	<i>Enamel changes during early caries lesion development</i>	55
	<i>The approximal white spot lesion</i>	58
	<i>Progression of the enamel lesion</i>	63
	<i>Arrest of the caries lesion</i>	63
	<i>Occlusal caries</i>	66
	<i>Dentin reactions to caries progression</i>	71
	<i>Pulpo-dentinal reactions</i>	71
	<i>Root-surface caries</i>	77
	<i>Background literature</i>	80
	<i>References</i>	80

Chapter 6	Saliva and caries development	83
	A. Bardow and A. Vissink	
	<i>Introduction</i>	83
	<i>Saliva and salivary glands</i>	84
	<i>Saliva and caries development: biological aspects</i>	87
	<i>Saliva and caries development: clinical aspects</i>	98
	<i>Evaluation of salivary gland function</i>	101
	<i>Management of salivary gland hypofunction</i>	104
	<i>Concluding remarks</i>	105
	<i>Background literature</i>	105
	<i>References</i>	105
Chapter 7	Biofilms in caries development	107
	P.D. Marsh, N. Takahashi, and B. Nyvad	
	<i>Introduction</i>	107
	<i>The resident microflora</i>	108
	<i>Dental biofilms: development, structure, composition, and properties</i>	111
	<i>Caries microbiology: a brief historical perspective</i>	121
	<i>Methodological problems in microbiological studies of dental caries</i>	121
	<i>Microbiology of caries</i>	122
	<i>Cariogenic features of dental biofilm bacteria</i>	125
	<i>The 'ecological plaque hypothesis' to explain the role of dental biofilm bacteria in the etiology of dental caries</i>	125
	<i>Concluding remarks</i>	129
	<i>Background literature</i>	129
	<i>References</i>	129
Chapter 8	Diet and dental caries	133
	C. van Loveren and P. Lingström	
	<i>History</i>	133
	<i>Early ecological studies</i>	134
	<i>Experimental human studies</i>	134
	<i>Influence of fluoride on the diet-caries relationship</i>	135
	<i>Measuring cariogenicity</i>	136
	<i>Sweeteners</i>	141
	<i>Protective factors in foods</i>	149
	<i>Diet and dental erosion</i>	150
	<i>Conclusion</i>	151
	<i>References</i>	151
Chapter 9	Demineralization and remineralization: the key to understanding clinical manifestations of dental caries	155
	O. Fejerskov and M.J. Larsen	
	<i>Introduction</i>	155
	<i>Enamel mineral</i>	156
	<i>Stability of calcium phosphates</i>	156
	<i>Crystal dissolution</i>	157
	<i>Why is apatite solubility increased by acid?</i>	158
	<i>Effect of carbonate and fluoride on apatite dissolution and growth</i>	159
	<i>Demineralization and remineralization of the dental hard tissues</i>	160
	<i>Caries demineralization</i>	162
	<i>Remineralization of enamel</i>	164
	<i>Remineralization of dentin</i>	166
	<i>Background literature</i>	169
	<i>References</i>	170

Part III	Diagnosis	
Chapter 10	The foundations of good diagnostic practice	173
	V. Baelum, B. Nyvad, H.-G. Gröndahl, and O. Fejerskov	
	<i>Introduction</i>	173
	<i>The making of a dentist</i>	174
	<i>The dental examination; in the best interest of our patients</i>	175
	<i>What are we looking for? What is caries?</i>	176
	<i>The wealth of caries diagnostic methods and criteria</i>	178
	<i>The evolution in caries diagnostic methods</i>	178
	<i>Diagnostic test assessment in the essentialistic gold-standard paradigm</i>	179
	<i>Evaluating caries diagnostic methods</i>	180
	<i>Leaps in the essentialistic gold-standard reasoning</i>	181
	<i>Diagnostic test evaluation in the nominalistic caries paradigm</i>	183
	<i>Inter- and intra-examiner errors in caries diagnosis</i>	184
	<i>How do we deal with the unavoidable diagnostic uncertainty?</i>	185
	<i>The additional diagnostic yield argument</i>	186
	<i>Concluding remarks</i>	187
	<i>References</i>	188
Chapter 11	Visual-tactile caries diagnosis	191
	B. Nyvad, V. Machiulskiene, V.M. Soviero, and V. Baelum	
	<i>Introduction</i>	191
	<i>The diagnostic process</i>	192
	<i>Why do we diagnose caries?</i>	193
	<i>Diagnosis from a dental caries perspective</i>	193
	<i>How early should caries lesions be detected?</i>	194
	<i>What are the best visual-tactile caries diagnostic criteria?</i>	195
	<i>Commonly used visual-tactile criteria</i>	197
	<i>Differential diagnosis</i>	201
	<i>Visual-tactile caries examination; a systematic clinical approach</i>	205
	<i>Additional aids in visual-tactile caries diagnosis</i>	207
	<i>Benefits and limitations of visual-tactile caries diagnosis</i>	208
	<i>References</i>	209
Chapter 12	Additional caries detection methods	211
	H. Hintze, A. Lussi, F. Cuisinier, and B. Nyvad	
	<i>Introduction</i>	211
	<i>Radiography</i>	211
	<i>Methods based on light and electrical current</i>	223
	<i>Are the additional methods suitable for use in clinical practice?</i>	229
	<i>Can the methods serve as adjuncts to a visual-tactile caries examination?</i>	229
	<i>References</i>	229
Part IV	Controlling dental caries	
Chapter 13	The caries control concept	235
	B. Nyvad and O. Fejerskov	
	<i>Why the caries control concept should replace caries prevention</i>	235
	<i>How caries control was managed in the past</i>	236
	<i>Arrest of active enamel caries</i>	237
	<i>Arrest of active root caries</i>	238

	<i>Arrest of active cavitated caries</i>	239
	<i>Role of fluoride in lesion arrest</i>	242
	<i>Benefits and limitations of the caries control approach – and some recommendations</i>	242
	<i>References</i>	242
Chapter 14	Fluorides in caries control	245
	O. Fejerskov, J.A. Cury, L.M. Tenuta, and V.C. Marinho	
	<i>Introduction</i>	245
	<i>Fluoride in caries prevention and control</i>	246
	<i>Cariostatic mechanisms of fluoride</i>	250
	<i>Dental fluorosis and metabolism of fluoride</i>	253
	<i>The effectiveness of fluorides in the control of dental caries: evidence from systematic reviews</i>	263
	<i>Rational use of fluorides in caries control in different parts of the world: recommendations</i>	271
	<i>Background literature</i>	272
	<i>References</i>	272
Chapter 15	The role of oral hygiene	277
	B. Nyvad	
	<i>Introduction</i>	277
	<i>Some theoretical considerations</i>	277
	<i>The biological effect of tooth cleaning</i>	278
	<i>The clinical effect of tooth cleaning</i>	278
	<i>The effect of professional tooth cleaning</i>	282
	<i>The effect of dental flossing</i>	283
	<i>Concluding remarks</i>	283
	<i>References</i>	284
Chapter 16	Are antibacterials necessary in caries prophylaxis?	287
	A.A. Scheie, H.V. Rukke, and E.C. Petersen	
	<i>The biofilm lifestyle and the rationale for antibacterial intervention</i>	287
	<i>Biological activity and mode of action</i>	288
	<i>Vehicles for caries prophylactic agents</i>	292
	<i>Specific agents</i>	292
	<i>Other agents proposed for caries prophylaxis, but without documented anticaries effects</i>	296
	<i>Risk of antibacterial resistance development?</i>	298
	<i>Concluding remarks and future approaches</i>	299
	<i>Background literature</i>	299
	<i>References</i>	299
Chapter 17	The principles of caries control for the individual patient	303
	B. Nyvad and E.A.M. Kidd	
	<i>Introduction</i>	303
	<i>How are current caries activity and risk of future caries progression assessed?</i>	304
	<i>How is the information used to categorize patients into risk groups?</i>	307
	<i>What nonoperative treatments are available?</i>	308
	<i>How is the individual helped to control disease progression?</i>	312
	<i>When should the patient be recalled?</i>	312
	<i>Caries control in children and adolescents</i>	314
	<i>Patients with a dry mouth</i>	317
	<i>Failure</i>	318
	<i>References</i>	319

Chapter 18	Caries control for frail elders	321
	M.I. MacEntee, S.R. Bryant, H. Keller, C.T. Nguyen, and C.S. Yao	
	<i>Introduction</i>	321
	<i>A conceptual model of oral health</i>	321
	<i>Frailty</i>	322
	<i>Physical characteristics of caries in elderly mouths</i>	323
	<i>Incidence of caries in frail adults</i>	323
	<i>Recognizing the risk of caries</i>	324
	<i>Impact of caries in frailty</i>	326
	<i>Management of caries in frailty</i>	327
	<i>Summary</i>	329
	<i>References</i>	330
Part V	Operative intervention	
Chapter 19	Classical restorative or the minimally invasive concept?	335
	E.A.M. Kidd, J. Frencken, B. Nyvad, C.H. Splieth, and N.J.M. Opdam	
	<i>Operative dentistry and caries control</i>	335
	<i>Sealants</i>	339
	<i>Atraumatic restorative treatment</i>	345
	<i>Conventional minimal intervention methods</i>	353
	<i>Minimal intervention and the deciduous dentition</i>	365
	<i>References</i>	370
Chapter 20	Caries 'removal' and the pulpo-dentinal complex	375
	E.A.M. Kidd, L. Bjørndal, and O. Fejerskov	
	<i>Introduction</i>	375
	<i>The pulpo-dentinal complex and caries</i>	376
	<i>Pulpitis and its clinical diagnosis</i>	376
	<i>Why are pulpo-dentinal reactions important to the choice of operative management?</i>	376
	<i>The infected dentin concept and its clinical consequence</i>	377
	<i>Studies placing fissure sealants over carious dentin</i>	379
	<i>Stepwise excavation studies</i>	380
	<i>Randomized controlled clinical trials on stepwise excavation outcome</i>	382
	<i>Do we need to reenter?</i>	382
	<i>What happens if we do not remove caries at all but seal it in the tooth permanently?</i>	384
	<i>Further consideration of deciduous teeth</i>	384
	<i>Conclusion on caries removal and the pulpo-dentinal complex</i>	385
	<i>References</i>	385
Chapter 21	Longevity of restorations: 'the death spiral'	387
	V. Qvist	
	<i>Introduction</i>	387
	<i>Clinical assessment of restorations</i>	388
	<i>Assessment of restoration longevity</i>	388
	<i>The amalgam debate and its consequences for restoration longevity</i>	391
	<i>Longevity of restorations in the primary dentition</i>	391
	<i>Longevity of restorations in the permanent dentition</i>	394
	<i>Longevity of fissure sealants</i>	395
	<i>Longevity of atraumatic restorative treatment restorations</i>	396
	<i>Factors influencing restoration longevity</i>	397
	<i>Consequences of restoration longevity for dental health and cost</i>	398
	<i>Concluding remarks</i>	399
	<i>References</i>	400

Part VI	From chair-side to population caries control	
Chapter 22	Caries prevention and control in low- and middle-income countries	405
	W. van Palenstein Helderman, C. Holmgren, B. Monse, and H. Benzian	
	<i>Introduction</i>	405
	<i>Caries: a public health problem in low- and middle-income countries</i>	406
	<i>Health and oral health systems in low- and middle-income countries</i>	408
	<i>Public health approaches to address caries in low- and middle-income countries</i>	411
	<i>Conclusions and recommendations</i>	417
	<i>References</i>	420
Chapter 23	How accurately can we assess the risk for developing caries lesions?	423
	H. Hausen and V. Baelum	
	<i>Introduction</i>	423
	<i>The risk of developing caries lesions cannot be observed directly for an individual patient</i>	424
	<i>The course of a typical study for evaluating the accuracy of a prediction</i>	425
	<i>A real-life example of using a single, dichotomous predictor</i>	427
	<i>Interpretation and use of the measures of prediction accuracy</i>	427
	<i>What level of accuracy would be sufficient in everyday practice?</i>	432
	<i>What level of accuracy can be achieved?</i>	433
	<i>Clinical caries risk assessment: is it possible?</i>	435
	<i>How valuable are the proposed measures?</i>	436
	<i>Concluding remarks</i>	436
	<i>Background literature</i>	436
	<i>References</i>	436
Chapter 24	Caries control in low-caries populations	439
	H. Hausen, M. Jøssing, and O. Fejerskov	
	<i>Introduction</i>	439
	<i>A low caries frequency entails the polarization of the caries problem</i>	439
	<i>Are effective and feasible measures available for protecting the high-risk individuals from dental decay?</i>	440
	<i>Noninvasive treatment of early caries lesions among teenagers exposed to community-wide oral health promotion</i>	441
	<i>A model for controlling caries in low-caries child populations</i>	442
	<i>A demonstration case in 0–18-year-old Danes</i>	443
	<i>Concluding remarks</i>	446
	<i>References</i>	447
Chapter 25	Epilogue. Controlling the global burden of dental caries: the evidence calls for a reorganization of the oral health-care system	449
	O. Fejerskov, V. Baelum, B. Nyvad, and E.A.M. Kidd	
<i>Index</i>		453

Contributors



Professor Vibeke Baalum
School of Dentistry
Health
Aarhus University
Aarhus, Denmark



Professor Frédéric Cuisinier
Université Montpellier I
Montpellier, France



Associate Professor Allan Bardow
School of Dentistry
Faculty of Health Sciences
University of Copenhagen
Copenhagen, Denmark



Professor Jaime A. Cury
Piracicaba Dental School
University of Campinas – UNICAMP
Piracicaba, SP, Brazil



Dr Habib Benzian
University College London and
The Health Bureau Ltd
Milton Keynes, UK



Professor Ole Fejerskov
Department of Biomedicine
Health
Aarhus University
Aarhus, Denmark



Associate Professor Lars Bjørndal
School of Dentistry
Faculty of Health Sciences
University of Copenhagen
Copenhagen, Denmark



Professor Jo Frecken
College of Dental Sciences
Radboud University Medical Centre
Nijmegen, The Netherlands



Professor S. Ross Bryant
Faculty of Dentistry
University of British Columbia
Vancouver
British Columbia, Canada



Professor Hans-Göran Gröndahl
Institute of Odontology
Sahlgrenska Academy
University of Göteborg
Göteborg, Sweden



Professor Hannu Hausen
Institute of Dentistry
University of Oulu
Oulu, Finland



Professor Mogens Joost Larsen
Emeritus, School of Dentistry
Health
Aarhus University
Aarhus, Denmark



Associate Professor Hanne Hintze
School of Dentistry
Health
Aarhus University
Aarhus, Denmark



Professor Peter Långström
Institute of Odontology
Sahlgrenska Academy
Göteborg University
Göteborg, Sweden



Dr Christopher Holmgren
Aide Odontologique Internationale
Merigny, France



Professor Adrian Lussi
School of Dentistry
University of Bern
Bern, Switzerland



Dr Marit Jøssing
Chief Dental Officer
Odder Municipal Dental Service
Denmark



Professor Michael I. MacEntee
Faculty of Dentistry
University of British Columbia
Vancouver
British Columbia, Canada



Dr Heather Keller
Department of Nutrition and Aging
University of Waterloo
Waterloo
Ontario, Canada



Professor Vita Machiulskiene
Faculty of Odontology
Lithuanian University of Health Sciences
Kaunas, Lithuania



Professor Edwina A.M. Kidd
Emerita, Dental School
King's College
London, UK



Professor Valeria C. Marinho
Institute of Dentistry
Barts and The London School of Medicine
and Dentistry
Queen Mary University of London (QML)
London, UK



Professor Philip D. Marsh
Microbiology Services Division
Public Health England
Salisbury, and
School of Dentistry
University of Leeds
Leeds, UK



Associate Professor Vibeke Qvist
School of Dentistry
Faculty of Health and Medical Sciences
University of Copenhagen
Copenhagen, Denmark



Dr Bella Monse
Fit for School Regional Programme
German Development Corporation (GIZ)
Manila, Philippines



Scientist PhD Håkon Valen Rukke
Nordic Institute of Dental Materials
Oslo, Norway



Dr Caroline T. Nguyen
Faculty of Dentistry
University of British Columbia
Vancouver
British Columbia, Canada



Professor Anne Aamdal Scheie
Faculty of Dentistry
University of Oslo
Oslo, Norway



Professor Bente Nyvad
School of Dentistry
Health
Aarhus University
Aarhus, Denmark



Professor Vera Mendes Soviero
Faculty of Dentistry
University of the State of Rio de Janeiro
Rio de Janeiro, Brazil



Dr Niek J.M. Opdam
University of Nijmegen Medical Centre
Nijmegen, The Netherlands



Professor Christian H. Splith
School of Dentistry
University of Greifswald
Greifswald, Germany



Professor Fernanda Critina Petersen
Faculty of Dentistry
University of Oslo
Oslo, Norway



Professor Nobuhiro Takahashi
Division of Oral Ecology and Biochemistry
Tohoku University Graduate School of
Dentistry
Sendai, Japan



Associate Professor Livia M.A. Tenuta
Piracicaba Dental School
University of Campinas - UNICAMP
Piracicaba, SP, Brazil



Professor Arjan Vissink
University Medical Center Groningen
Groningen, The Netherlands



Professor Cor van Loveren
Academic Centre for Dentistry Amsterdam
(ACTA)
University of Amsterdam and VU University
Amsterdam
Amsterdam, The Netherlands



De Chao Shu Yao
Q & M Dental Group
Singapore

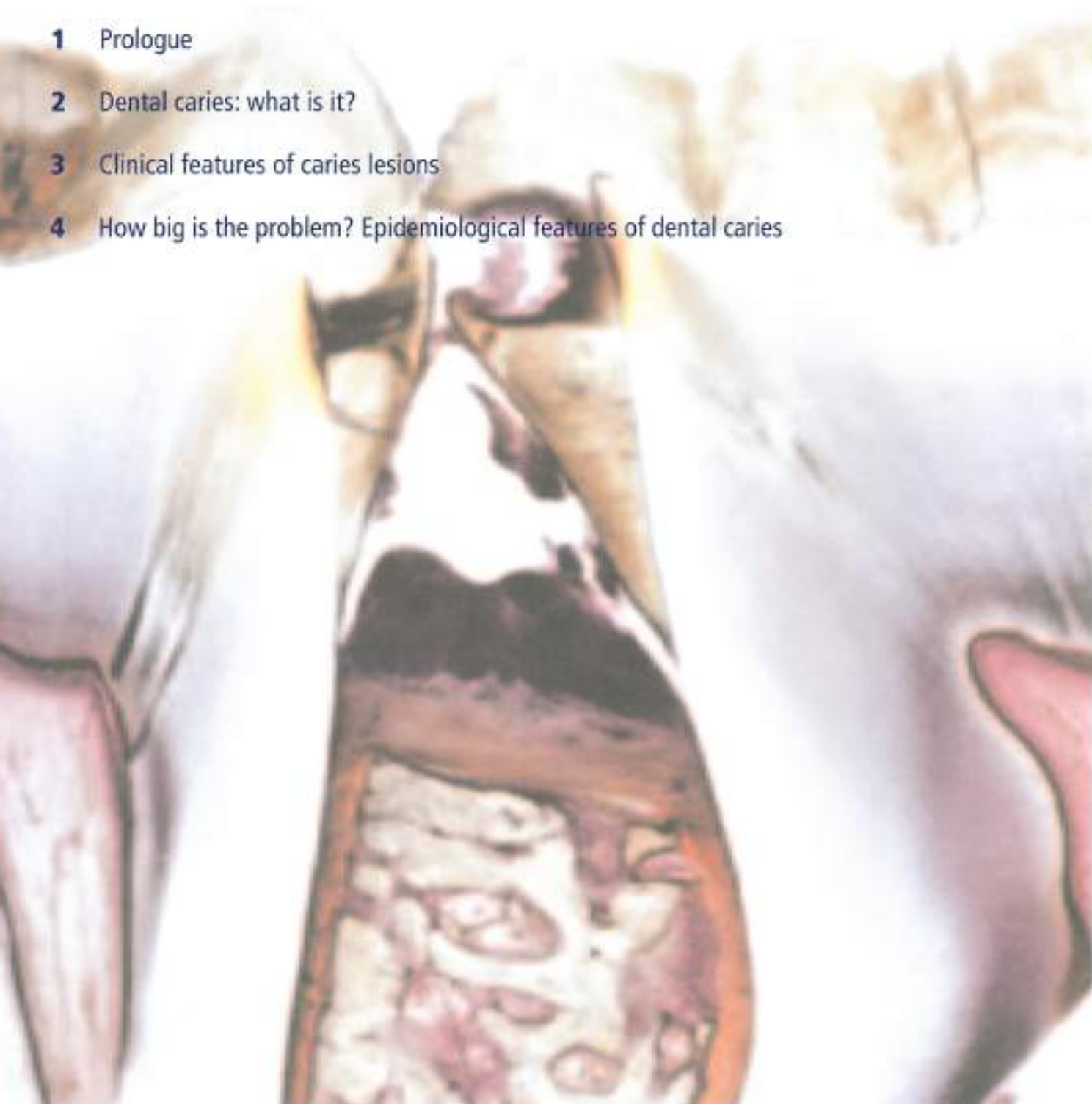


Professor Wim van Palenstein Helderma
Dental Health International Netherlands
Linschoten, The Netherlands

Part I

Dental caries: what is it and how widespread is it globally?

- 1 Prologue
- 2 Dental caries: what is it?
- 3 Clinical features of caries lesions
- 4 How big is the problem? Epidemiological features of dental caries



1

Prologue

O. Fejerskov, B. Nyvad, and E.A.M. Kidd

Introduction	3
The role of cariology in restorative dentistry	3
The content of this textbook	4

Introduction

Dental caries is ubiquitous – it is omnipresent in all populations and is as old as mankind. The caries incidence rate varies extensively between and within populations. With increasing age, signs and symptoms of dental caries accumulate, and in most adult populations the caries prevalence approaches 100%. Prevention and operative treatment of caries lesions and its sequelae occupy the majority of the dental profession lifelong around the world, and the cost of dental health care is a major societal burden.

The majority of dental restorations are made because of dental caries. Caries and failed restorative care are the main causes for tooth loss in all contemporary populations. So, it is hopefully obvious that there are good reasons for advancing an international textbook on *Dental Caries: The Disease and Its Clinical Management*.

It has been a great pleasure for us that the first two editions of this book have found their way all over the world. In the genesis of this third edition, we have realized that Edwina Kidd has been emerita professor for several years and soon Ole Fejerskov will follow, so it is time for a new generation to take over. Therefore, Professor Bente Nyvad is now a full member of the editorial team.

From the first to the second edition, the book 'put on weight,' becoming much more extensive. However, we have no wish for it to become 'morbidly obese' by continuing to

add to the length of text and topics. Rather, we have aimed in this third edition to make a 'slimmer' volume that hopefully makes it more readable for the main target: dental students and practitioners in public health and dental practice. We have invited 34 international colleagues to join us, many of whom are new, to ensure both continuity and novelty in lines of thinking throughout the book.

The role of cariology in restorative dentistry

The content of the book reflects our wish to make the basic knowledge about the bio-physiology of the oral cavity and dental caries applicable in daily clinical practice. When G.V. Black published his comprehensive textbook in 1908 he emphasized that clinical diagnosis and treatment decisions should have a sound biological rationale. Although it became appreciated by the middle of the 20th century that dentistry is a biomedical speciality, the technical advances with high-speed drilling and so on distorted the true application of biological knowledge in optimal treatment of dental caries.

Dental caries became synonymous with 'a cavity' in the tooth – and the automatic reaction was that the treatment should be 'drill and fill.' In the growing field of caries epidemiology, dental caries was recorded as DMF teeth/surfaces, where D stood for decay and decay meant a cavity. The knowledge about etiology and pathogenesis of caries was

often taught in dental curricula in departments of microbiology, pathology, and physiology, as well as in the growing disciplines of the 1950s and 1960s of dental public health and departments of pediatrics and preventive dentistry – but the clinical relevance of ‘cariology’ to restorative clinical departments was minimal, and knowledge to be applied at the chair-side fragmented. To some extent this was understandable, because in a clinical department the students were supposed to produce fillings, and crowns and bridges. *So the appreciation of the need for a concomitant disease control as part of any long-term successful restorative treatment was limited.*

The Keyes so-called triad stressed the components as being: (1) the tooth, (2) the diet, and (3) the microflora.

1. **The tooth.** In the last half of the 20th century much focus in caries research was on improving the ‘resistance’ of the tooth. Logically, there was an enormous interest in the role of fluoride in the prevention and control of dental caries. There were many attempts to introduce artificial water fluoridation worldwide in order to repeat the very impressive caries reduction that Trendley Dean and collaborators had documented in the USA. However, it proved difficult in many populations to introduce systemic fluoride programs, and by 1980 it became evident that the mechanism of action of fluoride in caries control was not a result of improving enamel resistance. Thus, the topical use of fluoride started to play a key role, particularly fluoride added to toothpaste.
2. **The diet.** The role of sugar in caries development had become evident by the middle of the 20th century, and much effort was invested in trying to reduce the intake, not least in children – with limited success. Total sugar consumption per capita stayed fairly constant and sugar substitutes were introduced. Then the dramatic caries decline became well documented, in particular in the Nordic countries, apparently without a significant reduction in total sugar consumption. So the relative role of diet was reconsidered.
3. **The microflora.** It was well known that ‘a clean tooth never decays’ – but the consequence of this statement was seldom fully appreciated. When rodent experiments around 1960 clearly indicated that dental caries was ‘an infectious and transmittable disease,’ extensive research focused on identifying one responsible microorganism: the caries pathogen. In the past, *Lactobacillus acidophilus* had been seen as the main bacterium causing caries, but the focus shifted towards mutans streptococci. This even led to attempts to introduce a caries vaccine against *Streptococcus mutans*! Extensive research was carried out without appreciating that there is a significant difference between the behavior of bacteria in a planktonic (free-floating) phase and the concept of oral ecology, where the single microorganism is part of a complex oral

microflora composed of more than 1000 different species. This was the focus around the turn of the century, where, gradually, dental plaque came to be considered an oral biofilm and dental caries seen as a biofilm-induced demineralization of the dental hard tissues.

But even at this stage many questioned the role of tooth cleaning in caries control!

This somewhat simplified overview of the major trends in dental caries research over the last 50 years is important because it will hopefully help new readers to understand how the very different concepts and ‘paradigms’ of today are influenced by historical tradition. The way in which scientific literature is selected and interpreted and its results introduced into diagnosis, prognostic assessments, treatments decisions, prevention, and public health strategies will profoundly influence how successful the dental profession will be in controlling dental caries and maintaining a functioning dentition in every patient from cradle to grave.

The content of this textbook

A textbook reflects the way in which the authors interpret scientific data on a given subject, but we do not pretend that this is the ‘truth’ about the complex disease called ‘dental caries.’ There are extensive data available on today’s Internet, and the stream of information will continue to grow. This is an enormous challenge to clinical students and practitioners. How can sense be made of the bombardment of information? The authors have been asked to carefully present their respective subtopics so that it is not just a compilation of data, but selected data critically brought together in order to explain why dental caries presents itself in the individual and in populations in the way it does in today’s world.

The aim of this book is to present the dental student and the dental practitioner with an update on the available knowledge about dental caries, and the consequences of this to its diagnosis, and how most appropriately and cost effectively to control caries progression. Clinical decision-making and the balance between nonoperative and operative treatments become even more important parts of daily life in clinical practice. An understanding of the caries process is needed to estimate the prognosis of treatment procedures and the possibility of assessing the risk of disease development in individuals and populations.

This book will demonstrate that in real life the processes involved in dental caries are highly complex. In an ideal world there would be a perfect deterministic model that could relate all the potential determinants perfectly to caries outcome. It will appear throughout the book that most of the determinants that influence caries can, at best, be measured only as proxy variables. The most we can hope for, therefore, is to develop probabilistic models that relate

determinants to risk of caries progression. However, even under such circumstances, caries would remain unpredictable. Such inputs as:

- variable exposure to fluoride,
- times, lengths, frequencies, and types of sugar consumption,
- quality of tooth cleaning,
- fluctuations in salivary flow rates and composition,
- quality and composition of biofilms,
- the behavior of the individual, and
- the societal context of the individual

are themselves highly variable. It is likely that this variability and unpredictability of the inputs may play a crucial role in the way in which the caries process develops. But all these factors make up the fascination and challenge of our profession.

It is our hope that this book will prepare the reader to become a less dogmatic and more knowledgeable health professional who strives to control dental caries in the most cost-effective way.

The *content* of the book is organized according to our wish to link theory with clinical performance. In other words, making prevention, diagnosis, and restorative procedures evidence based.

In Chapters 2–4 we define what dental caries is and how it most often manifests itself on different tooth surfaces.

Then we ask the question 'How big is the caries problem in different parts of the world?' and present basic epidemiological tools.

Part II, 'The caries lesion and its biological determinants' (Chapters 5–9), basically covers the aspects of the Keyes triad. Then this knowledge is applied in Part III, 'Diagnosis' (Chapters 10–12), and Part IV, 'Controlling dental caries' (Chapters 13–18). When caries lesions require 'Operative intervention' (Part V, Chapters 19–21), biological knowledge is essential to provide the most careful intervention and not to make unnecessary replacement of restorations. Finally, Part VI, 'From chair-side to population caries control' (Chapters 22–25), brings the principles of dealing with the single tooth surface or the individual patient forward by approaching caries control in whole populations of different kinds. Moreover, this part addresses the very important questions about risk assessment versus prediction. In the final chapter we ask the question: If we were to apply the knowledge we have today on how to control dental caries most cost-effectively in various parts of the world and maintain a functional dentition lifelong, what recommendations would we then formulate?

We hope you will enjoy the book – and interact with us, whether agreeing or disagreeing. As Charles Darwin said: 'All observations must be for or against some view to be of any service.'

2

Dental caries: what is it?

O. Fejerskov, B. Nyvad, and E.A.M. Kidd

The disease	7
Terminology	9
Background literature	9
References	10

The disease

The term *dental caries* is used to describe the results – the signs and symptoms – of a localized chemical dissolution of the tooth surface caused by metabolic events taking place in the biofilm (dental plaque) covering the affected area. The destruction can affect enamel, dentin and cementum. The lesions may manifest themselves clinically in a variety of ways, as will be dealt with in Chapter 3.

In principle, dental caries lesions may develop at any tooth site in the oral cavity where a biofilm develops and remains for a period of time. It is therefore a misconception to talk about more- or less-susceptible surfaces as this may erroneously give rise to the belief that certain parts of a tooth are more 'resistant' or 'less susceptible' to developing caries lesions due to variations in the chemical and structural composition [1, 3].

This is not to say that all tooth surfaces within the oral cavity of an individual develop caries lesions at the same rate. Dental caries lesions develop at relatively 'protected sites' in the dentition where biofilms (dental plaque) are allowed to accumulate and mature over time. Such sites include pits, grooves, and fissures in occlusal surfaces, especially during eruption, approximal surfaces cervical to the contact point/area, and along the gingival margin. Obviously, insertion of foreign bodies to the dentition (e.g. fillings with inappropriate margins, dentures, orthodontic bands) may also result in such 'protected' sites. These areas

are relatively protected from mechanical influence from the tongue, the cheeks, abrasive foods, and, not least, tooth-brushing. Thus, these are the sites where lesion development is more likely to occur because the biofilm is allowed to stagnate there for prolonged periods of time.

This knowledge is very important, and it is 100 years ago that Black [1] stated:

...the beginning of caries of the teeth occurs at such points as will favour such lodgement or attachment in which the microorganisms will not be subject to such frequent dislodgement as would prevent a fairly continuous growth. This is the cause of the localisation of the beginnings of caries on particular parts of the surface of the tooth.

Dental caries lesions, furthermore, do not develop at the same rate in all parts of the mouth. Thus, openings of the major salivary glands represent areas with a special salivary composition that favors a relative protection towards chemical dissolution because of buffering capacity and chemical composition of the secretory product (see Chapter 6).

Dental caries lesions result from a shift in the ecology and metabolic activity of the biofilm (Chapter 7) whereby an imbalance in the equilibrium between tooth mineral and biofilm fluid has developed. It is important to appreciate that an oral biofilm, which forms and grows ubiquitously on solid surfaces in the oral cavity, does not

necessarily result in the development of *clinically visible* caries lesions when grown on dental hard tissues. Thus, a biofilm grown on tooth enamel inserted in palatal devices (so-called in-situ models) has to be 'protected' from abrasion from the tongue movement. Similarly, in-situ models for studying caries lesion development under controlled conditions have preferably to be located so that microbial stagnation areas are created. But the biofilm is a prerequisite for caries lesions to occur. The biofilm is characterized by continued microbial activity, resulting in metabolic events in the form of continuous, minute pH fluctuations. The metabolism may be dramatically enhanced by changing the nutritional conditions (e.g., by adding fermentable carbohydrates) and the outcome of the metabolism can be recorded as pH fluctuations. Any shift in pH will influence the chemical composition of the biofilm fluid and the relative degree of saturation of this fluid with respect to the minerals that are important for maintaining the chemical composition of the tooth surface (see Chapter 9). From the very moment of eruption into the oral cavity, the tooth surface apatite will continue to be subject to these chemical modifications on innumerable occasions. Most of these modifications are so subtle that they can only be recorded at the nano-level. Surfaces that are frequently covered by biofilm (e.g., a cervical enamel surface) will gradually accumulate fluoride in the very surface layers (outermost 100µm) (see Chapter 9, Fig. 9.11). Thus, the enamel surface is in a state of dynamic equilibrium with its surrounding environment. *When the cumulative result of the numerous pH fluctuations over months or years is a net loss of calcium and phosphate of an extent that makes the enamel sufficiently porous to be seen in the clinic, we may diagnose it as 'a white spot' lesion* (see Chapters 3 and 5). It is important to appreciate, however, that, although the metabolic events may result in detectable caries lesion formation, most sequences of metabolic events tend to cancel each other, which is why the metabolic events should be considered intrinsic to biofilm physiology (Chapter 7). The caries lesions arise when there is a drift in the metabolic events; that is, when the pH drops result in a net loss of mineral. *Thereby, the dental caries lesions are a result of an imbalance in physiologic equilibrium between tooth mineral and biofilm fluid.*

These considerations lead to some important points:

- The dissolution (demineralization) when pH drops below a certain level in the biofilm and the redeposition (remineralization) of minerals when pH goes up (see Chapter 9) takes place in the enamel surface at the interface between the biofilm and the tooth surface. These processes occur numerous times during a day and can be modified extensively. If, for example, the biofilm is partly or totally removed, mineral loss may be arrested

or even reversed towards mineral gain because saliva is supersaturated with respect to the enamel apatite. This will result in arrest of disease progression – and may even result in some redeposition of minerals in the very surface of the tooth.

- Any factor that influences the metabolic processes, such as the composition (e.g., content of buffering proteins) and thickness of the biofilm, the salivary secretion rate and composition (Chapter 6), the diet (Chapter 8), and the fluoride ion concentration in the oral fluids (Chapters 9 and 13), will contribute to the likelihood of a net loss of mineral – and the *rate* at which this occurs. Figure 2.1 indicates how the many biological determinants of the caries process may act at the level of the individual tooth surface (inner circle). At the individual/population level (outer circle) the behavior, education, knowledge, and attitudes will have a strong influence on some of the biological determinants (quality of oral hygiene, choice of foods, use of fluorides, salivary flow from chewing gum, etc.)
- At any given point in time the net mineral loss or gain is part of a continuous spectrum of events. The absence of a clinically detectable caries lesion does not necessarily

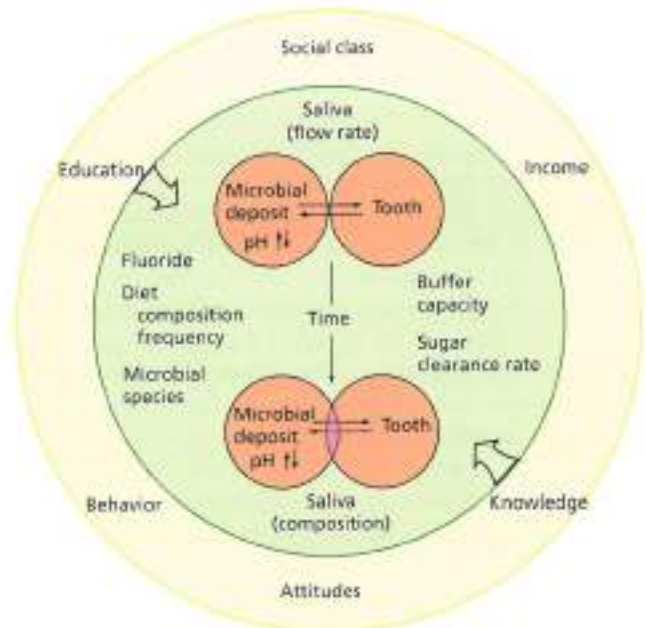


Figure 2.1 Schematic illustration of the determinants of the carious process. Those that act at the tooth surface level are found in the inner circle. With time, an ecological shift in the composition and metabolic activity of the biofilm ('microbial deposit') may result in an imbalance in the equilibrium between biofilm fluid and the mineral of the tooth. Thus, a net loss of mineral results in formation of a caries lesion (overlap of the two small circles). In the outer ring are listed more distant determinants that influence these processes at the individual and population levels. Adapted from [2]. Reproduced with permission of the University of North Carolina School of Dentistry.

mean that no mineral loss has occurred (Chapter 5); it only means that it could not be discerned clinically. If this concept of a continuum is appreciated it will immediately be understood why diagnosis of various stages of lesion progression is a question of defining certain 'cut-off' points (Chapter 4).

Terminology

Caries lesions may be classified in a number of ways. Unless the student is familiar with this terminology it can be difficult to understand what is written. This section introduces and defines various terms that will trip off the writers' pens in subsequent chapters.

Cariou lesions can be classified according to their *anatomical site*. Remember, there is nothing chemically special about these sites. Thus, lesions may commonly be found in *pits and fissures* or on *smooth surfaces*. Smooth surface lesions may start on enamel (*enamel caries*) or on the exposed root cementum and dentin (*root caries*).

Primary caries is used to differentiate lesions on natural, intact tooth surfaces from those that develop adjacent to a filling, which are commonly referred to as *recurrent* or *secondary caries*. These two latter terms are synonyms, but in this textbook we will use the term *recurrent caries* throughout. Recurrent caries is simply a lesion developing at a tooth surface adjacent to a filling. As such, its etiology is similar to primary caries.

Residual caries (as the term implies) is demineralized tissue that has been left behind before a filling is placed.

An important classification is whether a lesion is *cavitated* or *noncavitated*. A *cavity* is a physical hole in the tooth, and it may impinge directly on the management of the lesion (Chapters 16 and 18).

Caries lesions may also be classified according to their activity. This is a very important concept and one that impinges directly on management, although it will be evident from the text that the clinical distinction between *active* and *inactive* (arrested) lesions is sometimes difficult.

A lesion considered to be progressing (you anticipate that the lesion would have developed further at a subsequent examination if not interfered with) would be described as an *active caries lesion*. This distinction is based on a judgment of the features of the lesion in question in combination with an assessment of the oral health status of the patient. In contrast to this is a lesion that may have formed years previously and then stopped further progression. Such lesions are referred to as *arrested caries lesions* or *inactive caries lesions*.

You may also meet the terms *remineralized* or *chronic lesions* used to signify arrested lesion; but, as you will appreciate later, the term remineralization should be used with caution (Chapters 5 and 9). The distinction between active and inactive/arrested lesions may not be totally

straightforward. Thus, there will be a continuum of transient changes from active to inactive/arrested and vice versa. A lesion (or occasionally part of a lesion!) may be rapidly progressing, slowly progressing, or not progressing at all. This will be entirely dependent on the ecological balance in the biofilm covering the site and the environmental challenge. Clinically, if in doubt, the dentist should always react as if they are dealing with an active lesion.

Despite the diagnostic difficulties, these distinctions are very important to the clinician because if a lesion is not active then no action is needed to control further progression. If, on the other hand, a lesion is considered active, steps should be taken to influence the metabolic activities and possibly the ecological balance in the biofilm in favor of arrest rather than further demineralization.

At this point it is also sensible to discuss a possible confusion in terminology. The first sign of a carious lesion on enamel that can be detected with the naked eye is often called a *white spot lesion*. This appearance has also been described as an early, *initial* or *incipient lesion*. These terms are meant to say something about the stage of lesion development. However, a white spot lesion may have been present for many years in an arrested state; and to describe such a lesion as early would be inaccurate. A dictionary definition of incipient is 'beginning'; an initial stage. In other words, an initial lesion appears as a white, opaque change (a white spot) – but any white spot lesion is not incipient!

Rampant caries is the name given to multiple active carious lesions occurring in the same patient. This frequently involves surfaces of teeth that do not usually experience dental caries. These patients with rampant caries can be classified according to the assumed causality; for example, *bottle or nursing caries*, *early childhood caries* (ECC) when observed in children, and *bakers' caries*, *radiation caries*, and *drug-induced caries* when seen in adults. ECC is simply caries on teeth that are not clean, exposed to carbohydrates, and located in an area of the mouth where oral clearance is low (for details, see Chapter 16).

Hidden caries is a term used to describe lesions in dentin that are missed on a visual examination but are large enough and demineralized enough to be detected radiographically. It should be noted that whether a lesion is actually hidden from vision depends on how carefully the area has been cleaned and dried and whether an appropriate clinical examination has been performed.

Background literature

Baelum V, Fejerskov O. Caries diagnosis: 'a mental resting place on the way to intervention'? In: Fejerskov O, Kidd EAM, eds. *Dental caries. The disease and its clinical management*, 1st ed. Blackwell/Munksgaard, 2003: 101–10.

Fejerskov O. Changing paradigms in concepts on dental caries: consequences for oral health care. *Caries Res* 2004; 38: 182–91.

References

1. Black GV. *Operative dentistry*. Vol. 1. *Pathology of the hard tissues of the teeth*. London: Claudias Ash, Sons & Co. Ltd, 1914.
2. Fejerskov O, Manji F. Risk assessment in dental caries. In: Bader J, ed. *Risk assessment in dentistry*. Chapel Hill, NC: University of North Carolina Dental Ecology, 1990: 215–17.
3. Weatherell JA, Robinson C, Halleworth AS. The concept of enamel resistance – a critical review. In: Guggenheim B, ed. *Cariology today*. Basel: Karger, 1984: 223–30.