

Restorative Management of the Worn Dentition: I. Aetiology and Diagnosis

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Abstract: In this, the first of a four-part series on the restorative management of the worn dentition, the aetiological factors, diagnosis of toothwear and preventive measures are summarized. Later papers will deal with the management of localized anterior and posterior toothwear, the use of 'Dahl type' appliances as an effective means for the restorations and the various treatment options for the management of the dentition showing generalized wear. The series will discuss the relative merits of the treatment strategies, clinical techniques and dental materials for the restoration of health, function and aesthetics for the dentition.

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Clinical Relevance: Management of patients with toothwear requires thorough understanding of the causes and their prevention. This paper reviews the aetiological factors and assists the readers in reaching a diagnosis.

It has been argued that the prolonged retention of cusps in the human dentition is a relatively recent evolutionary aberration due to the low abrasiveness of modern diets, and that human teeth should be worn down to achieve maximum masticatory efficiency, as occurs in the dentitions of many herbivorous mammals.¹ Although some degree of toothwear is accepted as a normal part of the ageing process, problems associated with toothwear have increasingly been attracting the

attention of the dental profession in the last few decades. A large dental health survey of middle-aged adults showed that increasing toothwear was observed with age for cervical, occlusal and incisal surfaces.² Epidemiological studies of young adults reported that prevalence of toothwear was in the range 6–45%.^{3–5} Several investigations have also provided useful information on the prevalence of toothwear in children in relation to dental erosion.^{6–8} Although comparisons of these epidemiological studies are not feasible because of the different criteria used, toothwear caused by different aetiological factors is prevalent in different age groups and populations.

This four-part series of papers aims to summarize current knowledge of the aetiology and diagnosis of toothwear, and to present logical steps for the management of different degrees of localized and generalized toothwear affecting the adult dentition.

AETIOLOGY

Toothwear has been defined as loss of tooth substance resulting from abrasion, attrition and erosion⁹ (Table 1) acting singly or concurrently.¹⁰ When wear is due to more than one predominant aetiological factor, special terms have been suggested to highlight the multiplicity of causes. For example:

- 'abrosion' describes enamel removed with a toothbrush after enamel has been softened by acid (abrasion and erosion);
- 'demastication' refers to enamel worn away by attrition (mastication) after erosion (demineralization).¹¹

Abrasion

The combination of a hard toothbrush, an abrasive toothpaste and an intensive horizontal brushing technique is believed to cause well-defined, V-shaped notches in the cervical regions of one or more facial tooth surfaces, where the dentine

Abrasion	Wear process involving foreign objects sliding or rubbing against the tooth surfaces.
Abfraction	Non-carious cervical lesions caused by tensile stress generated from occlusal loading, and microfracture of cervical enamel rods.
Attrition	Wear process of the tooth substance by tooth-to-tooth contact.
Erosion	Loss of dental hard tissues by non-bacteriogenic acid etching.

Table 1. Definitions.^{9,11,13,28}

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Figure 1. Non-carious cervical lesions on buccal surfaces of I1/2.

and cementum are less wear-resistant than coronal enamel (Figure 1). Location of the abrasion (three-body wear) lesions depends on tooth alignment and/or which hand is holding the toothbrush – more lesions occur on the left side with right-handed persons, and vice versa. Other habits causing abrasion include the misuse of dental floss and toothpick, and pipe-smoking. Thread biting, and holding hair-grips between the teeth can lead to abrasion defects of incisal tooth edges in seamstresses and hairdressers, respectively.

Abfraction

Abrasion alone cannot satisfactorily explain how every cervical non-carious lesion occurs. The concept of 'stress-induced cervical lesions' was introduced to explain how wedge-shaped Class V lesions can be created by repeated compression and flexure of the teeth under occlusal loading. Dentine is more elastic than enamel,¹² and enamel rods can be fractured in such situations. In older adults, enamel crazing and microfractures are more common. This may explain why such lesions are more prevalent in older age groups. The term abfraction was used to describe this 'stress corrosion' mechanism (Table 1). However, the physiochemical effects of an acidic environment may also be responsible.¹³

The 'stress corrosion' theory has been supported by a number of observations:

- *in vitro* evidence of tensile stresses created in the cervical region under occlusal loading;¹⁴
- a high incidence in bruxists;¹⁵

- lesions can be found on only one tooth in one segment;
- lesions found in subgingival regions;
- the presence of such lesions in animals.

An SEM study reported that the tip of such lesions can be rounded or sharp, and the mesial and distal angles were connected by one or more internal grooves. The rounded-tip lesion is thought to have an erosion component.¹⁶

Attrition

Attrition resulting from tooth-to-tooth contact (two-body wear) produces well defined wear facets on the functional surfaces of teeth in one jaw which match corresponding lesions on teeth in the other jaw (Figure 2). While 'cupping' of the incisal edges (a shallow concavity of the incisal edge surrounded by enamel) could also be due to acid attack on dentine, it was postulated that this appearance could be a result of three-body wear or abrasion (as in the presence of food) because dentine has lower wear and erosion resistance than enamel.¹⁷ With improved life expectancy and

control of dental caries and periodontal disease, it is likely that retention of natural teeth into older age will lead to a higher prevalence of worn dentition as a result of attrition.¹⁸

Parafunctional habits such as bruxism and clenching were also believed to be important factors in causing accelerated attrition. However, short-term studies showed that toothwear was not significantly different between bruxists and non-bruxists.^{19,20} The difference between clinical impressions and research reports may be because the effects of bruxism are slow and short-term studies were not sensitive enough to detect the small changes.

Other factors predisposing to attrition include developmental dental defects,²¹ coarse diet, natural teeth opposing coarse porcelain (Figure 3), pseudo-Class III incisal relationship (Figure 4) and lack of posterior support (Figure 5).

Erosion

Erosion of tooth substance may be caused by intrinsic or extrinsic acids, and modified by changes of salivary flow and constituents.

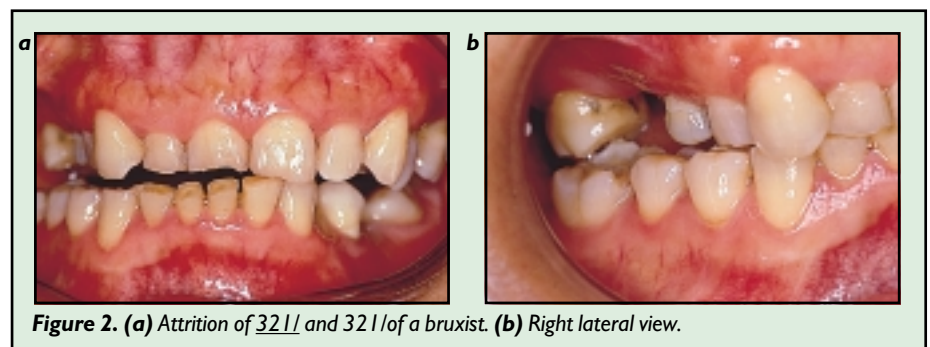


Figure 2. (a) Attrition of 321/1 and 321/2 of a bruxist. (b) Right lateral view.

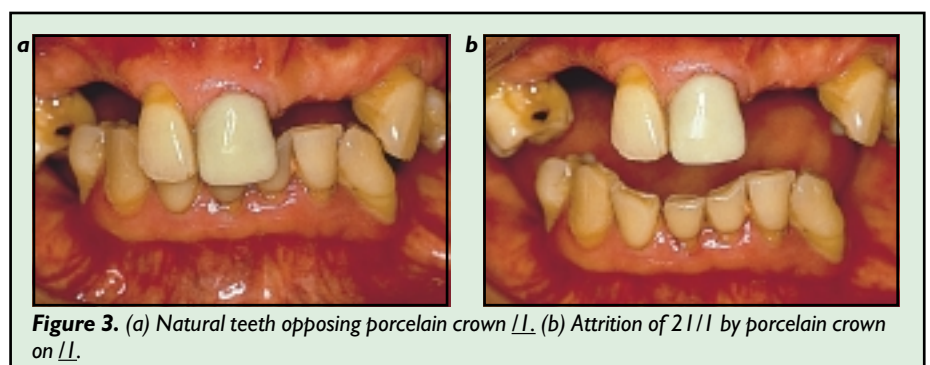


Figure 3. (a) Natural teeth opposing porcelain crown I1. (b) Attrition of 21/1 by porcelain crown on I1.

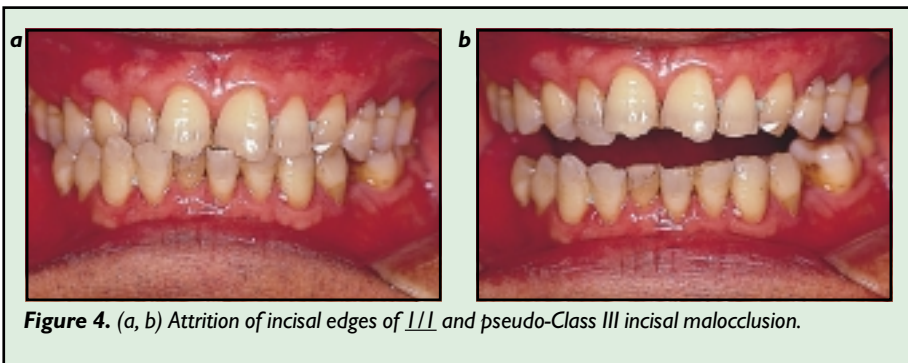


Figure 4. (a, b) Attrition of incisal edges of I11 and pseudo-Class III incisal malocclusion.

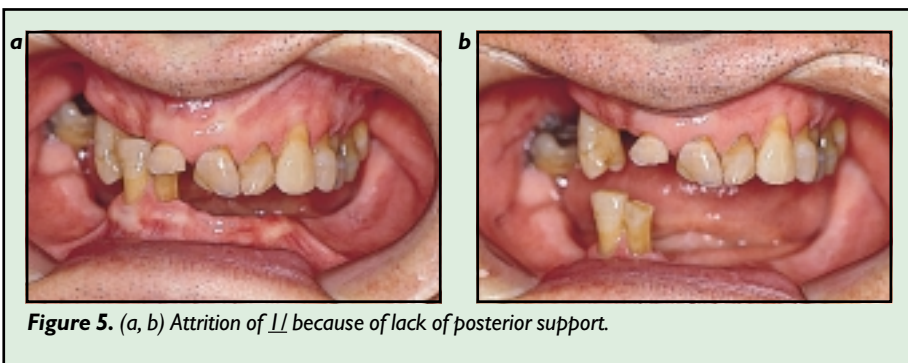


Figure 5. (a, b) Attrition of I1 because of lack of posterior support.

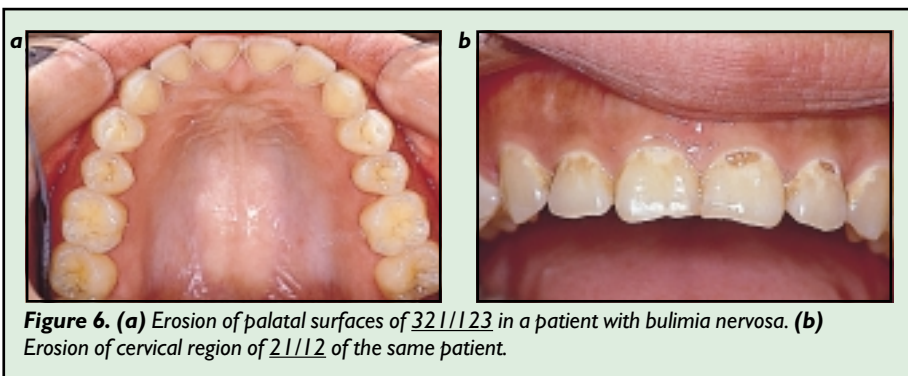


Figure 6. (a) Erosion of palatal surfaces of 32 I11 23 in a patient with bulimia nervosa. (b) Erosion of cervical region of 2 I11 2 of the same patient.

Acid erosion

Gastric juice, an intrinsic acid containing a high concentration of hydrochloric acid, is normally confined to the stomach by the gastro-oesophageal sphincter, although medical problems such as alcoholism-induced gastritis, pregnancy sickness and hiatus hernia, and eating disorders such as anorexia nervosa or bulimia nervosa, may lead to gastro-oesophageal reflux disorders and voluntary regurgitation.

Extrinsic acids are found in the diet (wine, soft drinks, preserved foods, medications, etc.) or the environment (e.g. vaporized sulphuric acids from batteries, poorly buffered chlorine in swimming pools). The increased

consumption of acidic beverages by children and adolescents during the past decade or so has had a significant effect on the incidence of dental erosion among young people: acidic beverages have been reported as major aetiological factors in 40% of young adult patients with anterior toothwear.²² Soft drinks may contain phosphoric and organic acids, and fruit juices contain citric and maleic acids.

An *in vitro* study reported that concentration, type of dietary acids, and the exposure time can greatly influence the erosion of enamel and dentine.²³ However, relationships between erosion and the pH values of foods and drinks,²⁴ the holding time of the foodstuffs in the

mouth, and the buffering effects of saliva and plaque are still not clearly understood.

Flow of Saliva

Reduced salivary flow following surgical excision of one or more major salivary glands, Sjögren's syndrome, drug intake (e.g. antidepressants, sedatives, tranquillizers), or radiotherapy in the head and neck region predisposes not only to rapid caries development but also to dental erosion. In addition to its diluting and flushing effects, changes in flow rate of saliva may also affect its buffering capacity, and concentrations of secreted ions available for remineralization.

Patterns of Toothwear in Erosion

Eroded tooth surfaces have less well defined extensions than attrition lesions. The enamel has a matted surface, and dentine may be exposed with continuous erosion. The distribution and severity of erosive lesions depend on how acidic substances were consumed or held in the mouth. Palatal erosion of upper anterior teeth has been attributed to intrinsic and extrinsic acids, which may be held by the tongue against the teeth (Figure 6). The acid may contact immediately with the palatal surfaces when it is regurgitated forcibly. Generalized but less severe toothwear may be seen with extrinsic acids, with labial/buccal and incisal/occlusal surfaces being more commonly affected. Erosion can lead to old amalgam restorations becoming 'outstanding' (Figure 7). Severe erosion may also increase the



Figure 7. Outstanding amalgam on occlusal surface of 6l because of erosion.

C/O:	Dental sensitivity	Y/N
	Tooth fracture	Y/N
	Poor masticatory function	Y/N
	Poor aesthetic of front teeth	Y/N
	Others: _____	
HPC:	Duration: _____ Years _____ Months _____ Days	
MH:	Intrinsic acid	
	Eating disorders	Y/N
	Gastritis: e.g. alcoholism	Y/N
	Medication: e.g. chewing vitamin C tablet (ascorbic acid), taking dilute HCl for hypochlorohydia	Y/N
	Pregnancy: related gastro-oesophageal reflux disorder	Y/N
	Idiopathic gastro-oesophageal reflux disorder	Y/N
	Reduced salivary flow	
	Dehydration	Y/N
	Surgical excision of salivary glands	Y/N
	Radiotherapy in head and neck region	Y/N
	Drug-induced xerostomia	Y/N
	Autoimmune disease, e.g. Sjögren's syndrome	Y/N
DH:	Construction of porcelain restorations	Y/N
FSH:	Bruxism/clenching	Y/N
	Extrinsic acid	
	Soft/sports drink consumption	Y/N
	Wine	Y/N
	Vinegar preserved food	Y/N
	Occupation: _____	
O/E:		
E/O:	TMJ pain	Y/N
	OVD satisfactory	Y/N
	RVD satisfactory	Y/N
	Amount of freeway space: _____ mm	
I/O:	Soft-tissue pathology	Y/N
	Periodontal charting	Y/N
	Dentine sensitivity	Y/N
	Endodontic treatment need	Y/N
	If yes, which tooth: _____	
	Coronal tissue loss (with dentine exposure = TWI 3/4):	
	Cervical lesions (teeth: _____)	Y/N
	Localized anterior (teeth: _____)	Y/N
	Localized posterior (teeth: _____)	Y/N
	Generalized (teeth: _____)	Y/N
	Occlusal relationship	
	Anterior crossbite (teeth: _____)	Y/N
	Defective contacts and mandibular deviation	Y/N
	Interocclusal clearance between worn teeth in ICP	Y/N
	Difference between ICP and RCP	Y/N
	Vertical	Large/Small
	Horizontal	Large/Small
	Functional need because of multiple missing posterior Teeth (according to SDA):	
	Aesthetics	
	Short clinical crown	Y/N
	Uneven fracture incisal edges	Y/N
	Satisfactory existing relation between incisal edges and lower lip line	Y/N
Ix	1. Study models were taken	Y/N
	2. Photos were taken	Y/N
	3. Salivary test	
	Resting salivary flow: _____ ml/min	
	Stimulated salivary flow: _____ ml/min	
	Buffering capacity: _____	
	4. Radiographs were taken	
	Bitewings (left/right)	
	Periapical (teeth: _____)	
Dx:	_____	

Figure 8. Examination form for assessing patients with toothwear.

translucency of anterior teeth, before thin enamel and dentine fracture.

Erosion as an aetiological factor of toothwear should not be overlooked: history taking and clinical investigations should attempt to reveal the possible presence of acid(s).

DIAGNOSIS

Before any intervention or restorative treatment, the nature and duration of a patient's chief complaints and expectations must be ascertained.

Diagnosis of the causes of toothwear may not be easy, partially because the patient may not recognize the signs or symptoms themselves, or might not want to volunteer sensitive information (such as an eating disorder).

Apart from using a routine medical questionnaire, emphasis may be placed on medical conditions predisposing to erosion because of gastro-oesophageal reflux and reduction of salivary flow (Figure 8). Close collaboration with medical colleagues is indicated for the investigation and management of underlying medical problems. For example:

- the gastroenterologist can place a 24-hour pH meter in the oesophagus to investigate the frequency and severity of reflux before medical and/or surgical treatment;
- a psychiatrist can counsel patients with eating disorders for correction of their misconceptions about self-image;
- a physician may replace drugs predisposing to xerostomia with appropriate alternatives.

Evaluation of the family and social history can reveal if the patient is under unusual stress, which may be related to bruxism, changes in diet, and regurgitation. A diet sheet is useful for recording the quantity and frequency of intake of citrus fruits and carbonated drinks.

Clinical examination of the dentition has two primary objectives:

1. To document and record the

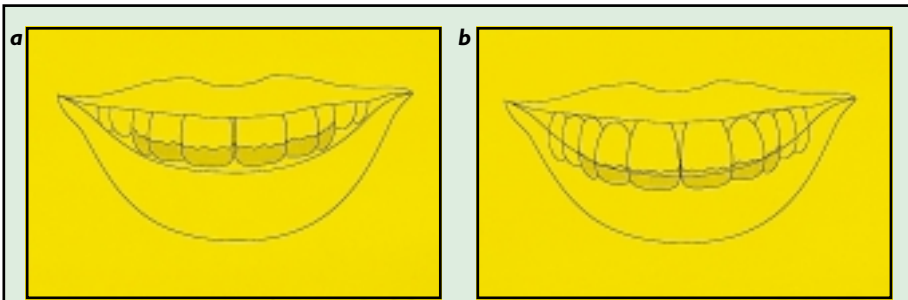


Figure 9. (a) Relationship between lower lip line and incisal edges of worn anterior teeth. Elongation of the worn anterior teeth is feasible. (b) Relationship between lower lip line and incisal edges of worn anterior teeth. Elongation of the worn teeth would lead to an excessively long clinical crown.

location, appearance and degree of toothwear.

2. To evaluate the progress of toothwear over time.

Toothwear does not preclude primary dental diseases such as periodontitis and caries. Therefore, full examination should be carried out. The modified toothwear index (TWI)^{2,25} had been proposed as a tool for quantifying the degree of damage to different tooth surfaces, and possible identification of aetiological factors. However, its clinical application is limited as the naked eye can detect only wear of more than 100 microns,²⁶ and it is impossible to identify minute changes over time without more sophisticated research methods unless toothwear is very rapid.

Clinical examination can be supplemented with high-density stone study casts, intra-oral photographs, radiographs, and salivary tests. The stone casts can be mounted in retruded contact position (RCP) on a semi-adjustable articulator for occlusal and space analyses. If a 'horizontal'

difference exists between RCP and intercuspal position (ICP), the available space between the anterior teeth may be useful for anterior restoration. In such cases, a new ICP must be provided by occlusal adjustments. Stone casts allow easier assessment of toothwear, while intra-oral photographs are helpful for identification of areas of dentine/pulp exposure. Direct clinical observation of the patient at rest and during speech, and the use of photographs for analysis of the smile line in relation to the level of incisal edges, are essential. If dentoalveolar compensation has occurred, further lengthening of the anterior teeth by restoration alone might not be acceptable, and clinical crown lengthening may be required (Figure 9). Periapical and bitewing radiographs are important for assessment of thickness of remaining enamel and dentine, as well as existing crown/root ratios, location of furcations and presence of periapical pathoses. Records of resting and stimulated salivary flow rates and salivary buffering capacity are relatively inexpensive investigations for patients suspected of suffering from reduced salivary flow (Figure 10).

PREVENTIVE AND INITIAL MANAGEMENT

Before any definitive restorative treatment is undertaken, plaque-induced dental diseases such as caries and periodontal disease should be controlled. The long-term success of rehabilitation is dependent on good oral hygiene and regular maintenance.

In addition to the relief of pulpal pain, the management of dentine sensitivity and the smoothing of sharp teeth, a number of preparatory procedures should be carried out to facilitate the restorative management of the worn dentition: for example, sensitive cervical cavities should be restored using resin composite, glass ionomer cements or compomer.

Efforts should be made to eliminate or control the aetiological factors. To prevent further cervical abrasion, a correct toothbrushing technique with suitable dentrifices has to be introduced as early as possible. Object-biting habits (once recognized) must cease. Patients with erosion caused by dietary acids should be instructed to reduce the quantity and frequency of such consumption, and to avoid abrasive toothbrushing immediately afterwards. Patients with eating disorders should be referred to a medical practitioner for advice and treatment. The use of soft vinyl mouthguards filled with magnesium hydroxide could be considered for use by bulimic patients before deliberate vomiting. Such mouthguards can be loaded with neutral fluoride gel (e.g. Karigel-N, Young Dental, Missouri, USA) to promote remineralization of tooth tissues and reduce tooth sensitivity.

To protect teeth from further attrition, an occlusal splint made in hard acrylic resin can be prescribed if most of the teeth are retained. The maxillary splint should have full occlusal coverage, multiple occlusal contacts on closure, and correct anterior guidance (Figure 11). For patients with attrition of anterior teeth and multiple missing anterior teeth, removable partial dentures are



Figure 10. Commercial product for testing salivary buffering capacity.



Figure 11. Hard acrylic maxillary occlusal splint.

recommended for protecting the anterior teeth. Correct polishing procedures can reduce the surface roughness of abrasive porcelain restorations.²⁷ Orthodontic treatment is recommended to correct Class II division 2 and Class III incisal malocclusion during adolescence.

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BOOK REVIEW

Antibiotic and Antimicrobial Use in Dental Practice, 2nd ed. M.G. Newman and A.J. van Winklehoff, eds. Quintessence Publishing Co. Ltd., New Malden, 2001 (304pp., £27.00 p/b). ISBN 0-86715-397-0.

Superbugs and antibiotic resistance are rarely out of the headlines these days and the dental profession, in line with other prescribers, have a duty to prescribe antibiotics judiciously. A text on antibiotics and antibiotic use in dental practice should therefore be a welcome addition to the practitioners bookshelf. This second edition, edited by Newman and Winklehoff, is certainly comprehensive. There are chapters on new and evolving clinical issues such as oral malodour, paediatrics, implant dentistry, legal

implications and even considerations for female patients. The editors rightly highlight the need for precise strategies and guidelines governing the use of antibiotics, however, I was unable to detect either of these in the text.

There are 21 contributors to the text and this has probably contributed to some confused messages and inconsistencies, for example, the use of tetracyclines in the prophylaxis of bacterial endocarditis. The layout of the text with key facts, clinical insights and important principles lost their impact on reading each chapter. Some important messages, although eloquently written, seemed to get lost in the text, for example, 'Antibiotics should be used very selectively in the treatment of periodontal disease. Narrow indications should replace the indiscriminate repeated use advocated by some manufacturers.' These key facts would

have been better placed at the end of each chapter.

Despite the prominent disclaimer about the differences in suggestions between chapters – I feel this text has lost an opportunity for consensus between such a distinguished group of contributors with some clear and concise guidelines for practitioners.

The book has a distinct North American feel to it which may or may not appeal to the reader. As a result, many of the drugs, for example, ciprofloxacin and dosages are either not in the Dental Practitioners Formulary or do not comply with current UK practice guidelines, for example 'loading doses of penicillin VK of 1000mg'. In view of these issues, I would find this text difficult to recommend to UK dental practitioners.

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