

## The oral medicine of tooth wear

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### Abstract

This review illustrates, through a series of case histories, how oral medicine insights aid the diagnosis and management of patients with excessive tooth wear. The cases reviewed are drawn from the records of 500 southeast Queensland patients referred to the author over a 12 year period. Patients most at risk of dental erosion have work and sports dehydration, caffeine addiction, gastro-oesophageal reflux, asthma, diabetes mellitus, hypertension or other systemic diseases or syndromes that predispose to xerostomia. Saliva protects the teeth from the extrinsic and intrinsic acids which cause dental erosion. Erosion, exacerbated by attrition and abrasion, is the main cause of tooth wear. These cases illustrate that teeth, oral mucosa, salivary glands, skin and eyes should be examined for evidence of salivary hypofunction and attendant medical conditions. Based on comprehensive oral medicine, dietary analyses and advice, it would seem patients need self-management plans to deal with incipient chronic tooth wear. The alternative is the expensive treatment of pain, occlusal damage and pulp death required to repair the effects of acute severe tooth wear.

Key words: Asthma, caffeine, cardiovascular, diabetes, gastro-oesophageal reflux, tooth wear.

(Received for publication February 2001. Approved March 2001.)

### INTRODUCTION

Tooth wear in Australia today is a totally different problem from that which, before European colonisation, affected the aboriginal population.<sup>1</sup> Dental erosion is now the most important clinical presentation, albeit that attrition and abrasion continue to contribute to the characteristics of tooth wear lesions. The aim of this review is to illustrate how both the correct diagnosis and management of most patients with excessive tooth wear require oral medicine insights. These insights have emerged from the investigation of 500 patients diagnosed by structured interview and clinical examination, managed by diet analysis and advice and by salivary stimulation with chewing gum, prior to

restorative treatment or occlusal therapy. Various aspects of aetiology, risk factors, pathogenesis and clinical features of the tooth wear in subgroups of these southeast Queensland patients have been reported.<sup>2-10</sup>

The risk factors in the 500 subjects' histories were reviewed to determine whether they had significant lifestyle-linked dehydration,<sup>2</sup> had a systemic condition or medication which predisposed to xerostomia<sup>11</sup> or had no risk factor. Dehydration, the commonest cause of salivary dysfunction,<sup>12</sup> was cited as the risk factor for 60.2 per cent of the sample. Systemic conditions or medications were identified in 24.4 per cent and only 15.4 per cent of the sample were found to have had no risk factor identified. Of the 60 per cent of subjects suffering dehydration, 35.2 per cent were 30 or younger (Fig 1). However, systemic conditions were more frequently found in older subjects (18.8 per cent). The greatest proportion of subjects (45.2 per cent) were otherwise healthy males suffering work- or sports-related dehydration. Of the 24.4 per cent of subjects with systemic conditions/medications predisposing to xerostomia, 12 per cent were women aged 30+ compared to 6.8 per cent males in this age group. Of the 15.4 per cent of subjects with no identifiable risk factor, the largest gender group, 6.4 per cent, were younger male subjects. It must be emphasised that these data are for a highly selected group of patients, referred with excessive tooth wear.

### At-risk groups

Analyses of the patients' lifestyles reveals there are six broad groups in southeast Queensland at risk of developing dental erosion and tooth wear. These groups are detailed in Table 1.

The first five of these groups are illustrated by case histories of patients from southeast Queensland. The sixth group has been reported elsewhere.<sup>10</sup> The cases document the wide range of complaints, symptoms and signs Australian dentists encounter in patients with excessive tooth wear. In order to address not only the source of acid but also the consequences of lack of salivary protection, dentists need to understand why each patient is at risk of developing dental erosion and incipient tooth wear. Understanding each patient's case requires a detailed dental and medical interview and an

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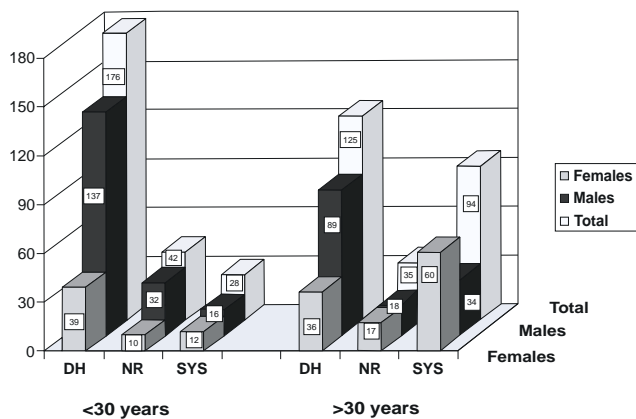


Fig 1. Frequencies with which 500 patients were at risk of excessive tooth wear from sports- or work-related dehydration (DH) from a systemic condition or medication associated with xerostomia (SYS) or from no identified risk factor (NR). Total <30 years compared to total >30 years.

examination that typically takes one hour. Referrals to oral medicine and pathology specialists may be useful.

Analysis of study models of the dentition concerning the site-specificity of the lesions of dental erosion is essential. The sites at which the lesions of dental erosion occur most commonly are those least protected by serous saliva from the parotid and submandibular glands, especially under conditions of reduced salivary flow.<sup>8</sup> Although dentists may not have the time to estimate resting or stimulated salivary flow rate, oral clearance or pH, they are expert at recording sites of wear from study models. The teeth bear the record of lifestyles with reduced salivary protection against damage by acids and thus can be analysed accordingly.<sup>7</sup> Analyses of study models of these subjects have shown their teeth are worn mostly by erosion, which softens the teeth, making them prone to wear by attrition, possibly from bruxism<sup>5</sup> and also by toothbrush abrasion. Thus diet analysis and advice and salivary stimulation for the protection and remineralisation of teeth must precede the restoration, or occlusal analysis and rehabilitation of lesions.<sup>11</sup> Only then can the patient be given the insights to manage their incipient tooth wear and avoid pain, occlusal damage, pulp death and the extensive treatments required for acute dental problems.

## CASE HISTORIES

### Group 1

For approximately 10 years before reporting for treatment, 28 year old male patient 'A' noticed the appearance of his upper central incisors had changed. The subject's upper anterior and lower posterior teeth were intermittently sensitive to hot and cold stimuli. Ten months previously, tooth 37 was restored, but 'never felt right' and eight months later, emergency root canal therapy was performed to resolve a dentoalveolar abscess. As a landscape worker, 'A' clenches his teeth when straining doing stonework. His former girlfriend complained 'A' ground his teeth at night; although 'A's' muscles and joints were not tender on waking or on palpation. Tongue indentations and linea alba of buccal mucosa were absent. No previous diagnosis or treatment for bruxism has been made. 'A's' work and hobby of working on car-bodies subjected him to abrasive dusts and the fumes of the hydrochloric acid he used to clean cement. His toothbrushing habits were not abrasive – 'A' did not use dental floss or a mouthrinse, but up until four years before presenting, he chewed gum regularly. Five years before presenting, 'A' drank as much as six litres of regular cola a day, saying he was addicted to this beverage. 'A' drank an average 12 beers a week and consumed one to two whiskies, with cola or ginger ale, when drinking spirits. 'A' continues to drink a variety of soft drinks on a regular basis but consumes less cola and no sports drinks.

### Medical history

'A's' general health is good, but he is frequently dehydrated when sun-exposed, working outside all day and suffering from a dry mouth daily. 'A' played rugby league for 10 years until the age of 20. During that time, for rehydration, cola beverages were used, both at work and after sports. Twelve months ago, 'A' experienced gastric discomfort and vomiting which was diagnosed as a bout of acute alcoholic gastritis, for which no medication was given. Otherwise, 'A' has had only occasional heartburn, but no reflux.

**Table 1. Groups of patients at risk of developing excessive tooth wear.**

Group 1	Healthy, active people whose sports or workplace dehydration reduces salivary protection of the teeth against acids in sports or other soft drinks. Addiction to caffeine in cola beverages.
Group 2	Patients with anxiety states, depression, anorexia or bulimia nervosa on tranquillising or antidepressant medications associated with xerostomia and reversible sialadenosis. The medication results in loss of salivary protection against both acid soft drinks and intrinsic acid vomiting.
Group 3	Patients with oesophagitis from GOR sometimes associated with alcoholism. Alcohol is a dehydrating drug that has long-term effects on salivary glands. Erosion is produced both by extrinsic acids in wines and spirits with mixes and by intrinsic acid from GOR.
Group 4	Asthma sufferers at risk either from the acidity of medications or from reduction in salivary flow induced by medications. Asthmatics' lifestyles and diet may include the risk factors of Groups 1 and 2.
Group 5	Diabetics and patients with other cardiovascular diseases suffer reduced salivary protection of the teeth as a result of their condition or from antihypertensive or diuretic medication.
Group 6	Patients with syndromes which may be genetic, epigenetic or iatrogenic which place them at risk of dental erosion by affecting salivation.

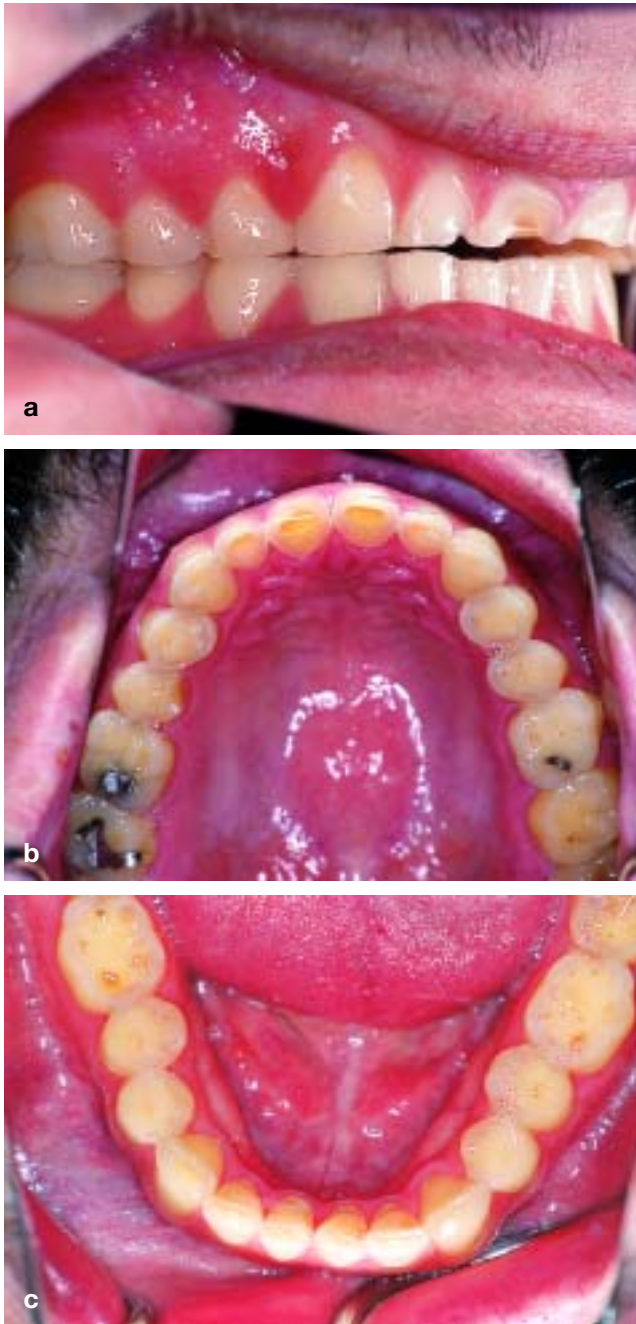


Fig 2. Group 1: A healthy 28 year old male with sports- and work-related dehydration and a caffeine addiction to cola beverages. (a) Loss of facial enamel and anterior vertical dimension; (b) loss of incisal and palatal enamel from maxillary incisors premolars and first molars; and (c) cuspal cupped lesions on mandibular premolars and molars.

#### Examination

Loss of incisal height and facial enamel were particularly evident on the maxillary incisor and canine teeth (Fig 2a). The maxillary incisors, premolar and first molar teeth had lost incisal cuspal and palatal enamel by moderate erosion (Fig 2b). The mandibular incisors and canine teeth had mild incisal attrition, however their lingual surfaces were stained and there was supragingival calculus (Fig 2c). Cuspal cupped lesions were evident on all mandibular premolar and molar teeth. The second molars were particularly affected and the restoration of the access cavity,

through which the left second molar had been root canal-filled, was surrounded by an area of cuspal erosion. Buccal shallow non-carious cervical lesions were present on maxillary and mandibular premolars (Fig 2a). This cervical pathology was associated with occlusal erosion on the same teeth.

Actinic cheilosis and melanosis were evident on the lower vermilion border. The tongue and oral mucosa were normal. However, the mouth was dry and the saliva stringy and frothy. Both parotid glands were slightly prominent and readily palpable but not tender or lumpy. The submandibular glands were readily palpable and slightly firm but not enlarged. A left submandibular node was tender. No cervical lymph nodes were palpable. The temporomandibular joints and muscles of mastication had no signs or symptoms of dysfunction.

#### Comment

This case illustrates the predominant group of subjects seen in this study (healthy young men with work and/or sports-related dehydration). In southeast Queensland, dehydration is the commonest risk factor for loss of salivary protection against the acids in the soft drinks and cola beverages which cause dental erosion.<sup>2,3</sup> Dehydration reduces the salivary flow rate and the ability, with its buffering capacity, to clear acids from the mouth.<sup>12</sup> It has been demonstrated that dehydration causes significant decreases in parotid salivary output<sup>12-13</sup> in healthy young and older subjects.<sup>14-15</sup> Therefore, particularly in climates such as those of Australia, a history of work and/or sports-related dehydration assumes considerable importance in the diagnosis of patients with tooth wear. Unfortunately, none of the metabolic indicators of hydration status have been found to be accurate predictors of salivary flow rates.<sup>16</sup>

The distribution of lesions in 'A's' mouth is common in this group. Facial, incisal and palatal lesions are found on maxillary incisors, canines and premolars.<sup>8-9</sup> The maxillary molars are less affected than the mandibular molars, which show extensive cuspal cupped lesions.<sup>4,7</sup> Loss of fissural enamel with dentine hypersensitivity resulted in loss of vitality of 'A's' lower second molar. The mandibular anteriors often show attrition or erosion on incisal edges and are associated with shallow cervical lesions on their buccal aspects.<sup>6</sup> Presence of lingual calculus indicates protection by submandibular saliva.<sup>8</sup>

The mild signs of sialadenosis and xerostomia are probably secondary to the subject's dehydration or can be attributed to alcohol consumption. Actinic cheilosis and melanosis confirm the extent of 'A's' sun exposure.

Evidence continues to mount that the dental erosion produced by soft drinks and sports-drinks depends not only on their pH, but on their buffering capacity and phosphate content.<sup>17</sup> Acid drinks pose a risk when consumed in the dehydrated state, when salivation is reduced.<sup>3</sup> Diet analyses of 120 young men in this study

revealed the acid beverage mainly consumed at work or after sports was cola (unpublished). Although the caffeine content of teas, coffees, chocolate and cola beverages varies considerably,<sup>18</sup> patients whose main source of caffeine is a cola beverage put themselves at risk of dental erosion from the orthophosphoric acid (food acid 338) contained in these socially acceptable, mildly addictive beverages. Thus, to the health consequences of caffeine addiction which have been investigated,<sup>19</sup> salivary dysfunction and dental erosion should be added.

### Group 2A

The 35-year-old male subject, 'B', was found to have tooth wear when he was 29. At that time, his mandibular left molar teeth were sensitive to brushing. 'B' had lived all his life in Brisbane where there is no water fluoridation and had been given no fluoride supplement before he was 12. 'B' gave no history of bruxism, either during the day or at night, nor of muscle or joint tenderness on waking or on palpation. No tongue indentations or linea alba on buccal mucosa were found. However, a dentist had suggested 'B's' tooth wear was due to bruxism during sleep. At age 31, over a five-month period, 10 appointments were used to construct two acrylic nightguards that were inserted and adjusted over that time. However, 'B' noticed his teeth continued to be sensitive and he ceased to wear the guards. There was no indication that abrasion, either in 'B's' work as a clerk or in toothbrushing, was responsible for his tooth wear. Dental floss, mouthwash or chewing gum were rarely used by 'B' and he did not suck lemons, drink lemon juice or take vitamin C tablets but took vitamin B tablets three times daily. 'B' consumed at least one piece of fresh fruit daily (apples, bananas, kiwi fruit), although oranges gave him mouth ulcers. 'B' drank two to three glasses of soft drink daily (cola beverages, ginger ale, lemonade) and lemon or orange mineral water alternatively. 'B' did not drink wine, beer or spirits.

### General health

'B's' general health was good, until at age 32 when, due to work-related stress, he left his employment and was unemployed for the next two years. In this two-year period, 'B' attended a psychiatrist, who prescribed Prothiaden (Dothep) 200mg for anxiety and to aid sleeping. 'B' suffered day and night with a dry mouth from this medication. 'B' did not complain of heartburn, sour mouth or gastritis and gave no history of frequent vomiting or of bulimia nervosa.

### Examination

The maxillary central incisors showed facial, incisal and palatal enamel and dentine loss, restored with composite resin (Fig 3a, 3b). The mandibular incisor and canine teeth were normal perhaps due to the anterior open bite. Occlusal cupped lesions were found particularly on buccal cusps of the maxillary premolars

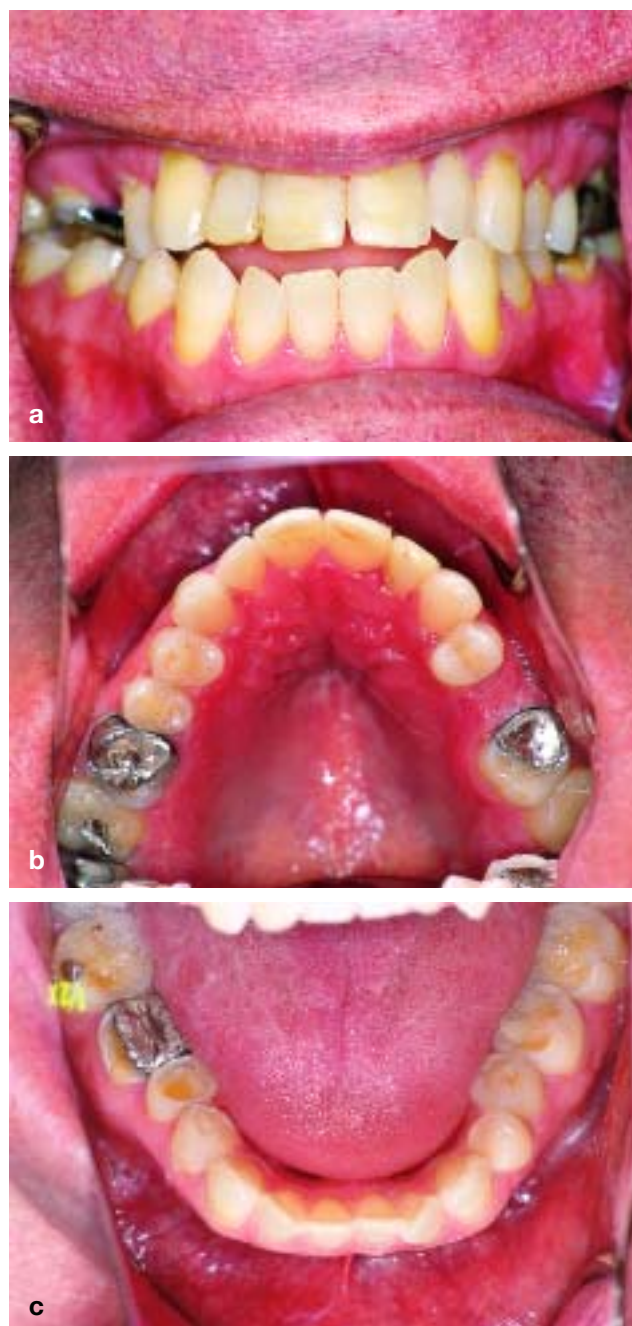


Fig 3. Group 2A: A 35-year-old male, on antidepressant medication, suspected of having bruxism. (a) Anterior teeth have restored facial maxillary lesions and no lesions on mandibular teeth due to anterior open bite; (b) palatal loss and cuspal cupped lesions on maxillary premolars; and (c) extensive cuspal cupped lesions on mandibular premolars and molars.

(Fig 3b), some of which also showed shallow non-carious cervical lesions on their buccal surfaces (Fig 3a). The mandibular premolar and molar teeth were particularly affected by extensive occlusal cupped lesions with high margins on amalgam restorations (Fig 3c).

The tongue and oral mucosae were normal but the saliva was ropery. Both parotid glands were prominent, slightly firm and readily palpable but not tender or lumpy. Both submandibular glands were enlarged slightly and firm but compressible with no tenderness (sialadenosis). A slightly firmer lump in the distal pole



Fig 4. Group 2B: A 19-year-old female suffering from bulimia nervosa. (a) Thinned and chipped incisal edges of maxillary incisors and arrested cervical caries on mandibular anterior teeth; (b) palatal aspects of the maxillary anterior teeth showed dentine exposure and chalky cervical enamel; (c) incisal edges of mandibular incisors were cupped and chipped, cuspal cupped lesions were evident especially on all cusps of the first and second molar teeth; and (d) lingual aspects of mandibular teeth were not involved and free of plaque or calculus, white plaque on the dorsum of the tongue was *Candida albicans*.

of the left gland was interpreted as an inflamed lymph node, although all other cervical nodes were normal. There were no signs or symptoms of pathology in temporomandibular joints or muscles of mastication.

#### Comment

Despite the lack of signs or symptoms of para-functional habits and the anterior open bite, this case illustrates the tendency to diagnose and treat bruxism preferentially in tooth wear patients.<sup>5</sup> The greater extent of cuspal cupped lesions on enamel and dentine of the mandibular premolar and molar teeth than in the maxillary teeth should alert the dentist that erosion has caused the lesions. The site-specificity of erosion lesions in patients on antidepressant medication does not appear to be different from that found in Group 1. The source of the acids was clearly the soft drinks taken to help the symptoms of dry mouth induced by the antidepressant medication, which also was responsible for the sialadenosis. Drug-induced xerostomia is a well established risk factor determining the occurrence and progression of dental erosion.<sup>20</sup> Viscous ropey saliva and swelling of the salivary glands were two important signs of salivary dysfunction observed in 'B'.

Medication with tricyclic antidepressants has been found to reduce salivary flow rate significantly.<sup>21-22</sup>

Drug-induced sialadenosis is apparently reversible, as it reduces on withdrawal of the drug.<sup>23</sup>

#### Group 2B

A 19-year-old woman, 'C', presented with loss of tooth structure and concern about hypersensitivity and the aesthetics of her teeth. 'C' admitted to being bulimic, her history revealing bingeing on fast foods such as hamburgers, chocolates and soft drinks, followed by induced vomiting. 'C' had been vomiting at least twice a day for the last four years. 'C' is a professional dancer and maintained this habit in an attempt to remain slim. The subject's oral hygiene was assessed as fair to poor – 'C' brushed her teeth once a day and did not use floss.

#### General health

When 'C' was 14, she consulted her general medical practitioner for treatment of amenorrhoea caused by her intense training at ballet school. At that time, examination revealed a slim athletic girl who was underweight for her age. In order to stay slim, 'C' had resorted to a diet low in fats and proteins but high in carbohydrates. 'C's' occasional binges were followed by self-induced vomiting twice a day. A faint heart murmur was detected and was ascribed to mild cardiac arrhythmia. A deficiency of potassium ( $K^+$ ) in the blood

was found to be the cause of the arrhythmia. Treatment was 80-120mEq potassium/day taken orally as a 10 per cent solution of potassium chloride in orange juice, particularly when 'C's' blood potassium fell below normal. 'C' attended counselling sessions for anorexics but these did not stop her bulimia.

#### Examination

The incisal edges of the maxillary incisors were thinned palatally and chipped (Fig 4a). Dentine was exposed on the palatal surfaces of these incisors and on the maxillary canines. The palatal cervical enamel was chalky (Fig 4b) and the mandibular incisors and canines showed incisal cupping (Fig 4c). Facial arrested carious cervical lesions, dark brown with borders of chalky enamel, were evident on the mandibular incisors, canines and premolars (Fig 4a, 4c). Cuspal cupped lesions were found on all buccal cusps of all premolar teeth, on mesiopalatal cusps of first maxillary molars and especially on all cusps of the mandibular first and second molar teeth (Fig 4b, 4c). The lingual aspects of mandibular teeth were normal and clean with no plaque or calculus (Fig 4d).

The oral mucosa were normal except for a small white pseudomembranous plaque found on the mid-dorsal surface of the tongue (Fig 4c). Histologically, pseudohyphae of *Candida albicans* were identified in an oral smear.

The parotid glands were moderately prominent, slightly firm and readily palpable but not tender or lumpy. Lanugo was present over the parotid areas. The submandibular glands were also enlarged, palpably firm but not tender or lumpy. The facial and cervical nodes were not painful or palpable. The temporomandibular joints and muscles of mastication were normal.

#### Comment

This case illustrates that in patients with eating disorders, the effects of these disorders on the hormonal states, salivary glands and the immune system predispose to dental caries, erosion and oral candidiasis. Dental caries and erosion are rarely active simultaneously in the same mouth, presumably as *Streptococcus mutans* cannot metabolise sugars at the low pH associated with dental erosion.<sup>24</sup> However, patients with eating disorders have significantly higher caries frequencies, very high *mutans streptococci* counts and very low unstimulated salivary flow rates.<sup>25</sup>

The distribution of the lesions of dental erosion, essentially incisal and palatal on maxillary anteriors and occlusal and buccal cervical on mandibular posteriors, did not necessarily indicate gastric acid regurgitation. Similar distribution of lesions can be found in non-acid regurgitators as in cases 1 and 2A. However, erosion of the lingual aspects of mandibular anterior teeth is found uniquely in chronic gastro-oesophageal reflux and in older bulimic patients.<sup>9</sup> This damage is not yet evident in 'C' (Fig 4d).

A retrospective study of 400 southeast Queensland patients identified nine self-confirmed bulimic subjects (0.02 per cent).<sup>9</sup> In western industrialised countries, the prevalence of bulimia nervosa has been estimated at 5 per cent among young women age 20-30.<sup>26</sup> In bulimic patients, on low acid diets, erosion lesions are initially confined to palatal and occlusal surfaces of maxillary teeth. However, bulimics who consume larger quantities of acid drinks, especially cola beverages, develop palatal and buccal erosions.<sup>25</sup> Accordingly, the initial lesions of bulimic subjects cannot readily be discriminated from those of non-bulimic subjects who drink cola beverages. However, erosive lesions on the lingual aspects of mandibular anterior teeth are found uniquely in some bulimics and some cases of chronic gastro-oesophageal reflux.<sup>9</sup>

In bulimic subjects, sialadenosis and xerostomia are of major importance. Sialadenosis and xerostomia can be attributed to medication, hormonal effects or from the effects of altered blood-ion metabolism secondary to vomiting.<sup>27</sup> Hypokalaemia in girls 'C's' age is most commonly due to prolonged absence of K<sup>+</sup> intake; from dietary fads, chronic laxative ingestion or chronic vomiting, as in bulimia and anorexia nervosa. More dental erosion has been found in bulimic subjects with salivary hypofunction than in bulimics with normal salivation.<sup>28</sup> Moreover, the source of acids producing erosion may not only be intrinsic; for extrinsic acids, cola beverages and orange juice in the potassium medication could have contributed in this case. Accordingly, patients with eating disorders should have regular dental checkups and adopt preventive programs tailored to their at-risk status.<sup>25</sup>

#### Group 3

Subject 'D', a 60-year-old man, had tooth wear identified 10 years previously. No teeth were sensitive and 'D' had no history, signs or symptoms of bruxism nor any history of tooth abrasion. 'D' regularly brushed his teeth twice a day with a non-fluoride toothpaste, with predominantly horizontal motions with his right hand, although he was ambidextrous. 'D' did not use dental floss, mouthwash or chewing gum. Amalgam restorations were placed many years ago and while a mandibular partial prosthesis had been provided at least 20 years before, 'D' rarely wore it.

For health reasons, 'D' consumed no lemon juice, vitamin C chewable tablets or apple cider vinegar. 'D' preferred apples, bananas, pineapple and oranges to grapefruit and lemons but not as freshly squeezed juice. 'D' regularly drank two glasses of cola beverage every second day and four cups of tea and four cups of coffee daily. Until age 56, 'D' drank beer heavily from 4.30-10pm every day. This practice ceased when he retired.

#### Medical history

'D' suffered mastoiditis as a child, leading to partial deafness that affected his learning. 'D' was given a hearing aid at 55 and at that time he learned to read

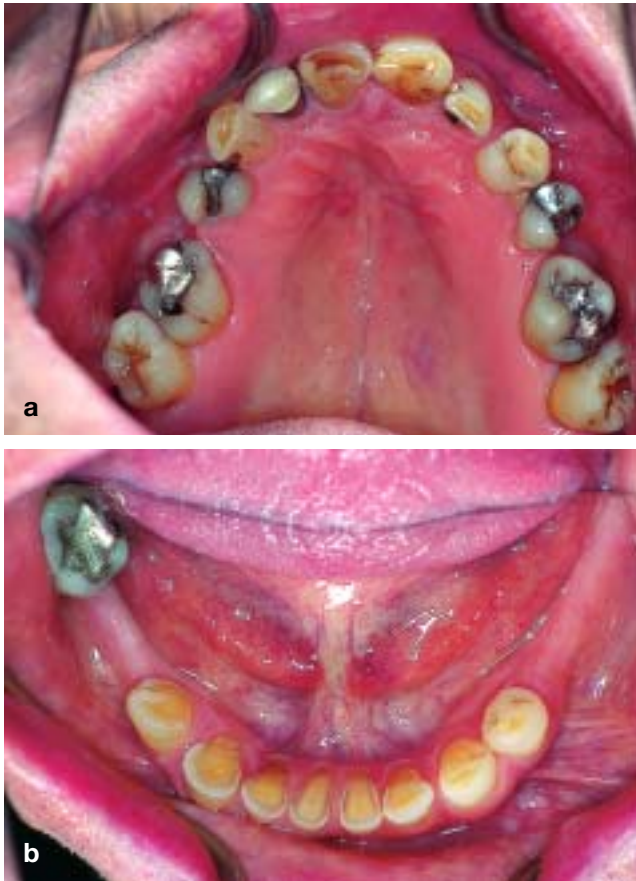


Fig 5. Group 3: A 60-year-old man with gastro-oesophageal reflux. (a) The maxillary anterior teeth had facial and incisal lesions, one lateral incisor was crowned and one canine unerupted; (b) mandibular anterior teeth had lost lingual enamel from the incisal edges to the gingivae, sublingual folds were prominent, erythematous and flabby.

and write. 'D's' health was good and he played rugby and cricket in his teens. From the age of 15 until his retirement at 55, 'D' worked as a general labourer and welder in foundries, manufacturing steel preforms for concrete construction. This work was very hot, sweaty and dehydrating and, when he was 54, 'D' woke with a throat so dry he could hardly talk and was suspected of having throat cancer. 'D' has controlled his heartburn with a fat free diet ever since but every morning has had a dry mouth and throat and suffers gastric reflux about twice a week. At 56, a hiatus hernia was diagnosed. Gastroscopy performed when he was 59 revealed no peptic ulcers but chronic gastritis, for which one tablet of Losec daily is prescribed.

#### Examination

The maxillary incisor and canine teeth showed facial and incisal, but not palatal, lesions of dental erosion (Fig 5a). One lateral incisor was crowned with porcelain-faced gold and one canine was unerupted. A cuspal cupped lesion was found on one first premolar, the other maxillary premolar and molar teeth were unaffected by tooth wear. All of 'D's' mandibular incisor and canine teeth showed loss of lingual enamel from their incisal edges extending to the lingual gingivae (Fig 5b). Cuspal cupped and lingual shallow

cervical lesions were present on the first premolars. One remaining molar was restored with amalgam but lingually was unaffected by erosion. The tongue and oral mucosa were normal, with the exception of the sublingual folds which were prominent and erythematous. Palpation of the parotid, submandibular and sublingual glands found them flabby, not firm, tender or lumpy, but palpable. The prominence of the parotid glands had obliterated 'D's' preauricular creases.

#### Comment

This lifestyle of hard physical labour in a dehydrating occupation has placed 'D' at risk of alcoholism, gastro-oesophageal reflux and the rarest form of dental erosion, erosion on the lingual aspects of anterior mandibular teeth (Fig 5b).<sup>9</sup> Chronic alcoholism is associated with greater tooth wear especially as erosion on the palatal surfaces of maxillary anterior teeth.<sup>29</sup> Moreover, dental erosion in alcoholics has been found to be associated with a long history of continuous alcohol abuse rather than with episodic binges of vomiting.<sup>30</sup>

The connections between tooth erosion and gastro-oesophageal reflux are by no means simple, for no changes were observed in oral pH in a total of 339 acid reflux episodes when oral and oesophageal pH were compared, even when the subjects were supine.<sup>31</sup> Significantly more of the patients with erosion had low salivary buffering capacity compared to the controls. Although extended periods of pH 4-5 were measured in the patients' mouths, these periods were not connected to the episodes of gastro-oesophageal reflux.<sup>31</sup> Several studies have considered the correlation of gastric acid reflux at different levels of the oesophagus with oral reflux and drop in pH.<sup>32-35</sup> The general conclusions of these studies is that palatal erosion is associated with chronic gastric reflux and that spicy meals eaten in the evening are possible causes of oesophageal reflux.

Insufficient attention has been paid to the possibility that although the pH of most beers is not low enough to produce dental erosion, both their alcohol and tannin content probably have effects on the salivary glands that ultimately result in the replacement of gland tissue by fat, hence subject 'D's' xerostomia, flabby glands and elevated sublingual folds.<sup>36</sup>

Subject 'D' was diagnosed with heartburn, gastric reflux, chronic gastritis and a hiatus hernia and the obvious assumption to make is that gastric acid was the main aetiology of the erosion on the lingual of his mandibular incisors. Of the 400 southeast Queensland subjects, 21 patients (0.0725 per cent) with gastro-oesophageal reflux disease (GORD) were identified. A case-control study showed lingual erosion on mandibular anterior teeth was found exclusively in the GORD cases and that minor lingual erosions could be detected in some bulimia cases.<sup>9</sup> Lingual erosion was only detected in 0.0725 per cent of all patients and was the rarest form of dental erosion. This is presumably because normally the major submandibular and

sublingual glands, and possibly the glands of Nunn-Blandin on the ventral of the tongue,<sup>37</sup> efficiently protect against all acids. However, in patients whose salivation is affected by work-related dehydration or alcoholism, these teeth are less well protected against gastric acid. Furthermore, the treatment of 'D's' GORD with the proton pump inhibitor Losec may further compound his dental erosion, as such drugs reduce salivation.<sup>11</sup>

Consequently, the diet analysis and advice given to 'D' must address not only the lifestyle-induced damage to his teeth but also the damage to his oesophagus and stomach. Normal saliva and mucus protect not only the teeth but also the oesophagus and stomach from irritants such as alcohol and tannins.<sup>38-39</sup> The value of salivary stimulation in the treatment of patients with GORD is only gradually being appreciated.<sup>40</sup>

#### Group 4

Severe tooth wear, noticed by a school dental nurse, led to an 11-year-old boy, 'E', being suspected as having amelogenesis imperfecta. 'E's' teeth felt sensitive after acid drinks and ice cream. In the few months before detection, 'E' could only chew minced beef and his teeth were sensitive to brushing. 'E' had no signs or symptoms of parafunctional habits or any indication of abrasion from toothbrushing or other sources. 'E' had a preference for regular cola beverages, as much as six glasses a week, and also drank chocolate milk on a daily basis.

'E's' father reportedly had no enamel on his teeth but his mother's teeth were normal. One brother, age 13, was asthmatic and was found to have severe dental erosion. 'E's' sister, age 12, had moderate dental erosion but was not asthmatic. No form of amelogenesis imperfecta was detected on the tooth enamel of any of 'E's' siblings.

#### Medical history

Subject 'E' has suffered severe asthma since he was two and uses Ventolin spray daily and Becatide twice daily, with prednisolone being administered for severe attacks. Although 'E's' asthma is sometimes exercise related, he had one training session of little athletics daily with water for refreshment afterward. 'E's' asthma was most frequent in dry, smoky weather and came on mostly at night.

Apparently, 'E' had gastric reflux when he was younger, but he reported this was no longer a problem. However, 'E' suffered dry mouth and lips.

#### Examination

The facial aspects of 'E's' maxillary and mandibular incisors were normal but not plaque-free (Fig 6a). The palatal aspects of the maxillary incisors showed thinned enamel and some dentine exposure (Fig 6b).

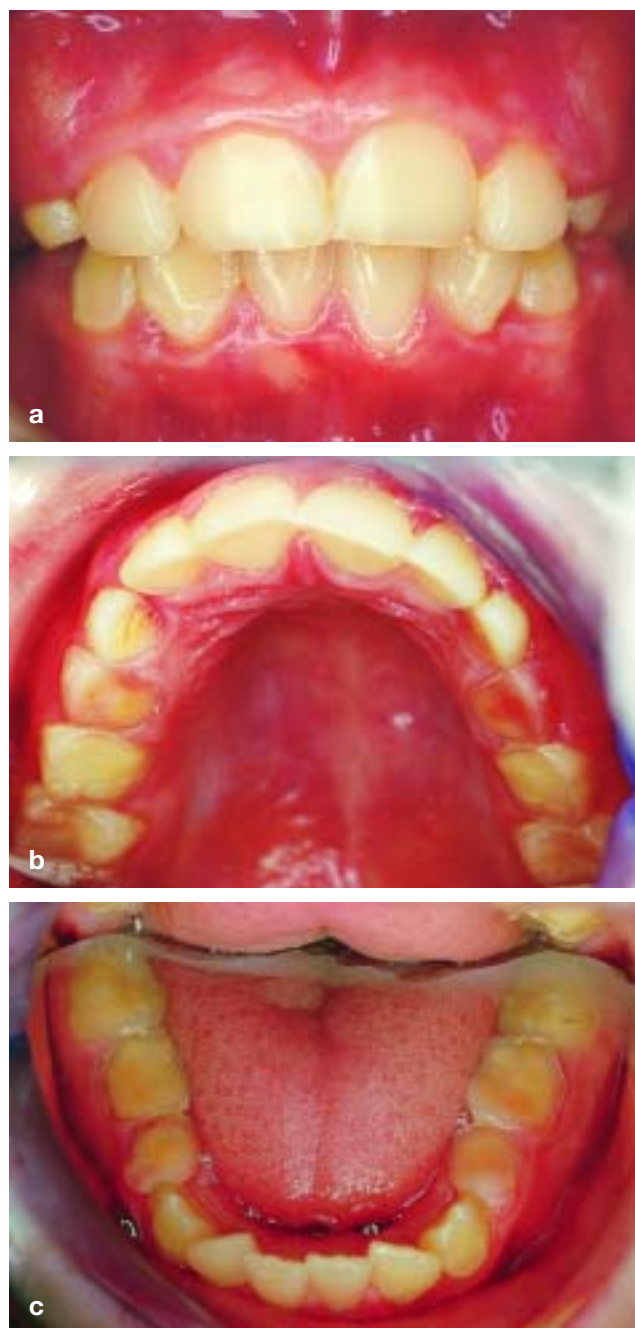


Fig 6. Group 4: An 11-year-old boy who had asthma since he was two. (a) Facial aspects of the permanent incisors were normal but not plaque-free, marginal gingivae were slightly hyperplastic; (b) enamel and dentine were lost from the palatal aspects of incisors, deciduous canines, molars and the first permanent molars in the maxilla – red patches of physiological resorption shine through the worn occlusal surfaces of deciduous molars; and (c) occlusal surfaces of deciduous and first permanent molars showed loss of buccal, cuspal and fissural enamel and dentine, the permanent incisors were normal.

Loss of occlusal and palatal enamel was marked on the maxillary deciduous canines and molar teeth. Erosion was also found on the palatal aspect of the palatal cusps of teeth 16 and 26. The mandibular deciduous molars showed occlusal and buccal loss of enamel and the lingual aspects of the mandibular teeth were normal. Red patches on dentine denoted physiological resorption from shedding of several deciduous molar teeth (Fig 6b). The occlusal surfaces of the mandibular deciduous and first permanent molars

showed cuspal cupped lesions and loss of buccal and fissural enamel (Fig 6c). No carious lesions or restorations were found.

The marginal gingivae showed mild hyperplasia of interdental papilla which bled on probing and were moderately inflamed in association with supra-gingival plaque. The other oral soft tissues were normal. The parotid and submandibular glands were slightly enlarged but fleshy and lumpy to palpation, but were not tender. No cervical nodes were palpable. The temporomandibular joints and muscles of mastication showed no signs or symptoms of dysfunction.

Orthopantomography revealed all premolar and second permanent molar teeth were normal in crown form and that their root development proceeded normally for his age. With the exception of one maxillary third molar, the third molar tooth germs were absent. Enamel thickness or radiopacity was normal and none of the unerupted teeth showed coronal resorption. Moderately dense radiopacities were seen within the maxillary antra.

#### Comment

Subject 'E', an asthmatic 11-year-old, has loss of salivary protection of his teeth against dental erosion from dietary and possibly gastric acids as a result of medications taken since he was two. The extent of dental erosion in the deciduous dentition is readily evident. Moreover, the loss of palatal and occlusal enamel on permanent molar teeth illustrates the process is active and continuing. It is not known if there is a site-specific pattern for the lesions of dental erosion in asthma sufferers.

In a retrospective analysis of 423 patients in south-east Queensland, 43 patients (10 per cent) were asthma sufferers (unpublished). The prevalence of asthma in Australian children is approximately 10 per cent.<sup>41</sup> However, several studies have found that asthma is a risk factor for both dental caries and erosion.<sup>42-43</sup> The increased risk of dental caries has been associated with the effects of asthma medications, principally the  $\beta_2$ -adrenoreceptor agonists, on salivation.<sup>44-48</sup> Young asthmatics are also at risk of dental erosion caused by their medications.<sup>49</sup>

One secondary effect of drug-related xerostomia is that asthmatic children are found to be thirstier than their non-asthmatic siblings.<sup>42</sup> Increased consumption of soft drinks, to wash away the taste of the medications, has been noted.<sup>43</sup> Some medications used to control asthma are themselves acidic.<sup>49-50</sup> It is of interest that, of three children in this family, the youngest was not asthmatic but suffered erosion as a result of family dietary habits established to cope with his siblings' dehydration.

The effect of chewing gum on the dental plaque pH of asthmatics has been investigated at intervals after various medications.<sup>51</sup> Plaque pH fell after medication to levels at which enamel demineralisation could occur (less than pH 5.5). Chewing gum restored the pH to

above 6.3. The study concluded that children with bronchial asthma, treated with  $\beta_2$ -adrenoreceptor agonists, should receive special caries-preventive attention.<sup>51</sup>

The frequent coexistence of asthma with GOR is well established.<sup>52</sup> The estimated prevalence of GOR in asthmatics is between 50-60 per cent in children and 60-80 per cent in adults. Four factors which may promote GOR in asthmatics are the use of bronchodilators, adopting the supine position, overeating and the presence of hiatus hernia.<sup>52</sup> However, GOR in asthmatics is not a result of bronchodilator-induced smooth muscle relaxation of the lower oesophageal sphincter. Nonetheless, when asleep in the supine position, asthmatics have significantly more acid reflux than non-asthmatics. It is also believed that inhalation of even small quantities of gastric acid can set off the symptoms of asthma.<sup>53</sup> However, it has not been resolved whether the pattern of dental erosion in asthmatics is due to extrinsic dietary acids or relates more to intrinsic acid from GOR. However, in the dental and dietary management of asthmatic children with excessive tooth wear, GOR must be taken into account.<sup>53</sup>

#### Group 5A

Fifteen years before presenting for treatment, 27 year old 'F' noticed his teeth had started to 'fall apart' – his front teeth became shorter and he panicked. 'F's' teeth had been sensitive to cold and sweet stimuli and, because his back teeth had been sore, he tended to chew on his front teeth. In addition, 'F's' gums bled sometimes even when eating a banana. There was no family history of a similar complaint. 'F' had lived in Brisbane most of his life and had no fluoride supplementation. From the age of three months, tetracycline antibiotics were prescribed for respiratory problems. These antibiotics were discontinued when 'F' was two. 'F's' teeth have always been discoloured.

Although 'F' clenches his teeth when concentrating, he does not believe he grinds his teeth at night. 'F's' occupation as a high school teacher does not expose him to industrial abrasives or acids. As a dentist had once suggested that paste was abrasive, 'F' brushes his teeth, without toothpaste, for two-three minutes at a time, with a medium toothbrush. His brushing was a vigorous scrubbing action and a toothbrush lasted only two-three weeks. 'F' used a small amount of fluoride toothpaste after brushing as a mouth/breath freshener. He also used Ultrafresh spray and occasionally an obtundent toothpaste (Sensodyne).

'F' drinks a lot of sugar-free diet cola (currently one litre/day) and diet lemon drink when he is dehydrated. Prior to being diagnosed with diabetes mellitus, 'F' preferred regular sugar-containing cola. 'F' has suffered from mild gastric reflux but not heartburn and vomited infrequently. 'F' did not suck lemons and drank tea and coffee in moderation.

Subject 'F' suffered from scoliosis, which was corrected by an operation when he was 19. The



Fig 7. Group 5A: A 27-year-old man diagnosed at 22 with diabetes mellitus. (a) Chipping and thinning of enamel and reduction in height evident on the maxillary incisors, shallow cervical lesions present on canines and premolars; tetracycline staining accounts for grey lines on lateral incisors, canines and premolars; and (b) loss of incisal edges, cusps and palatal surfaces of maxillary teeth were severe, and margins of all restorations were high.

operation required a blood transfusion. Diabetes mellitus was diagnosed when 'F' was 22 and prior to that diagnosis he had insatiable thirsts. 'F' controlled his diabetes by injection, as required, for four hours with 14 units of short-acting insulin (Velosulin) and 10 units of slow release Insulatard with maximum effect for nine hours. If 'F' neglected to inject insulin, he became dehydrated and drank a lot as the subjective symptoms of dry mouth increased. 'F' suffered nausea, headaches and lethargy. 'F's' weight fluctuates – he can gain and lose 4-5kg rapidly over a few days – but his weight is relatively stable at 64kg. 'F' is the only diabetes sufferer in his family.

#### Examination

Chipping and thinning of the incisal edge and marked loss of palatal enamel and dentine were visible on maxillary incisor, canine, premolar and anterior molar teeth (Fig 7a, 7b). Past resin and amalgam restorations on several teeth had elevated margins. Moderate plaque and calculus were present, particularly on the lingual aspect of the mandibular incisors, and crowding of the incisor-canine region was evident. Incisal loss of enamel and dentine on the maxillary incisors gives rise to an open bite but an edge-to-edge

occlusion could be achieved by the patient (Fig 7a). Tetracycline staining was most apparent on the canine teeth (Fig 7a) and his tongue and oral mucosa were normal. The parotid glands were not palpable, swollen or tender and normal salivary flow was elicited. The submandibular glands were palpable but not firm. Facial nodes bilaterally were firm but not enlarged or tender and no cervical nodes were palpable. There was slight tenderness on palpation of the left temporomandibular joint but no tenderness, clicking or deviation on opening was found.

#### Comment

The profound effects of diabetes mellitus on the lifestyle of 'F' placed him at considerable risk of developing dental erosion. 'F's' thirst and a craving for sugar initially resulted in an increased consumption of regular cola. Although diet cola does not contain sucrose, its pH has been tested at 3.45 compared to 2.40 for regular cola.<sup>54</sup>

The pattern of dental erosion on 'F's' dentition does not appear to be linked to diabetes mellitus in a site-specific manner. This pattern may be indistinguishable from that of severe dental erosion in healthy dehydrated subjects drinking cola beverages. Nor does the pattern appear to be related to the linear pattern of 'F's' tetracycline stained teeth. With the exception of bleeding on probing of the gingivae, the oral soft tissues were not affected by 'F's' diabetes. The saliva was apparently normal in quantity and in texture and the major salivary glands were not enlarged. However, tests of salivary flow rate were not performed. After the diagnosis was made at age 22, acidic soft drinks continued to be drunk. Patients whose diabetes is difficult to control have to be more concerned with control of their blood sugar than with the prevention of dental decay or erosion. One year after examination, 'F' had all of his remaining teeth extracted and prostheses placed.

Diabetes insipidus sufferers' excessive intake of acidic juices and soft drink plays a pivotal role in producing dental erosion.<sup>55</sup> Diabetes mellitus may similarly affect patients' drink-selection, ultimately adversely affecting their oral health. 'F' experienced xerostomia, the effect of juvenile onset diabetes type 1 on salivary function has not been well characterised.<sup>56</sup> However, patients with poorly controlled type 2 diabetes mellitus have lower stimulated parotid flowrates than patients with well controlled diabetes and non-diabetic control subjects, although no significant differences in xerostomic symptoms were found in type 2 diabetes.<sup>56</sup>

No prospective study of types 1 and 2 diabetes mellitus, to determine the prevalence of dental erosion and whether diabetes mellitus sufferers are a major at-risk group, have been performed.

#### Group 5B

Subject 'G', a 60-year-old man, presented with extensive tooth wear that had been evident for at least

12 years. 'G' had occasional tooth sensitivity to cold, which he treated with an obtundent toothpaste (Sensodyne) but he was not experiencing this at the time of presentation. 'G' reported clenching his teeth when stressed and often bit his cheek. Unilateral linea alba was found on his right buccal mucosa but tongue indentations were absent. Although 'G' had been told by a dentist that he must grind his teeth overnight, because of the wear on them, no one had observed this, nor were his muscles or joints tender on waking or palpation.

'G' had lived in Brisbane all his life and had no fluoride supplements as a child. For 40 years he was a bricklayer – dusty, sun-exposed, dehydrating work, with exposure to abrasive carborundum saws and hydrochloric acid. 'G's' oral hygiene was good, brushing twice daily for one-two minutes with a medium bristle brush, with right handed, circular or vertical strokes, using a non-abrasive fluoride-containing toothpaste. Although 'G' smoked cigarettes until he was 50, he had never used an abrasive smoker's toothpaste or any tooth whitening procedure. 'G' did not use mouthwash, dental floss or chewing gum.

'G' consumed 10 pieces of fruit a week – apple, banana, orange or pineapple in season – as part of his fat- and meat-free diet. Fruit drinks, two litres over three days, were taken periodically for 'healing' but he did not drink or suck fresh lemons or take a vitamin C supplement. Soft drinks and cola beverages were very rarely part of 'G's' diet, although he would have five-six 10oz glasses of beer a week.

#### Medical history

'G' was generally healthy up to the age of 45, although he had an appendectomy when he was 12. 'G' played hockey and soccer at school. When 'G' was 45, he had a bilateral inguinal hernia repair. However, when he was 49, a cervical lymph node was found to be the secondary spread of a squamous cell carcinoma from the skin on his left cheek. This was treated with 24 doses of radiotherapy. Laminectomies for back pain were performed on his lower lumbar spine when 'G' was 50 and 55. Hypertension was first diagnosed when 'G' was 57. Chronic renal failure necessitated four months of dialysis and, when he was 58, 'G' had a kidney transplant. Anti-rejection therapy consisted of cyclosporin (neoral) 7.5-100, prednisolone 7.5, Sandoz phosphate 1 and Metoprolol 100. 'G' continues to take diltiazem 180 (Cardizem CD) for hypertension, Adalat (Oros) 30 for angina pectoris and 100mg aspirin daily as an anticoagulant. 'G' also takes the diuretics Frusemide 40 PRN, Simvastatin 10 and Acitretin 10, tid.

At 59, over a three-month period, 'G' experienced gastro-oesophageal reflux investigated by gastroscopy. 'G' was found to have oesophagitis, which was brought under control by a three-month course of Zantac.

#### Examination

The maxillary incisors and canines were reduced in height with loss of incisal and palatal enamel and

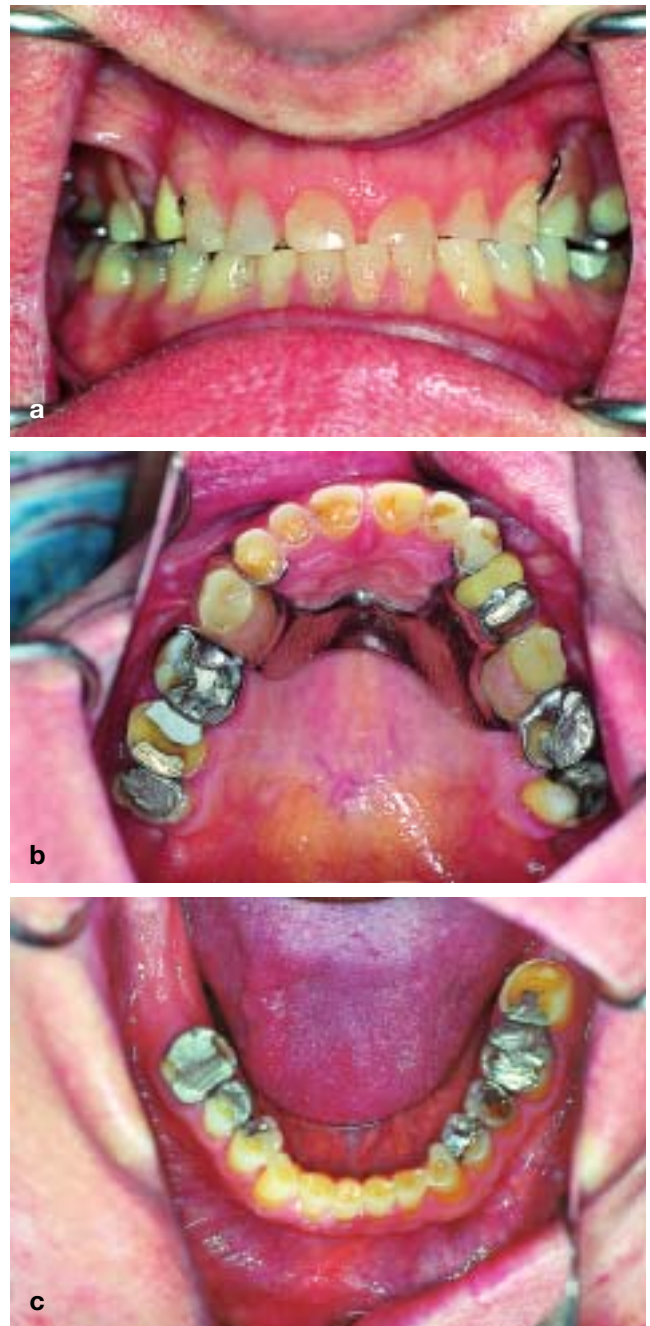


Fig 8. Group 5B: A 60-year-old man with a complex history of radiotherapy, chemotherapy and antihypertensive medications. (a) The predominant pattern is vertical loss of height of both maxillary and mandibular teeth; (b) shallow nature of the incisal lesions suggests attrition rather than erosion has been responsible for loss of maxillary tooth tissue; and (c) severe attrition evident on mandibular incisors. However, occlusal cuspal cupped lesions around the amalgam restorations with high margins denote erosion.

dentine (Fig 8a). The mandibular incisors, canines and first and second premolars showed horizontal loss of occlusal surfaces and minor shallow cervical lesions on their facial aspects (Fig 8b) but no lingual enamel loss. The remaining molars showed occlusal cupped lesions around amalgam restorations with raised margins (Fig 8c).

The gingiva and oral mucosa were normal with the exception of mild unilateral linea alba on the right buccal mucosa and mild atrophic glossitis of the anterior third of the tongue. Both the parotid and

submandibular glands were palpable but not tender and saliva could readily be expressed from the right duct but not the left. The cervical lymph nodes were not palpable, although those on the left hand side were obscured by scarring. No abnormal signs or symptoms were demonstrable in the temporomandibular joints or the muscles of mastication.

#### Comment

The attritional patterns of tooth tissue loss suggested, to 'G's' dentist, that bruxism was the cause of 'G's' tooth wear. It seems more probable that both attrition and the cuspal cupped lesions on his lower molars were the result of enamel softening by extrinsic and intrinsic acids, as previously discussed.<sup>5</sup> Subject 'G' has several potential reasons for loss of salivary protection of the teeth and the gastroesophageal mucosa. First, the radiotherapy to his head and neck has known associations with decreased salivary flow and altered sialochemistry.<sup>57</sup> Second, anti-hypertensive therapy – diltiazem enalapril and nifedipine and the diuretic frusemide – is associated with dry mouth and dehydration.<sup>11</sup> Third, after kidney transplantation, the anti-rejection therapy, utilising cyclosporin and prednisolone, has affected 'G's' salivation. All three of these therapeutic modalities are risk factors for dental erosion and are implicated in loss of salivary protection of the teeth.<sup>11</sup> The parotid glands are the most radio-sensitive of all the major salivary glands.<sup>57</sup> Importantly, they produce the greatest amount of salivary epidermal growth factor (EGF) in humans.<sup>58</sup> Concentrations of EGF in total salivary protein are reduced during and after radiation to the head and neck.<sup>59,60</sup>

The patient's beer drinking habits may also have affected both the composition of his saliva and its EGF content.<sup>61</sup> As saliva and EGF are known to protect and heal the oesophagus and gastric mucosa,<sup>62</sup> loss of salivary protection was probably responsible for the oesophageal erosion and GOR symptoms which 'G' developed when he was 59. As in the previous case, it is evident that loss of salivary protection from dehydration and excess alcohol consumption can predispose to GORD. However, in mouths unprotected by saliva, stomach acids can then attack the teeth.

#### Group 6

Although rare, the patients with syndromes associated with hyposalivation in Group 6 are at risk of developing dental erosion.<sup>10</sup> Paradoxically, children with Down's syndrome have been observed to have a copious flow of saliva; the saliva has been found to be more alkaline than the saliva of non-Down's syndrome control subjects.<sup>65</sup> As the buffering capacity of saliva is dependent on flow rate, the teeth of Down's syndrome subjects should normally have adequate natural protection against extrinsic and intrinsic acids. However, a recent case-control study of Down's syndrome subjects has found that tooth wear is more frequent and severe in the Down's syndrome subjects

than in age- and sex-matched controls.<sup>66</sup> Medications that induce xerostomia were taken by 37 per cent of the Down's syndrome subjects. Thirty-one per cent of the Down's syndrome subjects were reported to engage in tooth-grinding habits. Although the consumption of dietary acids could not be correlated with the severity of tooth wear, the incidence of gastric reflux and vomiting was apparently higher in the Down's syndrome cases.<sup>66</sup> Clearly, the dental care of patients with such syndromes should take into account that, because of diminished salivary protection, they may be at risk of developing dental erosion.

#### DISCUSSION

This review is predicated on the assumption that patients are mainly at risk of developing dental erosion because they suffer from a general systemic condition associated with salivary hypofunction or are on medications which induce xerostomia. Loss of salivary protection of the teeth against acids, extrinsic or intrinsic, predisposes to the lesions of dental erosion, attrition and abrasion. These lesions develop site-specificity, because sites of acid demineralisation are influenced by lack of the buffering capacity and clearance functions of serous saliva, derived mostly from the major salivary glands, and the capacity for remineralisation of enamel and dentine that is the property of a normal flow of whole saliva, supersaturated with respect to calcium phosphate and fluoride.<sup>8</sup> Tooth hypersensitivity was generally found in younger subjects. The lesions of dental erosion develop as cupped lesions on cusp apices, incisal edges and marginal ridges and as shallow cervical lesions in the absence of attrition or abrasion, as illustrated in the above cases. Wear of demineralised enamel at sites of tooth-to-tooth interactions results in wear facets on opposing teeth. Cervical demineralisations on enamel and dentine may be physically modified by abrasion into grooved lesions. These further develop into wedge-shaped lesions by the interaction of continuing erosion/abrasion,<sup>6</sup> with the physical presence of tracts of sclerotic dentinal tubules formed in reaction to stimuli which affect vital dentine and cause hypersensitivity.

Occlusal cupped lesions separate from well marked facets are more commonly found on mandibular molars and on second premolars than on the opposing maxillary molars and second premolars. Normally, the serous saliva from the parotids and the dorsolingual glands of von Ebner seems to be more effective in protecting the occlusal surfaces of maxillary molars. The facial and palatal surfaces of maxillary anterior teeth from the first premolar forward are common sites of cervical lesions, presumably due to the lack of buffering and oral clearance capacity of minor salivary gland mucus.<sup>8</sup> Moreover, there is lack of salivary tissue on the anterior two-thirds of palate and of the tongue dorsum.

The buccal surfaces of mandibular molars and premolars are the sites with the highest frequency of cervical lesions, the facials of lower anterior teeth being slightly less so, again due to lack of buffering capacity

of and clearance by minor gland mucus.<sup>6</sup> In contrast, all types of non-carious cervical lesions are very rare on lingual aspects of all mandibular teeth, presumably due both to the buffering capacity of submandibular serous saliva and to the rapid clearance rate of mixed submandibular and sublingual saliva from the lingual sulcus.<sup>8</sup>

The strong associations between cervical and occlusal pathology coexisting on the same tooth are also relevant to site specificity of dental erosion. Studies of these associations suggest cervical lesions are very rarely found without occlusal pathology.<sup>6</sup> The shallow cervical erosion is the commonest lesion found at all sites but is as commonly associated with occlusal attrition as with occlusal erosion. The wedge-shaped cervical lesion, in some sites, is more commonly found associated with occlusal erosion rather than attrition.<sup>6</sup> This finding suggests that wedge-shaped lesions are therefore primarily due to erosion and not due to putative stress at cervical margins caused by the strong occlusal forces that result in occlusal wear facets, as envisaged by the abfraction hypothesis.<sup>6,3</sup> Wedge-shaped defects are found more commonly at buccal than at palatal sites and hardly at all on mandibular lingual surfaces, further reducing the likelihood of the stress-corrosion hypothesis.<sup>8,6,3</sup>

However, the rarest cervical lesion, the lingual cervical lesion on mandibular anterior teeth, is more commonly associated with coexistent incisal cupping. It is rare because it depends on chronic gastric acid regurgitation for its aetiology and on reduced sublingual salivary protection for its pathogenesis.<sup>9</sup>

Thus, when perceptions of the aetiology and pathogenesis of tooth wear are changed from local effects on teeth to the more general perception of loss of salivary protection, lifestyles and oral medicine considerations become paramount. Our understanding of the risk factors for tooth wear is greatly expanded when work- and sports-related dehydration and caffeine addiction are considered as conditions of oral medicine importance in the diagnosis and management of dental erosion. Dehydration is acknowledged to be the commonest cause of salivary hypofunction,<sup>12</sup> but, in the southeast Queensland climate, lifestyles with dehydration are commonplace and may be overlooked in understanding why the number of healthy young Queenslanders affected by dental erosion is so high. Dehydration emerges as the most important risk factor for tooth wear in southeast Queensland and is undoubtedly the reason that the highest proportion of subjects seen on referral are healthy young males (Fig 1).

Superficially, it might seem that the lifestyles of the healthy young males in the first group involve a high consumption of acid soft drinks, citrus drinks, sports drinks and cola beverages. However, from an oral medicine perspective, the identification of the caffeine addiction that leads to monotonous consumption of cola beverages explains not only the aetiological factor (orthophosphoric acid) but also points to strategies of

diet analysis and advice for behavioural change, for example, substituting several alternative sources of caffeine for the monotony of cola. Caffeine addiction to cola beverages may also play an important part in the lifestyles of the patients in Group 2, as do other drugs of addiction.<sup>6,4</sup>

Equally pertinent to the oral medicine perspective is the importance of salivary gland dysfunction not only in drug-induced sialadenitis but also in bulimia, asthma, alcoholism, GOR and post-irradiation treatment. Loss of saliva is clearly not only important to protect against tooth erosion by intrinsic acid regurgitation but also may be important for the protection of the health of the oesophagus and stomach.<sup>40,62</sup> Superficially, gastric acid may be the cause of tooth erosion, but as the histories of bulimic, asthmatic and alcoholic cases illustrate, xerostomia and GORD occur more commonly than by chance in such patients.

## CONCLUSIONS

Based on a highly selected patient population suffering from excessive tooth wear in southeast Queensland, six broad groups were identified, from their lifestyles and due to loss of salivary protection against extrinsic or intrinsic acids in the mouth, as being at risk of developing the incipient condition of dental erosion.

The nature and site-specificity of the lesions of tooth wear can be better explained by loss of salivary protection against acids rather than by bruxism or toothbrush abrasion.

Loss of salivary protection of the teeth is caused by work- and sports-related dehydration, drugs and medications and certain medical conditions and syndromes.

In younger subjects, sources of oral extrinsic acids are acidic soft drinks, especially cola beverages, which are consumed for their caffeine content. In older subjects, alcoholic drinks such as wines are acidic, the alcohol is dehydrating and has potential long term effects on salivary function and gastric reflux.

Vomiting and chronic gastric reflux are sources of oral intrinsic acid, not only in bulimic and asthmatic younger subjects but also in alcoholic patients and sufferers from gastro-oesophageal disease. Loss of salivary protection of the upper gastrointestinal tract may contribute to gastro-oesophageal disease.

The diagnosis and management of tooth wear must incorporate oral medicine perceptions as well as preventive strategies.

Proper diet analysis and advice should be given to patients with excessive tooth wear after full consideration of the contribution of their oral medicine to their complaint.

Screening of subjects for dental erosion should be targeted at the preceding at-risk groups.

## ACKNOWLEDGEMENTS

The author acknowledges the help and assistance rendered by Associate Professor Neil Savage, the case referees, colleagues and the Australian Dental Research Foundation.

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