TOOTH WEAR IN YOUNG PEOPLE:

PREVALENCE AND RISK FACTORS

Thesis submitted in accordance with the requirements

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by

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To my wife, Ann Marie

and our children, Ivana, Sophie and Nicholas.

We have lost our certainties but kept our illusions.

François Truffaut

The Green Room

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ABSTRACT

Tooth wear in young people: prevalence and risk factors.
A Milosevic

This thesis aims to assess the prevalence of tooth wear in 14 year old school children and in the eating disorders (anorexia and bulimia nervosa). Those children with tooth wear into dentine on anterior palatal and occlusal surfaces were deemed to be the most severely affected and formed the case group in a follow-up case control study. This case control study assessed the odds ratios (OR) for caseness from the responses to questions regarding aetiological risk factors. The prevalence of pathological tooth wear and its association with vomiting variables and oral hygiene habits in the eating disorders was also investigated. Those bulimic subjects who indulged in self-induced vomiting were grouped according to the presence or absence of pathological tooth wear. The flow rate, pH, buffering capacity and viscosity of gum-stimulated saliva, as well as the calcium concentration dissolved in enamel biopsies, were determined in these vomiting bulimics. Throughout these studies, tooth wear was measured with the Tooth Wear Index (Smith & Knight, 1984a).

A total of 1035 children were examined in ten schools, of whom 307 (30%) had dentine exposed, mainly incisally. Of these children, 80 (8%) exhibited exposed dentine on the anterior palatal surfaces and on the first molar occlusal surfaces. A small positive correlation was found between tooth wear and Jarman score of the electoral ward in which the school was located (rho= +0.2, $p<0.05$). There was an increased risk of tooth wear when 'grinding or clenching' (OR= 2.5, 95% CI 0.94, 6.69) or when drinking fizzy drinks 'on most days' (OR= 2.0, 95% CI 0.77, 5.06). Multivariate logistic regression analysis revealed no significant interaction between these two factors in predicting tooth wear caseness.

Pathological tooth wear was significantly greater in anorexics and bulimics than in controls, although tooth wear was not linearly associated with vomiting frequency, duration or total number of vomiting episodes. Pathological tooth wear was likely to occur above a threshold of 1100 vomiting episodes ($p<0.01$). Gum-stimulated whole salivary flow rates were low but pH and buffering capacity were all within the normal range and were not significantly different in bulimics with and without pathological tooth wear. However, the salivary viscosity was significantly greater ($p<0.05$) in the tooth wear present group, whilst the dissolved calcium in enamel biopsies was significantly lower ($p<0.05$).

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CHAPTER 1

INTRODUCTION AND BACKGROUND

1.0 INTRODUCTION AND BACKGROUND

The seed for the research presented in this thesis was sown with a lecture, given in 1984, to the hospital staff by Professor P D Slade, Head of the Department of Clinical Psychology. His talk on Anorexia and Bulimia Nervosa prompted me to carry out a literature review on the dental aspects of the eating disorders. Planning for a collaborative study followed, which culminated in my first publication, "The oro-dental status of anorexics and bulimics" in the British Dental Journal (1989). On appointment to the department in 1989, Professor M A Lennon suggested that tooth wear should be the main thrust of my research. His interest had been aroused by the 6% prevalence of pathological tooth wear in the control group of healthy young subjects included in the aforementioned paper. Coincidentally, myself and other clinical colleagues were seeing more cases of tooth wear. In many cases, not only was the aetiology difficult to diagnose, but the management often presented a major restorative challenge. Research in the field of tooth wear seemed relatively sparse, despite many case reports associating various factors with erosion, abrasion and attrition. Indeed, the accepted view that self-induced vomiting in bulimia automatically led to dental erosion was not our finding. This raised the fundamental question, why did certain individuals get erosion or tooth wear and not others ?

The prevalence of tooth wear amongst certain population groups or age groups was not clearly known, hence the recent studies by Lussi et al. (1991) and Robb (1992). This thesis presents prevalence studies in 14 year old school children and eating disorder groups.

The age related increase in tooth wear is established. An increasingly older population keeping more teeth into old age may result in dentitions 'wearing out' over a lifetime. Even before birth, guinea pigs are born with worn molars (Ainamo, 1971). Apart from the biological reasons for the occurrence of such an intriguing phenomenon, the study of tooth wear in humans is clinically relevant and, therefore, important to dentists. Hopefully, our better understanding of the processes involved in tooth wear will lead to an effective preventive philosophy and improved treatment.

CHAPTER 2

LITERATURE REVIEW

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2.1 TOOTH WEAR TERMINOLOGY AND DEFINITIONS

The terms attrition, abrasion and erosion have been used extensively in the dental literature to describe the wear of the dental hard tissues.

The Heinemann Dental Dictionary (1987) defines attrition as the mechanical wearing down of the tooth surface in mastication, and abrasion as the wear caused by a pathological condition such as bruxism. These definitions seem to have been based on the British Standard Glossary of Dental Terms BS.4492 (1983) which has been withdrawn and replaced by European Standard EN 21942-1 (1992). This later standard has the status of a British Standard but is not comprehensive since the definitions for the clinical terms attrition, abrasion and erosion do not appear.

Attrition has also been defined as the wear of teeth induced by direct tooth to tooth contact e.g. bruxism. Abrasion is the wear caused by objects other than another tooth (Smith, 1989) e.g. toothbrushing or habits. Finally, dental erosion has been defined as the acid dissolution of enamel and dentine by a chemical process which does not involve bacteria (Pindborg, 1970). The distinct definitions of these three modes of dental wear tended to reinforce the traditional view that only one of these wear processes was occurring at any one site, or indeed within any one individual. However, a combination of any two or

all three modes of wear probably reflects the true clinical situation and this has been termed tooth surface loss (Eccles, 1982a) and more recently tooth wear (Smith & Knight, 1984a). Therefore, the three components of tooth wear are not taken in isolation but in combination. The terms attrition, abrasion and erosion are in themselves aetiologies, yet in many clinical situations the cause for the wear is not apparent from the history or the clinical examination. In such cases, tooth wear is a more appropriate term. Confusion, therefore, exists in the dental literature. It is apparent that Pollmann et al. (1987) described combined aetiology not just abrasion, Woda et al. (1987) equated tooth wear with abrasion only, and Xhonga and Valdmanis (1983) assessed cervical defects describing them purely as "erosions" of different type. English may not have been the first language for these authors and hence errors in translation or usage may be forgiven. Kerr (1988) postulated that the marked degree of "attrition" in medieval British dentitions was caused by crushing bones between the teeth. Dietary abrasion would perhaps be a more appropriate aetiology although this is an undefined term.

The term, tooth wear, allows for combined aetiology and does not presuppose the aetiology (ies) and will, therefore, be used throughout this thesis.

2.1.1 Tribological terms and fundamental wear mechanisms

The fundamental wear processes and their relationship to the dental terminology have been reviewed by Mair (1992). Pugh (1973) stated the generally accepted definition of wear is "the progressive loss of substance from the operating surface of a body occurring as a result of relative motion at the surface". The parent discipline for the study of wear, friction and lubrication is tribology. The fundamental wear mechanisms in tribology are: abrasion, adhesive wear, fatigue wear, fretting wear, erosive and corrosive wear (Pugh, 1973). Pugh further makes the point that each is seldom met in isolation and that in practice two or more of these occur in combination.

Abrasion may result from loose, hard particles sliding between two mating surfaces (three-body abrasion) or from the particles rubbing or cutting against one surface (two-body abrasion). The loose particles may be environmental contaminants or wear debris itself. The rate of abrasive wear is proportional to the load and to sliding distance and inversely proportional to the hardness of the material scratched (Pugh, 1973).

Adhesive wear arises when junctions cold weld together and become broken by relative motion to produce wear particles.

Some wear particles from a soft metal may transfer and adhere to a harder surface, thus preventing further cold welding.

High alternating stresses above a material's endurance limit will result in small cracks. These small cracks may either start at the surface or below the surface; hence the term surface fatigue wear. The material will appear to be unaffected for some time until a piece breaks away to be followed by rapid and catastrophic failure.

Fine wear debris resulting from small relative slipping motion between two surfaces is designated fretting wear.

Erosive wear is that due to impact of particles. An example of erosion by solid particles is sand-blasting. The process is a combination of deformation and cutting. The impact angle and the material involved influence the wear rate, low angles being most effective with ductile materials while 90^0 impact angles are most effective with brittle materials. Pitting and roughening of a surface under the continuous impact of liquid drops is called fluid erosion.

Chemical wear as a consequence of environmental interaction with sliding surfaces is termed corrosive wear. The initial chemical attack may be rapid, but any reaction film formed on the surface may retard or cease the corrosive

process. If the reaction products are non-adherent or the film is porous then pure corrosion occurs independently of any sliding or rubbing contact.

The relationship of the dental terms with these fundamental wear processes will be discussed briefly.

2.1.1a Attrition

Attrition has no counterpart in tribological terms since sites of tooth wear from direct tooth to tooth contact would be attributed to two-body abrasion, fatigue or adhesive wear (Mair, 1992). The notion is that contacting apatite crystal asperities on opposing teeth would adhere (local cold welding) and deform in the direction of movement resulting in sub-surface cracks and loss of the fragment (fatigue). Studies have not been carried out to show these features under physiologically relevant conditions. <u>In vitro</u> wear of single enamel crystals under sliding conditions resulted in both adhesive and fatigue wear (Powers & Craig, 1972). The mode of failure was dependent on load and slider design, but the enamel was worn against diamond sliders under dry conditions. Further research is needed in this area.

2.1.1b Abrasion

Dental abrasion has been defined as wear occurring as a result of contact by surfaces other than teeth and includes habits such as pipe smoking and holding hairpins or nails between the teeth. This is two-body abrasion with the object having one surface in contact with the upper dentition and another surface contacting the lower dentition. Three-body abrasion describes the wear caused by abrasive particles between two surfaces. Dietary wear, in the form of the masticatory slurry, and toothbrush, or more precisely toothpaste abrasion are examples of three-body wear.

2.1.1c Erosion

Dental erosion describes tooth surface loss resulting from non-bacterial acid dissolution. The process of dental erosion is a surface phenomenon as opposed to the sub-surface demineralisation occurring in enamel caries (Sognnaes, 1963). The subsequent demineralised surface is more susceptible to further wear by abrasion and/or attrition (Davis & Winter, 1980). Erosive wear in tribological terms results from the impact of particles or fluid under pressure (Pugh, 1973). The fundamental feature is that the wear medium (sand or salt in sea air) forms the second surface. Boyde (1984) described the wear of teeth caused by air-propelled abrasive particles from an

airpolishing device as erosion. Although tribologically correct, dentists and oral biologists would not agree with this usage of the term erosion.

2.2 AETIOLOGY OF TOOTH WEAR

The aetiology of tooth wear will be discussed under the headings of physiological, erosion, abrasion, attrition and combined aetiology. Although the causes for erosion, abrasion and attrition have been studied and reported on, less is known about the variables or co-factors influencing the physiological tooth wear process. A host of genetic, cultural or environmental co-factors may affect the rate or pattern of tooth wear in 'normal' individuals (Molnar, 1971). Furthermore, these factors may act protectively or co-destructively in 'at risk' individuals. For instance, a vomiting bulimic suffering with erosion may reduce the acid attack by chewing gum, so stimulating salivary buffering, or by rinsing the mouth with antacids. Conversely, the erosion on the palatal surfaces of the upper central incisors in the vomiting bulimic may be exacerbated by a Class II Division II incisal relationship.

Within the normal population very little is known about what constitutes acceptable or normal tooth wear. What is an aesthetic and functioning worn dentition to one may not be acceptable to another individual. Adaptation within the stomato-gnathic system by tooth eruption, alveolar bone

growth, secondary dentine deposition, and altered chewing patterns can all potentially compensate for tooth wear (Berry & Poole, 1976). This adaptive capacity may be exceeded by rapid wear rates or be inherently poor because of age or disease. It is against this complex background of dynamic factors and co-factors that the process of tooth wear continues throughout life.

2.2.1 Physiological

Tooth wear is not necessarily a pathological condition, since some tooth wear is an inevitable consequence of normal function (Molnar, 1971; Luke & Lucas, 1983). Although not aetiological factors in themselves, age, gender, saliva and dental/medical status may act as co-factors or determinants (Burt, 1991) influencing the occurrence, pattern or severity of physiological tooth wear.

2.2.1a Age

The process of tooth wear continues throughout life and for any given population age group there is a normal or physiological level of tooth wear. Therefore, a cumulative effect occurs with age. Few studies have attempted to ascertain what the physiological level of tooth wear should be for specific age groups. Smith and Knight (1984a) proposed threshold acceptable levels of tooth wear in six

age groups on the basis of clinical experience. These threshold levels were modified after a pilot study and two studies, each of 100 patients. The authors erred on the side of caution by proposing threshold levels for their tooth wear index (TWI) that were too high. Consequently the amount of pathological tooth wear would be underestimated rather than exaggerated. Other workers have since suggested increasing the thresholds for the two oldest age groups (Poynter and Wright, 1990; Donachie and Walls, 1991). Robb (1992) commonly found certain sites to be more worn than the threshold TWI values. In the youngest age group ($\leqslant$ 25 years) these were the buccal and lingual surfaces of the upper first molars, the lower first molars buccally and the lower canines incisally. He also reported alteration to the thresholds in the two oldest age groups, but concluded that pathological levels of wear increase with age.

Hand et al. (1987) examined 520 non-institutionalised elderly people for occlusal/incisal wear. Eighty four per cent exhibited some wear into dentine with an average of 6 teeth affected. Only 4% were found to have "severe attrition" where the tooth was worn to the gingival margin.

The rate of tooth wear may vary throughout life (Smith, 1989). An accelerated rate of tooth wear early in adult life may not necessarily manifest as pathological at that time, but because of cumulative wear it may prove

unacceptable later. Lambrechts et al. (1989) reported mean steady state wear rates over a 4 year period for occlusal enamel to be 29μ per year for molars and 15μ per year for premolars. The small sample of 21 subjects, average age 20 years, were not subdivided by sex. Lambrechts et al. further reported higher wear rates over a 2 year 'running in' period after restoration placement, and postulated that restorative treatment may interfere with the balance of the occlusal environment.

2.2.1b Gender

The influence of this determinant on tooth wear remains inconclusive. In all seven age groups men exhibited significantly more teeth with incisal/occlusal wear but not necessarily of greater severity (Hugoson et al., 1988). However, attritional wear scores were more severe in males according to Seligman et al. (1988). Upper incisal edges and lower anterior cervical areas were the only sites to show significantly greater wear in males than in females (Robb, 1992). Using the same index, Poynter and Wright (1990) found significantly more pathological wear on occlusal/incisal sites in males. Anthropologists have studied tooth wear in Aboriginal groups. Heithersay (1960) found greater "attritional values" in Aboriginal females, whereas Richards and Brown (1981) reported no difference in the wear rate between the sexes in two Aboriginal groups. Aboriginal females had a significantly lower rate of cusp

height loss than the males, but this was attributed to differences in diet and behaviour (Molnar et al., 1983). These conflicting results may be attributed in part to different methods of measuring tooth wear.

2.2.1c Saliva

The salivary functions of lubrication and maintenance of a neutral pH (the buffering capacity) are probably of most importance with respect to tooth wear.

The primary rheological function of saliva is lubrication (Schwarz, 1987). The main purpose of lubrication is the minimisation of friction and wear with the secondary roles of cooling and cleansing (Baker & Davies, 1983). Friction is defined as the force resisting motion when two contacting surfaces are moved relative to each other. A lubricant will reduce this frictional force, the extent to which is dependent on the lubricant viscosity (Baker & Davies, 1983). The lubricating properties of saliva have been ascribed to the mucin glycoproteins, because they can provide fluid layers with high film strength (Mandel, 1987). These rheological properties of mucins include low solubility, high viscosity, elasticity and adhesiveness (Tabak et al., 1982). Hatton et al. (1985) showed that the proline-rich glycoprotein of parotid saliva, when complexed with albumin was an extremely effective lubricant. The distribution of this complex in the oral cavity remains to

be established, but it should be functional on teeth as part of the acquired salivary pellicle and on mucous membranes (Mandel, 1987). More recently, the relative lubricating ability of the various salivary glycoproteins has been established, such that the high molecular weight mucins (MG1) are greater than the low molecular weight variety (MG2) which are in turn greater than the parotid proline-rich glycoproteins (Tabak, 1990). The concentration of the mucin glycoproteins is high in the minor mucous and sublingual gland secretions, intermediate in the submandibular and very low in parotid saliva (Edgar, 1992). The viscosity and therefore the lubricating property of the different secretions varies. Saliva is visco-elastic and thus exhibits different viscosities at different shear rates. In other words, as the shear rate of saliva increases, a concomitant decrease in the viscosity of saliva occurs. Saliva is thus non-Newtonian (Edgar, 1992). This shear-thinning nature of saliva imparts several useful functions: draining of saliva from surfaces is reduced, and saliva appears "thin" during swallowing (Schwartz, 1987). Saliva is a complex system of mucins (glycoproteins), electrolytes, proteins, sugars, bacteria, enzymes, etc. (Shellis, 1978), which accounts for the non-homogeneous nature of saliva. A liquid phase, a gaseous phase (bubbles) and a gel phase may co-exist simultaneously (Schwarz, 1987). Therefore, the rheological properties of whole saliva are highly variable; averaged values must be considered as crude estimates. The inter-

relationship of salivary lubrication and tooth wear has hardly been investigated.

Major salivary gland ligation in 16 rats produced significantly more occlusal wear than in the nonligated 16 controls on the same abrasive diet, the authors attributing this to the reduced salivary lubricating action (Carlsson et al., 1965). However, Billings (1993) reported no association of saliva flow with erosion or abrasion in 710 subjects aged from 20 to over 80 years old.

The major regulation of oral pH especially during eating and drinking is the salivary bicarbonate, the level of which varies directly with flow rate. At rest the unstimulated flow rate for whole saliva ranges between 0.1 and 0.5 mls/minute. This basal flow rate occurs the majority of the time and is important for oral health. It is influenced by circadian rhythms, state of arousal, hydration level and stress. However, salivary stimuli will lead to an increased flow rate (1.1 to 3.0 mls/min) with a dramatic rise in bicarbonate levels and hence buffering capacity. Poor salivation manifests orally as a dry mouth (xerostomia). This salivary gland hypofunction may be caused by the side effects of drugs, organic disease, head and neck irradiation, auto-immune disease (Sjögren's Syndrome), psychogenic disorders and decreased mastication. Ageing does not lead to reduced salivary secretion (Heft & Baum, 1984), but the above factors and especially

polypharmacy are prevalent in the elderly (Scully & Cawson, 1983). The most common cause of salivary gland hypofunction is as a result of drug therapy (Sreebny, 1989). Many classes of drugs cause dry mouth (see Table 2.1).

Anorectics
Anticholinergics
Antidepressants
Antipsychotics
Sedatives and hypnotics
Antihistamines
Antiparkinsonism
Antihypertensives
Diuretics

Table 2.1 Drugs which cause dry mouth
(From Sreebny, 1989)

Drugs with a hyposalivatory side effect were taken by 71% of institutionalised elderly (Handelman et al., 1989). Root caries, poor denture retention, cracks at the mouth commissures, dysphagia and loss in taste acuity have been linked to xerostomia in the elderly (Sreebny, 1989). Dry mouth and its effect on tooth wear in the elderly or in other age groups has not been assessed. Stimulated salivary flow rates and pH were not significantly different between subjects with extensive occlusal tooth wear and

controls (Carlsson et al., 1985). However, these authors found increased calcium concentrations and significantly lower buffering capacity, phosphate and sodium levels in the tooth-wear group. These conflicting results were not discussed despite the acceptable salivary flow rates and pH indicative of normal buffering capacity. Normal stimulated salivary flow rate, pH and buffering capacity in 18 year olds with incisal tooth wear was reported by Nystrom et al. (1990).

2.2.1d Dental status

The influence of the oro-dental status on physiological tooth wear will be discussed under this section. The factors include missing teeth, bite force, malocclusion, skeletal relationship and chewing pattern.

Loss of posterior support has been regarded as the cause of migration, loosening and wear of anterior teeth; the rationale being that all occlusal loads are placed on the anterior teeth. However, loss of posterior teeth was poorly correlated with incisal tooth wear (Robb, 1992). The influence of the incisal relationship was not assessed. Poynter and Wright (1990) found no relationship between occlusal/incisal tooth wear and denture status or occlusal contact area. They concluded that a reduced occlusal table may lead to increased wear with a concurrent increase in occlusal contact area on the remaining teeth, both effects

negating each other. Alternatively, bruxism, diet or cleaning habits may be of overriding importance. The relationship between bite force and reduced periodontal ligament surface area has been assessed by Nyman and Ericsson (1984). Teeth with reduced but inflammation free periodontal ligaments can act as successful bridge abutments. They reasoned that the reduced number of periodontal mechanoreceptors acted to reduce masticatory forces imposed across these abutments. Anaesthesia of periodontal ligaments of teeth decreased maximal biting force (Lund & Olsson, 1983). This positive feedback mechanism within the stomato-gnathic system may similarly work in mouths with few posterior teeth and thus explain the lack of correlation between posterior tooth loss and anterior tooth wear.

Tooth contacts frequently occur during mastication. As teeth contact they glide together in a pathway dictated by cusp morphology and thus do not produce pure axial loads but multidirectional loads (Moxham & Berkovitz, 1982; Faulkner & Atkinson, 1983). Studies using intra-oral occlusal telemetry indicated that teeth do not habitually contact into the Intercuspal Position during mastication (Glickman et al. 1974; Bates et al. 1975).

The forces which develop between the teeth have been measured using dynamometers or transducers incorporated into crowns, inlays or dentures. The former technique

measures the maximum static closing force and the latter technique allows assessment of masticatory forces during function. Biting loads of between 70-150N were recorded during mastication and swallowing by Anderson (1956). However, maximum biting loads may be more than ten times greater (Gibbs et al., 1986). Chewing forces are highest at the Intercuspal Position in the molar region so aiding food comminution, but are significantly lower near the incisors (Rugh & Smith, 1988). "Light", "masticatory" and "powerful" bite forces were greater in tooth-wear subjects compared to wear-free controls (Carlsson et al., 1985) and maximal bite force between the incisors was correlated with incisal wear in 18 year olds (Nystrom et al., 1990). Conversely, no differences were recorded in maximal bite force between tooth-wear groups and controls (Dahl et al. 1985; Lyons & Baxendale, 1990). The different methods of measuring bite force, the different age groups assessed and the small sample sizes could account for these conflicting results. A low Frankfort Mandibular Plane Angle and low gonial angle, but increased inter-incisal angle was reported in twenty subjects with severe attrition (Krogstad & Dahl, 1985). These authors postulated that increased masticatory function during growth interfered with mandibular development as evidenced by the reduced lower face height.

Attrition was not associated with the presence of unilateral or bilateral Retruded Contact Position (RCP)

premature contacts nor with the presence of TMJ clicking (Seligman et al., 1988). Interpretation of these results must be tempered by the fact that the Mann-Whitney U-test for differences was carried out, not statistical tests for association. Male subjects with an incisal classification of Class II Division II had significantly less wear than did Class II Division I and Class III subjects (Seligman et al., 1988). Lower incisal 'attrition' increased with overbite depth, but decreased with an initial increase in overjet (Ritchard et al., 1992). Inter-incisal angle, Angle's molar classification and anterior or posterior cross-bite was thought not to be associated with the lower incisor wear (Ritchard et al., 1992).

Further research aimed at establishing the influence of skeletal bases, dynamic and static occlusal/incisal relationships, tooth loss and restorations on tooth wear is still indicated.

2.2.1e Medical Status

The effect of drug therapy on salivary function has been discussed in Section 2.2.1c on saliva and will be elaborated further under medication (Section 2.2.2d). Medical conditions such as gastro-intestinal disorders, alcoholism and eating disorders will be discussed in the next section under dental erosion. Excessive drinking of squashes and fruit juices by an individual with diabetes

insipidus resulted in severe dental erosion (Finch, 1957) and Insulin Dependent Diabetes Mellitus was mildly associated with non-pathological tooth wear (Robb, 1991). "Erosions" occurred in 14% of teeth in 30 hyperthyroid subjects compared to 5% of healthy controls (Xhonga & Van Herle, 1973). This tenuous link between elevated thyroxine and erosion has not been reported elsewhere. A patient on renal dialysis was reported to suffer with dental erosion as a consequence of dialysis-induced regurgitation (Sampson & Meister, 1984). Interestingly, the clinical presentation of excessive occlusal wear in this dialysed patient would be more likely to have resulted from a combination of attrition and abrasion.

2.2.2 Erosion

The aetiological factors associated with dental erosion are shown in Table 2.2.

1.	Acidic Beverages	- natural or reconstituted fruit juice - squashes/cordials requiring dilution - carbonated drinks - "sport" drinks
2.	Acidic Foods	- pickled foods - relishes and ketchups
3.	Regurgitation and vomiting	- gastro-intestinal disorders - alcoholism - anorexia and bulimia nervosa
4.	Certain Industrial Processes	- galvanising/pickling - battery manufacturing
5.	Medication	- Vitamin C (ascorbic acid) - Aspirin (salicylic acid) - Hydrochloric acid replacement

Table 2.2. Factors associated with Dental Erosion

2.2.2a Acidic beverages and food

Dental erosion is not a new disorder since both the effects of fruit juices and industrial processes on the teeth were reported in the first half of this century (Berenzon, 1931; McClure, 1943; Lynch & Bell, 1947; Stafne & Lovestedt, 1947). An acidic atmosphere of 1mg/m^3 of tartaric acid prompted Elsbury (1952) to study the erosive potential of various acids on extracted human teeth. He found citric

acid to be twice as destructive to enamel as hydrochloric or nitric acid, and believed dental erosion was controlled by the hydrogen ion concentration.

Wynn and Haldi (1948) reported the lack of relationship between the extent of erosion and the degree of acidity of different juices indicating that other factors were involved in enamel erosion. Buffering agents or preservatives such as the sodium salts of fluoride, benzoate, alginate or oxalate reduced the acid erosion (Restarski et al., 1945; Holloway et al., 1958). Conversely, sweetened acid solutions increased the erosion, either because of reduced salivary stimulation and buffering capacity or because the sweetened drinks were retained in the mouth for longer (Holloway et al., 1958). These authors emphasised that the pH would have a less significant role in determining erosive potential than titratable acidity.

The titratable acidity relates to the volume of alkali which needs to be added to an acid to bring the pH to neutrality. Conversely, it is a measure of the acid's ability for hydrogen ion dissociation and subsequent donation to neutralise the added hydroxyl ions. Whereas the pH measures the hydrogen ion concentration at a point in time, the titratable acidity indicates the potential quantity of hydrogen ions available for acid attack.

In vivo studies on rats confirmed that a malic acid/ phosphate solution produced no erosion (Hartles & Wagg, 1962). Addition of calcium phosphate to melted ice lollies or their feed reduced erosive potential in rats (Wagg *et al.*, 1965; McDonald & Stookey, 1973) as did the cola drink supplemented with fluoride (Shabat *et al.*, 1975). Reussner *et al.*, (1975) reported that monocalcium phosphate supplements added to low pH powdered beverages resulted in strong protection against enamel erosion, but that total acidity or pH could not be used with reliability to predict erosive potential.

Recent case reports (Levine, 1973; Reuter, 1978; Eccles, 1982b; Mueninghoff & Johnson, 1982; Smith & Shaw, 1987; Asher & Read, 1987) of dental erosion in children and young adults associated with low pH drinks prompted Grenby *et al.*(1990) to study the dental effects of adult and infant fruit drinks. They confirmed the view of Holloway *et al.*(1958) that titratable acidity was a better predictor of erosive potential than pH, and implicated a possible role for ascorbic acid (Vitamin C) which was present in all the infants' drinks in varying amounts. Fresh fruit juices were potentially more erosive than carbonated beverages (Grobler & Van der Horst, 1982; Birkhed,1984) although Rytomaa *et al.* (1988) found highest erosion depths in bovine enamel after a four hour immersion in a cola beverage. The dissolution rate of enamel increases with decreasing acid dissociation constant (Gray, 1966).

Therefore, a ranking of the erosive potential of various acids can be made, such that maleic > fumaric > citric > meso-tartaric > malic (CRC, Handbook of Chemistry and Physics, 1983). Any one of these acids may be present in still or carbonated beverages. Uniquely citric acid is a triprotic acid with three carboxyl groups per molecule. Each citrate molecule can therefore donate three hydrogen ions, see Figure 2.1.

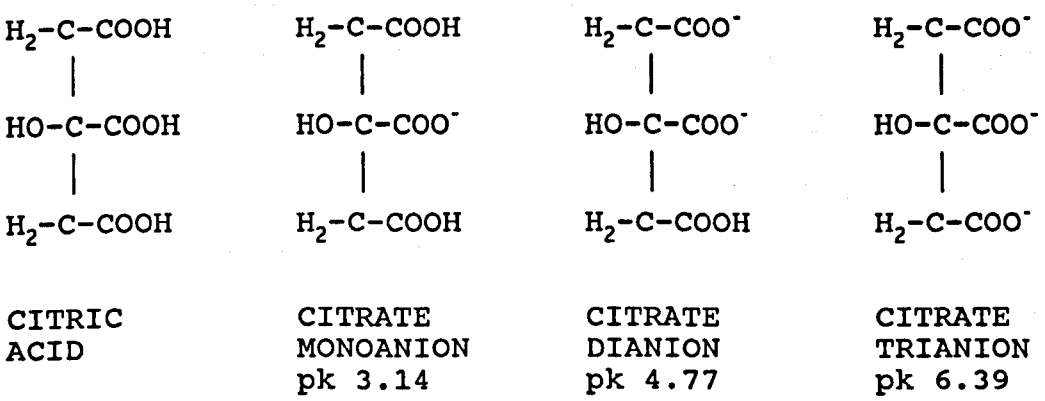


FIGURE 2.1 The citric acid molecule and dissociation constants at 20°C for the three citrate anions.

Furthermore, citrate can chelate to calcium to form calcium citrate independent of the pH of the solution thus increasing its damaging potential even further (Elsbury, 1952; Meurman et al., 1990).

Interestingly, a diet cola was not only less cariogenic but also less demineralising than other acid drinks and the

authors found no significant pH difference before and after degassing carbonated beverages (Grobler et al., 1990).

The erosiveness of foods has not been researched as much as that of drinks. The enamel etch depth produced by minced apricot and grape was greater than by guava, apple and orange (Grobler et al., 1989). These authors concluded that because of the variations in the pH, acid type and acid ratios in different fruits, it was difficult to predict the erosive potential. Erosion did not occur on extracted teeth despite them being immersed in several low pH foods such as yoghurt, tomato juice and strawberry jam (McIntyre, 1992). This confirmed, according to McIntyre (1992), that pH itself does not provide an indicator of erosive potential. Studies into the abrasivity of foods have hardly been carried out.

From the clinical cases reported and laboratory-based animal or in vitro studies described there is clear evidence that a vast range of beverages has the potential to erode dental hard tissues. The reduction of dental erosion from beverages may occur with a change in acid type and concentration, and with the incorporation of calcium or phosphates (Hay et al., 1962; Hartles & Wagg, 1962; Wagg et al., 1965; McDonald & Stookey, 1973; Reussner et al., 1975), fluoride (Sorvari et al., 1988) and other constituents such as buffers and preservatives. Obviously, the soft drinks industry should be aware of the dental

problems associated with these products, particularly since their consumption has greatly increased both in the USA (Morgan et al., 1985) and in the UK (National Association of Soft Drinks Manufacturers, 1985).

2.2.2a.i Saliva and dental erosion

Rats weaned on citrate fluids at pH 5.5 to 7.2 showed marked lingual erosion of the lower molars (McClure & Ruzicka, 1946). Citric acid concentrations under 2mg per 100cc (0.1mM/L) have been found in stimulated saliva (Zipkin, 1947). There was no difference in salivary citrate concentration between Ohio University students with and without erosion (Shulman & Robinson, 1948). It is not clear in the study whether the erosion group did indeed have erosion. In a more comprehensive study a positive association between citric acid in saliva and dental erosion was reported (Zipkin & McClure, 1949). Staff at the National Institute of Dental Health, Bethesda, acted as the non-erosion control group. Unfortunately, the dietary intake of citrate or the salivary bicarbonate concentration in either the erosion or control group was not considered. Ericsson (1953) determined salivary citrate concentrations and enamel solubility after ingestion of citrus fruit. Salivary levels of citrate were raised up to ten minutes after eating oranges or drinking grapefruit juice. Her experimental results on enamel solubility found no prolonged solubility increasing effect after ingestion of

citrus fruit. Ericsson (1953) concluded that the erosive effect of these fruits and beverages would be limited to the time of chewing.

The importance of the saliva buffer to acidic beverages has been alluded to previously. Holloway et al. (1958) believed that the pH of the beverage and that at the tooth surface may be very different due to salivary buffering action. However, Hay et al. (1962) stated that the volume of liquid taken into the mouth was very large compared with salivary volume, and hence any salivary buffering effect was probably insignificant. Mannerberg (1963) analysed stimulated whole saliva in an erosion and control group. No differences in stimulated flow rate, pH, calcium or phosphate concentrations were reported although proper statistical testing was not carried out. The mucin concentration was higher in the erosion group and Mannerberg postulated that this prevented calcium phosphate from reprecipitating and thus remineralising eroded teeth. Mannerberg cited the work of Kusunoki (1940, 1941). Kusunoki suggested that saliva oversaturated with calcium phosphate was due to mucin colloids covering the tooth and so retaining calcium phosphate in solution. Student's t test on Mannerberg's (1963) data carried out by this author reveals significantly higher calcium values in the erosion group but no difference in phosphate values. Significantly lower values for unstimulated resting saliva flow rate and significantly higher values for glucose clearance, calcium

and phosphate concentrations were found in erosion patients (Woltgens et al. 1985). However, there were no differences in the stimulated flow rate, buffering capacity and viscosity between the control and erosion groups. Woltgens et al. (1985) found an elevated lactobacilli count (Snyder test) in the erosion group. They suggested that the resultant enamel demineralisation accounted for the higher calcium and phosphate in the saliva.

Human saliva, a calcifying solution and 0.5% sodium fluoride had little beneficial effect on eroded enamel in vitro (Kelly & Smith, 1988). Erosion alone produced nearly six times more surface loss than abrasion alone but moreover, the surface loss from erosion and abrasion in combination was significantly greater than the sum loss from erosion alone and abrasion alone.

Salivary pellicle was found to protect enamel from erosion in vitro (Meurman & Frank, 1991; Millward et al. 1992). In both these studies pellicle was grown for seven days and, therefore, it was probably much thicker than intra-oral pellicle.

Enamel micro-hardness decreased significantly after subjects wearing enamel specimens in removable orthodontic appliances drank four cups of Coca Cola over one hour (Gedalia et al., 1991). The same specimens were rehardened to their original values after the subjects drank the same

quantity of milk or were exposed to resting salivary flow for one hour. The milk had a significantly greater rehardening effect than the resting saliva. Unfortunately, the effect of stimulated saliva on this rehardening was not assessed. This simple *in vivo* experiment could be refined and repeated in an *in vitro* model system in order to compare the erosive and subsequent abrasive/attritive changes in surface and possibly sub-surface enamel.

2.2.2b Gastric reflux, regurgitation and vomiting

Allan (1969) described a case of vomiting secondary to a duodenal ulcer resulting in severe tooth wear especially in the upper anterior region. A similar appearance was reported in a male with hiatus hernia (Howden, 1971). A cross-sectional study of various gastro-intestinal disorders detected seven cases of dental erosion in oesophagitis and duodenal ulcer sufferers, but because of a reduction in gastric acidity not those who had undergone cholecystectomy (Jarvinen *et al.*, 1988). Interestingly, the duration of gastro-intestinal symptoms did not show an association with the severity of erosive lesions. From these reports the dental sequelae of gastric acid induced erosion may include poor appearance of the upper anterior teeth with chipping and shortening, dentine sensitivity, burning mouth syndrome, tongue sensitivity, parotid gland enlargement and oral ulcers.

Severe dental erosion caused by subclinical regurgitation in alcoholics with chronic gastritis has been reported (Simmons & Thompson, 1987; Smith & Robb, 1989). Significantly more pathological tooth wear occurred in alcoholics than age-matched controls, with a significantly greater prevalence in continuous drinkers than binge drinkers (Robb & Smith, 1990).

Gastro-intestinal reflux in children resulted in erosion of both primary and secondary dentitions (Gallo & Randel, 1981; Taylor et al., 1992; Aine et al., 1993). It seems that gastro-oesophageal reflux in children is fairly common and presents with a variety of respiratory symptoms such as asthma, bronchitis or pneumonia as well as gastro-intestinal symptoms including recurrent abdominal pain and dysphagia (Aine et al., 1993).

It was as early as 1689 that Richard Morton published his 'Treatise of Consumptions', describing a case of 'nervous consumption' caused by 'sadness and anxious cares' (Morton, 1689). It was much later, in 1874, that Sir William Gull named the disorder as anorexia nervosa (Gull, 1874). Anorexia nervosa and the more recent category of bulimia nervosa are collectively termed the eating disorders. Diagnostic criteria are revised with the definition of new variants (Walsh, 1992). The classification presented in Table 2.3 is based on the subtyping described by Fairburn et al., (1993) and Garner et al., (1993).

Anorexia Nervosa	Pure restricting (restrictors/ fasters who do not binge)
	Restricting and purging (engage in purging behaviour)
	Binge eaters
Bulimia Nervosa	Purging
	Non-purging
	Eating disorder not otherwise specified (EDNOS)

Table 2.3 Classification of eating disorders after Fairburn *et al*. (1993) and Garner *et al*., (1993)

It would appear that there are overlapping areas and the distinction between anorexic and bulimic behaviour is not always clear cut.

The incidence of anorexia nervosa has risen dramatically over the past 50 years with variant forms of the disorder having emerged over the past 20 years (Russell, 1979). The

likeliest explanation for the increased incidence and prevalence may be heightened social and cultural pressures acting on young, impressionable individuals to gain a slim body. The complex psychological aspects of anorexia nervosa have been established, and may include obsession, depression, rejection of sexuality and the emphasis on the morbid pre-occupation with body weight, including the dread of obesity (Russell, 1986). Anorexics have been shown to have a distorted view of their body size (Slade & Russell, 1973).

The sufferers with bulimia nervosa or the binge-purge syndrome, are within 80% of mean weight for age, but binge eat and may then induce vomiting, abuse laxatives or exercise strenuously. Indeed this behaviour may be commoner than anorexia, with prevalence ranging between 1-2% of British women (Fairburn & Beglin, 1990).

The association between eating disorders and tooth wear was first brought to the attention of dentists by Holst and Lange (1939). They describe several cases of upper lingual erosion, termed perimylolysis, as a consequence of gastric reflux or actual vomiting. One female, diagnosed with "chronic obstipation or vomitus nervosus" had marked erosion of the upper teeth, but not the lower teeth. Many subsequent research papers and case reports reiterate the occurrence of dental erosion in sufferers with anorexia or bulimia nervosa (Hellstrom, 1977; Hurst et al., 1977;

Brady, 1980; Barkmeier et al., 1982; Stege et al., 1982; Wolcott et al., 1984; Abrams & Ruff, 1986; Roberts & Li, 1987; Cowan et al., 1991; Philipp et al., 1991). The putative cause for this tooth wear, primarily erosion, was self-induced vomiting and the high consumption of acidic beverages after vomiting (Hellstrom, 1977; Hurst et al., 1977). Gastric secretions include hydrochloric acid at a maximal concentration of approximately 0.16 mol/L with a pH of 0.8 (Guyton, 1991). However with other gastric secretions and stomach contents this pH increases and indeed pepsin has an optimum pH of 1.8-3.5. This is still well below the critical pH 5.5 for enamel demineralisation. Hellstrom (1977) grouped the 39 anorexic cases into 12 non-vomiters and 27 vomiters. Twenty three of the latter group exhibited lingual and buccal erosion, often into dentine, whereas 3 non-vomiters exhibited superficial enamel wear only. Hurst et al. 1977 divided their 17 cases into vomiters (5), regurgitators (5) and non-vomiters (7); a precise distinction between the first two groups was not made. They reported 7 cases to have erosion, including only 1 non-vomiter. Roberts and Li (1987) studied 17 anorexics and 30 bulimics, also without a control group. Although Roberts and Li found that 35% of anorexics and 33% of bulimics showed lingual erosion of the maxillary anterior teeth, an index to assess the degree of erosion was not used, nor did they assess the influence of vomiting frequency on the erosion, merely stating that all bulimics in the study practised frequent vomiting. Robb (1992)

found significantly more pathological tooth wear in Abstaining Anorexia Nervosa, Vomiting Anorexia Nervosa and Bulimia Nervosa than in the control group.

2.2.2c Industrial processes

The British Dental Association Memorandum on the Erosion of Teeth (Boyes et al., 1959) listed scores of occupations with a high risk of dental erosion associated with an acidic work environment.

Over 30% of acid process workers examined in three British industrial cities exhibited dental erosion which particularly affected the labial surfaces of the upper and lower incisors (ten Bruggen Cate, 1968). Battery formation workers, galvanisers and picklers were particularly implicated, with factors influencing the degree of erosion being length of exposure, lip level and acid concentration in the air. Surprisingly, despite this knowledge and modern health and safety standards, a significantly high prevalence of dental erosion was reported in such workers both in Finland (Tuominen et al., 1989) and Germany (Petersen & Gormsen, 1991). Conversely, erosion was absent in workers exposed to hydrofluoric acid fumes whilst engaged on the Manhattan project at Rochester University (Dale et al., 1948). The high urinary fluoride and mottled looking enamel suggested a protective effect to erosion by fluoride.

2.2.2d Medication

A study of 42 children with juvenile arthritis found high levels of dental erosion in the 25 children prescribed chewable Aspirin (Sullivan & Kramer, 1983). It was believed that salicylic acid had the potential to produce a low pH in the mouth, although the influence of any possible abrasive effect from other tablet constituents was not considered. Similar cases of "erosion" associated with chewable Vitamin C tablets have also been published (Giunta, 1983; Passon & Jones, 1986). All Vitamin C preparations on the Scandinavian market had a pH below the critical value for enamel demineralisation (Meurman & Murtomaa, 1986), whereas only a 500mg 'megadose' vitamin C tablet produced a sustained pH fall below 5.5 in an American study (Hays et al., 1992). Zander (1946) described two cases of palatal erosion associated with the drinking of dilute hydrochloric acid in achlorhydria. Apart from the direct effect drugs may have on salivary flow or consistency and hence indirectly on tooth wear, certain drugs such as oestrogens, chemotherapeutic agents and digitalis may cause nausea or vomiting as a side effect (Kleier et al., 1984). Although Kleier et al., (1984) list a group of ten drugs with such a possible side effect, the author is unaware of any documented association between erosion and drug-induced vomiting.

2.2.3 Attrition

Attrition has been regarded as a natural, physiological process which needed artificial replication in modern unworn dentitions (Klatsky, 1939; Murphy, 1968; Mills, 1975; Neiburger, 1977). Sicher (1953) stated that in many animals attrition was an indispensable stage in the development of the masticatory apparatus. The low abrasivity of modern diets with concomitantly little or no attrition has led to "locked occlusions" and inefficient mastication (Murphy, 1968; Berry & Poole, 1974). This was pure conjecture, since there was no evidence to support occlusal locking or inefficient mastication. Murphy (1968) believed that tooth wear in Aborigines provided a blueprint for artificial, prophylactic tooth grinding. He even proposed that prophylactic tooth grinding should be commenced at 6 years of age and carried out at 6 monthly intervals thereafter. Berry and Poole (1974) also advocated "planned occlusal modification" and interstitial contact point stripping. They felt this resulted in less plaque, caries and periodontal disease interproximally. Kirveskari (1979) dismissed prophylactic grinding to mimic tooth wear as irrational but advocated removal of retrusive interferences.

Weinberger (1955) agreed that attrition in primitive and modern man could be caused by emotional or stress-related factors. Mammalian tooth sharpening, termed thegosis by

Every and Kühne (1971) and honing by Zingeser (1969) resulted in attritional wear which was deemed necessary to maintain masticatory efficiency (Mills, 1975). Lumsden and Osborn (1977) posed the question "which is more efficient, the unworn, the partially worn or the flattened tooth ?" They related form to function, stating that a cusp to fossa relationship suited crushing, whereas in the flat tooth the sharp leading edges of enamel tore food effectively; albeit the flat occlusal surface would not retain food. However, they felt omnivores would adapt to wear changes by altering their diet.

Luke and Lucas (1983) suggested that the degree of tooth wear found in the dentitions of the higher primates and man was best regarded as a product of wear and tear: as an age change rather than an attempt to improve the efficiency of the dentition.

There is no evidence that masticatory efficiency is improved in the worn human dentition. Anatomists and anthropologists continually state that the diet of primitive man, ancient civilizations, Eskimos, Aborigines and other ethnic groups is 'natural'. The modern Western diet is somehow deemed 'unnatural'. It is perhaps true to state that it can be unhealthy and that generally it is not abrasive.

Uvula tongue malposture consequent to mandibular retrusion resulted in breathing and swallowing difficulty according to Cooperman (1992) and was putatively prevented by "organic" tooth wear. Cooperman believed that "organic" tooth wear resulted in mandibular protrusion, so reducing the risk of the uvula impinging on the dorsum of the tongue. This bizarre hypothesis does not recognise that rotation of the mandible was more likely, should other compensatory mechanisms not occur.

Pindborg (1970) distinguished between physiological and intensified attrition according to the rate of wear. However, attrition caused by abnormal function or malocclusion was termed pathological attrition. The diagnostic distinction between physiologic and pathologic was noted to be difficult. Often opposing wear facets cannot meet in the Intercuspal Position (Centric Occlusion) or in excursive mandibular positions. Furthermore, the wear facets themselves can be either flat with even wear of enamel and dentine or cupped, manifest as a higher enamel rim. For cupping or notching to occur an abrasive/erosive component must be present. Only 11% of hospital specialist referral cases could be ascribed a diagnosis of attrition as opposed to 35% diagnosed dietary or regurgitation erosion (Smith & Knight, 1984b). Thus attrition may not be as common as supposed. Interproximal wear at the contact points seems to be less of a clinical problem than occlusal/incisal wear.

Such empty mouth tooth to tooth contacts that lead to wear occlusally and incisally may occur during bruxism. Bruxism is defined as non-functional jaw movements with or without audible sound and can occur during the day or night (Ramfjord & Ash, 1971). Bruxism implies muscle hyperactivity with increased length of contact time but not necessarily increased maximal bite forces.

As mentioned in Section 2.2.1.d, under dental status, no differences in maximal bite force were found in tooth-wear and non-tooth-wear groups (Dahl et al. 1985; Lyons & Baxendale, 1990). Xhonga (1977) reported significantly greater tooth wear in bruxers than non-bruxers. This result is not unexpected since she grouped her 30 subjects into non-bruxers or bruxers according to whether tooth wear was minimal or advanced. Reliance on tooth wear as the sole diagnostic indicator of bruxism was not recommended by Pavone (1985).

2.2.4 Abrasion

Pindborg (1970) classified abrasion into five categories:

Toothbrush abrasion

Clasp abrasion

Habitual abrasion

Occupational abrasion

Ritual abrasion

Cervical areas are commonly said to be susceptible to abrasion particularly first molars and premolars where thin buccal plates or dehiscences, gingival recession and exposed root surfaces may predispose to cervical notching (Radentz et al. 1976). Toothbrushing technique, brushing frequency, nylon filament/bristle stiffness and dentifrice abrasivity have all been associated with cervical abrasion. Of these four variables horizontal brushing technique and brushing frequency resulted in the greatest occurrence of deep cervical defects (Bergstrom & Lavstedt, 1979). Tooth powders were markedly more abrasive than tooth pastes (Kitchin & Robinson, 1948). Fifty six percent of an elderly dentate population had some cervical abrasion, mainly in the maxillary arch, but the abrasion was not extensive since only 16% of all teeth were affected with 5% having lesions deeper than 1mm (Hand et al., 1986).

Tensile stresses concentrated at the cervical fulcrum area on teeth subjected to lateral forces were postulated to

result in enamel fracture and the formation of narrow, wedge-shaped cervical defects (Lee & Eakle, 1984).

Abrasion from various habit patterns such as holding hair-grips and pipe stems between incisors has been documented (Pindborg, 1970). Contemporary dietary abrasion may not be problematic with modern Western diets tending to be soft. Primitive man's diet caused tooth wear and was thus termed abrasive, however extraneous material became included in the food during preparation, or indeed was retained on roots or shoots eaten raw and unwashed, thus the food *per se* was not necessarily abrasive. Grit from millstones was incorporated into flour up until the last century. Kerr (1988) believed that the crushing and pulverising of small bones between the teeth produced marked "attrition" in medieval Anglo-Saxons. This habit is still practised by Asians who value the nutritious bone marrow.

Industrial processes can cause not only dental erosion but abrasive tooth wear as well. Significantly more occlusal toothwear was reported in miners than controls (Enbom *et al.*, 1986) and similarly a group of cement factory workers showed more wear than a control group although the authors reported that dentine was not exposed in any of the 36 cases (Tuominen & Tuominen, 1991).

Tobacco chewing did not lead to more occlusal wear, possibly because the increased salivary flow improved oral

buffering and lubrication (Magnusson, 1991).

2.2.5 Combined Aetiology

The clinical procedure of enamel etching, usually with phosphoric acid, results in the enamel surface becoming frosty and white. However, this appearance is not present in clinical erosion. An additional factor, such as abrasion and/or attrition, might remove the softened, decalcified surface (Smith, 1989). Even though the principal aetiology is erosion, a combination of aetiologies is likely to be in operation. The wear produced during mastication is also of combined aetiology, yet Walls (1989) stated that mastication caused attrition. Both static and gliding tooth contacts do occur during mastication (Graf & Zander, 1963), but abrasive and/or erosive elements in the masticatory slurry would be of greater influence on the tooth-wear process during chewing. Gibbs et al. (1981) reported that the time and force of tooth contacts during chewing were not damaging to the dentition.

The presence of erosive, abrasive and attritive elements in a patient's history is a common finding. Smith & Knight (1984b) reported 23% of referred tooth-wear cases to be of combined aetiology. Ninety six per cent of subjects aged 46-85 were allocated to combined aetiological groupings (Poynter & Wright, 1990).

2.3 THE MEASUREMENT OF TOOTH WEAR

The earliest measure of tooth wear was devised by Broca in 1879. This very early index had its merits, grading horizontal or oblique patterns of occlusal wear without presupposition into attrition, abrasion or erosion. Restarski et al., (1945) assessed enamel destruction by various acid beverages on lingual surfaces of rat and puppy molars. They allocated six scores for the severity of wear. A total score per experimental animal was assigned by averaging the grades in each quadrant and summing them.

Parma's index cited by Pollmann et al. (1987) described scoring criteria for "abrasion" but only applied the index to occlusal surfaces, hence limiting its use.

Anthropological research into primate mastication has led anthropologists and anatomists to develop measures of tooth wear. Lavelle (1970) had a different approach in his study of cadaveric dentitions of different races by grading according to the number of cusps with exposed dentine on each molar, and whether or not these discrete areas had coalesced. Further, "intermolar and interjaw gradients of tooth attrition" were compared.

Eccles (1979) surveyed 72 cases of non-industrial dental erosion utilising a three-tiered classification. Enamel lesions were categorised as Class I; localised lesions

involving dentine for less than one third of the surface as Class II; and generalised lesions where dentine exposure was more than one third of the surface as Class III. Further subdivision of this latter category was made according to the surface involved, into either facial, lingual/palatal, incisal/occlusal, and severe multisurface involvement.

Smith and Knight (1984a) devised the Tooth Wear Index (TWI) which seems a natural extension of the Eccles classification. Basically, the TWI classifies tooth surfaces as cervical, buccal, occlusal/incisal and lingual with scoring criteria which are an amalgam of the Parma and Eccles criteria. The occlusal/incisal surfaces have scoring criteria based on whether less than or greater than one third of the dentine is exposed, hence scores of 2 and 3 respectively, and a score of 4 assigned to surfaces where enamel is completely lost or where pulp or secondary dentine is exposed. Complete enamel loss may be misleading since an enamel rim at the margin of the worn surface would always tend to be present. This index is designed to measure and monitor all wear. Furthermore, the authors distinguish between acceptable and pathological levels of wear by giving maximum acceptable levels of toothwear according to age. The six age groups were 25 years or less, the four decades from 26 to 65 years and greater than 65 years.

Hand et al. (1987) studied gross occlusal wear in 520, over 64 year old dentate individuals, categorizing enamel, dentinal and severe attrition when dentine was exposed, when enamel was completely lost, or when wear occurred to the gingival margin respectively.

Oilo et al. (1987) devised a wear index based on treatment need. Three satisfactory categories called Romeo, Sierra and Mike were further subdivided according to the degree of dentine exposed with concomitant lack of dentine sensitivity. Two further unacceptable categories 'Tango and Victor' with several subdivisions according to the degree of sensitivity, pain, softening or fractures of tooth/restorations makes this index somewhat unwieldy. Furthermore, the satisfactory category Mike is given the "operational explanation"/criterion as "considerable wear with obvious change of anatomical form, but without need for treatment". The next category, Tango, is unacceptable being given the criterion "considerable wear with marked change in anatomical form, further damage to the tooth and/or its surrounding tissues is likely to occur". The clinician or epidemiologist may find the decision, whether or not further damage is likely to occur, somewhat difficult. Furthermore, patients would not usually accept considerable wear with obvious anatomical change, thus treatment may well be demanded.

In two highly confused publications, a French group, Woda et al. (1987) and Gourdon et al. (1987) described an index applied to study casts based on the coronal distribution of wear facets. They proposed separate indices for anterior and posterior teeth and further differentiated between working and non-working side facets. How this differentiation was made was not divulged. A similar approach to scoring dentinal exposure on study casts was noted to be difficult (Seligman et al., 1988). Nonetheless, these authors scored working and non-working enamel attrition facets on posterior teeth without having articulated the study casts on a semi-adjustable instrument and, furthermore, compounded their imprecision by not having a clinical examination to check the contacts.

Hugoson et al. (1988) surveyed the prevalence of incisal and occlusal toothwear in 585 adult Swedes utilizing an index assessing the amount of crown height loss. This is a difficult clinical interpretation to make, more so than visualising the area of worn enamel or exposed dentine. Fareed et al. (1990) also studied the prevalence of occlusal wear but on study casts rather than directly in the mouth.

The problem with any wear index is that the qualitative data obtained cannot be used to measure a rate of wear. The prevalence of wear on a specific surface and its severity, as defined by the criteria may be made, but

whether any index is sensitive enough to monitor all but the most severe change in tooth wear is doubtful. The ability of an index as a predictor of wear is a function of the index sensitivity, the rate of wear itself, the time interval between successive examinations, and the accuracy of the examiner. Nonetheless, such tooth wear indices have their uses and can be successfully applied in clinical practice and epidemiological studies.

Material scientists have developed sophisticated techniques to measure wear of restorative filling materials either in laboratory-based wear tests or indirectly from patients. Lambrechts et al. (1989) measured vertical enamel wear on serial study casts over a four year period with a computerised three-dimensional measuring microscope. The 21 subjects were young individuals with an age range of 18-23 years at the beginning of the study. After the four years, the total vertical enamel wear was 153μ on molars and 88μ on premolars, but the vertical wear rate decreased over the 48 months.

Nystrom et al. (1990) calculated the wear areas on upper anterior teeth from dental casts of 39 individuals at five years then at 10, 14 and 18 years of age. The technique involved outlining the facet areas by pencil, photographing and enlarging them, then calculating the areas with a drawing tablet connected to a micro-computer. The authors realised that the greatest error lay in the subjective

outlining of the wear facet margins. However, they reported tooth wear at 5 years of age was a poor predictor for later wear although at 18 years of age associations between tooth wear, anterior bite force and gonial angle size were found.

Teaford and Tylenda (1991) devised a novel method of assessing tooth wear, by taking scanning electron micrographs of epoxy resin casts of the buccal and lingual inclines of the buccal cusps on the lower first and second mandibular molars. Rates of wear were expressed as the proportion of new features, such as microscopic pits and scratches created in a week. Baseline and follow-up micrographs were magnified three times to aid counting of these new features, and the authors concluded that with the aid of stereo photogrammetrics, actual measurements of the amount of dental tooth substance lost per unit of time may be made.

Digital image analysis was reported to be a quick yet accurate method of determining the eroded surface area of rats' teeth (Mistry & Grenby, 1993). This technique, though expensive, has the potential to measure areas of tooth wear directly in the mouth. Furthermore, once worn area measurements are stored in the computer memory, monitoring the progress of tooth wear in an individual is possible.

2.4 THE PREVALENCE OF TOOTH WEAR

Few studies have actually assessed the prevalence of tooth wear as such, but many have assessed the prevalence of either erosion, abrasion, or attrition. It seems that researchers have identified a wear problem in a specific population group, then devised their own index to assess the extent of either erosion, abrasion or attrition.

That these aetiologies could have acted in combination was often not appreciated. Additionally, the appearance of wear lesions may be ascribed incorrectly. Sognnaes et al. (1972) reported that "erosion-like" lesions were present in 18% of 10,827 extracted teeth. However, Figure 8 in their paper shows a worn buccal class V amalgam restoration flush with the surface of a cervical defect. This seems to be an abrasion lesion. Furthermore, these authors reported a greater frequency of erosion in mandibular teeth than maxillary teeth. This is contrary to common clinical presentation.

2.4.1 The prevalence of tooth wear in children

Recent case reports of British teenagers have described severe tooth wear of the upper palatal surfaces said to be associated with erosion from acidic beverages (Eccles, 1982b; Asher & Read, 1987). Scandinavian researchers have tended to focus on the possible association of dental wear

with functional disturbances of the stomato-gnathic system such as occlusal interferences and bruxism in children aged from 6 to 18 years (Nilner, 1981; Nilner & Lassing, 1981; Egermark-Eriksson, 1982; Ingerslev, 1983; Nilner, 1983). Three different indices were used in these studies, none of which enabled wear analysis on individual tooth surfaces. Comparison of results is thus difficult, but dentine exposure in the anterior sextant ranged from 14% of 15 year olds (Egermark-Eriksson, 1982), to 78% of 15-18 year olds (Nilner, 1981). Wear of one third of the crown height or "noticeable wear on the facial or lingual surface" was reported in 18% of 15-18 year olds (Nilner, 1981) and 39% of 7-14 year olds (Nilner & Lassing, 1981). Anterior attrition was observed in 13% of 8-11 year olds, but decreased over the four age groups (de Boever & van den Berghe, 1986). Conversely, molar attrition increased from 14% of 8 year olds to 38% of 11 year olds. Unfortunately the diagnostic criteria for attrition were not given.

Lindqvist (1971) diagnosed bruxism from atypical wear facets and found them to be present in 47% of 10-13 year olds. Facets were described as atypical if they were into dentine or if they occluded in extreme mandibular excursions. The multifactorial aetiology of bruxism in children was reviewed by Cash (1988) who also reported on the wide ranging prevalence of between 7% and 88%. Bruxism has been reported in youngsters taking the amphetamine stimulant drug, "Ecstasy" (Henry, 1992). Moreover, 3,4

methylenedioxymethamphetamine (MDMA) causes xerostomia which, exacerbated by dehydration from vigourous activity, is relieved by taking soft drinks (Duxbury, 1993). The limited information on MDMA abuse suggests 10% of teenagers have taken the drug. Frequent MDMA use may lead to increased tooth wear from both erosion and attrition. Bruxism leading to severe dental attrition has also been reported in Rett's syndrome, a neurological disorder affecting females (Peak et al., 1992). Grinding of the teeth is a common feature of many other autistic disorders. Another developmental defect, arthrogryposis multiplex congenita has been associated with regurgitation erosion of the deciduous dentition (Gallo & Randel, 1981).

2.4.2 The prevalence of tooth wear in adults

Few epidemiological studies of wear in different age, ethnic and occupational groups have been published. The variety of indices used in these epidemiological studies makes a meaningful comparison of results difficult. A further difficulty arises with interpretation of the type of wear. Authors whose first language is not English have used the terms abrasion, attrition and erosion rather loosely. Hansson & Nilner (1975) assessed abrasion yet tooth wear would have been a better term. They examined 1,069 shipyard workers and reported severe abrasion in 12%, even in some 20 year olds. A third of their sample had anterior wear into dentine as opposed to 13% with similar

involvement of the posterior teeth. Hugoson et al. (1988) found the prevalence of incisal or occlusal wear in the six age groups from 30 years onwards remained remarkably static with a range of 20.2% at age 30 to 23.7% at 80 years. In young Saudi students, anterior wear was a frequent finding, but the levels of occlusal wear were stated to be greater than in similar Western groups (Fareed et al., 1990). These authors believed that environmental factors could account for this.

Dimmer (1986) reported that 85% of Korean manual workers exhibited excessive attrition compared to 14% of Chinese, but made no mention of the diagnostic criteria utilised.

Exposed occlusal dentine caused by erosion occurred in 30% of 26-30 year olds and 43% of 46-50 year olds (Lussi et al., 1991). Lingually eroded dentine was found in only 2% of the older age group with this site affected in the maxillary arch only. Unfortunately, these authors do not state their diagnostic criteria for erosion.

Although tooth wear affected 99% of 1000 adult dental attenders in Southern England, only 6.7% had pathological tooth wear on more than 20% of available tooth surfaces (Robb, 1992).

2.5 CONCLUDING REMARKS AND AIMS OF THE PRESENT INVESTIGATION

The foregoing chapter reviews the terminology, determinants, aetiological risk factors and measurement of tooth wear. The ill-disciplined usage of the terms attrition, abrasion and erosion has complicated a clear understanding of the prevalence and distribution of these conditions. Tooth wear is, therefore, a preferable term. The multiplicity of aetiological factors has also complicated the study of tooth wear. Despite our knowledge regarding aetiological factors, the cause for tooth wear in many individual cases cannot be determined. The dental literature on erosion, abrasion and attrition, is replete with case reports describing the clinical appearance of the dentition and linking this to aetiology (ies). However, very little is known about the prevalence of tooth wear in children, teenagers and adults or the inter-relationship, if any, between the aetiological factors. Indeed, do we know what are all the aetiological factors ? This could account for our diagnostic failures. The possible role of individual susceptibility or resistance to tooth wear has hardly been assessed. The fundamentally important role of salivary lubrication and buffering in the mouth has been discussed, but few researchers have examined its contribution to the problem of tooth wear.

Rather than develop yet another index, it was decided to use the Tooth Wear Index (TWI) of Smith and Knight (1984a) throughout these studies. This index has clear diagnostic criteria and has been used recently by Robb (1992) and other workers. The TWI reproducibility is good to excellent with a mean reproducibility score of 79.5% achieved by seven examiners in the original study (Smith & Knight, 1984a). The author is grateful to Dr Nigel Robb for coming to Liverpool, discussing the Index, and taking part in a calibration exercise.

With the foregoing in mind, the aims of this thesis were:

1. To establish the prevalence of tooth wear in a sample of 14 year old school children in Liverpool.

2. To investigate the factors associated with tooth wear in 14 year old school children in Liverpool by means of a case control study.

3. To assess the prevalence and association of tooth wear with the eating disorders (anorexia and bulimia nervosa).

4. To determine the possible differences in salivary factors of those bulimics with and without pathological tooth wear who indulged in self-induced vomiting.

CHAPTER 3

THE PREVALENCE OF TOOTH WEAR

IN 14 YEAR OLD SCHOOL CHILDREN

CONTENTS

3.1 INTRODUCTION

The prevalence of tooth wear in children was discussed in the literature review in section 2.4.1. In a typical and recent case report Asher & Read (1987) demonstrated that the palatal surfaces of the maxillary incisors, followed by the occlusal surfaces of mandibular molars, were most at risk to erosion in a group of 12 children aged from 9-15. These same authors found that a concentrated fruit flavoured drink (Vimto) was drunk relatively undiluted by all these children as well as other fruit juices, cordials, squashes and carbonated beverages. They also suggested that newly erupted teeth were particularly susceptible to acid erosion: however, both this and other comparable British studies were based on small numbers of patients and gave no indication of the prevalence and distribution of tooth wear within the population.

The Scandinavian research reviewed in section 2.4.1 which linked tooth wear with bruxism normally involved population samples of 400 subjects (Nilner, 1981; Nilner & Lassing, 1981; Egermark-Eriksson, 1982; Ingerslev, 1983; de Boever & van den Berghe, 1986). However, the wide age range studied, the involvement of both deciduous and permanent dentitions, and the use of different wear indices make interpretation difficult.

A basic tenet of epidemiological analysis is that crude rates should not be used to compare populations of different age structure (Rose & Barker, 1992). Crude rates may be misleading and, therefore, age specific rates should be reported in epidemiological studies.

Furthermore, because of the different conclusions concerning the aetiology of tooth wear from the Scandinavian and British literature, a case control study was planned as a follow-up to the prevalence study to determine the relative impact of erosive, abrasive and attritive factors. The prevalence study will be described in this chapter while the case control study will be described in Chapter 4.

3.2 METHOD

The Community Dental Services in Liverpool were planning a dental survey of 14 year old school children in 1991 as part of the British Association for the Study of Community Dentistry (BASCD) national caries epidemiology programme. A total of over 4,000 children in 36 schools were to be examined. The tooth wear study was conducted as part of this survey on a subsample of 1 in 4 of the main sample. The 15 single sex schools and the seven schools with less than one hundred pupils in the year were excluded. Ten schools were then selected at random from the remaining 14 schools to provide the overall sample of 1 in 4 children.

The examinations were carried out by the author at the schools, usually in the medical room. Tooth wear was scored according to the criteria for the Tooth Wear Index (TWI) with the aid of a dental mirror and intra oral fibre optic light tip. (Eurotec Optical Fibres Limited, Shaw Lane Industrial Estate, Ogden Road, Doncaster, DN2 4SQ). The depth of worn cervical areas was measured with a Williams 14W periodontal probe.

A small 2 litre aqualung with a regulator to reduce the pressure to 40 PSI, to which was attached a triple syringe, acted as a compressed air source enabling teeth to be dried as necessary during the examination. Cross infection control measures were those prescribed by Mersey Regional Health Authority Protocol for dental epidemiological studies.

Apart from tooth wear scores, the only details to be entered on the chart by the assistant were the school name and full name with date of birth for each child. Any child who had not attained their 14th birthday or had achieved their 15th birthday on the day of examination was not examined.

The ten schools examined were stratified according to the socio-economic status of the electoral ward in which they were located. This was achieved by reference to the underprivileged area score calculated from the Jarman eight

census variables (Jarman, 1983) obtained from the Liverpool Health Authority Community Health Services Headquarters. The Jarman scores vary from +60 in extreme deprivation to -60 in extreme affluence. The Jarman variables take into account within each electoral ward the numbers of pensioners living alone and single parent families, the number of unskilled and unemployed, the number of people living in over-crowded conditions or who had moved in the past year and the number of children under five years of age.

The Survey Plus computer programme was used to store and analyse the data. (Survey Plus: Providence Software, Bristol). Categorical data, such as sex, were analysed by Chi Square. The strength of any association between the prevalence of tooth wear and the Jarman Index of the electoral ward in which the school was located was determined by Spearman's rank order coefficient of correlation (rho). This prevalence study was carried out between 26 March 1991 and 14 May 1991.

Diagnostic consistency for TWI scoring

Assessment of intra-examiner scoring consistency was carried out on 15 tooth wear subjects referred to the author and not included in the main study. They were examined and TWI scored by the author. At a subsequent follow-up, a minimum of six months later, they were re-

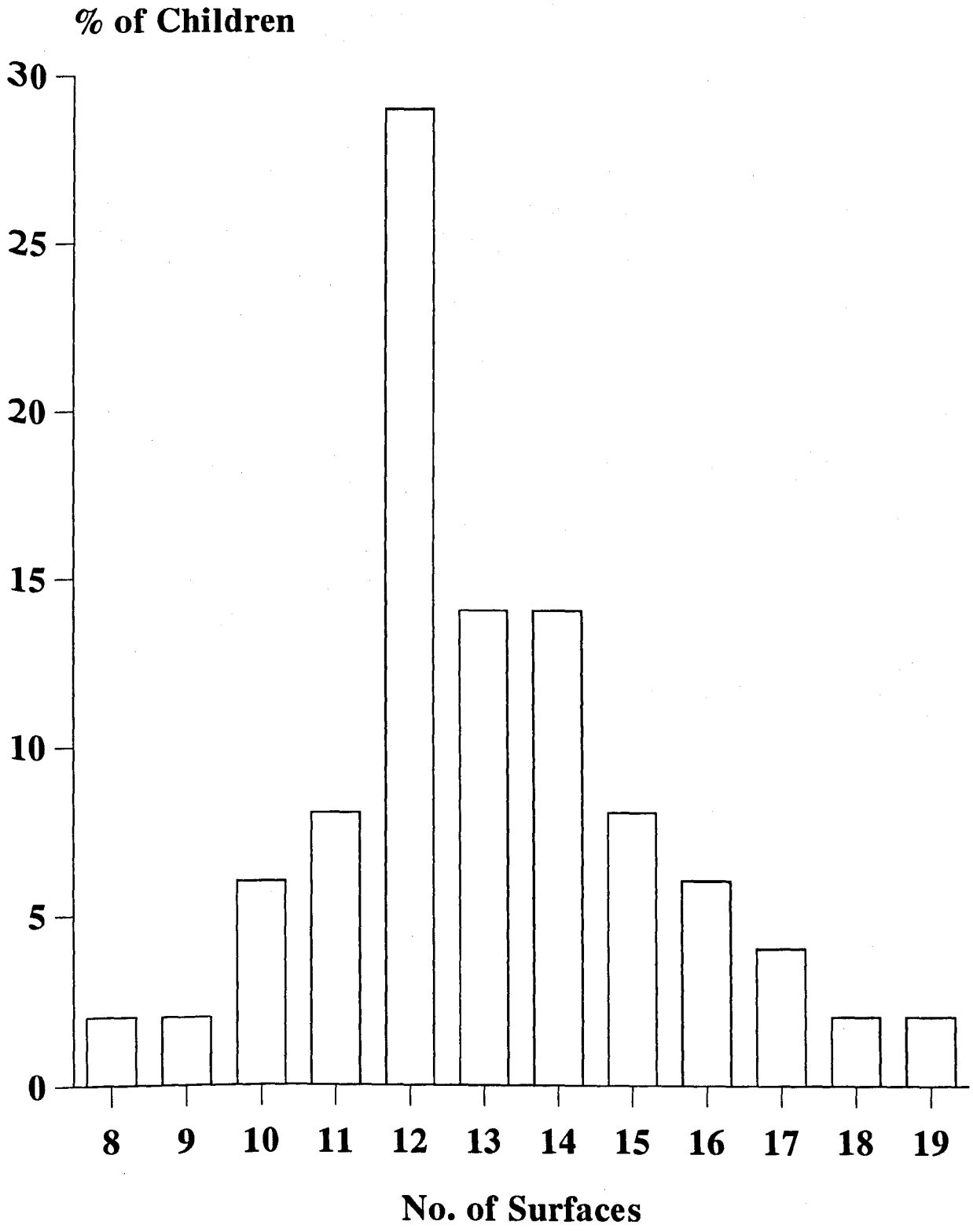
assessed with the TWI. The scores were counted at both the first examination and the subsequent examination, and placed into a contingency table utilising the Survey Plus software.

3.3 RESULTS

An unweighted kappa score of 0.85 for scoring tooth wear was achieved, which is an indicator of good agreement (Bulman & Osborn, 1989).

Seven children were excluded from the study because they had at least one fixed orthodontic appliance precluding scoring of tooth wear, and one child declined the examination. A total of 1035 children were examined for tooth wear representing 62% of the total number of 3rd and 4th year school children in the ten schools selected for the examination. Of these, 526 (50.8%) were male and 509 (49.2%) were female. All the children exhibited some tooth wear (Score ≥1), with 12 being the modal value of teeth affected. The modal number of worn surfaces per child was also 12 (Figure 3.1). This is accounted for by all 12 anterior teeth exhibiting incisal tooth wear in 30% of the children sampled. Three hundred and seven children (30% of the total sample) had at least one tooth with dentine exposed, (a score of 2 or 3). Significantly more males had tooth wear into dentine ($p<0.01$) than females (Table 3.1).

Fig. 3.1 Distribution of ALL WEAR according to the number of affected surfaces



Male		Female	
Number (and %) with exposed dentine		Number (and %) with exposed dentine	
185	(35.2%)	122	(24.0%)

Total number of children with tooth wear into dentine = 307
Total number of males = 526
Total number of females = 509

Gender Chi Square = 15.03, $p<0.01$.

Table 3.1 The number and percentage of children with tooth wear into dentine on at least one tooth

Analysis according to the surface affected revealed no child with tooth wear into dentine on the buccal or cervical surface. Three hundred and seven children (30%) had a score of 2 or 3 on occlusal and/or incisal surfaces, of whom 79 (7.6%) exhibited wear into dentine on the occlusal surface of one or more molars or premolars. Of these 79 children, three scored 3 (>1/3 dentine exposed) on a total of 4 occlusal surfaces. The prevalence of lingually exposed dentine was low. Six children scored 2 on 8 lingual surfaces including one child who also scored 3 on two lingual surfaces. Only one child exhibited lingually exposed dentine without similar involvement on at least one occlusal surface. Altogether, therefore, 80 children exhibited dentinal exposure on occlusal and/or lingual surfaces, and Table 3.2 categorises these children according to the number of surfaces involved.

The mean DMF index (Decayed, Missing, Filled teeth) for all the children sampled was 2.83. Eighty two per cent of the children had no missing teeth, whilst 92 children (8.9%) had one missing molar, and a further 96 children (9.3%) had two or more molars missing.

Incisal or occlusal wear into dentine was seen most frequently on central incisors and lower first molars (Figure 3.2). Lingual wear into dentine was seen only on the palatal aspects of upper incisors (Figure 3.3).

	Male	Female	Total
1 Surface	29	20	49
2 Surfaces	17	6	23
≥3 Surfaces	5	3	8
TOTAL	51	29	80

No significant difference between males and females

TABLE 3.2 Number of children with tooth wear into dentine on at least one occlusal and/or lingual surface according to the number of surfaces affected

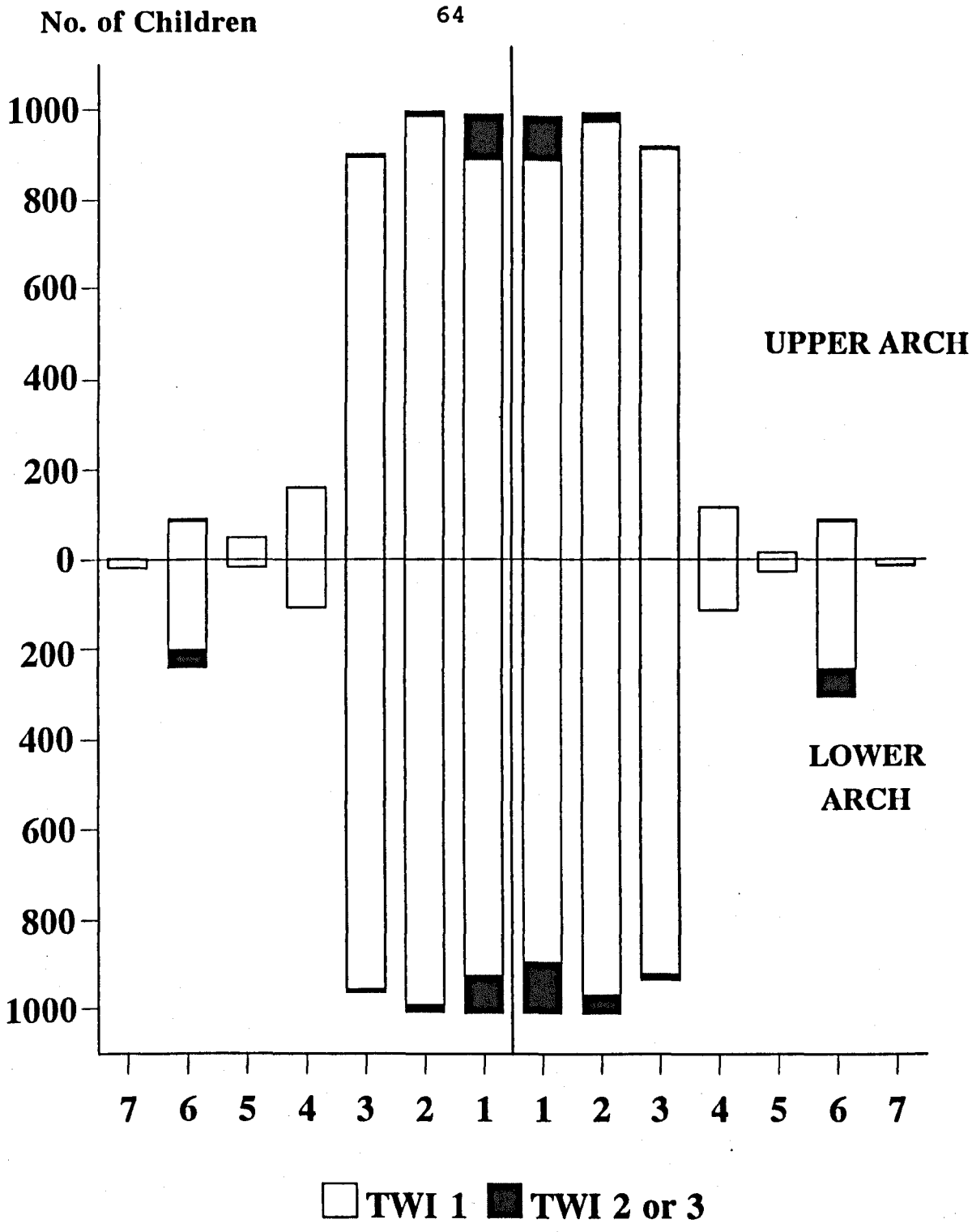


Figure 3.2 Intra-oral distribution of incisal and occlusal wear according to the number of children scoring TWI 1 and TWI 2 or 3

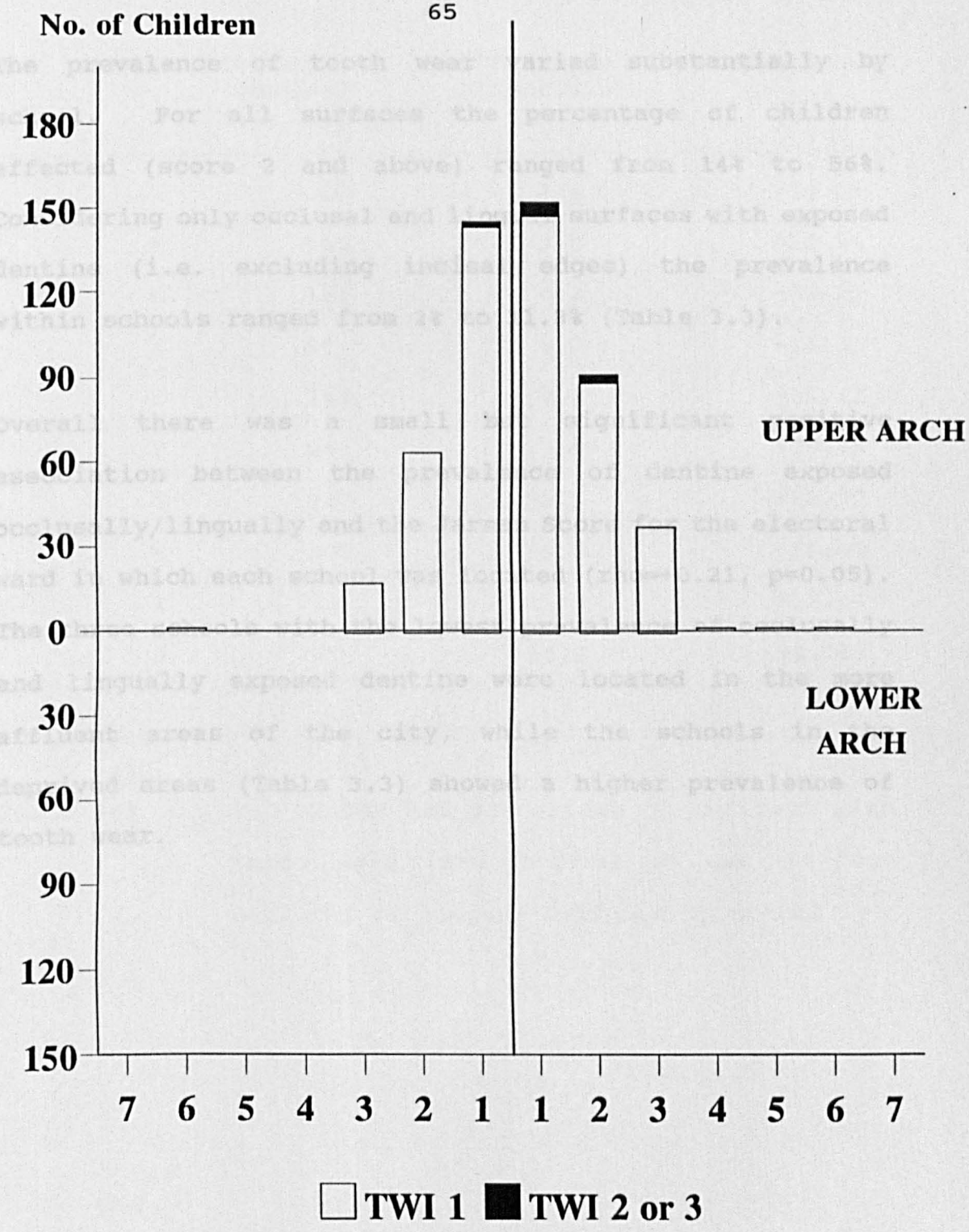


Figure 3.3 Intra-oral distribution of lingual TWI scores.

The prevalence of tooth wear varied substantially by school. For all surfaces the percentage of children affected (score 2 and above) ranged from 14% to 56%. Considering only occlusal and lingual surfaces with exposed dentine (i.e. excluding incisal edges) the prevalence within schools ranged from 2% to 11.8% (Table 3.3).

Overall there was a small but significant positive association between the prevalence of dentine exposed occlusally/lingually and the Jarman Score for the electoral ward in which each school was located (rho=+0.21, p=0.05). The three schools with the lowest prevalence of occlusally and lingually exposed dentine were located in the more affluent areas of the city, while the schools in the deprived areas (Table 3.3) showed a higher prevalence of tooth wear.

School	Number Examined	% Children affected	Jarman Score
A	82	2.4	-10.95
B	146	3.4	-10.04
C	158	6.3	-10.95
D	98	8.2	+17.11
E	143	8.4	+17.11
F	64	9.4	+25.88
G	98	10.2	+18.32
H	105	10.5	-11.45
I	73	11.0	+13.64
J	68	11.8	+8.79

TABLE 3.3 The number and percentage of children with tooth wear into dentine on one or more occlusal or lingual surfaces by school

3.4 DISCUSSION

The amount and nature of data generated in this study is potentially confusing since the results can be expressed as the number of individuals affected, the number of teeth affected or the number of surfaces affected. Furthermore, a distinction can be made according to the severity of tooth wear occurring in any of the above categories. This study presents the results as the number and percentages of individuals affected with tooth wear into dentine by tooth and by surface.

Observing, and thus scoring, exposed dentine is an easier undertaking than judging whether perikymata, mammelons or tritubercular ridges have been worn away leaving a smooth enamel surface. However, it should not be considered that score 1 is unimportant since the early diagnosis of tooth wear has significant implications for monitoring and prevention. It is perhaps unfortunate that the most readily recognizable change occurs after enamel loss.

That 307 (30%) children exhibited some exposed dentine is surprising, but this is accounted for mainly by the 227 children with exposed incisal dentine only. The 79 children that exhibited wear into occlusal dentine is cause for concern, particularly as 31 subjects have two or more surfaces so involved, mainly the first mandibular molars.

Of these children, 5 also had exposed dentine on the palatal aspects of the upper incisors.

Egermark-Eriksson (1982) found 14% of 135 15 year olds to have at least one incisor with exposed dentine, whereas Ingerslev (1983) reported incisal dentine exposure in 42% of 366 children aged 6-16. Wear into dentine was present in the anterior teeth in 78% of 309 15-18 year olds (Nilner, 1981) and in 61% of 440 7-14 year olds (Nilner & Lassing, 1981). The high prevalence of incisally exposed dentine in the latter two studies may be explained by the wide age ranges studied. The 30% prevalence of wear into dentine in this sample of 1035 Liverpool school children is nearer to the results of Egermark-Eriksson (1982) and Ingerslev (1983).

Hugoson et al. (1988), assessing only incisal and occlusal surfaces, reported 6% of 20 year olds to have lost up to 1/3 of crown height with dentinal exposure. Their study found males had significantly more wear than females at this age. The results presented here show a similar prevalence for exposed occlusal dentine (7.6%) after removing the incisal scores. Nystrom et al. (1990) analysed anterior tooth wear in 14 year olds, measured as facet area on casts, and reported wear at this age to be a good predictor of wear in the same 42 subjects four years later, but reasons for the wear in this group were not discussed.

School H in Table 3.3 seems to be different from the other schools, in that the Jarman score of -11.45 places it in an affluent area yet it has a high proportion of children with exposed dentine (10.5%). Removing this school from the calculation of rho increases the correlation coefficient to r=+0.33, p=0.005.

The aetiology of the tooth wear has not been elucidated in this study. However, there are many reports in the dental literature suggesting an association between consumption of acidic beverages and dental erosion (Levine, 1973; Eccles, 1982; Asher & Read, 1987; Jarvinen, Rytomaa & Heinonen, 1991) while other studies have found a wide range of drinks to have an erosive potential (Rytomaa et al., 1988; Grobler et al., 1990; Grenby et al., 1989). The consumption of soft drinks in the UK has increased tenfold since 1939 (National Association of Soft Drink Manufacturers Ltd. Factsheet No. 8. Twickenham, Middlesex, 1985) and seemingly for many children these drinks act as the main source of fluid intake (The Healthy Drinks Report, 1991). Interestingly, boys drink more fizzy drinks than girls (Balding, 1992), the latter showing preference for low calorie fizzy drinks which are not only less cariogenic but also less erosive (Grobler et al., 1990). This may account for the sex difference in those with occlusally/ incisally exposed dentine.

Apart from the type of fluid intake, other dietary factors may promote tooth wear, such as pickled foods, relishes or stoneground bread. Although this is a young age group, adolescent psychological pressures or occlusal interferences may predispose to parafunctional habits (Lindqvist, 1971) and some children especially females may have a tendency towards an eating disorder (Whitaker et al., 1989).

CHAPTER 4

FACTORS ASSOCIATED WITH TOOTH WEAR

IN 14 YEAR OLD SCHOOL CHILDREN

A CASE CONTROL STUDY

CONTENTS

4.1 INTRODUCTION - STUDY DESIGN, SOME GENERAL PRINCIPLES

The preceding study of tooth wear prevalence in 14 year old school children found 8% of the sample with wear into dentine on occlusal and lingual surfaces. The high proportion of young teenagers so severely affected was both surprising and disturbing. This prompted further investigation into the possible aetiology.

To test hypotheses about suspected causes of disease two kinds of epidemiological observations may be made on groups of individuals rather than whole populations. One method would be to compare two groups, one with disease and the other without and retrospectively assess the antecedent exposure to suspected aetiological risk factors, the case control study. The second method defines groups with different levels of aetiological exposure and determines differences in the subsequent development of disease over time. This latter study, the cohort study, necessitates larger sample sizes particularly if the disease is rare, and if the latent period between exposure and disease is long, then the study may have to continue for some years.

The major problem in a case control study relates to the fact that both exposure and disease have already occurred.

Consequently, the study design is susceptible to bias (Hennekens et al., 1987; Rose & Barker, 1992), including selection, investigator, recall and ascertainment biases.

Selection of controls in the design of a case control study is difficult and critical (Hennekens et al., 1987). The main objective in selecting controls is to select subjects who represent those who might have become cases in the study (Rothman, 1986). The controls do not represent the entire non-diseased population but rather the individuals who would have been identified and included as cases had they also developed the disease.

The possible sources of controls include groups of patients with other diseases, the general population, neighbours, friends and relatives. The choice of source is partly determined by the need for cases and controls to be matched in respect of confounding variables. In other words, the controls should be selected to be comparable to the cases. However, if family members or friends are used as controls and have similar exposure to the causative factor under investigation as the cases, an underestimate of the true effect of exposure may result.

The problem of matching controls may be overcome by increasing the control to case ratio from 1:1 to a maximum of 4:1, so reducing error and increasing the power of the study (Hennekens et al., 1987).

The analysis of a case control study is basically a comparison between cases and controls with respect to the frequency of an exposure whose potential aetiologic role is being evaluated. This comparison is made primarily by estimating the relative risk as calculated by the odds ratio. The odds ratio is a measure of association and the data are presented in the form of a two by two contingency table (Table 4.1).

The odds ratio for a set of case control data is the ratio of the odds in favour of exposure among the cases to the odds in favour of exposure in the non-cases (Last, 1988). A value greater than 1 indicates a positive association or an increased risk among those exposed to a factor. A relative risk of 2 or greater is considered important evidence of an exposure effect (Lemeshow et al., 1990). The advantages and disadvantages of a case control study are summarised in Table 4.2.

Exposure	Disease		
	Yes	No	Total
Yes	a	b	a+b
No	c	d	c+d
Total	a+c	b+d	a+b+c+d

a = the number of individuals who are exposed and have the disease

b = the number exposed that do not have the disease

c = the number not exposed that have the disease

d = the number who are both non-exposed and non-diseased

The odds ratio (OR) is calculated from the observed numbers in each cell using the formula:

$$OR = \frac{ad}{bc}$$

Table 4.1 Presentation of data from a case control study

Advantages

Quick and inexpensive compared with other analytic designs.

Ideal for evaluation of diseases with long latent periods.

Optimal for the evaluation of rare diseases.

Ability to examine multiple aetiological factors for a single disease.

Disadvantages

Inefficient for the evaluation of rare exposures.

Disease incidence rates cannot be calculated unless study is population based.

The temporal relationship between exposure and disease may be difficult to establish, especially prone to selection and recall bias.

Table 4.2 Advantages and disadvantages of the case control study design (After: Hennekens et al., 1987)

4.2 METHOD

The case control study needed careful planning prior to actual execution. The planning involved questionnaire design, the selection of cases and controls, gaining Ethical Committee approval, gaining school, parental and subject approval, and the piloting of the questionnaire.

4.2.1 Questionnaire Design

The design of the questionnaire fell into two parts. Firstly, which questions to include in the questionnaire and secondly, the design or layout of the questions. Most of the questions aimed to ascertain which aetiological factors were associated with tooth wear. A questionnaire that has been used on over 125,000 children is the Health Related Behaviour Questionnaire. This questionnaire was developed by the Schools Health Education Unit at Exeter University in order to derive a 'lifestyle' profile of school children. The Director of the unit, Mr John Balding was contacted and kindly sent a copy of the questionnaire which is now in its 15th version having been used between 1984 and 1990 inclusive. It was, therefore, decided to format the tooth wear questionnaire on the Health Related Behaviour Questionnaire. Indeed some of the proposed questions regarding risk factors were identical and permission to use the format and certain questions was sought and gained from the unit Director. Copies of the

correspondence are in Appendix 1.

The first questions were designed to establish general medical and dental status with particular reference to prescriptions and gastro-intestinal disorders (see Appendix 1 for the Tooth Wear Questionnaire). Tooth wear has been associated with certain medications and gastro-intestinal disorders. Question 7 relates to clenching and grinding of teeth in order to ascertain the role of attrition. Questions 8 to 13 relate to toothbrush and dentifrice abrasion and questions 14, 15 and 16 attempt to identify children with a tendency towards body shape/weight dissatisfaction and who may be susceptible to an eating disorder. The remaining questions are intended to establish any link between dietary or drinking habits and tooth wear. The frequency of intake and the type of erosive/abrasive food are itemised in question 17. If drinks are chilled or drunk with a straw, erosive potential is reduced (Grobler et al., 1985). Between meal consumption is probably more deleterious than at meal times. These issues are covered in questions 19 to 28.

To reduce observer variation the questionnaire was self administered rather than 'interviewer' based. The latter method could lead to bias should the interviewer know who were the cases and controls. More than one interviewer would necessitate training in order to keep strictly to the questions as printed and avoid supplementary questions.

Furthermore, self administration would be less time consuming (Rose & Barker, 1992).

Questionnaire design is aimed to help standardisation, speed and accuracy in recording under field conditions as well as coding and retrieval of results afterwards (Rose & Barker, 1992). These epidemiologists advocated numerical coding with results separated from questions and answers.

4.2.2 The selection of cases and controls

The cases were those children identified from the prevalence study to have lingually or occlusally exposed dentine. The 80 children in this category were deemed to be the most severely affected. It was hypothesised that they would have significantly different dietary and behavioural habits to the control group. The choice of controls was discussed with the senior lecturers in Statistics and in Public Health Medicine at The University of Liverpool. The cases represented nearly 10% of the 1035 children sampled in the prevalence study. Since all the schools had at least 100 pupils in the year, the 10% control sample was picked from every tenth name on the class register.

The class or year registers were obtained at a preliminary meeting with the teachers (see next section) and every tenth child was selected. If this child was a case or the

wrong sex, then the next name was accepted. Occasionally the teacher would advise that the named subject was a poor attender and suggest the selection of another individual. This advice was resisted, as non-responders might be different, even though this meant a lower than optimal response rate.

4.2.3 Approval and Consent from Ethical Committee, Schools, Parents and Subjects

The local dental Ethical Committee approved the protocol for the case control study. Copies of the relevant letters are in Appendix 1.

The headteachers or year heads of all 10 schools were contacted by telephone to arrange a meeting at the respective school. The purpose and nature of the case control study was subsequently discussed. A list of those children identified with tooth wear from the previous prevalence study was handed to the teacher. Two separate covering letters asking both the child and parent/guardian for consent to take part in the study were also given to the teacher.

4.2.4 Piloting the questionnaire

Questions in a questionnaire may seem clear and concise to the person setting it, but not necessarily to the target subjects. Pretesting or piloting any questionnaire is essential (Rose & Barker, 1992). The questionnaire was pretested both in the field on representative subjects and subsequently for coding and data extraction. The pretest was carried out at the Blue Coat School in Liverpool. A class of 15 year olds, the same age as those in the case control study, was requested to take part. At the commencement of the pretest the author gave instructions to the class. It was emphasised that should any problem occur the children should feel free to ask for help. The questionnaire took up to 15 minutes to complete. Since none of the children asked for help, after the pupils had filled out the questionnaire they were asked whether there were any problems. All the pupils felt the questionnaire was straightforward.

4.2.5 Photographic records and execution

Apart from the questionnaire, there were two other aspects of this follow-up study. A further tooth wear charting was carried out on both cases and controls. This would verify that cases had wear into dentine on occlusal and palatal sites and that controls did not. Additionally, case children with worsening tooth wear could be identified.

This was carried out using the same instrumentation as in the prevalence study.

A photographic record was also taken. The photographic views decided upon differed from the suggestions of Smith & Knight (1984c). The first view was in Intercuspal Position and the second view was a maxillary occlusal view of the anterior palatal surfaces using a mirror. The third and fourth views were left and right mandibular occlusal views centred on the first molars, without the use of a mirror. These specific views were taken because the prevalence study showed that these surfaces were most frequently worn. A photographic assistant retracted the lips. The colour transparencies were taken with the Pentax dental close-up system. This included the SMC Pentax-M Dental Macro 100mm f/4 lens and AF080C ring flash unit.

The study was carried out on the pre-arranged dates during April 1992. This was one year after the prevalence study.

All the data were entered into and analysed by the SPSS PC+ software. Odds ratios with confidence limits were calculated with the Epi Info Version 5.01a software. The categorical or nominal data were analysed by the Chi square test for significance.

4.3 RESULTS

The results are presented in three main sections. In the first section (4.3.1) each of the variables evaluated in the questionnaire is compared for case and control. Those values which reach (or approach) statistical significance are presented in both text and tabular form. It will be remembered that in the case control study conducted in 1992 the wear status of the subjects' teeth was re-evaluated. In six of the control subjects the wear status had changed from that recorded in the 1991 epidemiological prevalence study. This complication is considered in section 4.3.2 of the results. Finally, a logistic regression analysis in which the combined effect of tooth grinding/clenching and the consumption of carbonated drinks on the risk of tooth wear is considered.

4.3.1 Response to the questionnaire

One hundred and two children participated in the study out of a possible 160. This is an overall response rate of 63.75%. A maximum of 80 cases and 80 controls were anticipated but the response was 48 cases and 54 controls. This represented a 60% case response rate and a 67.5% control response rate as shown in Table 4.3.

There was no difference in the frequency of males and females between cases and controls (Table 4.4).

		Target sample	Effective sample	
	SCHOOL	(TW into dentine*)	CASES	CONTROLS
A		12	4	9
B		8	8	6
C		10	7	6
D		11	5	8
E		8	2	4
F		10	4	6
G		5	5	4
H		2	2	2
I		6	6	4
J		8	5	5
	TOTAL	80	48(60%)	54(67.5%)

* The 80 children identified from the prevalence study with wear into dentine occlusally and palatally. Eighty controls matched by school and gender were also selected (see text).

Table 4.3 The numbers of cases and controls in the study target sample and the number who actually provided data (i.e. the effective sample) by school.

	Cases	Controls	
MALE	33	39	72
FEMALE	15	15	30
TOTAL	48	54	102

Table 4.4 The frequency of males and females in the case and control groups.

Each question provided observed frequencies in the case and control groups. Questions 1 to 5 relating to medical history and medication yielded no significant differences between the two groups.

The raw data for clenching or grinding habits are shown in Table 4.5. Twenty two cases (47%) claimed that they occasionally or regularly clenched or ground their teeth as against 16 controls (30%). In order to calculate the odds ratio in a two by two table, the category "occasionally" was combined with "regularly" and the "not noticed" groups were excluded. The combined category formed the positive exposure group and, by convention, forms the first row in the table, as shown in Table 4.6. Fifty eight per cent of the cases and 36% of the controls claimed to grind or clench their teeth occasionally or regularly. These differences were statistically significant (x^2=4.14, DF=1, $p<0.05$) if the uncorrected chi square value is calculated. However, the Yates correction, applied whenever any cell is less than 20 in a 2 x 2 table only, resulted in a nearly significant Chi value of 3.29, p=0.07. The calculated odds ratio was 2.5 with 95% confidence interval ranging between 0.94 and 6.69.

	Cases	Controls	
Never	16 (34%)	29 (54%)	45
Occasionally	21 (45%)	15 (28%)	36
Regularly	1 (2%)	1 (2%)	2
Not Noticed	9 (19%)	9 (16%)	18
Total	47*(100%)	54 (100%)	101

Table 4.5 The number and percentage of cases and controls who claimed to clench or grind their teeth.
Response to Q.7. Would you say that you clench or grind your teeth ?

* One missing value in case group

		Disease (Tooth Wear)	
		Yes (Cases)	No (Controls)
Exposure	Yes	22 (58%)	16 (36%)
	No	16 (42%)	29 (64%)
		38	45

Odds ratio = 2.5

95% Confidence interval (0.94, 6.69)

Table 4.6 Odds ratio for clenching or grinding of teeth. (Exposed groups are the combined categories "occasionally" and "regularly". The group "not noticed" has been excluded from the table).

Questions 8 through to 13 relating to oral hygiene and brushing habits revealed no significant differences between the two groups. Likewise questions 14, 15 and 16 referring to dissatisfaction with body shape/weight found no differences. Question 17 with the itemised list of drinks/foods did reveal some differences.

The raw data for carbonated or fizzy drink consumption are shown in Table 4.7. Thirty six cases (75%) and 32 controls (60%) consumed carbonated drinks on most days. For the calculation of odds ratio, the three cells 'rarely or never', 'less than once a week' and 'at least once per week' were combined into a single cell (Table 4.8). These differences were not significant (Yates corrected x^2=1.83, DF=1, p=0.18). The calculated odds ratio was 2.0 with a 95% confidence interval of 0.77, 5.06.

	Cases	Controls	
Rarely or never	1 (2%)	6 (11%)	7
Less than once a week	4 (8%)	3 (6%)	7
At least once a week	7 (15%)	12 (23%)	19
On most days	36 (75%)	32 (60%)	68
Total	48 (100%)	53* (100%)	101

Table 4.7 The number and percentage of cases and controls according to the frequency of consumption of carbonated drinks.

* One missing value in control group

		Disease	
		Yes (Cases)	No (Controls)
Exposure	Yes	36	32
	No	12	21
		48	53*

Odds ratio = 2.0

95% Confidence interval = (0.77, 5.06)

Table 4.8 Odds ratio table for carbonated drink consumption
(Non-exposed groups are combined categories "rarely or never"; "less than once a week" and "at least once a week").

* One missing value in control group

There were no differences in purchase or consumption of fizzy drinks in school, (questions 18 and 19) and most drinks were chilled (question 22).

Only 5 children drank these drinks with a straw whereas 94 children did not with no difference between cases and controls (Q.23). The purchase and consumption of fruit juices in cartons was analysed in questions 24 to 27 inclusive. Whereas 44 children purchased fizzy drinks in cans or bottles at school only 21 children purchased fruit juice cartons at school. Sixty five children drank fizzy drinks in bottles or cans at school but only 37 drank fruit juice in cartons. Cartons were not usually chilled.

Question 28 ascertained the time of day when these drinks were consumed. Most children (60) drank these items at lunchtime, but again there was no difference between cases and controls.

The response to the question (17p), about pickled onion consumption, did not result in any significant differences between the case and control group, however question 17q asked about other pickled food intake with a significant difference (p=0.01, Table 4.9) between the groups. Odds ratio calculation could not be determined as none of the controls consumed "at least once a week".

	Cases	Controls	
Rarely or never	40	42	82
Less than once a week	3	10	13
At least once a week	5	0	5
Total	48	52*	100

$x^2=8.67$ df=2 p=0.01

Table 4.9 Response to the question regarding consumption of other pickles (Q.17q)

* Two missing values in control group

4.3.2 The problem of defining controls

A check of the tooth-wear status of all cases and controls was also carried out. It will be remembered that cases were defined according to their status in the descriptive epidemiological survey conducted in 1991. The tooth-wear status was re-evaluated in all subjects (both cases and controls) one year later during the case control study conducted in March 1992. All of the cases had tooth wear into dentine either occlusally or palatally but six of the control group also had developed tooth wear into dentine. Of the six subjects (all males), five had developed dentine exposure on the lower first molars and one subject on an upper first molar. Four of these individuals had a score of 1 (i.e. tooth wear in enamel but not into dentine) on these sites previously. In view of this, advice from the Department of Statistics and Public Health Medicine was sought. Apparently the occurrence of the disease under investigation amongst controls is not uncommon, especially if matching criteria for the controls are good (personal communication, Dr L Williams, Senior Lecturer in Public Health Medicine). The advice given, was to re-analyse the data in two additional ways; first, by re-allocating the six into the case group and second, by excluding them altogether. Table 4.10 recalculates the odds ratio for grinding/clenching with the "rogue" controls re-classified as cases.

		Disease (Tooth Wear)	
		Yes (Cases)	No (Controls)
Exposure	Yes	23	15
	No	19	26
		42	41

Odds ratio = 2.1

95% Confidence interval = (0.80, 5.58)

Table 4.10 The number of cases and controls claiming that they clenched or ground their teeth with the "rogue controls" reallocated to the case group. Odds ratio and 95% confidence intervals are also provided.

Two of the children that had not noticed whether they ground/clenched their teeth are not included as in Table 4.6. The odds ratio decreases slightly to 2.1 (Yates corrected x^2=2.08, p=0.15). Excluding the controls from the analysis leads to a slight improvement of the odds ratio with a Yates corrected Chi square value of 2.79, p=0.09 (Table 4.11).

The same approach was carried out with the six subjects for carbonated drink consumption. Table 4.12 shows the number of children in each category but with the six controls reallocated to the case group. Reference back to Table 4.8 will show that there has been a slight increase in odds ratio from 2.0 to 2.34 (Yates corrected x^2=3.11, p=0.08).

The exclusion of the six from the odds ratio calculation for fizzy drink consumption (Table 4.13) changed the odds ratio from its original value of 2.0 to 2.22 with a 95% confidence limit of 0.85, 5.85 (Yates corrected x^2=2.54, p=0.11).

		Disease (Tooth Wear)	
		Yes (Cases)	No (Controls)
Exposure	Yes	22	15
	No	16	26
		38	41

Odds ratio = 2.38

95% Confidence intervals = (0.88, 6.54)

Table 4.11 The number of cases and controls claiming that they clenched or ground their teeth with the "rogue controls" excluded from the analysis. Odds ratio and 95% confidence interval are also provided.

		Disease (Tooth Wear)	
		Yes (Cases)	No (Controls)
Exposure	Yes	41	27
	No	13	20
		54	47

Odds ratio = 2.34

95% Confidence interval = (0.92, 5.99)

Table 4.12 Odds ratio contingency table for fizzy drink consumption re-allocating six controls to the case category

		Disease (Tooth Wear)	
		Yes (Cases)	No (Controls)
Exposure	Yes	36	27
	No	12	20
		48	47

Odds ratio = 2.22

95% Confidence interval = (0.85, 5.85)

Table 4.13 Odds ratio contingency table for fizzy drink consumption excluding the six controls with tooth wear

4.3.3 Logistic regression analysis

In the previous sections the relationship between the independent variables (clenching/grinding and consumption of carbonated drinks) and the dependent variable (tooth wear) have been considered separately. In this section, the interplay between these variables is considered, firstly, by examining a simple three-way cross-tabulation and then by considering a multi-variate logistic regression analysis.

The cross-tabulation is presented in Table 4.14. Grinders were the "occasional" and "regular" category as in Table 4.5, non-grinders were the "never" and "not noticed" categories. Drinkers were defined as those claiming to consume fizzy drinks "on most days", the non-drinkers were the combined three categories as in Table 4.7. The relationships in Table 4.14 were not significantly different ($x^2=6.40$, $p<0.09$). However, a significant difference occurred when the six controls with exposed dentine were re-allocated or excluded as shown in Tables 4.15 and 4.16.

	Cases	Controls	
Grinders+Drinkers+	15	11	26
Grinders+Drinkers-	7	5	12
Grinders-Drinkers+	20	21	41
Grinders-Drinkers-	5	16	21
Total	47	53	100

$x^2=6.40$, df=3, $p<0.09$

Table 4.14 Crosstabulation of the original responses to clenching and grinding (Q.7) and for drinking carbonated drinks (Q.17f) by group

	Cases	Controls	
Grinders+Drinkers+	16	10	26
Grinders+Drinkers-	7	5	12
Grinders-Drinkers+	25	16	41
Grinders-Drinkers-	5	16	21
Total	53	47	100

$x^2=9.13$, df=3, $p<0.05$

Table 4.15 Crosstabulation of the responses to Q.7 and Q.17f by group with re-allocation of six controls with tooth wear into the case group

	Cases	Controls	
Grinders+Drinkers+	15	10	25
Grinders+Drinkers-	7	5	12
Grinders-Drinkers+	20	16	36
Grinders-Drinkers-	5	16	21
Total	47	47	94

$x^2=7.54$, df=3, $p<0.05$

Table 4.16 Crosstabulation of the responses to Q.7 and Q.17f by group excluding the six controls with tooth wear

The hypothesis that fizzy drink consumption and tooth grinding may interact and promote tooth wear was further tested by logistic regression analysis (Tables 4.17 to 4.19).

Table 4.17 considers cases and controls as originally defined, Table 4.18 considers the situation when the six "rogue controls" are reallocated to the case group and Table 4.19 considers the situation when the six controls are excluded. Table 4.19 is considered the most valid comparison (see discussion) and, therefore, this table will be the main focus of attention.

The overall Chi square value for the total deviance is 7.85 with three degrees of freedom. The remainder of the table partitions the deviance between the independent variables, grinding and drinking and their interaction. The first step assesses whether grinding alone or drinking alone accounts for possible differences in observed numbers between cases and controls. The Chi square values of 2.20 for grinding alone and 3.82 for drinking alone (highlighted yellow) compare with the critical Chi square value of 3.84. 3·84
The second step assesses the effect of drinking controlling for grinding (D/G on left side of table) and grinding controlling for drinking (G/D on right side of table). Comparing the two values of Chi square (highlighted pink) suggests that the effect of drinking (x^2=3.65) may be greater than the effect of grinding (x^2=2.03), the critical

Chi square value is 3.84. The third and final step considers the interactive effect of grinding and drinking (G.D) on case prediction, which produces a Chi square value of 2.00 (highlighted green).

Table 4.18 provides the equivalent data for the situation where the six "rogue controls" are re-allocated to cases. The overall effect of partitioning the Chi square values is similar to that in Table 4.19 although the table does suggest the possibility that the interaction of grinding with drinking (G.D) (x^2=2.56) may be greater than the effect of grinding when controlling for drinking (x^2=1.35).

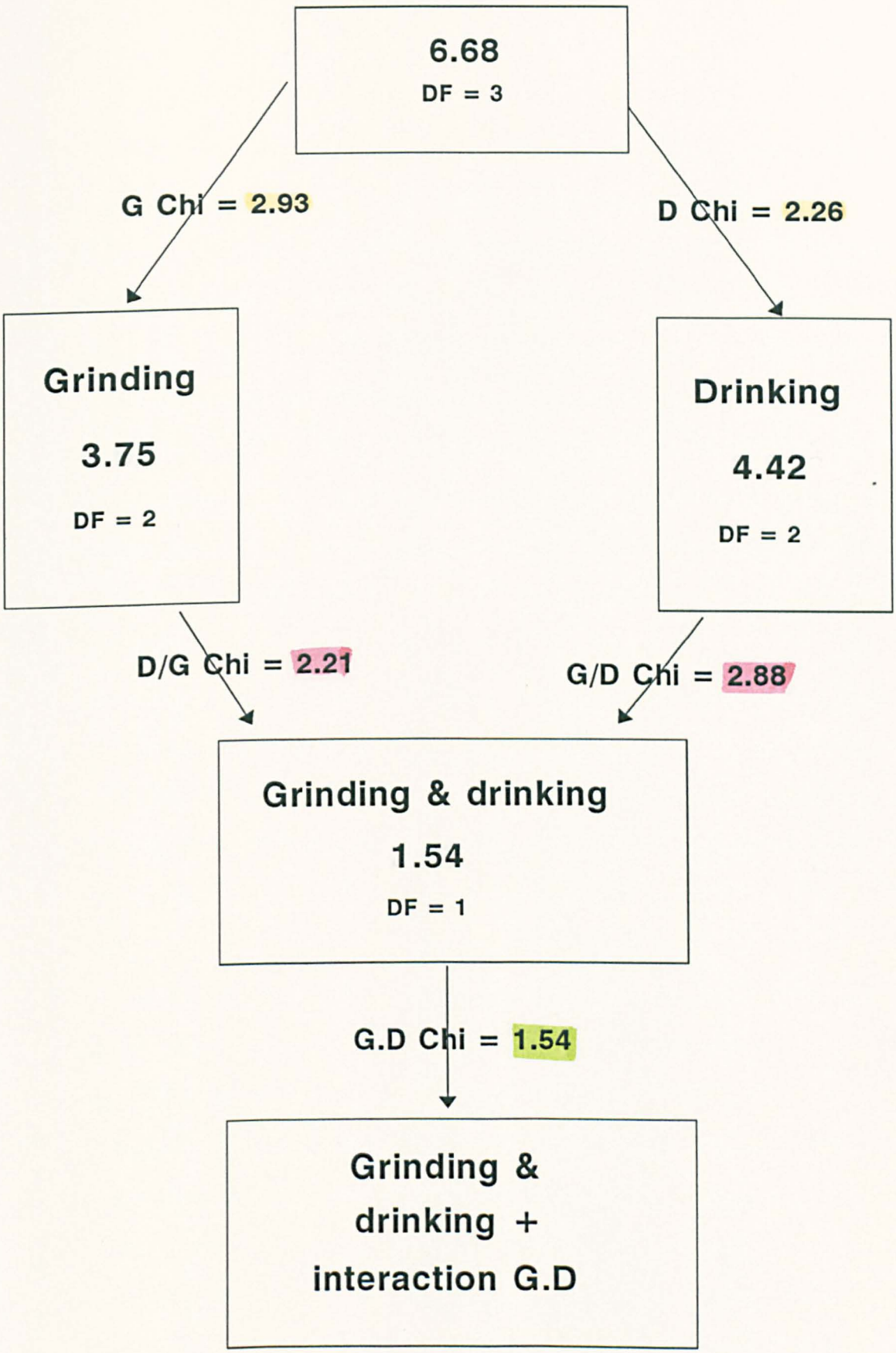


Table 4.17 Steps in logistic regression analysis for tooth wear prediction from fizzy drink consumption and tooth grinding

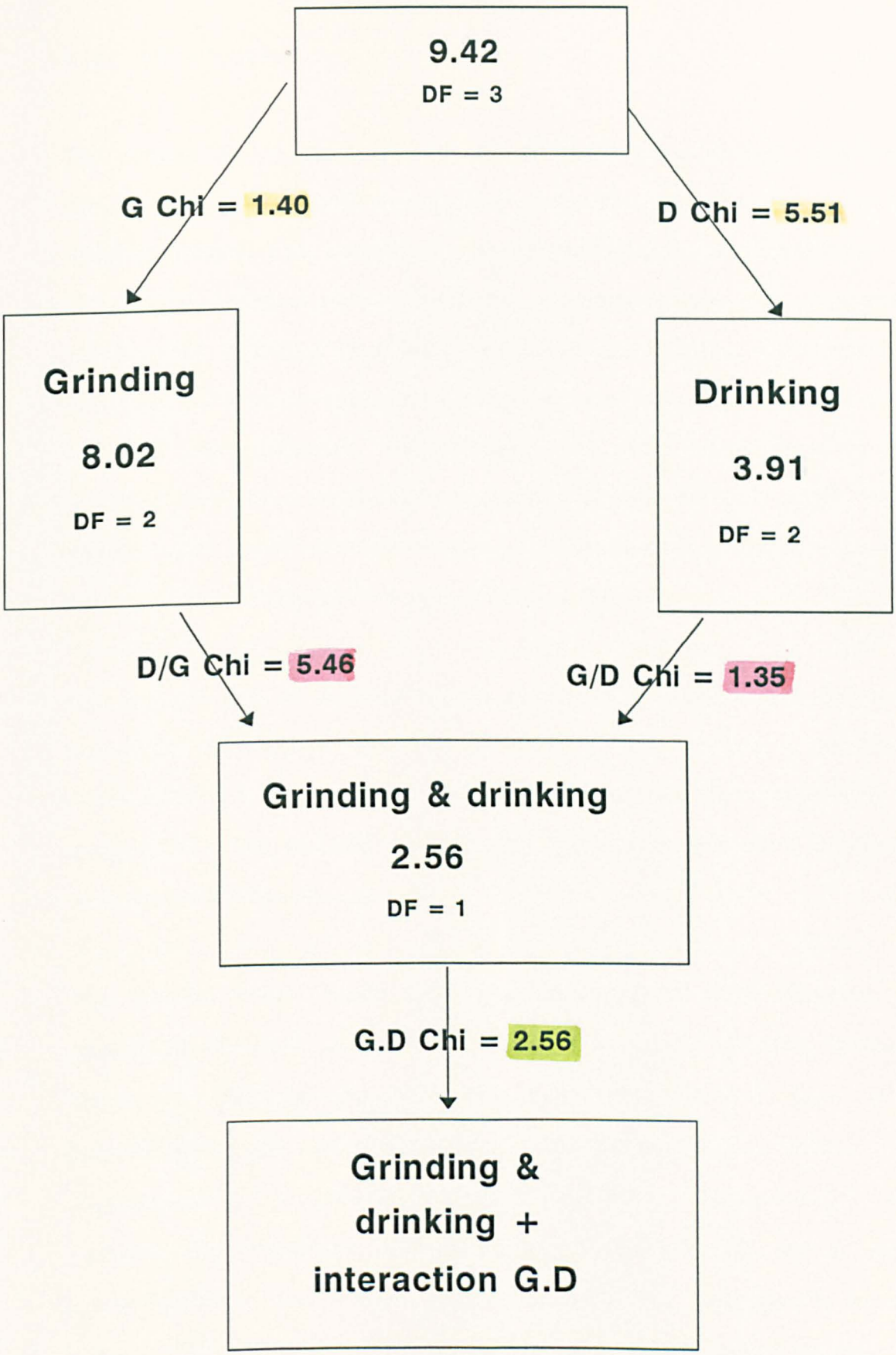


Table 4.18 Steps in logistic regression analysis for tooth wear prediction re-allocating the six controls with tooth wear

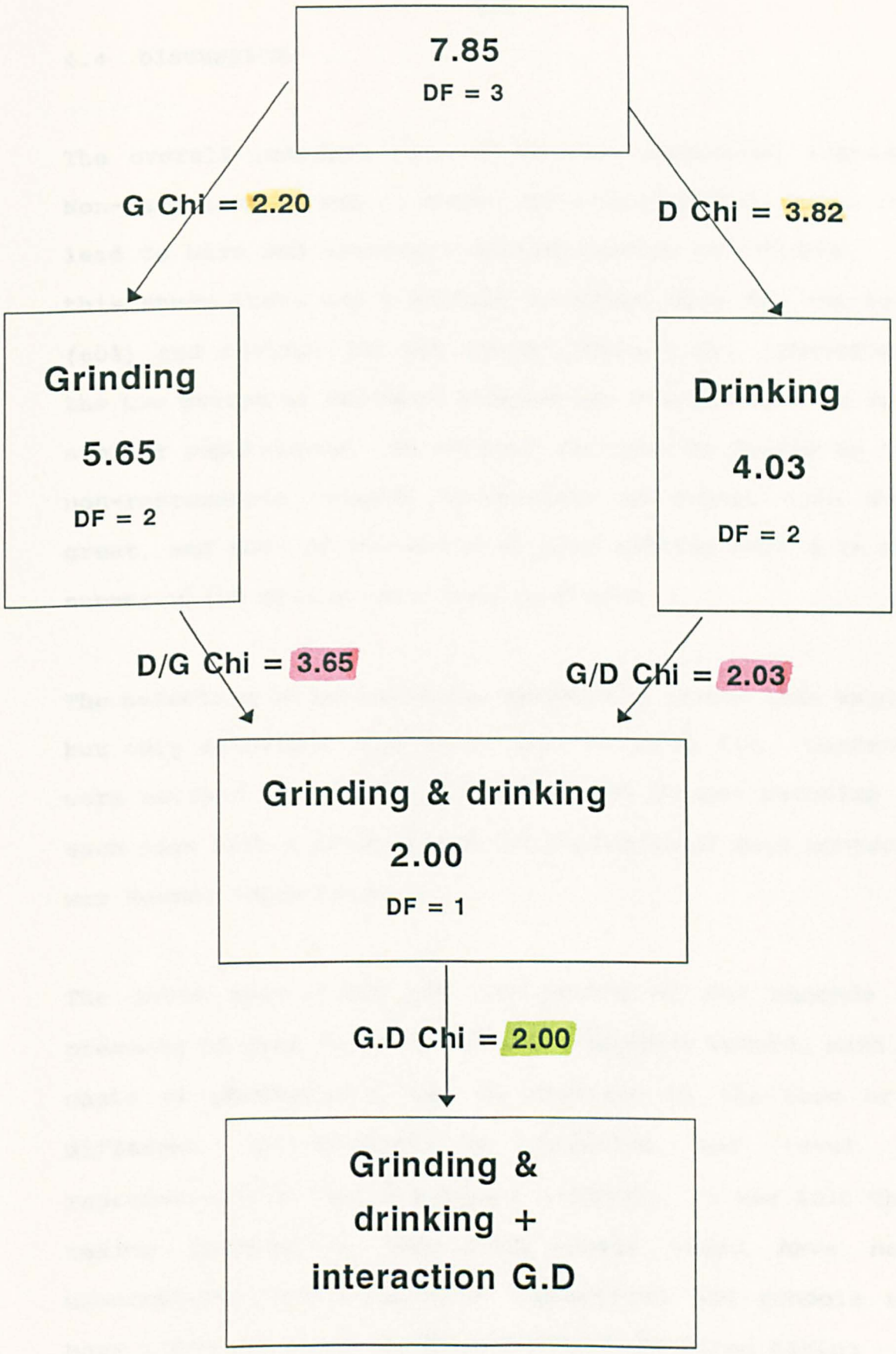


Table 4.19 Steps in logistic regression analysis for tooth wear prediction excluding the six controls with tooth wear

4.4 DISCUSSION

The overall response rate of 64% is considered average. Non-respondents tend to differ from respondents, which can lead to bias and incorrect interpretation of results. In this study there was a similar response rate for the case (60%) and control (67.5%) group (Table 4.3). Therefore, the two groups of children sampled are assumed to come from similar populations. No attempt was made to follow up the non-respondents because constraints on school time were great, and some of the children were sitting GCSE's in the summer while others were leaving school.

The selection of controls was systematic rather than random but only allowed a case to control ratio of 1:1. Controls were matched for gender and school but proper matching of each case with a control and the inclusion of more controls was deemed impractical.

The tooth wear chart was one record of the absence or presence of wear in an individual. Another record, such as casts or photographs, can be rescored by the same or a different investigator to indicate the level of reproducibility (Smith & Knight, 1984c). It was felt that taking impressions for study casts would have been unacceptable and relatively impractical and schools may have taken exception to an invasive impression taking

procedure. However, photographs were deemed a worthwhile exercise.

None of the children stated that they had difficulty in understanding the questionnaire. The results of every question on the questionnaire have not be tabulated, since in many instances there were no differences between the groups. Certain results were of interest.

Question 7 asked about clenching and grinding. The odds of developing tooth wear in a child who claims to grind his/her teeth is presented in the odds ratio contingency table (Table 4.6). The grinder or clencher is at a 2½ times greater risk of developing tooth wear than the non grinder/ clencher.

The results in Table 4.7 regarding frequency of fizzy drink consumption were disappointing. It was hoped that this question would elicit an obvious difference between the cases and controls in that fewer controls would have been drinking in the most frequent category "on most days". However, as in many biological and social science investigations results are not clear cut. The frequency category "on most days" may not have been sensitive enough to discriminate between those children drinking <u>several</u> cans or bottles daily and those drinking just <u>one</u> can every day. A fluid intake diet sheet would be a more sensitive method of assessing drinking habits. In order to obtain an

odds ratio for fizzy drink consumption a 2 x 2 contingency table needed to be constructed. "Rarely or never", "less than once a week" and "at least once a week" were therefore combined and compared to "on most days" (Table 4.8).

The recommended sample size for an odds ratio of 2.0 at the 5% level of significance is 563 (Lemeshow et al., 1990). This may explain the lack of a significant difference between the two groups with respect to fizzy drink consumption. A larger sample is needed.

Odds ratios associated with various aetiological factors for dental erosion have been published (Jarvinen et al. 1991). Drinking apple vinegar once a week or more often resulted in an odds ratio of 10. The case control study carried out by Jarvinen et al. (1991) used unmatched controls. A control was the first patient to attend a dentist on the next morning following the visit of a case. This sample of dental attenders is not representative of the general population. Nonetheless, the Jarvinen odds ratio of 3.5 for soft drinks is comparable to the odds ratio of 2 for fizzy drinks consumption reported in this thesis. Unfortunately, Jarvinen et al. (1991) did not state whether 'soft drinks' included fresh fruit juices, fizzy drinks, squashes and cordials. If this was the case then the greater odds ratio reported by Jarvinen et al. (1991) may be expected. These authors stated that "structured medical and dental histories were taken" with

"emphasis placed on" various putative aetiological factors. This history may have been taken after the dental examination for erosion. This opens to question possible interview bias introduced by the dentist in ascertaining exposure frequency in those cases. A further problem when completing a questionnaire is subjects' recall of exposure frequency. This can be inaccurate, especially in a hospital-based case control study when cases may provide more accurate answers to questions regarding the cause of their disease. Recall bias was less of a problem in this school based study. Systematic bias with respect to the frequency of fluid consumption could have been introduced had the case control study been carried out in summer. More children would be expected to drink these items in summer leading to a possible underestimate of the association with tooth wear.

Gum chewing immediately after an acid attack would stimulate saliva and so neutralise intra oral acids. It was hypothesised that there may have been a difference in gum chewing frequency between cases and controls. In fact there was no difference in gum chewing frequency between the two groups. This would reinforce that the case and control groups came from the same source population. The children probably do not chew gum after drinking carbonated or other beverages.

The temperature of the drink could influence erosive potential. The acid solubility rate of enamel increased by 20% for the 10°C temperature rise from 27°C to 37°C (Gray, 1962). This variation of the acid dissociation constant by temperature may have an important influence on dental erosive potential. Question 22 asked whether cans or bottles were chilled and question 26 asked the same regarding fruit drinks in cartons. Most cans or bottles were drunk chilled (68 children, 70% of both cases and controls) whereas cartons were not (78 children, 85% of both cases and controls).

A further two questions elicited the way cans, bottles or cartons were drunk. Question 23 asked whether a straw was used to drink from the can or bottle with question 27 asking similarly for cartons. The overwhelming majority of children (94) drank without a straw from a can or bottle (5 children used a straw) with no difference between cases and controls. However, many more children used a straw when drinking from a carton (38 children used a straw; 56 children did not use a straw). The hypothesis here was that fluid drunk by straw would not be in contact with the teeth and thus there would be a reduction in erosive potential. It was, therefore, discouraging that there were no differences in the method of intake between the two groups. Although the cans/bottles of carbonated beverages may bathe the teeth, because they are cold, erosive potential may be limited. Conversely the consumption by

straw of juices in cartons, albeit not chilled, may also reduce erosive potential. These influences are worthy of further investigation.

Question 28 asked about the time of day drinks were consumed. Sixty children drank cans, bottles or cartons at lunch time, with no difference between the groups. Stimulated saliva during lunch would buffer any intra oral acid and possibly account for the non-significant difference between cases and controls.

The six controls with tooth wear complicated the analysis and interpretation of the results. Since these subjects were originally selected to be controls, their re-allocation into the case group was deemed less acceptable than their outright exclusion. Nonetheless, odds ratio analyses for grinding/clenching and drinking after both re-allocation and exclusion are shown in Tables 4.10 to 4.13. There are, however, only minor variations in the odds ratios; for clenching/grinding this was 2.5 (originally), 2.1 (re-allocation) and 2.38 (exclusion) and for drinking it was 2.0 (originally), 2.34 (re-allocation) and 2.22 (exclusion).

Logistic regression analysis of the data in Tables 4.17, 4.18 and 4.19 produced interesting results. Overall the data suggest that the effect of drinking may be greater than grinding and that the effect of grinding may be

enhanced when combined with drinking. However, from the data presented in this thesis we cannot be confident about such a conclusion and we must leave these suggestions as interesting hypotheses. A further study on similar lines to that outlined in this chapter would be appropriate but with the following modifications:-

* increase the sample size of cases to around 165; this could be achieved by extending the case search to all 4000 14 year olds in Liverpool rather than a 1 in 4 sample.

* extend the range of the carbonated drink consumption index by including an option "more than 1 drink per day". The use of a 24 hour dietary recall might also be of value.

* define "case" as the confirmed tooth-wear status on the date of interview.

CHAPTER 5

TOOTH WEAR IN THE EATING DISORDERS

CONTENTS

5.1 INTRODUCTION

As discussed in Section 2.2.2b of the literature review there have been numerous case reports describing dental erosion occurrence in the eating disorders. Hellstrom (1974) associated self-induced vomiting with the erosion. In her subsequent more detailed paper Hellstrom (1977) compared the dental status of 27 vomiters and 12 non-vomiters. Hellstrom stated that she used the diagnostic criteria of Eccles and Jenkins (1974) to score erosion, whereby she graded erosion into light, moderate and severe. She reported that nearly all the vomiters exhibited severe lingual erosion with lesser involvement buccally. A significant positive association ($p=0.02$) between duration of vomiting and erosion was found, but not the frequency of vomiting. The degree of association between the two variables of erosion and duration or frequency of vomiting was not reported, nor the actual correlation coefficient employed.

Hurst et al. (1977) assessed dietary habits and dental status in seventeen cases; five regurgitators, five vomiters and seven non-vomiters. They reported that dental erosion was more common in the vomiters and regurgitators than in the non-vomiters ($p<0.04$). The vomiters were more severely eroded. However, only 3 out of 5 vomiters exhibited erosion as did 3 out of 5 regurgitators. The intake of citrus fruits seemed high but this influence was

not statistically related to the erosion. The index of erosion they used is unclear since they scored "++" for full thickness enamel loss and "+++" for loss of enamel and dentine. Full thickness enamel loss without dentine loss is difficult to judge.

Roberts and Li (1987) reported 35% of 17 anorexics and 33% of 30 bulimics showed palatal erosion of the maxillary anterior teeth. An index was not used to measure erosion, the authors merely noting the presence or absence of erosion on "maxillary lingual surfaces". All the bulimics practised frequent self-induced vomiting (SIV) with the result that a third of their sample exhibited erosion, albeit localised to the anterior palatal aspect. These authors felt, without supporting evidence, that oral hygiene and rinsing practices after vomiting may have reduced the erosive damage.

The presence of erosion was found to be significantly greater ($p=0.05$) in those bulimic females who had been vomiting for four years or more (Simmons et al., 1986). Overall, 25 subjects (38% of 66 participants) had "significant erosion". Their diagnostic criteria for erosion included shiny, smooth enamel, loss of vertical dimension, proud amalgam margins and exposed dentine. It seems any one of these criteria constituted "significant erosion". In which case, they may have overestimated the prevalence of 'significant' erosion since, in the author's

opinion, enamel surface wear alone would not be significant or severe. Nonetheless, these authors pose the question of why certain bulimics do not develop erosion despite chronic frequent vomiting.

Sixty nine per cent of teeth in bulimics were eroded bucccally or lingually (Jones & Cleaton-Jones 1989). They scored erosive lesions (by depth and area) on buccal or lingual surfaces only. Although two controls were matched for each bulimic, only 11 bulimics were included in the study. They reported 50% of the bulimics had erosion of the upper canines and premolars palatally, but did not relate this to vomiting variables.

Spigset (1991) analysed the answers to a 72 item questionnaire sent to 34 bulimic females. There was only one group in this study, but Spigset stated that differences between groups were evaluated by Fisher's and Wilcoxon's tests. Additionally, he reported no association between dental symptoms such as hypersensitivity and the duration or frequency of vomiting but omitted to state which correlation tests were employed or how vomiting frequency was gauged.

Hellstrom (1977) analysed the saliva in her two groups and found 'subnormal values' for buffering capacity of resting saliva. Student's t test on her data carried out by the author found no difference between vomiters and non-

vomiters; the mean of pH values indicated that buffering capacity was not poor but intermediate. Alteration in salivary composition secondary to electrolyte imbalance was postulated to lower buffering and remineralising capacity of saliva in anorexics (Hurst et al., 1977). Additionally, the enlargement of parotid glands has been reported in anorexics and bulimics (Levin et al., 1980; Hasler, 1982; Taylor & Sneddon, 1987).

None of these studies utilised established wear indices. Additionally, the statistical analysis of the data was often incorrect. The aim of the present study was to ascertain the prevalence of dental wear in anorexics and bulimics compared to age-matched controls. The association between wear, vomiting variables and salivary buffering capacity of resting unstimulated saliva was also investigated. Ethical Committee approval was gained for this study.

5.2 METHOD

The dental status of 106 individuals referred from Clinical Psychology was assessed by the author, who was blind to any eating disorder. A further two individuals who attended the dental hospital for routine treatment were included, as they admitted to bulimia. The total sample studied was therefore 108. The nature of the dental examination was explained to all participants, who signed a consent form;

a standard proforma was used for the assessment. The medical history gained was not fully comprehensive, but sufficient for examination purposes; otherwise, blind assessment of those referred from Psychology may not have been possible. Patient complaints such as pain, sensitivity and bleeding gums were noted, as were any abnormal extra-oral features. Particular reference was made to possible salivary gland swelling, especially of the parotids, and to excess facial hair (lanugo). Individuals were also asked whether they clenched or ground their teeth, either during the day or at night. The intra-oral soft tissues were carefully examined. The restorative status and any caries was charted, and all participants had left and right bitewing radiographs taken. The degree of tooth wear was assessed using the tooth wear index (TWI) devised by Smith and Knight (1984a).

A simple chairside technique assessing the buffering capacity of saliva was carried out with the Dentobuff kit (Orion Diagnostica, Espoo, Finland). Individuals were asked to dribble approximately 2 mls of unstimulated whole saliva into a disposable, sterile galley pot. An indicator dye in 3 ml of 0.005M HCl is the basis of the Dentobuff kit, which is an accurate, yet fast and simple, chairside technique. Resting saliva was used, as it was felt that some of the individuals with an eating disorder might be averse to chewing a sterile paraffin block. Intermediate and normal buffering capacity is determined from a final pH

range of 4.5 to 5.5 and >5.5, respectively. Alginate impressions of both arches were taken for study casts. These could be used for future baseline reference to assess any longitudinal changes.

Those individuals who had an eating disorder were assessed in Clinical Psychology. Careful, sympathetic questioning elicited a thorough medical and psychological history. Height was measured and all subjects were weighed. They were asked about their eating patterns and whether or not they abused laxatives. Normal heights and weights for age were obtained from The Metropolitan Life Insurance Scales (1959). Those who indulged in self-induced vomiting (SIV) were asked for how many months they had been vomiting and the frequency of this per month. The product of these two figures gave the estimated total number of vomiting episodes those individuals had experienced.

The diagnostic criteria used for categorisation into the anorexia nervosa group or the bulimia nervosa group are based on the Diagnostic and Statistical Manual III revised diagnostic criteria of the American Psychiatric Association (1987), and are summarised in Tables 5.1 and 5.2. These diagnostic criteria were present at the time of the study but are currently in the process of revision. The bulimics were further divided into those who indulged in self-induced vomiting and those who did not.

(1) Refusal to maintain body weight over a minimum normal weight for age and height (usually >15% below expected body weight).

(2) Intense fear of gaining weight or becoming fat, even though underweight.

(3) Disturbance in the way in which one's body weight, size or shape is experienced (the person claims to feel fat even when emaciated).

(4) In females, absence of at least three consecutive menstrual cycles when otherwise expected to occur.

Table 5.1 Diagnostic criteria for anorexia nervosa

(1) Recurrent episodes of binge eating (rapid consumption of large amounts of food).

(2) A feeling of lack of control over eating behaviour during the binges.

(3) The person regularly engages in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain.

(4) A minimum average of two binge eating episodes a week for at least three months.

(5) Persistent overconcern with body shape and weight.

Table 5.2 Diagnostic criteria for bulimia nervosa

The control subjects were volunteers, recruited through Clinical Psychology. They were all female and were selected in order to provide an age-matched comparison sample to the patients suffering from an eating disorder. The sample comprised nurses, psychology and physiotherapy students, technicians, secretaries and members of the university academic and research staff. Control subjects were asked not to reveal their occupation or their status as control subjects (i.e. that they did not have any eating disorder) when being examined dentally. The socio-economic mix of the control sample was similar to that of the eating disorder patients.

Between-group statistical comparisons were conducted in one of two ways. For the buffering capacity pH measurements, which represented a continuously-distributed interval scale, group comparisons were made by means of univariate analysis of variance (ANOVA). For the variable which, in practice, was not continuously distributed, tooth wear (i.e. TWI), nominal scaling was assumed and the appropriate Chi square analysis duly performed. The relationship between tooth wear and cut-offs, in terms of estimated total number of vomiting episodes, was also evaluated using a Chi square analysis.

5.2.1 Follow-up study

A follow-up analysis of 20 participants was carried out 3-4 years after the original study. A further 13 new cases, all bulimics with SIV were also included in this follow up study. Diagnostic criteria for the categorisation into the eating disorder groupings were exactly the same as before. All participants underwent a TWI charting. For the 20 original participants, the procedure could potentially identify deterioration of the tooth wear. A questionnaire designed to identify the oral hygiene practices of these 33 participants is shown in Appendix 2. The questions were posed to ascertain whether there were any different oral hygiene habits between those cases with tooth wear and those without tooth wear. Additional questions targeted at vomiting bulimics asked about oral hygiene practices with respect to the time of vomiting. Brushing immediately after vomiting may abrade away demineralised/softened enamel or dentine, but rinsing with a fluoride mouthrinse could potentially remineralise the eroded surfaces. Were these oral hygiene practices any different after vomiting than at other times ? Questions 17, 18 and 19 were aimed at answering these considerations.

Although gastric acid has a pH of 1-2, regurgitated or vomited gastric contents may have a higher pH value. Foodstuffs could buffer gastric secretions, which if regurgitated rapidly after ingestion may not have reached

a low pH. Not surprisingly perhaps, the pH of vomit from bulimics has not been analysed! The 20 follow up participants were asked whether they would be prepared to bring a sample of their vomit for pH analysis. Those subjects that agreed were instructed to note the time interval between last food intake and collecting the sample. The subjects were then asked to freeze the contents in their home freezer. The subjects brought the frozen vessels at a subsequent appointment when the pH of the thawed vomitus was measured in an Orion pH meter (Orion Research Inc., Boston, USA).

5.3 RESULTS

A total of 108 individuals participated in the first (original) study, of which 58 had an eating disorder (i.e. bulimia with SIV, bulimia without SIV and anorexia) and 50 were age-matched controls (Table 5.3). One-way analysis of variance (ANOVA) showed no statistical difference between the groups for age.

The 33 bulimics who induced vomiting (SIV) had a mean duration of vomiting of approximately 6 years, at a mean frequency of approximately 42 times a month, giving an estimated mean total number of vomiting episodes of 3037. However, the standard deviations are high (Table 5.4) with one female vomiting three times a month for 42 months and another vomiting 112 times a month over an 11 year period.

The mean resting salivary pH values by group are shown in Table 5.5. There were no significant pH differences between the four groups.

The number of cases showing an unacceptable level of tooth wear is shown in Table 5.6. Chi square analysis of the nominal data in this contingency table gave a highly significant result ($P<0.001$). All of the eating disorder groups contained a significant number with tooth wear as opposed to only 6% of the control group.

Eating Disorder Group	M	F	Total	Mean Age (Years)	SD (Years)	Age Range (Years)
Group 1: Bulimia with SIV	1	32	33	25.0	4.9	19-43
Group 2: Bulimia without SIV	0	7	7	24.7	6.8	20-36
Group 3: Anorexia	1	17	18	23.6	5.9	16-36
Group 4: Controls	0	50	50	23.1	4.9	15-39

One-way ANOVA for age F = 0.9181 Not Significant
SIV = self induced vomiting

Table 5.3 Numbers of participants by disorder group, gender and age

	Mean (SD) SIV duration in months	Mean (SD) frequency per month	Mean (SD) total number of vomiting episodes
Bulimics with SIV	71.5(55)	42.48.(43.46)	3037 (4054)

Table 5.4 Vomiting variables of bulimics with self-induced vomiting (means and SD)

Group		pH
Group 1:	Bulimia with SIV	5.76
Group 2:	Bulimia without SIV	5.14
Group 3:	Anorexia	5.78
Group 4:	Controls	5.66

F = 0.57 Not Significant
Normal buffering capacity pH > 5.5, intermediate 4.5-5.5, low <4.5

Table 5.5 Mean values of the resting salivary buffering capacity (pH)

Pathological level of tooth wear	Group 1	Group 2	Group 3	Group 4
Absent	19 (58%)	5 (72%)	12 (67%)	47 (94%)
Present	14 (42%)	2 (28%)	6 (33%)	3 (6%)

Table 5.6 Number of cases within groups showing presence or absence of pathological tooth wear

Group 1 = Bulimics with SIV
Group 2 = Bulimics without SIV
Group 3 = Anorexics
Group 4 = Controls

Pearson's correlation coefficients were carried out to assess any association between the 5 variables of age, SIV duration, SIV frequency, SIV total, TWI, and pH. No association was found between these variables.

The total number of vomiting episodes was estimated from the product of the vomiting frequency and duration. A significantly different frequency of pathological tooth wear occurred in the vomiting bulimics with a cut-off threshold of 1100 vomiting episodes (Table 5.7).

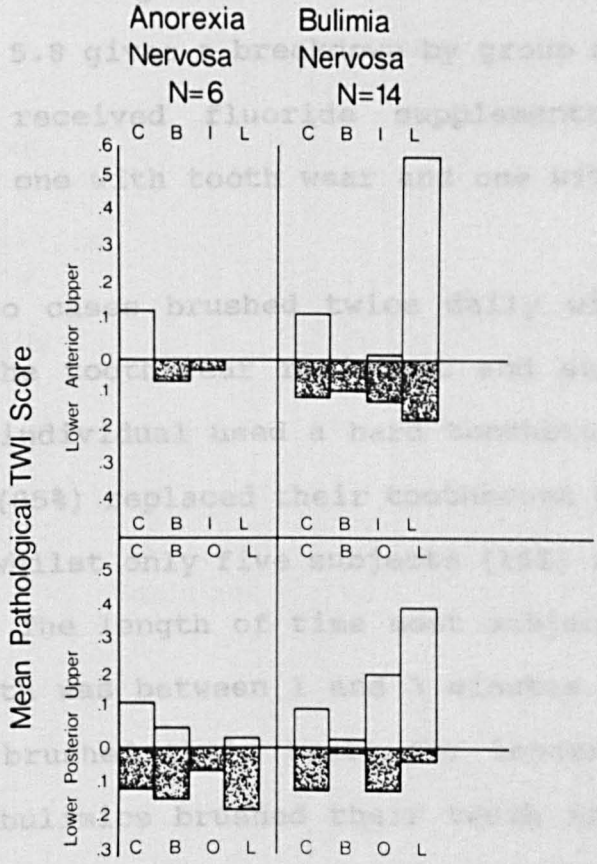
An average tooth-wear profile was produced for the six anorexics and fourteen bulimics with pathological tooth wear (Figure 5.1). These TWI profiles are attained from individual mean pathological TWI scores as described by Smith and Knight (1984c). Cervical, buccal, incisal/ occlusal and lingual surface mean scores for the upper and lower, anterior and posterior teeth are presented. The upper anterior and posterior lingual surfaces are severely worn in the bulimic group.

Pathological level of tooth wear	Total number of vomiting episodes	
	<1100	≥1100
Absent	15 (83%)	4 (27%)
Present	3 (17%)	11 (73%)

x^2=8.56, p<0.005

Sensitivity = 73% Specificity = 83%

Table 5.7 Presence or absence of tooth wear in vomiting bulimics dependent on total vomiting episodes



C = Cervical, B = Buccal, I = Incisal, O = Occlusal, L = Lingual

Fig. 5.1 Mean pathological TWI profiles for anorexics and vomiting bulimics

5.3.1 Results of the follow-up study

A total of 33 subjects were included in the follow up study and Table 5.8 gives a breakdown by group and age. Only two subjects received fluoride supplements, both vomiting bulimics, one with tooth wear and one without.

Twenty two cases brushed twice daily with an even split between the tooth-wear resistant and susceptible groups. Only one individual used a hard toothbrush. Twenty eight subjects (85%) replaced their toothbrush every three months or more, whilst only five subjects (15%) replaced the brush monthly. The length of time most subjects spent brushing their teeth was between 1 and 3 minutes (67%) whilst five subjects brushed their teeth for longer than 3 minutes. Nineteen bulimics brushed their teeth after vomiting, and five did not. However a further breakdown of this group into those with pathological tooth wear (susceptible) and those without (resistant) is presented in Table 5.9, with no significant differences.

Group	N	Mean Age (yrs)	Standard Deviation (yrs)
1: Vomiting bulimics	26	28.3	8.3
2: Non-vomiting bulimics	2	23.0	1.4
3: Anorexics	5	22.6	5.3

Total N=33 Mean age=27.1 (7.9) years

Table 5.8 Mean age values of the follow up groups

Immediate tooth brushing after vomiting	Tooth Wear Status		
	Susceptible	Resistant	Total
Yes	7	12	19
No	2	3	5
Total	9	15	24*

Table 5.9 The number of vomiting bulimics responding to Q.12. Do you brush your teeth immediately after vomiting ?

*Two missing values

Nine individuals brushed within one minute of vomiting and a further eight up to 5 minutes afterwards. Only eight bulimics (33%) spent more time brushing after vomiting than at other times. Seven subjects used a mouthrinse after vomiting with no significant differences between those with and without tooth wear. Again there were no differences between the tooth-wear groups in their response to the length of time between vomiting and brushing (Q.13) and the length of time spent brushing (Q.17).

Six subjects returned samples of frozen vomitus. None of these individuals exhibited tooth wear. The mean pH was 3.8 (SD 0.89) with a range of 2.9-5.0 (Table 5.10). One subject vomited four hours after eating, but the others ranged from 10-20 minutes, with one missing value.

Table 5.11 presents the total pathological TWI scores and the number of surfaces scored 2 or more for the 20 subjects who were recalled in the follow up study. The total individual pathological TWI score was obtained by adding the scores for each of the 16 sets of tooth surfaces (Smith & Knight, 1984b,c). The total pathological TWI score increased in 5 subjects in Group 1, in both cases in Group 2, and in 4 anorexics (Group 3). Four cases in Group 1 exhibited a decrease in pathological TWI score because having crossed into an older age group, the acceptable threshold TWI levels changed, rendering fewer surfaces

Subject	Time after eating (mins)	pH
1	Not known	3.3
2	240	3.5
3	10	2.9
4	15	3.4
5	20	5.0
6	10	4.9

N=6 Mean time=59 mins Mean pH=3.8 (SD 0.89)

Table 5.10 Vomitus pH values and times after eating in six vomiting bulimics

pathological. This is the reason why the number of surfaces scored 2 or more are also presented. Seven vomiting bulimics had more surfaces with exposed dentine at recall as did both non-vomiting bulimics (Group 2) and three anorexics (Group 3). Subject No. 6 in the vomiting bulimic group exhibited a reduction in pathological TWI score from 5.29 to 1.84 and 16 fewer surfaces with exposed dentine because she had virtually all her upper teeth crowned as a result of the erosion. Restored surfaces are not scored.

GROUP 1 Vomiting bulimics	Total individual pathological TWI Scores at Baseline	Total individual pathological TWI Scores at Recall	Number of TWI Scores ⩾ 2 at Baseline	Number of TWI Scores ⩾ 2 at Recall
1	0	0.67	1	12
2	3.68	0	0	7
3	0	0	1	5
4	0	1.00	0	2
5	2.67	4.78	11	11
6	5.29	1.84	19	3
7	0	0	0	0
8	13.34	10.74	38	38
9	0	0.37	5	11
10	0	0	2	11
11	0.17	0	1	0
12	0	0.17	4	4
13	0	0	0	2
GROUP 2 Non-vomiting bulimics				
14	0	2.0	0	12
15	0	2.25	0	3
GROUP 3 Anorexics				
16	0	0	0	0
17	0	0.43	0	0
18	0	1.9	0	2
19	0.33	0.8	3	7
20	0	0.86	2	7

Table 5.11 The total individual pathological TWI scores and the number of surfaces with exposed dentine at baseline and at recall

5.4 DISCUSSION

The most commonly reported dental manifestation of vomiting is acid erosion of enamel and dentine. Hellstrom (1977) concluded that erosion occurred mainly in the 27 vomiters, with three of the 12 non-vomiters having erosion of buccal surfaces only. A difference in the tooth wear profile between the vomiters and non-vomiters was noted. The former group suffered mainly from lingual/palatal erosion. In the Hurst et al., (1977) study, ten subjects vomited/regurgitated within three years of the study date, of which six had erosion of varying degrees of severity. One individual without erosion had mottled enamel caused by living in a highly fluoridated area, but why some patients had erosion and others did not was unclear.

The frequency of pathological tooth wear between the various groups presented in this study is significantly different (Table 5.6). The individual sets of pathological tooth-wear scores were added together to give a total for that individual. The Pearson correlation coefficient shows no continuous association between either the SIV duration, SIV frequency or SIV total and the pathological level of tooth wear. This is interesting, as it would be expected that the more vomiting episodes undergone, the greater would be the erosion present. However, closer analysis of the vomiting group does show a trend. Of the 14 cases with

pathological tooth wear, 11 had had 1100 or more vomiting episodes. This threshold is the basis of Table 5.7 with a significant difference in the observed frequencies between the groups ($P<0.005$).

The individual profile of tooth wear may enable allocation to aetiological categories if the mean pathological TWI profiles described by Smith and Knight (1984b) are diagnostic (Fig.5.1). The mean pathological TWI scores in the 18 subjects with regurgitation erosion was approximately 1.7 on the upper anterior lingual surfaces (Smith & Knight, 1984b). Robb (1992) reported a mean pathological TWI score on the upper anterior lingual surfaces of 0.56 in vomiting anorexics and 0.47 in bulimics. From Fig. 5.1., the mean pathological TWI score in this area is 0.55 in the bulimic group. Further similarities with the results of Robb (1992) are found in the upper lingual posterior sites with mean TWI scores of 0.4 presented here and 0.43 (Robb, 1992). Some dissimilar results have occurred, in that very little buccal tooth wear was noted in this study, but significantly more in the study by Robb (1992). However, reference must be made to Table 5.6, in order to appreciate that only 42% of vomiting bulimics and 33% of anorexics showed unacceptable tooth wear levels.

Twelve of the 20 follow up subjects did have more scores graded 2 or more at the follow up assessment (Table 5.11). The increase in tooth wear occurred mainly on the upper and lower incisal edges, but because a TWI score of 2 on canine and incisal edges is acceptable at all ages, this did not manifest as pathological wear in any subject. Four subjects produced a lower total pathological TWI profile because they had changed from a younger to an older age group with an increased maximum acceptable score for the worn surface.

The influence of parafunctional habits on tooth wear has not been objectively determined in this study. Participants were asked whether or not they ground their teeth. Of those with pathological tooth wear, only one non-vomiting bulimic and two controls responded positively. Lanugo and parotid gland swelling were not common findings in the eating disorder groups.

Reference to Table 5.5 shows that the mean buffering pH for resting saliva in groups 1,3 and 4 falls within the normal range, and that the non-vomiting bulimics (group 2) have intermediate salivary buffering capacity. This is contrary to the conclusions and summaries reported by Hellstrom (1974 and 1977). However, on close inspection of her data there are several imprecisions. The summary, in English, of her 1974 paper stated "all patients had very low

secretion, pH and buffering of both resting and stimulated saliva". The paper is in Swedish, but Table 2 is understandable with only 3 out of 9 subjects exhibiting low stimulated salivary buffering. Unfortunately, her more extensive follow up paper of 1977 does not clarify the issue. The mean resting buffering capacity for vomiters (N=17) and non-vomiters (N=11) was normal. The stimulated flow rate and salivary pH means were normal in both groups yet the stimulated buffering capacity was low in both groups. This is surprising since the normal stimulated salivary flow rates and pH values ought to indicate a raised bicarbonate level manifest as acceptable buffering capacity. Although Hellstrom (1977) found no association between dental erosion and the salivary factors, the wrong statistical test (Chi square) was applied for correlation analysis. The result for resting salivary buffer capacity presented in this thesis is within an acceptable range for both the bulimics and anorexics.

The results of the follow up study analysing oral hygiene practices does not shed further light as to why some of the vomiting bulimics have severe tooth wear and others not. The upper palatal surfaces are not readily brushable, but neither the pattern of brushing nor time spent brushing nor mouthrinse use was any different between those vomiting bulimics susceptible to tooth wear and those without. Most of the subjects (87.5%) used a soft or medium toothbrush,

replacing it between 3 and 6 months in 60% of cases. This would be considered acceptable 'brush behaviour'.

Another aspect to this follow up was the pH analysis of vomiters. Although the procedure was rather indelicate, it does support the demineralising potential of vomited gastric contents since all 6 samples were below the critical pH of 5.5 for enamel demineralisation to occur. Furthermore, since all these 6 subjects did not have pathological levels of tooth wear, it supports the main result that tooth wear is not linearly correlated with vomiting variables. Other factors, as yet unidentified, must come into play in addition to vomiting and regurgitation.

CHAPTER 6

SALIVARY FACTORS AND TOOTH WEAR

IN BULIMIA NERVOSA

CONTENTS

6.1 INTRODUCTION

The previous study found no linear correlation between vomiting variables and pathological tooth wear. In this group of vomiting bulimics, other factors that could influence the wear process, such as saliva, warranted investigation.

The role of salivary lubrication and buffering in maintaining oral health was discussed in the literature review chapter. However, there is a dearth of information on saliva in the eating disorders and those results that are available are contradictory or uncorroborated. Thus, although Hellstrom (1977) found stimulated flow rate, unstimulated pH and buffering capacity, stimulated pH and buffering capacity all to be normal in her group of 39 vomiters and non-vomiters, she believed the low resting flow rate was important and occurred secondary to dehydration or xerostomia induced by medication. Analysis of hydration levels or drug therapies was not undertaken in her study, and furthermore since stimulated flow rates were normal, the subjects were probably not dehydrated or xerostomic. She reported no significant association between erosion (tooth wear) and any of the salivary factors.

Unstimulated and stimulated salivary concentrations of potassium, chloride, calcium, urea nitrogen and salivary

albumin were no different between vomiting bulimics and age matched controls (Tylenda et al., 1991). Unfortunately, these authors did not state what was the tooth-wear status of their 15 bulimics. Moreover, less than half the bulimics provided salivary data and then the incorrect statistical tests were applied. Recalculation of their stimulated potassium concentration data by this author revealed significantly higher parotid potassium concentration but significantly lower submandibular concentration in the bulimics.

Liew et al. (1991) compared stimulated salivary flow rates and salivary pH between 15 anorexics and 15 controls. They reported no significant difference in flow rates but a significantly lower pH in the anorexics, although the mean pH value of 7.1 is within the normal range (pH 5.0-8.0, Edgar, 1992). Only one of their subjects exhibited erosion, which was confined to enamel.

Stimulated salivary flow rates in bulimics and controls were no different, although the rates were at the lower end of the range (Touyz et al., 1993).

One result, reported in the previous chapter was that unstimulated salivary buffering capacity was normal. However, unstimulated salivary flow is not relevant to the act of vomiting. Oesophageal distension after binge eating and chemical irritation to the gastric mucosa leading to

nausea are powerful stimuli to salivation (Jenkins, 1978). Additionally, the vomiting centre in the medulla is connected to salivary nuclei giving rise to copious reflex salivation prior to vomiting (Edgar, 1992). Unstimulated flow rates vary widely between subjects and the bicarbonate ion concentration is too low to be an effective buffer (Edgar & O'Mullane, 1990). Therefore, in vomiting bulimics, it is more important to investigate salivary factors after maximal salivary stimulation.

The aim of this study was to compare the stimulated salivary factors of flow rate, pH and bicarbonate in vomiting bulimics with and without pathological tooth wear. The importance of intra-oral lubrication to maintain oral health was discussed in Section 2.2.1c. The viscosity of saliva is a measure of the mucin content. Another aim of this study, therefore, was to assess salivary lubrication by viscosity measurement. It was also decided to assess the influence of the salivary pellicle on enamel acid attack since pellicle protected enamel from erosion *in vitro* (Nieuw Amerongen *et al*. 1987; Meurman & Frank, 1991; Millward *et al*. 1992). The susceptibility to acid erosion may be different in individuals with tooth wear. To this end calcium determination in surface enamel was carried out. Local Ethical Committee approval was gained for this study. Copies of the documentation, patient information sheets and consent forms are presented in Appendix 3.

6.2 MATERIALS AND METHOD

6.2.1 Subjects

Nineteen bulimics all of whom practised self-induced vomiting took part in this study. All subjects were appraised of the purpose and the nature of the study. Informed consent to participate was gained from subjects after they had read the information sheets and discussed the procedures with the author. One subject was on hormone replacement therapy and 15 were taking the oral contraceptive pill. No other medications were taken.

The subjects were separated into two groups (1) pathological tooth wear present (N= 9) and (2) pathological tooth wear absent (N= 10) on the basis of the Tooth Wear Index (TWI).

6.2.2 Saliva Collection

Saliva collections were made between 11.00 a.m. and 3.00 p.m. Subjects had not eaten, drunk, smoked or vomited for at least an hour prior to saliva collection. They were asked to chew a stick of spearmint flavoured gum (Orbit Sugar Free, Wm. Wrigley Co. Ltd., Plymouth) and gently dribble into a preweighed Sterilin pot. This method of stimulating saliva by chewing gum has been established in the Department of Clinical Dental Sciences at Liverpool by

Professor W M Edgar and Mr L Dawson. Each subject chewed the gum for a total of nine minutes, with three, three minute collections having been made. The pots were stoppered at the end of each time interval. Flow rate, pH and bicarbonate analyses were performed within fifteen minutes of saliva collection and enamel biopsies. Thereafter, the saliva samples were frozen at $-5^{0}C$ until viscosity measurements were undertaken.

6.2.3 Enamel biopsy technique

In order to determine the calcium content of surface enamel and, therefore, susceptibility or resistance to acid erosion an enamel biopsy technique was developed. A modification to the technique described by Spector & Curzon (1978) was devised by the author.

Whatman filter paper discs (Whatman Scientific Limited, Springfield Mill, Maidstone, Kent, UK) were cut with a leather punch into 3mm diameter circular discs. A 0.1mol/L solution of perchloric acid ($HClO_4$) was made as the enamel etchant. Perchloric acid is non-volatile, has a low fluoride concentration and does not leave a precipitate of calcium phosphates on the tooth surface. The chosen concentration was within the range for gastric acid (Guyton, 1991). From laboratory experiments on extracted teeth, the optimum volume of acid was found to be 1 μL with an etch time of 60 seconds. A greater volume of acid ran

beyond the paper disc and the 60 second time resulted in good calcium estimates (see later).

6.2.4 Enamel biopsy - clinical procedure

Since the susceptibility of enamel to acid erosion was to be determined in this procedure the teeth to be biopsied had to be accessible, have unworn enamel and be readily remineralizable/cleansable. The labial surfaces of the lower central incisors fulfilled all these criteria. All subjects were supine. The lower right central incisor was isolated with cotton wool rolls in the labial sulcus, the filter paper placed centrally on the labial surface and the tooth biopsied by dropping 1μL of $HClO_4$ from a Hamilton syringe onto the disc. After etching for 60 seconds the disc was removed and stored in a stoppered bottle. The procedure was repeated on the lower left central incisor, but, in order to remove the salivary pellicle, the labial surface was thoroughly cleaned by pumice in a rotating bristle brush. After etching, the surfaces were thoroughly washed with water from the triple syringe, dried and Duraphat fluoride varnish (Inpharma AS, Drammen, Norway) applied in order to aid remineralization.

6.2.5 Saliva flow rate, pH and bicarbonate analysis

The three stoppered Sterilin pots with saliva were reweighed and the flow rate calculated. The salivary pH was measured with an Orion pH meter (Orion Research Inc., Boston, USA). The salivary bicarbonate was measured with a Model 600 Natelson Microgasometer. Natelson (1951) described a micromanometric method for the estimation of plasma carbon dioxide based on the principles of the Van Slyke-Neill method (1925). Gas pressure (CO_2) is measured under constant volume, thus results are independent of atmospheric pressure. Temperature is the only operating variable which is corrected for by a set of conversion factors. The carbon dioxide is released from bicarbonate (HCO_3) by introducing 0.03 mls of lactic acid in the reaction chamber. The microgasometer was calibrated before use with a standard 30 mmol/L sodium bicarbonate. Then 0.03 mls of each saliva sample was pipetted followed by 0.03 mls of lactic acid, 0.01 mls of anti-foaming agent and 0.1 mls of distilled water according to manufacturer's instructions. The average water pressure at the ambient room temperature was subtracted from the observed pressure and this figure was multiplied by the factor to gain the bicarbonate (HCO_3^-) concentration.

6.2.6 Viscosity Measurements

The viscosity measurements were made in a Model DV-I Brookfield digital viscometer (Brookfield Engineering Laboratories, Inc. Stoughton, Massachusetts, USA). The spindle rotational speed was set according to manufacturers' instructions at 60 r.p.m. utilising the small sample chamber (SC4 18/13 R) at a shear rate of 1.32N. The minimum sample volume in this chamber is 8ml, and therefore saliva samples were pooled. The saliva samples were carefully poured into the chamber to avoid bubble formation and all measurements were made at room temperature. Relatively constant viscometer readings were obtained after a few minutes and this value was multiplied by the factor for the spindle/speed combination. For the saliva values this proved to be 0.5.

The viscosity of gum-stimulated saliva from two healthy volunteers was measured in the viscometer in order to determine technique reproducibility. To check the effect, if any, of sorbitol on viscosity, plain gum base was also chewed by the volunteers. The viscosities of gum base and Orbit stimulated salivas were compared by Student's t-test and found not to be different. It was felt that study acceptability and compliance by the bulimics would be greater with a proprietary brand of gum such as Orbit. Furthermore, the gustatory stimulus would be greater from flavoured gum rather than gum base or paraffin wax. This

would hopefully, produce the optimal flow rate. Unfortunately, an international standard stimulus has not been deployed despite its recommendation (G N Jenkins in Edgar & O'Mullane, 1990).

6.2.7 Atomic absorption spectrophotometry (AAS) of enamel biopsy samples

This has become a well accepted method for determining the concentration of elements in solution. The main advantage of using AAS is that it is possible to quantify ultra-trace and minor concentrations with high precision and accuracy at relatively low cost.

Radiation of a wavelength characteristic of the element is generated by a hollow cathode lamp and passed through a flame. A solution of the sample, in the form of an aerosol, is sprayed into the flame, vaporised, and the degree of absorption of the radiation is measured.

Filter digestion was carried out by placing each Whatman filter paper sample and blanks in an acid washed polytetrafluoroethylene (PTFE) beaker to which was added 5 mls of concentrated nitric acid. Filter digestion was aided by placing the beakers on a hot plate at 120^{0}C for one hour which was followed by overnight evaporation down to a "bead". This was placed in a 10 mls volumetric flask made up to the mark with 0.1N nitric acid. Each subject had two

enamel biopsies giving a total of 38 samples.

A Perkin Elmer AA 2380 "flame" spectrophotometer with an Intensitron "multi-element" lamp and a nitrous oxide/acetylene flame was used for all analyses. The six Whatman filter papers used as blanks produced negligible calcium readings ranging from 0.03 to 0.08 μg/ml (mean = 0.05 μg/ml, SD = 0.02).

Calcium standards were prepared in 0.2, 0.5, 1.0 and 2.0ppm concentrations and a calibration curve plotted. All standards, blanks and samples were run three times through the spectrometer, and an average taken for each calcium reading. The dissolved calcium concentration in the enamel biopsies was determined from the calibration curve.

6.3 RESULTS

The mean age of the tooth-wear present group was 28.6 years and in the tooth-wear absent group it was 27.3 years (Table 6.1). Student's t-test found no significant difference between the ages of these groups.

The flow rates, pH and bicarbonate concentrations are presented in Tables 6.2 and 6.3. The flow rates for each three minute collection are presented in the first three columns. The mean overall flow rate for the 9 minutes is shown in the fourth column. These figures are within the normal range for all subjects with tooth wear (Table 6.2). Three subjects without tooth wear (K,M,O) have a lower than normal flow rate (Table 6.3). However, no significant flow rate differences were found between the two groups ($t=1.54$, $p=0.14$). Additionally, there were no significant differences between the groups for the initial flow rate ($t=1.47$, $p=0.16$).

The pH results are presented in the second set of columns in Tables 6.2 and 6.3. All pH results are within the normal range for stimulated saliva of 5.0-8.0 (Edgar, 1992).

The pH is reflected in the bicarbonate concentration of the saliva, which of itself is an expression of buffering capacity. The bicarbonate concentrations for all three

TOOTH WEAR	M	F	TOTAL	MEAN AGE (Years)	SD (Years)	INTERQUARTILE AGE RANGE (Years)
PRESENT	1	8	9	28.6	3.01	26-31
ABSENT	0	10	10	27.3	12.37	18-30

t=0.31, df=17, p=0.76 No significant difference

Table 6.1 Age and gender by tooth-wear group

SUBJECT	FLOW RATE (mls/min)			INDIVIDUAL SUBJECT'S MEAN OVERALL FLOW RATE	pH			INDIVIDUAL SUBJECT'S MEAN OVERALL pH	BICARBONATE (mmol/l)			INDIVIDUAL SUBJECT'S MEAN OVERALL BICARBONATE
	1ST COLLECTION	2ND	3RD		1ST COLLECTION	2ND	3RD		1ST COLLECTION	2ND	3RD	
A	1.54	0.98	0.98	1.17	6.44	6.82	6.82	6.69	6.30	6.50	6.50	6.43
B	4.11	2.06	2.38	2.85	7.18	7.11	7.15	7.15	16.40	10.20	9.50	12.03
C	2.90	1.08	0.91	1.63	6.82	7.17	6.86	6.95	8.20	5.40	1.70	5.10
D	2.39	1.37	1.24	1.67	6.65	6.76	6.69	6.70	6.67	3.46	2.10	4.08
E	2.50	1.50	1.60	1.87	6.74	7.12	7.21	7.02	3.10	6.20	9.40	6.23
F	2.38	1.23	1.41	1.67	7.00	7.19	7.12	7.10	9.10	7.00	5.10	7.06
G	1.90	1.60	1.50	1.67	7.05	7.46	7.49	7.33	8.90	12.60	10.80	10.77
H	3.12	3.09	2.34	2.85	6.88	7.15	7.17	7.07	6.30	10.20	7.80	8.10
I	2.71	1.67	1.27	1.88	6.94	7.26	7.19	7.13	8.50	11.20	8.90	9.53
N = 9												
Mean collection values	2.62	1.62	1.51	1.92	6.86	7.12	7.08	7.02	8.16	8.08	6.87	7.70
(Standard deviation)	(.74)	(.64)	(.53)	(.57)	(.22)	(.21)	(.25)	(.21)	(3.6)	(3.06)	(3.29)	(2.64)

TABLE 6.2 Individual values for salivary factors in the tooth-wear present group with means by subject and by collection.

SUBJECT	FLOW RATE (mls/min)			INDIVIDUAL SUBJECT'S MEAN OVERALL FLOW RATE	pH			INDIVIDUAL SUBJECT'S MEAN OVERALL pH	BICARBONATE (mmol/l)			INDIVIDUAL SUBJECT'S MEAN OVERALL BICARBONATE
	1ST COLLECTION	2ND	3RD		1ST COLLECTION	2ND	3RD		1ST COLLECTION	2ND	3RD	
J	2.19	1.73	1.41	1.78	7.02	7.19	7.33	7.18	9.30	9.70	10.0	9.67
K	0.89	0.44	0.39	0.57	6.96	7.09	7.13	7.06	5.60	2.50	2.00	3.37
L	2.81	0.83	1.41	1.68	6.65	7.19	6.73	6.86	5.60	4.70	3.40	4.57
M	0.77	0.55	0.51	0.61	6.97	7.29	7.30	7.19	5.20	10.50	10.60	8.77
N	3.71	2.46	2.26	2.81	7.21	7.36	7.36	7.31	14.40	17.30	15.80	15.83
O	1.16	0.49	0.63	0.76	7.18	7.36	7.35	7.30	8.90	7.30	7.30	7.83
P	3.04	1.90	1.70	2.21	6.56	6.89	7.00	6.82	4.30	5.40	5.90	5.20
Q	2.57	1.39	1.48	1.81	6.53	6.96	7.26	6.92	3.10	5.50	6.50	5.03
R	1.34	0.82	1.00	1.05	5.89	6.40	6.51	6.27	<1.00	1.00	1.11	1.04
S	1.69	0.83	1.10	1.21	7.43	7.98	7.89	7.77	15.90	20.00	17.50	17.80
N = 10												
Mean collection values	2.02	1.14	1.19	1.45	6.84	7.17	7.19	7.07	7.33	8.39	8.01	7.91
(Standard deviation)	(1.0)	(.69)	(.58)	(.74)	(.44)	(.40)	(.38)	(.39)	(4.8)	(6.19)	(5.51)	(5.36)

TABLE 6.3 Individual values for salivary factors in the tooth-wear absent group with means by subject and by collection.

collections and individual overall means are shown in the final set of columns in Table 6.2 and 6.3. Only subject R in the tooth-wear absent group had a low bicarbonate concentration. Once again there were no significant bicarbonate concentration differences between the two groups.

The salivary viscosities, measured in Centipoise (cps), are shown in Table 6.4. There was a significantly greater viscosity in the tooth-wear present group ($t=2.30$, $p<0.05$). Three subjects (A,E,I) in the tooth-wear group had very high viscosity values. Six tooth wear subjects had viscosity values ≥ 5.0 cps as compared to only one non tooth-wear subject. Only two tooth-wear cases exhibited intermediate viscosity in the range 4.5 to 4.9 cps whereas four non tooth-wear subjects were in this category. Finally, only one tooth-wear subject (G) had a viscosity less than 4.5 cps, whereas three subjects in the non tooth-wear group (N,O,S) were in this category. This frequency distribution is presented in tabular form in Table 6.5. Two subjects (K and M) had a low volume of collected saliva and hence a low flow rate. Consequently there was insufficient saliva for viscosity analysis.

Table 6.6 presents the calcium dissolved in perchloric acid during the enamel biopsy procedure. These results represent the calcium concentration released from surface enamel taken from the labial uneroded/unworn surface of the

TOOTH WEAR PRESENT GROUP	SUBJECT	VISCOSITY (Centipoise)
	A	11.0
	B	5.6
	C	6.0
	D	4.6
	E	13.1
	F	5.1
	G	4.4
	H	4.7
	I	12.0
	N = 9	Mean=7.39 (SD=3.56)
TOOTH WEAR ABSENT GROUP		
	J	4.9
	K	-
	L	5.1
	M	-
	N	4.2
	O	4.3
	P	4.5
	Q	4.5
	R	4.5
	S	3.7
	N = 8	Mean=4.46 (SD=0.43)

Table 6.4 Viscosity measurements of saliva in 17 vomiting bulimic subjects

		TOOTH WEAR	
		PRESENT	ABSENT
VISCOSITY cps	≥ 5.0	6	1
	4.5-4.9	2	4
	<4.5	1	3

Table 6.5 Frequency distribution of whole saliva viscosity in 17 vomiting bulimic subjects

TOOTH WEAR PRESENT GROUP	CALCIUM CONC. WITH PELLICLE µg/ml	CALCIUM CONC. WITHOUT PELLICLE µg/ml
SUBJECT		
A	0.11	0.20
B	0.28	0.29
C	0.29	0.29
D	0.10	0.20
E	0.28	0.24
F	0.30	0.28
G	0.31	0.28
H	0.28	0.25
I	0.28	0.28
N = 9	Mean=0.25 (.08)	Mean=0.26 (.04)
TOOTH WEAR ABSENT GROUP		
J	0.26	0.30
K	0.33	0.28
L	0.35	0.31
M	0.28	0.33
N	0.31	0.29
O	0.33	0.24
P	0.29	0.31
Q	0.34	0.29
R	0.34	0.36.Mean=0.30(.03)
S	0.35	0.79
N = 10	Mean=0.32 (.03)	Mean=0.35 (0.16)

Table 6.6 Dissolved calcium concentrations of surface enamel by tooth wear group with and without pellicle

lower right central incisor (with pellicle *in situ*) and the lower left central incisor (with pellicle removed). Comparison of the results with and without pellicle *within each tooth wear group* was not statistically significant. However, a significantly higher amount of dissolved calcium occurred in the tooth wear absent group with pellicle compared to the tooth wear present group with pellicle ($t= 2.52$, $p< 0.05$). The high value for the "outlier" subject S (0.79 μg/ml) probably reduced the level of significance to $p= 0.11$ when comparing the two groups without pellicle. Exiting this one data point resulted in a significantly higher dissolved calcium concentration in the tooth wear absent group ($t= 2.40$, $p < 0.05$).

6.4 DISCUSSION

The main results in this chapter are that in a group of vomiting bulimics with tooth wear gum-stimulated salivary flow rate is low but pH and buffering capacity are normal. A significantly higher salivary viscosity was found in bulimics with tooth wear compared to those without tooth wear, whereas dissolution of enamel calcium was significantly lower.

The higher salivary flow rate for the first collection is both predictable, given the initial gustatory (taste) and masticatory (mechanical) stimuli. The masticatory stimulus from chewing is prolonged compared to the diminishing gustatory drive which, as the gum contents leach out reduces, but is not entirely lost. The initial three minute mean flow rates of 2.62ml/min for the tooth wear present group and 2.02ml/min for the tooth wear absent group are lower than the 4ml/min reported by Dawes and Macpherson (1992). This was for a similar sorbitol mint flavoured chewing gum (gum no. 2), and an average rate calculated from their initial three 1 minute collections taken from 22 "normal" subjects of similar age.

Stimulated salivary flow in 11 healthy volunteers carried out at Liverpool using the same method resulted in a mean initial 3 minute flow rate of 3.63 ml/minute. However, the

subsequent 6 and 9 minute flow rates (1.84 and 1.55 ml/min) are similar to the results presented in this thesis.

The mean pH and mean bicarbonate results for both groups are within the normal range but lower than the means obtained from the 11 "normal" subjects (personal communication, Luke Dawson, 1993).

The eight bulimics without tooth wear had a mean viscosity of 4.46 centipoise (cps) which is similar to the 4.2 cps reported by Veerman et al. (1989) for fresh, unhomogenised human whole saliva. It has been well established that the submandibular-sublingual (SM-SL) salivary secretions are more mucinous and thus more viscous than the parotid secretion. The significantly higher viscosity in those bulimics with tooth wear is an interesting result. The frequency of pathological tooth wear was significantly greater above a threshold of 1100 vomiting episodes (see previous Chapter, Table 5.10). Histopathological changes occurring in the salivary glands after this number of vomiting episodes could result in a difference in salivary secretion and thus viscosity. One of two possibilities may account for this difference. Either the serous parotid secretions are reduced or conversely the SM-SL (mucin) secretions are increased. There may be a difference in the pattern of gland stimulation, since the sympathetic nerves stimulate mucin and amylase production, and the para-sympathetic supplies water and electrolytes. Bilateral

parotid enlargement has been reported in bulimics (Levin et al., 1980; Hasler, 1982; Burke, 1986; Taylor & Sneddon, 1987; Philipp et al., 1991). Although none of the subjects in this study had complained nor presented with obvious parotid swelling, subclinical parotid change may have occurred. Moreover, parotid enlargement was episodic (Levin et al., 1980) and resulted in reduced parotid salivary flow in malnourished African subjects (Du Plessis, 1956).

Reduced salivary flow in the bulimics did not result in reduced buffering capacity. Reduced stimulated salivary flow with normal pH and buffering capacity was reported in malnourished Indian children compared to Swedish controls (Johansson et al., 1992). Conversely, the Swedish children had a significantly lower buffering capacity despite a normal stimulated secretion rate. These authors believed dietary composition affected the salivary buffering ability. Dietary factors have not been assessed in relation to tooth wear and bulimia in this thesis. The frequency of fizzy drink consumption as well as the quantity and the type of foods eaten may influence salivary composition and gastric acidity. Further work is needed in this area.

Some salivary gland structural change may be induced by metabolic disturbance but only after a certain duration of bulimic behaviour. The commonest metabolic change

associated with bulimia is hypokalaemia. Serous acini are more susceptible to metabolic disturbance than mucous acini (Du Plessis, 1956; Batsakis & McWhirter, 1972). A threshold of 1100 vomiting episodes could be related to both increased frequency of tooth wear and metabolic disturbance leading to parotid gland change with a reduction in serous output. Unfortunately, the flow rates in the wear and non-wear groups were not significantly different, albeit the initial flow rate in both groups was low compared to healthy volunteers.

Although Mannerberg (1963) measured viscosity by a relatively crude method, significantly greater salivary viscosity was found in subjects with 'local erosions' (observed on anterior labial surfaces) compared to controls. Presumably these sites were eroded because of exogenous acid attack but nonetheless, the results presented here are supported by Mannerberg (1963). Conversely, salivary viscosity from 11 subjects with labial erosion was not significantly different to 39 healthy controls as measured by a viscometer (Woltgens et al., 1985).

Apart from the metabolic effect bulimia nervosa may have on the parotid salivary glands, the increased salivary viscosity in bulimics with tooth wear could be the cause of the tooth wear or a direct effect from vomiting. The viscosities of dog tracheal mucin and rat goblet cell mucin

increased in the presence of calcium (Marriott et al., 1979; Forstner & Forstner, 1976) whereas human salivary viscosity decreased slightly with increased calcium concentration in vitro (Veerman et al., 1989). During vomiting, the demineralised calcium from the teeth may bind to the sialic acid in the mucin and so increase viscosity. Sialic acid in mucin has been shown to bind calcium (Jaques et al., 1977). However, this process would probably only occur during the vomiting period and for a short time afterward. The effect of lowering salivary pH resulted in increased mucin viscosity in vitro (Veerman et al., 1989). Whether the saliva undergoes a prolonged viscosity increase as a result of these processes secondary to vomiting is unknown. The more viscous saliva could, therefore, be a consequence of the vomiting process, the subsequent acid erosion, or salivary gland change secondary to metabolic disturbance.

Furthermore, the potential for enamel and dentine remineralisation may be reduced as a consequence of the calcium binding to salivary mucin. Salivary mucin has also been reported to be an effective inhibitor of dental calculus formation (Kusunoki, 1940). Kusunoki stated that salivary glycoprotein acted to inhibit precipitation of calcium phosphates. Presumably the more viscous the saliva the greater the potential for saliva to bind demineralised calcium from the tooth surface and the less the propensity for remineralisation.

Could the viscosity account for the site distribution of the erosion ? Intra-oral acid clearance and the site specificity of caries has been related to salivary film velocity (Dawes & Macpherson, 1993). The normally high salivary film velocity in the upper palatal region may be significantly reduced by the viscosity increase leading to erosion particularly in the palatal region. Whether the more viscous saliva in the tooth-wear group has a profound influence on the site distribution as well as the incidence of tooth wear is a matter of conjecture.

The enamel biopsies yielded puzzling results which are difficult to account for. The mean lower calcium dissolution in the tooth wear present group could indicate (1) that the enamel had reduced solubility in acid, or (2) that the enamel had less calcium i.e. was hypo-mineralised. Obviously, the tooth wear group did not have reduced enamel acid solubility. Hypo-mineralised enamel would however reflect an increased solubility because of increased protein content and greater porosity volume at prism junctions. Prism borders provide the main entry route of acid and exit path of dissolved hydroxyapatite (Robinson, 1987). In the more porous regions, located at prism borders and tail areas, the higher ratio of inter-crystalline pore volume to crystal surface area resulted in faster dissolution of mineral (Shellis & Hallsworth, 1987). Although the lower incisor surfaces appeared unworn, some erosion may have taken place, causing enamel porosity.

Salivary protein deposited in the porous enamel would protect the enamel from further acid attack and thus the enamel biopsy data may not reflect the enamel status when the tooth wear started. This result is difficult to interpret and this part of the study needs to be replicated.

Slightly more calcium was removed from the pellicle-free surface in both groups but this was statistically non-significant. In vitro pellicle from clarified human whole saliva protected enamel against acid erosion from a cola beverage (Meurman & Frank, 1991). Their pellicle was grown for seven days and they admitted to having a much thicker pellicle than would occur in vivo. Nonetheless, it was postulated that salivary pellicle would be protective for erosion as is the case with in vitro caries (Zahradnik et al., 1976 & 1977). The labial surfaces of the lower central incisors were chosen because they appeared unworn/uneroded. The pellicle from unworn/uneroded sites may be different in thickness or quality from pellicle on eroded/worn surfaces. Affected sites, such as the palatal aspects of the upper anterior teeth, may have thinner pellicle as a consequence of tongue rubbing. The combined effects of thin pellicle and tongue rubbing act as vulnerability/ susceptibility factors, the acid attack being the precipitating factor leading to wear in this region.

CHAPTER 7

GENERAL DISCUSSION

GENERAL DISCUSSION

That tooth wear has become a major concern to dentists is emphasised by its inclusion in the recent National Child Dental Health Survey. General dental practitioners, community dentists and hospital based clinicians are seeing more cases of tooth wear, not just in the elderly but also in the young. Irrespective of which end of the age scale, the treatment can be complex and, therefore, expensive. Furthermore, once restorative treatment has been carried out, maintenance of orodental health is made more demanding. Knowledge of prevalence rates, aetiological factors and associated risk, predisposing conditions (e.g. medical and dental status), tribology, and the compensating mechanisms of the tooth wear process is relatively limited.

The aim of this thesis has been to assess the prevalence and the risks of getting tooth wear from the known tooth wear aetiologies in a sample of 14 year old school children. The association of tooth wear with vomiting, oral hygiene practices and salivary factors in the eating disorders has also been investigated.

Much of the information obtained from the results in the foregoing chapters has already been discussed in detail.. It is intended in this final section to present an overall picture of the results obtained, together with the general conclusions, implications for management and some

recommendations for future research.

The sites of dentine exposure in 30% of the sampled 1035 14 year olds were palatal on the upper anteriors, occlusal on the lower first molars and incisally on the upper and lower anteriors. A similar pattern of wear into dentine is also seen in the vomiting bulimics. If we accept that upper palatal wear is primarily of an erosive nature, although the contribution by solid foods or the tongue to an abrasive process is still not clear, then it seems that the susceptible sites are the same whether from exogenous or endogenous acid attack. Indeed, Jarvinen et al. (1992) came to the same conclusion.

Rugg-Gunn (1993) cited the variation in resistance to acid attack between teeth in vitro reported by Davis and Winter (1980) and mentioned the unknown role that water fluoridation may play in reducing susceptibility to erosion. A 19 year old vomiting bulimic, with 'mild' dental erosion, had significant levels of fluoride in surface enamel biopsies and in stimulated saliva after daily use over three weeks of fluoride mouthrinse (Jensen et al., 1987). Perhaps more significant, was the high value for salivary fluoride before the introduction of the rinse regimen and the normal values of fluoride obtained from the one mildly eroded palatal aspect of an upper incisor. These authors concluded, from this single case, that good oral hygiene and use of fluoride toothpastes in

bulimics minimised the erosive effect of gastric contents. However, worn sites on teeth from an area with a highly fluoridated water supply did not have a high surface fluoride content (Weatherell et al., 1973) and the protective effect from topical fluoride on erosion sites was judged to be minimal (Xhonga & Sognnaes, 1973). It is intended to analyse the fluoride concentration in saliva from the vomiting bulimics who participated in this study.

The follow up study of individuals with an eating disorder reported in Chapter 5 found no difference in fluoride toothpaste use between subjects with and without pathological tooth wear. This is perhaps not surprising, since most toothpastes contain fluoride and eating disorder subjects tend to obsessive behaviour with good standards of plaque control (Milosevic & Slade, 1989). Fluoride mouthrinses, supplementary to toothpaste, will promote remineralisation of eroded surfaces with fluorapatite but will not prevent erosion continuing in those individuals where other susceptibility factors are present.

The oral hygiene practices were not different between case and control group in the 15 year old school children. The influences of oral hygiene practices on tooth wear were reported by Robb (1992). Brushing more than twice a day produced more tooth wear on posterior buccal and cervical surfaces (both upper and lower) than in those who brushed less than once a day. Sensodyne users had significantly

less wear than those using ordinary toothpaste although toothbrush texture (softness/hardness) did not result in any wear difference. It would seem that the influence of oral hygiene practices on tooth wear is limited and in any event the upper palatal sites are not those normally associated with tooth brushing.

An explanation for the pattern of dentinal exposure follows. The upper and lower incisal edges have a small surface area, are the first teeth to erupt and provide, in an Angles' Class I incisal relationship, the guiding surfaces for protrusive mandibular movement. The canines provide canine guidance on lateral mandibular excursions. Furthermore, this wear would probably be accelerated by grinding and drinking acidic beverages. The influence of occlusal relationships on tooth wear has, by and large, not been assessed. The reasons mentioned above would probably account for the incisally exposed dentine in the 30% of sampled children. The occurrence of dentinal exposure on the upper palatal surfaces and on the occlusal surfaces of the lower first molars in 8% of sampled children is more difficult to explain. Drinks and regurgitated/vomited gastric contents probably flow over the upper anterior palatal surfaces. Cineradiographic studies of the intra oral path taken by such activity have not been undertaken. The site specificity of tooth wear requires further investigation.

Interestingly, unilateral mastication, known as the 'preferred side of chewing' (Rugh and Smith, 1988) does not seem to manifest as unilateral tooth wear. The examination of 856 individuals from Britain (including British, Asians and Chinese) found no evidence for left or right-sided occlusal tooth wear (Cruwys, 1987). Although Robb (1992) found tooth wear to be an almost universal condition he did not state whether the occlusal wear was more prevalent on one side of the mouth. Furthermore, despite the majority of the population being right-handed, cervical or buccal wear was not greater on the left side of the mouth. The incisal/occlusal and lingual tooth wear distribution is symmetrical in the sample of 1035 school children (Figures 3.2 and 3.3) presented in this thesis.

'Grinding and clenching' and 'drinking fizzy drinks on most days' resulted in increased risk of tooth wear in 15 year old school children followed up in the case control study. This increased risk, expressed as an odds ratio (O.R.) with the 95% confidence interval, was 2.5 (0.94-6.69) for 'grinding and clenching' and 2.0 (0.77-5.06) for 'drinking fizzy drinks on most days'. The confidence intervals represent a range of plausible values for the population (Gardner & Altman, 1990). The wide confidence intervals presented in this thesis range from no risk (O.R.≤1) to a significantly increased risk. This wide range is reflected in the relatively small size of both the case and control group (50). Greater precision in the determination of the

odds ratio and decrease in the width of the confidence interval is achieved by increasing the sample size (Gardner & Altman, 1990). This case control study should be repeated with larger sample sizes, possibly as a multi-centre study. The frequency categories for food/drink consumption used in the questionnaire may not be sufficiently sensitive to identify those subjects most at risk of getting tooth wear. A diet sheet recording every fluid intake over a certain period would probably have been more precise in identifying frequent fizzy drink consumption.

The lack of association between the frequency and duration of vomiting with tooth wear in bulimia nervosa was an unforseen result. The reasons for this result are unclear. Dentists probably automatically associate vomiting with erosion and may forget that other factors such as frequent fizzy drink consumption could also be at play.

The time interval after finishing a meal up to the act of vomiting may be an important factor in determining gastric acidity, as may the buffering of gastric acid by the food itself or conversely, any additive effect from acidic foods. The bulimics participating in the studies for this thesis have completed diet sheets, but dietary analysis has not yet been undertaken. The uneven distribution of pathological tooth wear in alcoholics, believed to be caused by sub-clinical regurgitation (Robb & Smith, 1990)

is further evidence of the complex nature of the erosive tooth wear process.

The site specificity of caries and supragingival calculus was related to salivary film velocity, sugar clearance and the local effect of the minor salivary glands (Dawes & Macpherson, 1993). Fluoride clearance was much more rapid lingually than buccally (Weatherell et al., 1986) and the half-life for the diffusion of potassium chloride was very short in the upper palatal areas (Lecomte & Dawes, 1987).

Although the buffering capacity for expectorated whole saliva reported in Chapter 6 was within the normal range, in vomiting bulimics with tooth wear, the more viscous saliva could have a slower film velocity with a concomitant reduction in acid clearance and buffering capacity on the palatal areas. To establish whether the increase in viscosity is due to a reduction in serous parotid output the parotid flow rate should be measured. The known lubricating ability of the proline-rich glycoprotein of the parotid saliva (Hatton et al., 1985) should be assessed as well as quantifying the MG1 fraction of unstimulated and stimulated whole saliva from tooth wear subjects and controls. Laboratory-based techniques for quantifying MG1 and MG2 have been devised (Denny et al., 1991). Whether there is a more viscous saliva in children or in adults with tooth wear is not known. 'Thick' dental plaque was proposed to protect from dental erosion as well as the

stored phosphate in the plaque matrix (McIntyre, 1992). However, plaque is not a feature of non-eroded sites in mouths with erosion elsewhere and the protection from plaque would be confined to the area where plaque accumulates, interproximally and above the gingiva.

The unstimulated and stimulated salivary buffering capacity reported in Chapters 5 and 6 of this thesis was normal. These results are in agreement with Jarvinen et al. (1991) who also reported normal mean unstimulated and stimulated flow rates in males and females with and without erosion. The stimulated salivary flow rates were low in the vomiting bulimics presented in this thesis. It is not clear why Jarvinen et al. (1991) believed that low unstimulated salivary flow rate was associated with dental erosion since only seven cases and six controls (of 106 and 100 subjects respectively) had this problem. As discussed in Chapter 6, the very act of eating, drinking and vomiting would stimulate saliva and, therefore, the factors associated with stimulated saliva are of more importance.

The aetiological factors associated with dental erosion having the highest odds ratios were eating citrus fruit more than twice a day (O.R.=37) and vomiting more than once a week (O.R.=31) (Jarvinen et al., 1991). Individuals indulging in self-induced vomiting are undoubtedly at higher risk of getting tooth wear, but the association, reported in chapter 5, is not linear.

Jarvinen et al., (1991) mentioned that their study may have overestimated the importance of some risk factors, but for reasons other than those stated. Their erosion criteria after Eccles and Jenkins (1974) probably included cases of abrasion and attrition. Furthermore, their cases were sent to the University Dental Clinic because they had erosion and presumably were told so, therefore, their recollection or awareness of risk factors would have been greater than the controls. This is another reason for replication of the case control study and validation of the odds ratios.

In order to relate and tabulate the various aetiological factors involved in tooth wear, a chart is presented in Figure 7.1. Despite the presence of one or more of these factors, some individuals remain free of pathological tooth wear. Conversely, it can be difficult to diagnose the cause in referred cases with severe tooth wear. Bizarre practices such as the recently described voluntary reflux phenomenon are coming to light (Gilmour & Beckett, 1993). Stress or drug therapy may produce subtle changes in saliva that either directly or indirectly lead to tooth wear. The inter-relationship of the various factors complicates our understanding of the tooth wear process. Furthermore, the progress of tooth wear is probably episodic and thus a prediction of future tooth wear status is difficult. In other words, an individual's rate of tooth wear will vary and a projection for tooth wear status from a tooth wear profile at one point in time may lead to an erroneous

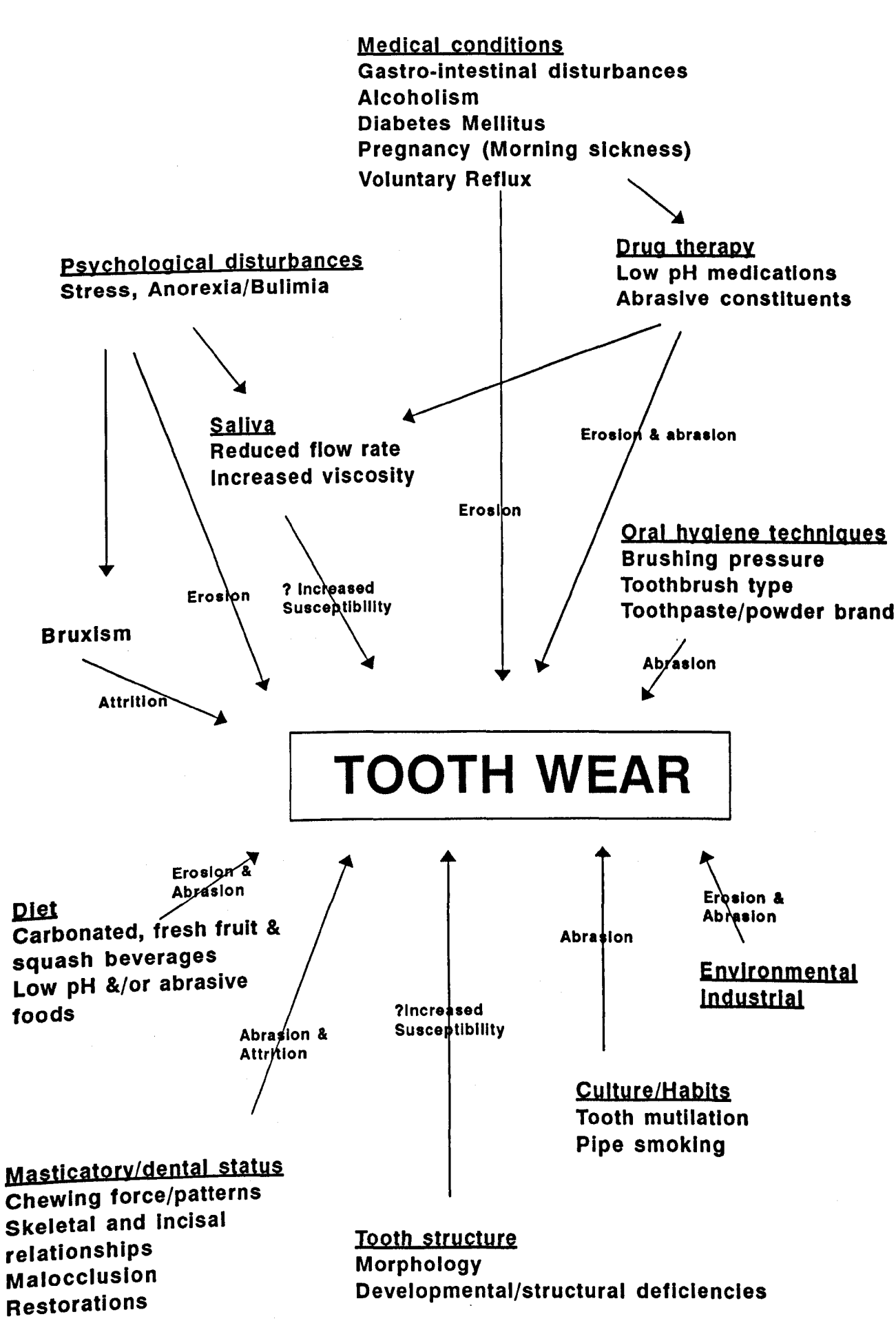


Figure 7.1 The aetiological and possible influential factors of tooth wear

prediction. Regular examination for and charting of tooth wear in order to identify early tooth wear and monitor it's progress may become routine dental practice especially for younger individuals. If the susceptible sites are palatal of the upper anteriors and occlusal of the first molars, then these should be the sites for preferential examination.

The problems surrounding the definitions of attrition, abrasion and erosion were discussed in the literature review section. The European Standards Committee for dental terminology should define these terms precisely and include them in a new standard.

The mechanisms of tooth wear require study with respect to the fundamental tribological processes. For instance, in vitro studies under both static and dynamic conditions may help to explain the influence of lubrication and loading on enamel and dentine wear. It may be difficult to reproduce physiologically relevant conditions, but at least some insight may be gained into surface phenomena such as cold welding, particle fracture or sub-surface fatigue.

Returning, therefore, to the aims of this thesis as determined on page 55 it may be concluded that:-

co-educational state schools in

1. Eight per cent of 14 year old children in Liverpool have evidence of tooth wear into dentine on the occlusal surfaces of lower molars and/or on the palatal surfaces of upper anterior teeth.

2. The daily consumption of carbonated drinks, the consumption of some pickled foods and grinding or clenching of teeth are the likely risk factors; it is possible that these dietary and masticatory factors act in concert.

3. Tooth wear is more frequent in anorexics (33%) and bulimics indulging in self-induced vomiting (40%) than in controls (6%). However, there is no linear association between pathological tooth wear and vomiting variables, although it was more likely to occur above a threshold of 1100 vomiting episodes. The pattern of tooth wear is similar to that seen in the sample of 14 year olds.

4. Gum-stimulated salivary flow rates were at the low end of the range but pH and buffering capacity were all within the normal range and were not significantly different in bulimics with and without pathological tooth wear. The vomiting bulimics with pathological

tooth wear have more viscous saliva than those subjects without pathological tooth wear. The dissolution of enamel calcium was significantly lower in those in the former group.

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APPENDIX 1

The University of Liverpool

Alex Milosevic BDS, FDS, DRD
Room 418

Dept. of Clinical Dental Science, P O Box 147, Liverpool L69 3BX Tel: 051 706 5221

AM/LJJ

13th December 1991

Mr J Balding
Schools Health Education Unit
School of Education
University of Exeter
Heavitree Road
Exeter
Devon EX1 2LU

Dear Mr Balding

Thank you very much for the Health Related Behaviour Questionnaire and other enclosures. I am writing to ask your permission if I may use the format and some of the questions in our questionnaire on toothwear. The questions are aimed to answer some of our hypotheses as to why certain children exhibit severe toothwear in a case control study. I enclose a draft of our questionnaire for your perusal and would welcome any comments or suggestions. Obviously, any papers published from this work will acknowledge your Health Related Behaviour Questionnaire.

Once again, many thanks for your help in this matter.

Yours sincerely

ALEX MILOSEVIC

Enc.

Schools Health Education Unit

Director: John Balding M.A. M.Sc. M.Med.Sci.

SCHOOL OF EDUCATION
UNIVERSITY OF EXETER
HEAVITREE ROAD
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UNIVERSITY OF EXETER

Alex Milosevic BDS FDS DRD
The University of Liverpool
Dept of Clinical Dental Science
Room 418
PO Box 147
LIVERPOOL
L69 3BX

19 December 1991

Dear Alex

Thank you for sending the draft of your questionnaire. I am delighted to give support to your work in this way.

We have an abundance of experience in examining those questions that you have selected from our Health Related Behaviour questionnaire in the context of all the other attitudinal and behavioural data from the other questions. If when you come to the time of your data analysis you would like information on any of these other connections from our databanks please ask.

We have recently published a book which might be of interest to you. I enclose a copy for your inspection.

Yours sincerely

John Balding

Enc

(jb/saj/milosevic)

LIVERPOOL HEALTH AUTHORITY
LIVERPOOL DENTAL HOSPITAL
Pembroke Place, Liverpool L3 5PS
Telephone 051 706 2000
Fax: 051 706 5807

Your Ref THMW/PT/ The person to please ask for 5020

ORTHODONTIC DEPARTMENT

27 April 1992

Mr A Milosevic
Lecturer in Restorative Dentistry
LIVERPOOL UNIVERSITY DENTAL HOSPITAL

Dear Mr Milosevic

Re: Case control study investigating the aetiology associated with tooth wear in fourteen year old school children

My committee have discussed your protocol for the above study and agree that you should proceed forthwith and we wish you well in your investigations.

Yours sincerely

DR T H M WYNNE
Chairman of Ethical Committee

cc Mr P Hardy
Secretary
Division of Dental Surgery
LIVERPOOL DENTAL HOSPITAL

AM/LJJ

28 January 1992

Dear Headteacher

You may recall that a year ago a dental survey of 14 year old children was undertaken at your School. We studied decay rates and a newly emerging problem, namely toothwear. Although decay rates are declining, our results found a surprising high level of toothwear.

In order to investigate the causes of the toothwear we would like to plan a follow up visit when the most severely affected children and pre-selected controls would be asked to fill in a questionnaire. Depending on the school we estimate a maximum of 24 children being involved, some schools much less. The questionnaire takes approximately 20 minutes to fill and there is no reason why the children could not be involved as a group with Alex Milosevic supervising and answering any questions. We are fully aware of the constraints of your busy GCSE curriculum and would not wish to disrupt your schedule unduly, therefore it is anticipated that this would be carried out when it is most convenient for you.

We do hope you will grant us this opportunity to carry out research which will benefit children in the future and hope to hear from you shortly.

Many thanks for your kind attention.

Yours faithfully

A MILOSEVIC

AM/LJJ

March 1992

Dear 5th former

Last year you kindly took part in a dental survey at your school. We would now like to arrange a follow-up study to provide a more detailed picture of teenagers dental health. This follow-up study would involve a questionnaire/interview which would take about 15 minutes to complete and a brief examination of your teeth by a dentist which will take about 2 minutes.

We would also like to take photographs of your teeth and this would only take a further 2 minutes.

All records are strictly confidential and are not divulged to anyone.

We do hope you will consent to this investigation, and have enclosed another letter requesting parental consent.

Thank you very much for your attention.

Yours sincerely

A MILOSEVIC
Lecturer

c.c. Professor M A Lennon, Head of Department

AM/LJJ

March 1992

Dear Parent

Last year you consented to your child having a dental examination and the information we gained was most helpful. With your permission we would like to carry out a follow up study of teenagers' dental health. This would entail the child filling in a questionnaire, having a dental examination, and having photographs taken of just the teeth. The questionnaire deals with dental and dietary habits and is based on the Health Education Authority School's Education Unit questionnaire. Answering the questionnaire may take 15 minutes, the dental examination 2 minutes and the photographs a further 2 minutes. We can assure you that all the information gathered is held in the strictest confidence.

We do hope you will consent to this investigation being undertaken.

Thank you very much for your attention.

Yours faithfully

A MILOSEVIC
Lecturer
Dentistry

PROFESSOR M A LENNON
Professor of Preventive

I give consent for _____ to participate in the above study

Signed_____
(Parent or Guardian)

Date_____

DENTAL QUESTIONNAIRE

NAME ..... DATE OF BIRTH / /

SCHOOL ..... HOME POSTCODE .....

PRIMARY SCHOOL .....

1. How often in the past 12 months have you attended the doctor ?

   0 = Never 1 = Once
   2 = Twice 3 = Three times
   4 = Four times 5 = More than four times 0 1 2 3 4 5

2. Do you take any medicines/prescriptions regularly ?

   0 = No 1 = Yes 0 1

3. Do you suffer with stomach upsets ?

   0 = Never 1 = Occasionally
   2 = Regularly 0 1 2

4. Would you say that generally you are fit and healthy ?

   0 = No 1 = Yes 0 1

5. How often in the past 12 months have you visited the dentist ?

   0 = Never 1 = Once
   2 = Twice 3 = Three times
   4 = Four times 5 = More than 4 times 0 1 2 3 4 5

6. This question is about your last visit to the dentist.

   0 = No 1 = Yes

   a Did you have a routine check-up ? 0 1
   b Did you have any fillings done ? 0 1
   c Did you have any teeth out (extractions) 0 1
   d Did you have toothache or sensitivity 0 1
   e Did the dentist make any remarks or comments about your teeth 0 1
   If so, what comments?

   _____

   f Did you receive any advice ? 0 1
   If so, what advice was given?

g Are you happy with the appearance of your teeth

0 = No 1 = Yes 0 1

7. Would you say that you clench or grind your teeth ?

0 = Never 1 = Occasionally
2 = Regularly 3 = Not noticed 0 1 2 3

8. How often do you brush your teeth ?

0 = Once a week 1 = Once a day
2 = Twice a day 3 = More than 2x a day 0 1 2 3

9. What sort of toothbrush do you use ?

0 = Hard 1 = Medium
2 = Soft 3 = Don't Know 0 1 2 3

10. How often do you change your toothbrush ?

0 = Once a year 1 = Every 6 months
2 = Every 3 months 3 = Monthly 0 1 2 3

11. Do the bristles on your brush flatten out ?

0 = No 1 = Yes 0 1

12. Which brand of toothpaste do you use regularly ?

.....

13. Do you use anything else to clean your teeth?

If Yes state .....

14. Which statement describes you best ?

0 = I would like to put on weight
1 = I would like to lose weight
2 = I am happy with my weight as it is 0 1 2

15. Are you happy about your body shape ?

0 = No 1 = Yes 0 1

16. Are you trying to change your weight or your body shape ?

0 = No 1 = Yes 0 1

17. Please study each item in this list of foods, decide how often you eat or drink them, and circle a number.

0 = Rarely or never 1 = Less than once a week
2 = At least once a week 3 = On most days

Fresh Oranges	0	1	2	3
Apples	0	1	2	3
Fresh grapefruits	0	1	2	3
Tinned fruits	0	1	2	3
Fruit juices	0	1	2	3
Fizzy drinks	0	1	2	3
Low-calorie drinks	0	1	2	3
Fizzy mineral water	0	1	2	3
Tomato ketchup	0	1	2	3
Other sauces	0	1	2	3
Coffee	0	1	2	3
Milk tea	0	1	2	3
Lemon tea	0	1	2	3
Yoghurt	0	1	2	3
Baked beans	0	1	2	3
Pickled onions	0	1	2	3
Other pickles	0	1	2	3
Relishes	0	1	2	3
Stoneground Bread	0	1	2	3
Peanut butter	0	1	2	3
Do you chew gum ?	0	1	2	3
Do you take vitamin supplements ?	0	1	2	3

18. Do you purchase canned or bottled fizzy drinks at school ?

0 = No 1 = Yes 0 1

19. Do you drink canned or bottled fizzy drinks at school ?

0 = No 1 = Yes 0 1

20. Are you allowed out of School during breaktime ?

0 = No 1 = Yes 0 1

21. If yes, have you purchased canned or bottled fizzy drinks off School premises ?

0 = No 1 = Yes 0 1

22. If you drink these cans or bottles are they chilled ?

0 = No 1 = Yes 0 1

23. Do you use a straw to drink from the can or bottle ?

0 = No 1 = Yes 0 1

24. Do you purchase fruit drinks in cartons at school ?

0 = No 1 = Yes 0 1

25. Do you drink fruit drinks in cartons at school ?

0 = No 1 = Yes 0 1

26. Are these cartons chilled ?

0 = No 1 = Yes 0 1

27. Do you use a straw to drink from the carton ?

0 = No 1 = Yes 0 1

28. At school, when do you drink any of these ?

Breaks between meals = 0
or at lunch = 1
or at both times = 2 0 1 2

29. What do you usually drink at teatime in your home ?

_____

30. What is your favourite drink at parties or discos ?

_____

APPENDIX 2

DENTAL HISTORY QUESTIONNAIRE

Individual History Name_____

D.O.B_____/_____/_____

Where were you born ?_____

How long have/had you lived in area of birth ? _____ years

1. Did you receive Fluoride supplements of any kind ? Yes/No/Don't know

2. How often do you attend a dentist ? Every 6m 12m 2yr >2yrs

3. Have you seen a dental hygienist ? Y/N

   If yes, how often ? Every 6m 12m 2yr >2 yrs

4. How often do you brush your teeth daily ? 0 1x 2x >2x

5. Do you floss between your teeth ? Y/N

6. How often to you use floss ?
   - Once a month
   - Once a week
   - Twice at week
   - Daily

7. What make of toothbrush do you usually use ?_____

8. Approximately, how often do you replace it ?
   - Once a month
   - Every 3 months
   - Every 6 months
   - Annually

9. Do you use toothpaste on your brush ? Y/N

10. If yes, which brand do you usually use ?_____

11. Approximately, how long do you spend cleaning your teeth at any one time ?
    - <1min
    - 1-3 mins
    - 3-5 mins
    - >5 mins

Name_____

12. Do you usually brush your teeth immediately after vomiting ? Y/N

13. If yes, when do you brush your teeth after vomiting ?

up to 1 min
1-5 mins
5- 15 mins
> 30 mins

14. Do you use a proprietary mouthrinse after vomiting ? Y/N

Which brand is it ? _____

15. Do you usually gargle with it ? Y/N

16. Do you use toothpaste on your brush after vomiting ? Y/N

Which toothpaste brand do you usually use ? _____

17. Do you spend more time brushing after vomiting than you would otherwise normally do ? Y/N

18. If yes, approximately how long in minutes ? _____

19. Do you brush any particular area of your mouth/teeth after vomiting ? Y/N

If yes, which area do you concentrate on ?

_____

_____

and why ?

_____

_____

APPENDIX 3

LIVERPOOL HEALTH AUTHORITY
LIVERPOOL DENTAL HOSPITAL
Pembroke Place, Liverpool L3 5PS
Telephone: 051 - 706 - 2000
Fax: 706 - 5801

Your Ref: Our Ref: THMW/PT/ If telephoning please ask for: 5020

8 November 1991

Mr A Milosevic
Lecturer in Conservation Dentistry
LIVERPOOL DENTAL HOSPITAL

Dear Mr Milosevic

A Study by enamel biopsy into the enamel acid solubility characteristics of individuals with an eating disorder

The Ethical Committee give you full approval to proceed with the above study and wish you well with your investigations.

Yours sincerely

T H M WYNNE
Chairman - Ethical Committe
Division of Dental Surgery

cc Mr P Hardy
Chairman - Division of Dental Surgery
LIVERPOOL DENTAL HOSPITAL

"Enamel biopsy" Participant information sheet

The aim of the study is to determine why some individuals have acid wear of their teeth and others not. A procedure to determine this is called "Enamel Biopsy". The procedure itself is painfree, simple and quick. You don't need local anaesthetic as only an extremely small fraction of the insensitive enamel is etched into a type of blotting paper disk of 2mm diameter. The whole time spent in the dental chair will be about 10-12 minutes or less and the usual hospital cross infection control measures will be employed. The biopsy specifically entails a small drop (1/1000 of a millilitre) of dilute acid be placed on the 2mm diameter absorbent disc for 60 seconds. Two lower front teeth are used but being behind the lower lip it is not normally visible. This area is not prone to decay, is easily polished and will be thoroughly washed afterwards. In the short term a whitish area the size of the disc will be present, but should disappear within 48 hours. Studies have shown that such acid etched areas heal without any detrimental consequences. This is the only simple yet accurate method to determine the tooth solubility in acid and will greatly help in the understanding of the tooth wear process, not only with those who have an eating disorder. I am available and only too happy to explain the procedure more clearly, but should you wish to decline participation without giving reasons I can thoroughly understand and support your decision.

A MILOSEVIC, Lecturer in Clinical Dental Sciences

Patient Consent Form

Enamel Acid Solubility

Characteristics

(The patient should complete the whole of this sheet himself/herself)

Please cross out as necessary

Have you read the Patient Information Sheet ? Yes/No

Have you had an opportunity to ask questions and discuss this study ? Yes/No

Have you received satisfactory answers to all of your questions ? Yes/No

Have you received enough information about the study ? Yes/No

Who have you spoken to ? Dr/Mrs/Ms.....

Do you understand that you are free to withdraw from the study:

at any time

without having to give a reason for withdrawing

and without affecting your future medical care Yes/No

Do you agree to take part in this study ? Yes/No

Signed..... Date.....

(NAME IN BLOCK LETTERS).....