

DEPARTMENTAL INITIATIVES TO ADDRESS EATING DISORDERS

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NSW Health is taking up the challenge to improve pathways to care for children and adolescents who exhibit the earliest symptoms of eating disorders. The aim will be to improve detection and strengthen local networks of professionals who have skills to implement effective early intervention, including those who can offer appropriate specialist child and adolescent mental health support. A sum of \$100,000 has been allocated to the Department of Psychological Medicine at the New Children's Hospital at Westmead to develop an effective early intervention model for identifying and treating children and young people with eating disorders.

Expansion of a successful shared-care pilot project involving general practitioners and specialist mental health services in Central Sydney Area has also been funded. This program includes a training component for general practitioners that is expected to equip them with skills to detect eating disorders earlier and to intervene more effectively.

In partnership with the former Ministerial Advisory Committee on Body Image and Disordered Eating, the Centre for Mental Health, NSW Health Department, convened workshops in mid-1998 involving several groups which provide support for people with eating disorders and their families.

NSW Health is collaborating with the NSW Department of Education and Training in the development of the School-Link Program. The current focus of that program is on the prevention and early detection of depression in adolescents; however, the inclusion of components to teach

positive coping and life skills has broader implications for mental health promotion.

The Centre for Mental Health and the Health Services Policy Branch are also assisting the Statewide Services Development Branch of the NSW Health Department in a project to develop a plan to provide services for people in NSW who have eating disorders. Consultants are currently working on a comprehensive service model that incorporates the full spectrum of care, from prevention and early intervention through to treatment for people with the most severe and complex problems. The process includes wide consultation with professionals, consumers and carers.

Editor's note:

The NSW Ministerial Advisory Committee on Body Image and Disordered Eating was formed in April 1997 on a recommendation of the Summit on Body Image and Eating Disorders held in Sydney in August 1996. The Summit was held in response to growing concern about health problems associated with disordered eating among young women, adolescents and children. It focused particularly on the effect of the media, fashion trends and other social pressures on women's body image and the relationship between those factors and such eating disorders as anorexia nervosa and bulimia nervosa. Following the release of the report from the Summit, the Minister announced the formation of an intersectoral Ministerial Advisory Committee to further examine the issues identified at the forum and to report back with recommendations for addressing those issues. The primary focus of the committee was on preventing disturbed body image and disordered eating.

DENTAL EROSION: MORE ACID MEANS FEWER TEETH

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The surfaces of the teeth are in a continuing state of flux throughout a lifetime. On contact with the tooth, a piece of grapefruit or a drink of orange juice will cause a demineralisation of the enamel or dentine surface. If no further acid attack occurs, the tooth can be remineralised over time by calcium and phosphate ions from the saliva. The balance of this dynamic ionic exchange can easily be upset resulting, for many patients, in irreversible and extensive tooth tissue loss (dental erosion) that may be difficult, if not impossible, to correct. As our knowledge of this form of tooth structure loss increases, there is a

growing awareness of the complexity of the erosion process. This article examines the prevalence of dental erosion, the usual sources of acid responsible for it, other physiological factors that determine the impact of acid on the teeth, and ways of both managing and preventing dental erosion.

Dental erosion is defined as loss of dental hard tissue by a chemical process that does not involve bacteria.¹ There is a perception among some dental clinicians that the occurrence and severity of dental erosion is increasing markedly. Epidemiological evidence now appears to confirm these observations. In a national survey of more than 2000 children in the United Kingdom,² as many as 50 per cent of the five- and six-year-old patients studied displayed erosion of their first teeth, and 25 per cent were

found to have had severe erosion involving the dentine or pulp (nerve) of the teeth. Evidence of tooth erosion near the roof of the mouth was detected in more than a quarter of the subjects over 11 years of age, rising to 32 per cent of 14 year olds. Erosion was sufficiently severe in 13 to 15 year olds to have penetrated to dentine in between two and three per cent of the subjects. These alarming statistics appear to indicate an increasing and significant problem for the individual and the community.

SOURCES OF ACID

In simple terms, the acid required for erosion to occur will either be acid going into the mouth (extrinsic) or acid coming out of the stomach (intrinsic). The effect of the acid will be determined by its volume, type and concentration, the length of time it is in contact with the tooth surface, and its ability to overcome the environment of the saliva and the resistance of the tooth.

Extrinsic acids

Dental students are familiar with dramatic textbook photographs, usually of workers in battery acid factories, who suffered from occupational exposure to acids in the air they breathed. No teeth could be seen in these subjects even when they were smiling because all the tooth surfaces exposed to the air had been lost. Before effective occupational health measures were adopted, exposure to acids was common in the chemical and metal industries. Nowadays, occupational exposure is rare, but it is still found routinely in some fields, such as professional wine tasting. Exposure to extrinsic acid can also be associated with such activities as frequent swimming in chlorinated pool water that has not been maintained at the optimal pH.³

Dietary considerations

Eating acidic foods and drinking acidic beverages are the most common sources of extrinsic acids that cause dental erosion. Some medicines, such as vitamin C preparations and iron tonics, also contribute acid. Some chewable vitamin C tablets have a pH of about 2.0.

A healthy diet may also contain a substantial quantity of acidic foods. Healthcare workers advocate fresh fruit as a component of a 'balanced' diet as a 'healthy' option, but some fruits have a high acid content. Additionally, in an attempt to control body weight, patients may consume acidic foods such as fruit juices and diet drinks in place of high-calorie alternatives. Diet foods and low-calorie beverages are invariably acidic. The potential for erosive damage by these foodstuffs is not well recognised by the public.

In one study, it was found that the erosion risk for individuals who consumed citrus fruits more than twice a day was 37 times greater than it was for those who consumed citrus fruits less often.⁴ Thus, the critical frequency of consumption was more than twice a day. For

soft drinks, the critical frequency of consumption was once a day or more. Many soft drinks contain citric, phosphoric, carbonic, and other acids, and their pH value is often less than 4.0. Soft drinks are marketed to the young adult, promoted as being healthy, and are linked with high-profile sportsmen and women. In the United Kingdom, 42 per cent of fruit drinks are consumed by children between the ages of two and nine.⁵ Adolescents and children account for 65 per cent of sales of acidic drinks. Sales of soft drinks have increased sevenfold since 1950.

Increasingly popular are the new 'sports drinks'. In a Scandinavian study, sports drinks had the same acidogenicity as fruit juice and carbonated beverages.³ Most contain citric acid.

The demineralising effect of citric acid is especially great because its chelating action on the enamel calcium continues even after the pH increases at the tooth surface (the citrate ions bind with the calcium in the tooth, thereby forming soluble citrates).⁶ It should be noted that the titratable acidity of a solution, that is, the total amount of H⁺ able to dissociate, is a better indicator of erosive potential than the pH value, which solely measures H⁺ concentration of the solution. Studies indicate that 5.5 is the critical pH for tooth enamel demineralisation.⁷

Intrinsic acid

There is increasing evidence that regurgitation erosion is a major contributor to tooth tissue loss.⁸ Acid moving through the lower oesophageal sphincter into the oesophagus is described as gastro-oesophageal reflux. In some patients, the acid movement becomes chronic and painful and requires treatment. This is termed gastro-oesophageal reflux disease. Regurgitation is the reflux of gastric juice through the upper oesophageal sphincter into the oral cavity. Many factors, including hiatus hernia, pregnancy, obesity, chronic alcoholism, certain types of exercise, and some foods, have been linked to gastro-oesophageal reflux. Reflux symptoms may include heartburn, epigastric pain and regurgitation, although many patients are symptom-free.

It is estimated that more than 60 per cent of the population suffer from gastro-oesophageal reflux at some time,⁹ and the abundance of antacid preparations in supermarkets is evidence of the widespread self-medication for this condition. If the reflux becomes longstanding, continual damage to the oesophageal lining can lead to oesophagitis, stricture, ulceration and, in some cases, malignant changes. Further, some medicines have the potential to provoke or increase gastro-oesophageal reflux. These include anti-spasmodic drugs, non-steroidal anti-inflammatory drugs and anti-cholinergics.

Eating disorders such as anorexia and bulimia nervosa have been shown to cause a similar pattern of dental erosion, which is often initially evident on the palatal

surfaces of the maxillary anterior teeth. Rumination (voluntary regurgitation), especially in patients who are mentally handicapped, can also be a possible cause.

The presence of palatal dental erosion and any symptom of reflux is indicative of pathological gastro-oesophageal reflux. In those patients without reflux symptoms but with significant unexplained palatal erosion, gastro-oesophageal reflux should always be suspected, and referral to a gastroenterologist will often be indicated.

CO-FACTORS

Saliva

It is possible that the quantity and quality of saliva may be as important in the development of dental erosion as the concentration or frequency of acid attack. At normal salivary flow rates, acidic drinks are eliminated from the mouth in about 20 minutes and the pH at the tip of the tongue remains low for only about two minutes after the drink has been consumed.¹⁰ In contrast, in patients with low salivary flow rates, the pH remains low for more than 30 minutes. Although the role of salivary calcium and phosphorous in remineralisation following acid attack is not yet fully understood, the buffering capacity of the saliva is of critical importance.

As discussed in the article by Mark Shifter in the March issue of the *NSW Public Health Bulletin*, many prescribed medications lead to xerostomia (dry mouth). This results in less dilution and buffering of the acid as well as less lubrication during mastication. This must exacerbate attritional and abrasive wear of eroded surfaces. Those people who moisten their mouths with acidic drinks will have a reduced buffering capacity and will suffer accelerated erosive damage.

Saliva is known to be important in protecting the oesophagus from gastric acid damage after reflux, and the introduction of acid into the oesophagus is known to stimulate salivary output and buffering capacity.

Teeth

Not all teeth are created equal. It would be expected that those teeth that have had the benefit of fluoride during development will resist acid demineralisation, while already hypocalcified teeth would be more at risk.

RECOGNITION

It is often difficult to differentiate between erosion, attrition and abrasion, as these conditions invariably occur in synergistic combination. Evidence now suggests that erosion is a major contributor in tooth wear. Clinically, lesions indicating an erosive cause are often characterised by:

- smooth enamel surfaces, with developmental ridges absent

SUGGESTIONS FOR PREVENTING DENTAL EROSION

It is advisable to provide patients with printed information giving dietary advice and oral hygiene hints when dental erosion has been detected. This can contain information such as:

- Be aware of the acidity of the foods and beverages you consume.
- Never brush your teeth immediately after eating or drinking anything acidic, otherwise there will be no opportunity for remineralisation.
- Try to always rinse with water after having anything acidic.
- Never have anything acidic last thing before going to bed without rinsing your mouth thoroughly with water because your swallowing frequency and saliva production decreases dramatically during sleep.
- Don't allow a toddler to drift off to sleep with a bottle containing fruit juice. Water is best.
- Don't sip acidic drinks. Belting down a Solo is less damaging to your teeth!
- Remember that fruit juices are generally more erosive than whole fruit, and dried fruits can be very acidic.
- When undertaking physical exertion (sport, gym, etc.), always rinse your mouth with water after drinking a sports drink or soft drink, especially when your mouth has become really dry.
- Never put a mouthguard into an unrinsed mouth.
- Avoid chewable vitamin C tablets.

- areas of exposed dentine, often with a fine margin of intact enamel at the gingival crevice
- restorations that are standing 'proud' of (higher than) the surrounding tooth surface
- depressions in the cusp tips of posterior teeth, creating a 'rock-pool' effect.

When erosion is suspected, it is essential to take a detailed history that includes an investigation of past and present diet, medical history, medications, slimming habits, occupation, sporting activities, and other factors. It should be remembered that the dentist may be the first to suspect certain psychological diseases, such as bulimia nervosa about which the patient may be secretive. Confrontation in such situations may be counterproductive and, because dentists are not qualified to treat these underlying conditions, a judicious referral to the patient's general medical practitioner is warranted.

MANAGEMENT

Following differential diagnosis, it is critical that the source of the acid be identified if at all possible. This

source can then be removed or modified, and preventive measures, such as the prescription of neutral fluoride mouth rinses, can be undertaken. It is important to monitor and record the rate of loss of tooth structure. This is best undertaken using accurate study models that are retained and compared over time. Intraoral photographs may be of some use. Emphasis should be on patient education, behaviour modification and careful monitoring. In most cases, active restorative treatment is not indicated until the preventive measures have been shown to be effective and, even then, only in certain circumstances. These circumstances include:

- significant aesthetic concerns
- loss of vertical dimension that will compromise any possible future treatment
- pain from dentine hypersensitivity that does not respond to conservative treatment.

If required, restorative treatment may vary from the simplest of bonding procedures to extremely complex rehabilitation comprising multiple crowns and onlays.

CONCLUSION

The increasing numbers of older people using medications associated with xerostomia, the increasing use of drugs that can promote gastro-oesophageal reflux and the increasing consumption of acidic soft drinks, fruit drinks and sports drinks all contribute to dental erosion becoming a larger public oral health problem. Just as dental erosion has many factors relating to its aetiology,

management and treatment, so there is no single solution. Product modification by the food and drink manufacturers, community education, and individual patient behaviour modification all are required to reduce the damage from this growing problem.

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INFECTIOUS DISEASES, NSW: APRIL 1999

TRENDS

Reports of infectious diseases followed largely expected trends through to early autumn (Figure 1). Statewide, **arbovirus infections** appeared to plateau in March, although late-season increases in **Ross River virus infections** (RRV) were reported in coastal regions. This was notably in the Hunter, Illawarra, Northern Rivers and Mid North Coast areas (Table 1). In inland NSW, increases in RRV were reported earlier, in late spring and early summer. **Barmah Forest infections** also have been reported from the Northern Rivers, Mid North Coast and Illawarra areas, albeit in fewer numbers.

There are encouraging signs that the epidemic of **gonorrhoea** (mainly affecting men in inner Sydney) may have levelled off, although it is probably too early to be sure, given reporting delays. The number of reports of **meningococcal infection** has fluctuated markedly in recent months, with 20 cases in January, seven in February, and 19 in March. This is a reminder that, while this disease

tends to peak in winter and spring, it can occur in any season. Reports of pertussis cases are again abating, following a peak last spring.

ASIAN TIGER MOSQUITO FOUND IN BOTANY BAY

A single 'Asian tiger' mosquito (*Aedes albopictus*) was identified in routine trapping carried out by the Australian Quarantine and Inspection Service (AQIS) at Botany Bay in early April. This species is a known vector for **dengue fever** in Asia, and has never before been reported in New South Wales. The *Aedes albopictus* probably arrived on a container ship, possibly as an egg laid in stagnant rainwater. In response to the finding, an intensive mosquito-trapping program was initiated in collaboration with the Medical Entomology Department at Westmead ICPMR and AQIS. No other mosquito of this species has been detected subsequently. ☒