The "Point of Care" section answers everyday clinical questions by providing practical information that aims to be useful at the point of patient care. The responses reflect the opinions of the contributors and do not purport to set forth standards of care or clinical practice guidelines. This month's articles were written by speakers at the Pacific Dental Conference, to be held in Vancouver, B.C., from March 6 to 8, 2008. For more information on the conference, visit www.pdconf.com.

Pacific Dental Conference

QUESTION 1

What do I need to know about oral cancer screening?

Background

Oral cancer is a devastating disease: approximately 3,200 new cases and 1,050 deaths from oral cancer occur each year in Canada.[1] Regrettably, many cases are diagnosed late and require aggressive treatment. Today, 50% of oral cancer patients die within 5 years of diagnosis. Those who survive often endure significant disfigurement, impairments in oral function and compromised quality of life. Of further concern, global survival rates have changed little over the last 3 decades.[2]

It is firmly believed that early detection of oral cancer can significantly reduce oral cancer deaths and morbidity.[3] The British Columbia Oral Cancer Prevention Program (BC OCPP) team thinks that dentists are ideally positioned to make this happen. Oral cancer is frequently preceded by an identifiable premalignant lesion — a white patch or, less frequently, a red patch — and progression from dysplasia to cancer occurs over years.[4] This allows clinicians the opportunity to detect early changes in the oral mucosa and intervene. However, a major challenge has been differentiating between benign and precancerous or early cancerous mucosal changes when there are often no distinctive clinical features that distinguish the conditions (**Fig. 1a**).

The BC OCPP team has embraced this challenge through a multifaceted program incorporating research, education and care. This program provides the scientific groundwork for oral cancer screening using a standardized clinical approach in conjunction with screening tools that include toluidine blue staining and direct fluorescence visualization using a number of devices including the VELscope (LED Dental Inc., White Rock, B.C.).

Toluidine blue has a long history of use as a vital stain to identify oral cancers and has been used sporadically in dental practice for many years. Research from an ongoing longitudinal study conducted at the British Columbia Cancer Agency has shown that oral premalignant lesions that stain with toluidine blue are 6 times more likely to become oral cancers than those that do not (**Fig. 1b**). This finding supports a new role for this vital stain in identification of high-risk oral lesions.[5]

Using evidence-based techniques, the BC OCPP team is working toward understanding the value of direct fluorescence visualization in the management of oral dysplasia and oral cancer. The team has used this technology in its highly specialized clinics to follow about 600 patients for more than 3 years.[6] This experience has provided sufficient evidence of added value to warrant use of this technique in specialized referral clinics for the management of oral dysplasia or in the follow-up

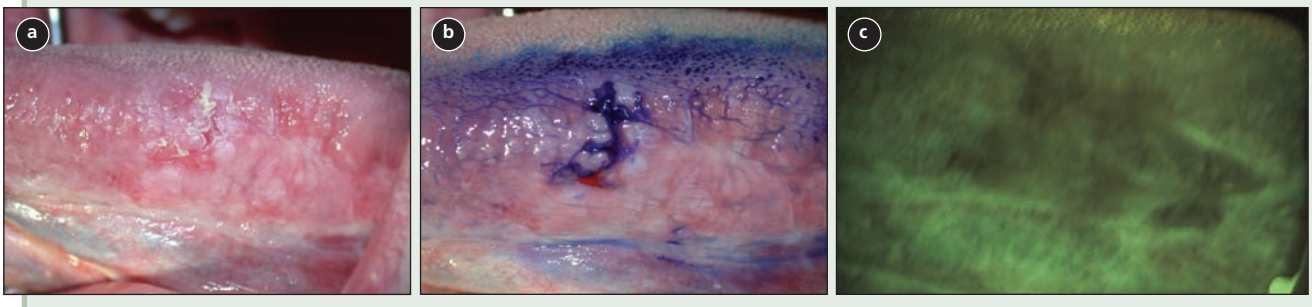

Figure 1: A painless, diffuse, red and white lesion on the left lateral area of the tongue of a 35-year-old man with a history of tobacco chewing. Diagnostic biopsy identified carcinoma in situ. **(a)** Lesion viewed with conventional white light showing a diffuse, predominantly white lesion. **(b)** Lesion viewed following application of toluidine blue showing a focal region of dye uptake. **(c)** Lesion viewed with direct fluorescence visualization showing loss of fluorescence.

of treated oral cancer patients (**Fig. 1c**). Efforts are being made to understand its use in community settings where evidence is still being collected.

Management Advice

In the specialized BC OCPP affiliated clinics, the team employs a standardized step-by-step approach to the evaluation of any mucosal lesion suspected to be premalignant or potentially malignant.[7]

- **Patient history —** including family history of head and neck cancer, habits and lifestyle, signs and symptoms
- **Visual inspection (general)** — including extraoral and intraoral examinations
- **Visual inspection (specific) —** location, size, colour, texture and outline of identified lesion(s)
- **Visualization aids —** direct fluorescence visualization; toluidine blue application
- **Clinical photos —** all visible lesions
- **Diagnostic biopsy —** as indicated

It is critical to note that toluidine blue staining and direct fluorescence visualization are not diagnostic in all settings. The impression is that these techniques are complementary to and not a replacement for a comprehensive history and conventional visual and manual examination of head and neck. The value of these techniques depends on the knowledge and training of the operator in their use and interpretation. Training and experience are important as a variety of benign and common mucosal changes may result in staining with the application of toluidine blue or show loss of fluorescence. These alterations are not restricted to potentially malignant or malignant disease. As always, good clinical judgement is indicated in all circumstances. The challenge to the dental profession will be to ensure that all adult patients receive a regularly scheduled comprehensive oral cancer screening examination. Working together with a strong commitment to change, dentists have the opportunity to make a dramatic difference.

THE AUTHORS

Dr. Michele Williams *is clinical professor in the faculty of dentistry, University of British Columbia, and staff in the oral oncology department, British Columbia Cancer Agency, Vancouver, B.C.* ***Email:*** *mwill@bccancer.bc.ca.*

Dr. Catherine F. Poh *is clinical assistant professor in the faculty of dentistry, University of British Columbia, and staff in th oral oncology department, British Columbia Cancer Agency, Vancouver, B.C.*

Dr. Lewei Zhang *is professor and director of oral medicine oral pathology in the faculty of dentistry, University of British Columbia, Vancouver, B.C.*

Dr. Miriam R. Rosin *is professor and director of the British Columbia Oral Cancer Prevention Program, British Columbia Cancer Agency, Vancouver, B.C.*

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References

1. Canadian cancer statistics 2007. Toronto: Canadian Cancer Society/National Cancer Institute of Canada; 2007. Available: www.cancer.ca/vgn/images/portal/cit_86751114/36/15/1816216925cw_2007stats_en.pdf (accessed 2007 Oct 17).

2. Cancer facts & figures 2005. Atlanta: American Cancer Society; 2005. Available: www.cancer.org/downloads/STT/CAFF2005f4PWSecured.pdf (accessed 2007 Oct 17).

3. Sankaranarayanan R, Ramadas K, Thomas G, Muwonge R, Thara S, Mathew B, and others. Effect of screening on oral cancer mortality in Kerala, India: a cluster-randomised controlled trial. *Lancet* 2005; 365(9475):1927–33.

4. Rosin MP, Cheng X, Poh C, Lam WL, Huang Y, Lovas J, and others. Use of allelic loss to predict malignant risk for low-grade oral epithelial dysplasia. *Clin Cancer Res* 2000; 6(2):357–62.

5. Zhang L, Williams M, Poh CF, Laronde D, Epstein JB, Durham S, and others. Toluidine blue staining identifies high-risk primary oral premalignant lesions with poor outcome. *Cancer Res* 2005; 65(17):8017–21.

6. Poh CF, Ng SP, Williams PM, Zhang L, Laronde DM, Lane P, and others. Direct fluorescence visualization of clinically occult high-risk oral premalignant disease using a simple hand-held device. *Head Neck* 2007; 29(1):71–6.

7. Poh CF, Williams PM, Zhang L, Rosin MP. Heads up! — A call for dentists to screen for oral cancer. *J Can Dent Assoc* 2006; 72(5):413–6.

A special edition of *JCDA* on oral cancer screening and early detection of oral cancer is planned for the spring of 2008. This edition will contain detailed information on the resources developed by the British Columbia Oral Cancer Prevention Program team, including practical and time-efficient clinical practice guidelines that incorporate these techniques into an already busy dental practice. The BC OCPP team will also be giving a presentation on this topic at the Pacific Dental Conference on Thursday, March 6 (session repeated Friday, March 7).

QUESTION 2

What is the role of the physiotherapist in managing the patient with complex temporomandibular disorder?

Background

Temporomandibular disorder (TMD) is a syndrome that is often misdiagnosed or even ignored by medical professionals. It may cause headaches, earaches, facial pain or sinusitis, and the afflicted are often left to suffer the sequelae of chronic pain. The etiology of TMD is multifactorial: trauma (a direct blow to the jaw or the result of a motor vehicle accident), stress,[1] forward head posture or dental work. TMD can also be psychosomatic,[2] which means that effective treatment should be directed at the mind as well as the body.

Physiotherapists are university-trained body specialists, who are educated in pathology, anatomy, physiology and kinesiology. In short, they are able to assess and treat the muscles and joints of the human body. Diagnosis and treatment of TMD requires a physiotherapist with additional postgraduate education in anatomy and physiology of the head, neck and maxillofacial region,[3] as well as postural and breathing analysis. Therapists who recognize the mechanical causes of TMD as well as psychosomatic problems can use sensorimotor techniques,[4] which help patients manage the effects of stress on both their mind and body.

How Do Dentists and Physiotherapists Work Together?

Dentists are often the first health care professionals to recognize TMD. Referrals to physiotherapists often come as a result of dental patients complaining of pain, particularly in the chewing muscles or jaw joint, limited movement or locking of the jaw, painful clicking, grating sounds or sudden changes in occlusion. Patients will self-refer to physiotherapists if they have other symptoms, such as headaches or neck and shoulder pain, which often lead to a diagnosis of TMD.

Assessment to Determine the Pathophysiology of TMD and Related Pain

Subjective assessment includes a TMD questionnaire, questions about past and present life history, direct triggers, such as dental work, increased stress, symptom behaviour (time of day, posture), medications, related medical history and other investigative tests.

Physical assessment includes an upper quadrant scan to determine which area and tissue require further investigation.

Biomechanical examination comprises:

- observation of the jaw, face, posture and breathing patterns (habitual and excessive head forward posture adversely alters the occlusal relationship and may lead to continual stress on the temporomandibular joint. Altered breathing patterns are an indication of stress[1])
- specific testing of the joint and its supporting structures for hypo- or hypermobility and disc derangements
- occlusal tests
- intraoral and extraoral palpation of the muscles of mastication and lateral aspects of the temporomandibular joint.

The results of the subjective and physical assessment can be used to determine the category of TMD: myofascial pain, internal derangement of the joint or degenerative joint disease. Once the category is determined, a treatment plan can be made.

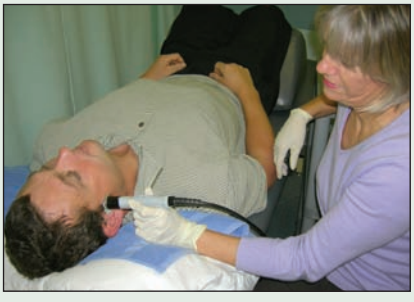

Figure 1: Patient receiving ultrasound treatment.

Figure 2: Transcutaneous electrical nerve stimulation helps relieve pain.

Treatment of TMD

If the condition is acute, treatment consists of:

- pain relief, including application of ice, rest and use of such methods as ultrasound, biofeedback and transcutaneous electrical nerve stimulation (**Figs. 1** and **2**)

Figure 3: Patient doing temporomandibular realigning exercises.

Figure 4: Pterygoid muscle release.

- education, including analysis of causes, diet guidelines, ergonomic advice, postural and breathing correction and the "12 self-care tips".[5]

If the condition is not acute, treatment includes:

- the application of moist heat
- teaching and tailoring a specific exercise program to the patient's condition (**Fig. 3**)
- manual therapy to restore glides (anterior, inferior, lateral and antero-inferior) and movement of the temporomandibular joint (**Fig. 4**) and cervical spinal areas
- myofascial release[3] of the muscles in the neck and facial areas to relieve pain, improve range of movement and correct forward head alignment.
- psychological support, which involves educating the patient on how stress can entail changes in physiology (heart rate, muscular tension, respiration and visceral feedback) and teaching the patient techniques to deal with these changes
- explaining to the patient the scope of the comprehensive treatment plan, including time commitment and cost.

Physiotherapy treatment for TMD patients requires one-on-one interaction; the course of treatment will depend on the gravity of the patient's condition and the techniques used by the physiotherapist. Each treatment session lasts about 1 hour. It is imperative that the patient feel an improvement in their symptoms, however small, at the end of their first visit.

TMD is difficult to diagnose and treat effectively.[6] As such, it is best managed through a multidisciplinary approach. Physiotherapy is most helpful if good rapport is established between patient and therapist. The sooner a patient believes that treatment can help, the more effective the treatment will be.

THE AUTHOR

Ms. Catherine Russell *is a physiotherapist specializing in temporomandibular disorder and the rehabilitation of sports injuries. She is based in West Vancouver, British Columbia. Email: info@cathymrussell.com*

Ms. Russell's session ***at the PDC, titled "The taming of the jaw: a tooth fairy's perspective" will be presented on Thurday, March 6, and repeated on Friday, March 7.***

References

1. van der Kolk BA, McFarlane AC, Weisaeth L, editors. Traumatic stress: the effects of overwhelming experience on mind, body and society. New York: Guilford Press; 1996.

2. Poveda Roda R, Bagan JV, Diaz Fernandez JM, Hernandez Bazan S, Jimenez Soriano Y. Review of temporomandibular joint pathology. Part I: classification, epidemiology and risk factors. *Med Oral Patol Oral Cir Bucal* 2007; 12(4):E292–8.

3. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual. Baltimore: Williams & Wilkins; 1992.

4. Ogden P, Pain C, Fisher J. A sensorimotor approach to the treatment of trauma and dissociation. *Psychiatr Clin North Am* 2006; 29(1):263–79, xi–xii.

5. Goddard G, Rudd P. Self-care tips for TMD. Included in an information package distributed by the National Oral Health Information Clearinghouse titled "TMD: Information for Professionals". June 1996; OP-32.

6. Medlicott MS, Harris SR. A systematic review of the effectiveness of exercise, manual therapy, electrotherapy, relaxation training, and biofeedback in the management of temporomandibular disorder. *Phys Ther* 2006; 86(7):955–73.

QUESTION 3

What role does dentistry play in the diagnosis and treatment of sleep-disordered breathing?

Background

Obstructive sleep-disordered breathing (SDB) is now recognized as a significant medical condition that is becoming more deadly as the population ages and gains weight. It is represented by a spectrum of conditions, ranging from the age-old nemesis of personal relationships — snoring — to the potentially life-threatening condition of severe obstructive sleep apnea. Obstructive sleep apnea may be appropriately described as temporary asphyxia, the word "apnea" being derived from the Greek term meaning "lack of breath." Sleep apnea may be caused by either a central nervous system disorder resulting in central sleep apnea or a narrowing of the airway, which leads to various degrees of obstructive sleep apnea. More specifically, airway collapse leads to a build-up of pressure within the airway, which in turn causes vibration of the pharyngeal tissues (snoring), the first and most obvious sign of an SDB problem. The extent of the collapse will determine the severity of sleep apnea: mild, moderate and severe. The lives of patients with severe sleep apnea may be in danger every time they go to sleep.

The primary causes of SDB are genetics, advancing age and weight gain. In terms of the genetic causes, patients may have inherited a narrowed airway and other predisposing craniofacial factors. With age, the body's tissues lose tone, and in the airway, this results in a greater propensity for collapse. Over time, a snoring condition may develop into sleep apnea that may eventually become severe. Weight gain can cause a further narrowing of an already compromised airway; additional weight in the abdominal area can affect breathing. Many other minor factors, such as smoking, drinking alcohol and eating before bedtime, may have an impact on an apnea condition.

The extent of sleep apnea is defined by the number of airway obstructions that occur during each hour of sleep. For reasons of training, experience and licensure, dentists have no role in the differential diagnosis of SDB, and a specific diagnosis along the spectrum of SDB must be made by a physician. In some jurisdictions in Canada, such a diagnosis is required by regulation before oral appliance therapy can be undertaken. This can be a constraint to the involvement of the general dentist, who will have to establish a relationship with either the patient's physician or another medical specialist who can make a diagnosis and a referral. Once a diagnosis has been made, however, dentists can make a difference in the lives of patients with obstructive sleep apnea by offering oral appliance therapy.

Screening and Management

Dentists have both an opportunity and a responsibility to become active in screening for obstructive sleep apnea. Dentists may participate in the field of SDB by undertaking the training necessary to effectively manage treatment of such patients or by adding a sleep apnea screening questionnaire to their standard medical history and then referring affected patients to another dentist with appropriate training and expertise or to the patient's physician.

In many Canadian communities, there are relatively few or no specialists involved in treating obstructive sleep apnea. In the absence of a medical specialist, the dentist should work directly with the patient's general practitioner to secure appropriate medical back-up before undertaking treatment. Even in communities where specialists are available, it is important to realize that under the structure of the Canadian health care system, a specialist may not be paid without the referral of the patient's medical general practitioner.

Screening for obstructive sleep apnea has benefits not only in terms of patients' general health but also in terms of protecting the work that dentists do to maintain good dental health. For example, there appears to be a correlation between bruxism and sleep apnea. It has been demonstrated that a patient's airway will increase in dimension during clenching. If airway collapse can be reversed to any degree by muscular activity, the patient will naturally respond to collapse by clenching and grinding to relieve the blockage. The forces that may be applied (e.g., to esthetic dental restorations) during this bruxism response to apnea can be extreme and damaging. As such, it is a long-established practice to prescribe night guards for patients who are vulnerable to bruxism. But do these appliances really represent the right answer?

Figure 1: The Silencer with the Halstrom Hinge.

In a recently published study,[1] Dr. Gilles Lavigne and colleagues from the University of Montreal found that the use of single-arch night guards by apneic patients can *worsen* the apnea by more than 50% in half of the patients. The same researchers found that the use of double-arch night appliances can be twice as effective in relieving bruxism as single-arch appliances. As such, especially if combined with mandibular advancement and support, double-arch appliances may be suitable for relief of bruxims as well as the symptoms of snoring and sleep apnea.

In 2006, in its position paper on oral appliance therapy, the American Academy of Sleep Medicine recommended oral appliance therapy as "first-line" therapy for the majority of obstructive sleep apnea patients.

A wide variety of appliances, many with adjustable features, are available (**Fig. 1**). Most appliances use double-arch units to manage the airway and support the mandible during sleep. Practitioners treating patients with SDB must be familiar with the range of options. The sophistication of the appliance has a direct effect on the dynamics of treatment. The use of precision attachments to control the mandibular positioning can be highly effective.

Snoring has long been considered a nuisance to be ignored or tolerated, but it should be recognized that in extreme cases, SDB may eventually threaten the patient's life. The first step in treatment is accurate diagnosis, which is the responsibility of the patient's medical team. The dentist may then have a role in providing oral appliance therapy.

THE AUTHOR

Dr. Wayne Halstrom *has been in practice since 1960 with the last 14 years limited to the treatment of snoring and sleep apnea at the West Coast Sleep and Breathing Centre. He is a diplomate of the American Academy of Dental Sleep Medicine. Email: lwh1@telus.net*

Dr. Halstrom will be giving 2 presentations at the PDC: "The role of the dental professional in the treatment of snoring and sleep apnea" (Thursday, March 6, and repeated Friday, March 7) and "Screening for sleep apnea — an opportunity and a responsibility" (Saturday, March 8).

Dr. Halstrom is the inventor of the Silencer and the Halstrom Hinge.

Reference

1. Gagnon Y, Mayer P, Morisson F, Rompré PH, Lavigne GJ. Aggravation of respiratory distrubances by the use of an occlusal splint in apneic patients: a pilot study. *Int J Prosthodont* 2004; 17(4):447–53.

QUESTION 4

How should I diagnose dental fluorosis?

Background

Ingestion of excess fluoride, especially in early childhood, increases the risk of dental fluorosis.[1] In most areas of Canada, the concentration of fluoride in untreated drinking water is below 0.3 mg/L (equivalent to 0.3 parts per million or ppm), but in some areas the water exceeds the maximum level, set by Health Canada, of 1.5 ppm. In the past, children living in areas of low fluoride exposure were given fluoride supplements, but this practice increased the risk of dental fluorosis. Similarly, more than half of Canadian communities have been fluoridating their drinking water for years; however, there has been a steady rise in the prevalence and severity of dental fluorosis in such communities.[2,3] Overall, 12.5% of children in communities with fluoridated water have objectionable dental fluorosis, which is often treated cosmetically.[4]

Diagnosis

When fluoride exposure occurs early in life (at 1–3 years of age) and then falls to a low level, only the anterior incisors and first molars are affected. Excess exposure occurring later (after mineralization of the incisors is complete) causes dental fluorosis only on the canines, premolars and second molars.[5] Increasing the fluoride level in water from 0.4 ppm to the typical level used for fluoridation (1.0 ppm) "would lead to one extra person with dental fluorosis for every 6 people."[4] Among susceptible children, continuous intake of fluoride at this level, from birth, results in fluorosis of all teeth (**Table 1**). Severe fluorosis, characterized by deep pitting and substantial loss of enamel tissue, rarely occurs in Canada but might be present in immigrants who previously lived in areas where fluorosis is endemic (e.g., India, Africa, China and the Middle East).

A medical history, including questioning about whether there has been excess fluoride exposure, should corroborate the appearance of the teeth. Dental fluorosis is a symmetric, systemic condition, occurring on pairs of teeth that develop at the same time. Depending on the timing of exposure, it can appear on the cusp tips only, on the incisal third of the teeth or on the entire surface

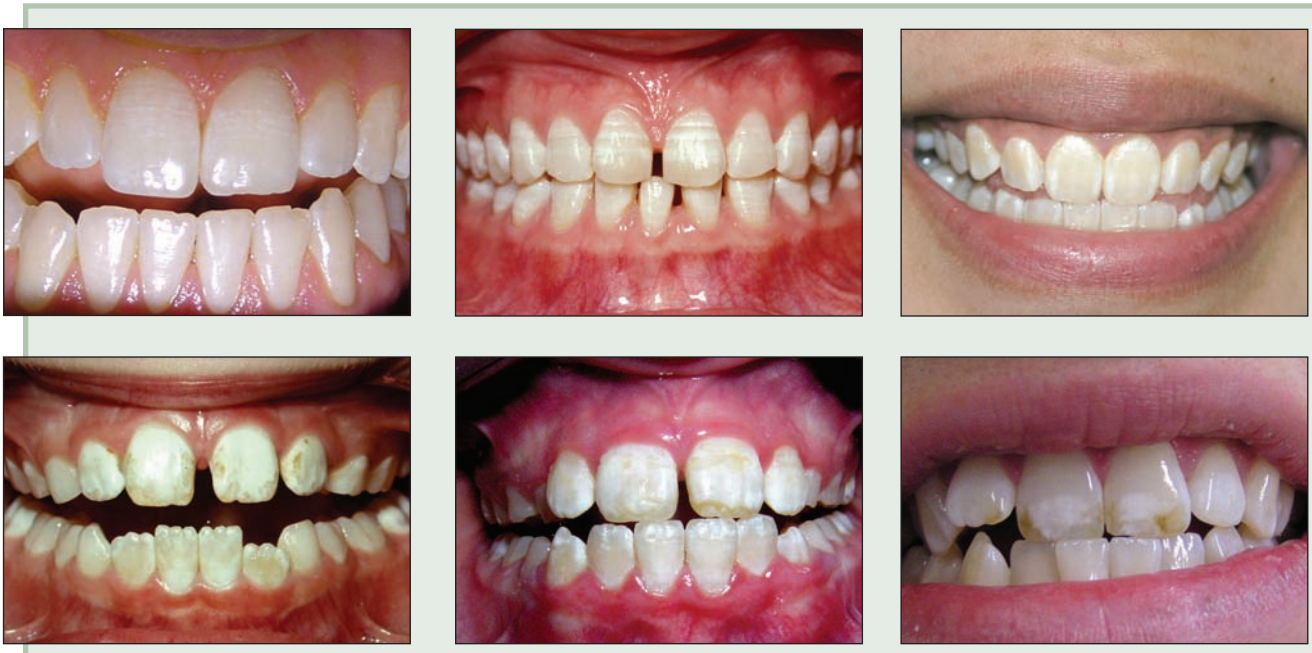

Figure 1: Six typical cases of mild to moderate dental fluorosis. The condition presents as various forms of white chalky spots and streaks, sometimes covering the entire tooth surface. In very mild cases (top left), these spots are barely noticeable. In moderate cases, areas of brown discolouration may occur. Among the patients depicted here, all but the patient at top left requested some form of treatment. All of the patients had nominal exposure to systemic fluoride (through fluoridated water, fluoride supplements or fluoridated toothpaste).

Table 1 Effects of the timing of fluoride exposure on the pattern of dental fluorosis

Daily fluoride intake[a] (mg/kg) and age at exposure	Severity of dental fluorosis	% prevalence[b] (95% CI)	Potential sources of excess fluoride	Permanent teeth usually affected
Low (< 0.05)	Mild	< 15 (10–22)		
0–3 years			Fluoridated tap water used for infant formula Early use of fluoridated toothpaste	Maxillary incisors, all first molars
3–6 years			Fluoridated water Minor ingestion of fluoridated toothpaste	Premolars, canines, second molars
0–6 years			Any combination of the above	All teeth
Medium (0.05–0.15)	Moderate	12.5 (7.0–21.5)		
0–3 years			Fluoridated tap water used for infant formula Early fluoridated toothpaste use General anesthetics Fluoride tablets Fluoridated water	Incisors, first molars and tips of canines and premolars
3–6 years			Fluoridated water Intentional fluoridated toothpaste ingestion General anesthetics Fluoride tablets	Cervical third of incisors, first molars and tips of canines and premolars
0–6 years			Any combination of the above	All teeth
High (> 0.15)	Moderate–severe	1–26[c]		
0–3 years			Early fluoridated toothpaste use Elevated fluoride in the drinking water (> 4 ppm fluoride) Pollution	All teeth
3–6 years			Pollution Elevated fluoride in drinking water Intentional toothpaste ingestion	All teeth
0–6 years			Any combination of the above May also be complicated by increased retention (e.g., kidney problems)	All teeth

CI = confidence interval
[a]Adapted from data published by the Committee on Fluoride in Drinking Water (U.S. National Research Council).[1]
[b]Prevalence estimates according to McDonagh and others.[4]
[c]Statistical analysis was not conducted for data from patients with severe fluorosis (U.S. National Research Council)[1]

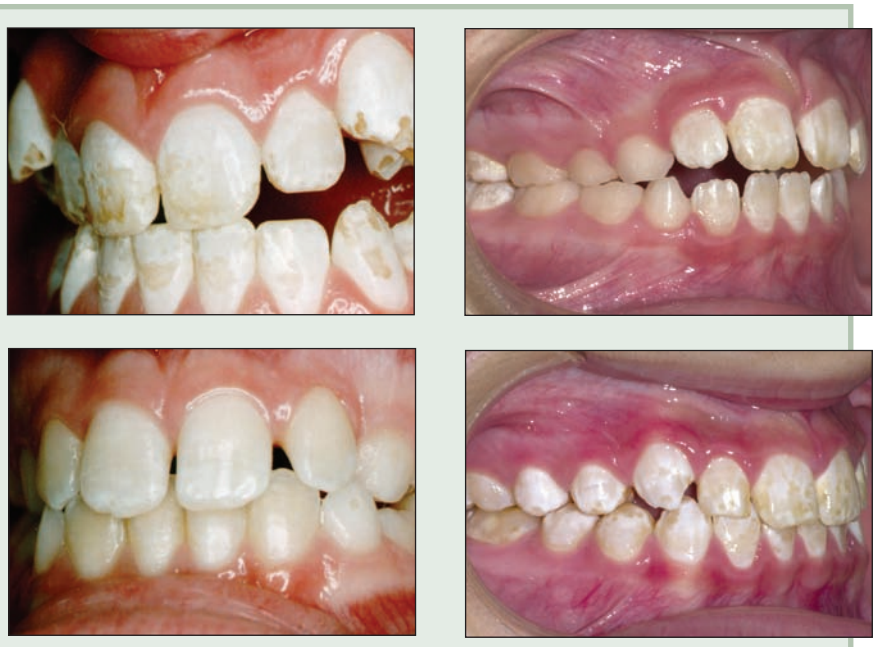

Figure 2: Family case studies of dental fluorosis. **Left:** Fraternal twins, 12 years of age, were exposed from birth to 1 ppm fluoride in drinking water. Regular use of fluoridated toothpaste from an early age resulted in extra fluoride ingestion in the girl (top left) because she was less efficient at expectorating the toothpaste than her brother, who had mild fluorosis (bottom left). **Right:** Siblings: 8-year-old girl (top right) and 12-year-old boy (bottom right). Both siblings had erosion of the enamel and white-spot mottling on all permanent teeth, accompanied by some staining. Both complained about the cosmetic appearance of their teeth. The patients and their parents stated that the problem had affected the children's self-image. The medical history was noncontributory. Several potential sources of excess fluoride were identified: general anesthetic used during caesarean birth, fluoridated tap water (although the mother breastfed each child for 6 months) and early (at 12 months of age) use of a "triple-swirl" of fluoridated toothpaste containing about 3 mg of fluoride.

of the tooth. The excess fluoride inhibits the final stages of tooth maturation. As a result, the surface enamel becomes hypercalcified while the subsurface layers are defective and hypocalcified, which makes bonding difficult. Mild fluorosis appears as chalky white spots or streaks, and moderate fluorosis may be associated with some structural loss of the surface enamel in thin layers, with or without accumulation of stain (**Fig. 1**). In more severe forms of fluorosis (such as those shown in **Fig. 2**), simple microabrasion (surface polishing) may be inadequate to remove the fluorotic enamel. More extensive treatment (involving composite resins, porcelain veneers and sometimes full-coverage restorations) is provided by most dentists.

Sources of Excess Systemic Fluoride

Foods and Beverages

Water containing fluoride at 1 ppm or higher (either naturally or artificially) and beverages made with fluoride-containing water contribute the most to daily fluoride intake. Because fluoride accumulates in bone, some foods (e.g. tinned salmon, mechanically separated chicken that contains ground bone) are rich in fluoride. Other foods also contain naturally high levels of fluoride. Dark tea,[6] for example, is rich in fluoride (3–6 ppm). Boiling water used for tea or cooking actually concentrates fluoride.

Fluoride Supplementation

Fluoride supplements are a major risk factor in dental fluorosis,[7] yet there is very little evidence that such supplements help to reduce dental caries.[8] The Canadian Dental Association (CDA), with input from various other health professionals, reached a consensus and modified its recommendations for the use of fluoride supplements in 1998.[9] The current protocol for fluoride supplement use was published in 2000.[10] If the current CDA guidelines (which state that total daily fluoride is not to exceed 0.05 mg/kg) are taken literally, physicians should abandon the prescription of supplemental fluoride altogether, since they are usually not prepared to estimate total fluoride intake from all sources. Fluoride works primarily by means of a topical effect,[11] so it may provide *some* benefit if given in lozenge form to patients at high risk for dental decay (i.e., those who have absolutely no topical exposure to fluoride and who are at high risk for caries because of their diet).

On the basis of average fluid intake and body weight, the daily fluoride intake of many infants exceeds 0.15 mg/kg.[1] To protect infants from ingesting too much fluoride, the American Dental Association now warns against using fluoridated tap water to make infant formula.[12]

Medicines

A large proportion of pharmaceuticals are fluorinated (e.g., Celebrex [celecoxib], Cipro [ciprofloxacin], Diflucan [fluconazole], Paxil [paroxetine], Dalmane [flurazepam], Lipitor [atorvastatin]). Furthermore, nearly all of the halogenated general anesthetics are fluorinated. Some

drugs, especially the general anesthetics, become defluorinated after administration and elevate serum levels of fluoride.[1]

Pollution

In the past, aluminum smelters produced a significant amount of fluoride pollution, but now the major fluoride polluters are phosphate fertilizer manufacturers and any industry that burns coal. Fluorosis associated with coal burning is well documented in China.[13]

THE AUTHOR

Dr. Hardy Limeback *is associate professor and head, preventive dentistry, University of Toronto, Toronto, Ontario. Email: Hardy.Limeback@dentistry.utoronto.ca.*

Dr. Limeback will be giving 3 presentations at the PDC: "New consumer products and fluorides," and "Cervical root hypersensitivity — ozone in dentistry," on Friday, March 7, and "New products, fluoride, root sensitivity and ozone," on Saturday, March 8.

References

1. Fluoride in drinking water: a scientific review of EPA's standards. Committee on Fluoride in Drinking Water, National Research Council, 2006. Available: www.nap.edu/catalog.php?record_id=11571.

2. Fomon SJ, Ekstrand J, Ziegler EE. Fluoride intake and prevalence of dental fluorosis: trends in fluoride intake with special attention to infants. *J Public Health Dent* 2000; 60(3):131–9.

3. Rozier RG. The prevalence and severity of enamel fluorosis in North American children. *J Public Health Dent* 1999; 59(4):239–46.

4. McDonagh MS, Whiting PF, Wilson PM, Sutton AJ, Chestnutt I, Cooper J, and others. Systematic review of water fluoridation. *BMJ* 2000; 321(7265):855–9.

5. Ishii T, Suckling G. The severity of dental fluorosis in children exposed to water with a high fluoride content for various periods of time. *J Dent Res* 1991; 70(6):952–6.

6. Whyte MP, Essmyer K, Gannon FH, Reinus WR. Skeletal fluorosis and instant tea. *Am J Med* 2005; 118(1):78–82.

7. Warren JJ, Levy SM. Current and future role of fluoride in nutrition. *Dent Clin North Am* 2003; 47(2):225–43.

8. Ismail AI, Bandekar RR. Fluoride supplements and fluorosis: a meta-analysis. *Community Dent Oral Epidemiol* 1999; 27(1):48–56.

9. Limeback H, Ismail A, Banting D, DenBesten P, Featherstone J, Riordan PJ. Canadian Consensus Conference on the appropriate use of fluoride supplements for the prevention of dental caries in children. *J Can Dent Assoc* 1998; 64(9):636–9.

10. Swan E. Dietary fluoride supplement protocol for the new millennium. *J Can Dent Assoc* 2000; 66(7):362–3.

11. Limeback H. A re-examination of the pre-eruptive and post-eruptive mechanism of the anti-caries effects of fluoride: is there any anti-caries benefit from swallowing fluoride? *Community Dent Oral Epidemiol* 1999; 27(1):62–71.

12. American Dental Association. ADA offers interim guidance on infant formula and fluoride. Posted November 9, 2006. Available: www.ada.org/prof/resources/pubs/adanews/adanewsarticle.asp?articleid=2212.

13. Feng YW, Ogura N, Feng ZW, Zhang FZ, Shimizu H. The concentrations and sources of fluoride in atmospheric depositions in Beijing, China. *Water Air Soil Pollut* 2003; 145:95–107.