

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. Readers are encouraged to do more reading on the topics covered. If you would like to submit or answer a question, contact editor-in-chief Dr. John O'Keefe at [jokeefe@cda-adc.ca](mailto:jokeefe@cda-adc.ca).

## QUESTION 1

Is it possible to salvage impacted strategic teeth associated with extensive dentigerous cysts?

### Background

**D**entigerous cysts constitute the second most common type of jaw cyst, representing 14% to 20% of all jaw cysts.

These cysts are more common in the mandible and occur more frequently in males. They are always radiolucent and are usually unilocular.<sup>1-5</sup> A dentigerous cyst results from proliferation of reduced enamel epithelium after formation of the enamel and is attached to an impacted tooth; such cysts are often discovered on routine radiographs or when films are obtained to determine why a tooth has failed to erupt.<sup>3-6</sup> Dentigerous cysts are common in children and are easy to treat when small (at which stage they are called eruption cysts) simply by unroofing. However, if the cysts become extensive, they are more difficult to manage. Associated teeth become impacted and may be displaced considerable distances (because of pressure from the cyst). Surgical management may require

the removal of several teeth or tooth buds and may endanger the vitality of adjacent teeth. However, enucleation of the cyst and extraction of associated teeth is often not in the patient's best interests. In particular, extraction of associated teeth in children may have functional, cosmetic and psychological consequences. The problem of how to replace dentition in growing children is also a concern. For adolescents, we feel that it is often inappropriate to extract affected anterior teeth, since combined surgical and orthodontic treatment can salvage deeply impacted strategic teeth (especially canines) associated with large dentigerous cysts.<sup>1</sup> Aggressive surgery is unnecessary, as recurrence seldom if ever occurs after enucleation.<sup>1-5</sup> The appropriate mode of treatment must take into account several clinical criteria.

### Specific Criteria for Management

Dentigerous cysts block tooth eruption, displace teeth when they become enlarged, destroy bone and encroach on vital structures (e.g., by encompassing or displacing the alveolar nerve or compressing the maxillary antrum).<sup>1-5</sup> One treatment consists of enucleation of the cyst and extraction of the tooth or teeth embedded in or impacted by the cyst.<sup>1-7</sup> This approach is favoured in cases involving impaction of a single tooth, such as a wisdom tooth in an adult, which has no function. However, removal of extensive cysts will lead to the loss of several teeth.<sup>1</sup> Conservative methods for eliminating cysts include decompression and marsupialization without removal of associated teeth.<sup>1-5</sup> Recently defined criteria for selecting the treatment modality (both indications and contraindications) refer to cyst size and site, patient age, the dentition involved and the involvement of vital structures.<sup>1</sup> Enucleation of the cyst without extraction of impacted teeth may be indicated for



**Figure 1a:** Orthopantomogram of an impacted canine displaced to the inferior border of the chin by a large dentigerous cyst of the mandible extending from the left canine to the right first premolar tooth.



**Figure 1b:** Lateral cephalogram obtained at the same time.



**Figure 2a:** Orthopantomogram 5 years after surgery. The tooth has been brought into occlusion after surgical exposure and orthodontic guidance, and the vitality of all of the teeth has been preserved.



**Figure 2b:** Lateral cephalogram obtained at the same time.

children and adolescents as a means of salvaging the involved dentition if the involved tooth is strategic. For instance, an 11-year-old boy had a swelling in the vestibular area of the left mandibular canine region; several teeth were impacted by a large dentigerous cyst in the symphysis from the right canine tooth to the left premolar (Figs. 1a and 1b). Aspiration of the lesion was performed first; in many cases, such aspiration reveals a clear yellow fluid. Next, the entity must be confirmed by biopsy. In this case, excisional biopsy was performed under local anesthesia via a submarginal mucoperiosteal trapezoid flap reflected from the right canine tooth to the left premolar from under the attached gingiva; the lesion was removed after the cyst had been separated from the bone and incised off the tooth surface with a #15 scalpel. The flap was sewn apically in the vestibule, which left the crown exposed for bracket bonding. Orthodontic treatment was started 2 weeks postoperatively. In most cases, the canine can be brought into occlusion within several years depending on depth of impaction, patient age and other factors (Figs. 2a and 2b).

The capacity to regenerate bone is greater among children than among adults, and teeth with open apices have great eruptive potential. Thus, large dentigerous cysts in children can be treated

differently, and conservative treatment with tooth preservation should be considered. However, the radiographic and clinical findings for dentigerous cysts are not diagnostic, and odontogenic keratocysts, unilocular ameloblastomas, and many other odontogenic and nonodontogenic tumours have similar features; thus, other lesions must be ruled out by histopathologic examination.<sup>3,5,8,9</sup> If other pathologic entities are reported, the treatment plan may be altered as appropriate for further pertinent treatment. ♦

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The authors have no declared financial interests.

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## QUESTION 2

Some of my patients are still having problems with dentinal hypersensitivity, even after conventional treatment. Are the new oxalate desensitizing agents the answer?

## Background

Dentin hypersensitivity, associated with either a restoration or exposure of the root surface, is caused by the rapid movement of fluid in the dentinal tubules. This phenomenon was first described as the hydrodynamic theory of dentinal pain by Brannstrom,<sup>1</sup> who attributed the fluid

flow to osmotic stimuli. It is widely accepted that this rapid flow of fluid creates a pressure change across the dentin, which stimulates the nerve fibres and results in the perception of pain.<sup>2</sup> It is therefore no surprise that the accepted treatments focus on occluding the dentinal tubules by various precipitates or covering the exposed dentin with an impermeable layer to prevent the osmotic gradient changes that create the painful stimuli.

Many products are available to decrease the sensitivity caused by exposure of the cervical dentin. One category of products consists of desensitizing toothpastes containing potassium nitrate, which penetrates the dentinal tubules and depolarizes the nerves, decreasing the painful stimulus.<sup>3</sup> Potassium nitrate gels that can be used in bleaching type trays for hypersensitive root surfaces include UltraEZ (UltraDent, South Jordan, Utah), Den-Mat Desensitize (Den-Mat, Santa Maria,

Calif.) and Relief (Discus, Culver City, Calif.). Another type of product is based on the fact that sclerotic dentin has dentinal tubules that are completely occluded by mineral deposits. To produce a similar clinical situation, fluorides are applied topically, creating precipitates of calcium fluoride, which can close the dentinal tubules.

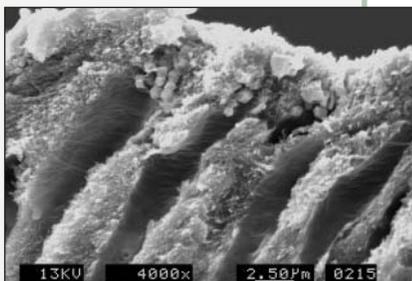
Products containing glutaraldehyde also work well for desensitization. Glutaraldehyde is an effective disinfectant. It kills bacteria and coagulates the plasma proteins within the dentinal fluids, forming a coagulation plug. Gluma Desensitizer (Heraeus Kulzer, Armonk, N.Y.), 5% glutaraldehyde with 35% hydroxyethylmethacrylate (HEMA) and water, is effective as a desensitizing agent under restorations and does not interfere with the bonding of resins to dentin. However, glutaraldehyde can be irritating to the soft tissues and should be used sparingly; it is applied with a microbrush, and the area is blotted to remove any excess. Hanks and others<sup>4</sup> reviewed the cytotoxic properties of glutaraldehyde, and Li and others<sup>5</sup> discussed its mutagenic potential. Like all products containing HEMA, glutaraldehyde can cause contact dermatitis, and it penetrates latex gloves.<sup>6</sup>

By their very nature, the many different types of dentin bonding agents currently available constitute a class of desensitizing agents because they form a hybrid layer. These bonding agents include total-etch 1-bottle and multi-bottle systems, and self etching 1-bottle and 2-bottle systems.

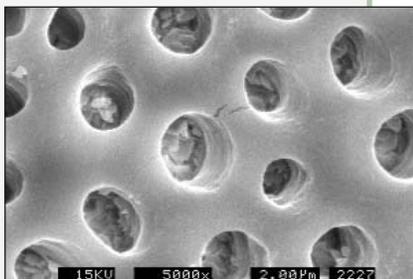
## Current Status of Oxalates

Whether used to treat exposed cervical dentin or exposed dentin under a restoration, application of oxalate desensitizing materials to the dentin results in precipitation of potassium oxalate or ferric oxalate crystals. Materials like Protect Drops (John O Butler, Chicago, Ill.) and Sensodyne Sealant (GlaxoSmithKline, Research Triangle Park, N.C.) are designed for application to exposed cervical dentin. Potassium oxalate has been used to occlude open tubules in sensitive cervical dentin, causing "instant sclerosis" of the tubules.<sup>7</sup>

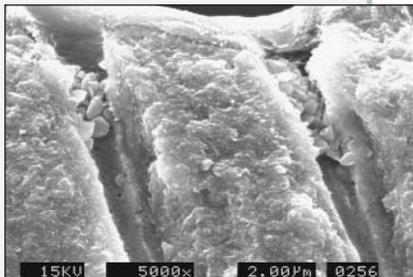
Intended for use under direct and indirect restorations, Super Seal (Phoenix Dental, Fenton, Mich.) is a potassium salt of oxalic acid; combined with water, it creates a calcium oxalate precipitate on the dentin, which affects the bond strength of



**Figure 1:** Scanning electron micrograph of typical deposition of oxalate crystals on the dentin surface. Courtesy of Dr. David Pashley.



**Figure 2:** Scanning electron micrograph of oxalate crystals that have precipitated in dentinal tubules. Courtesy of Dr. David Pashley.



**Figure 3:** Scanning electron micrograph (lateral view) of dentinal tubules, showing deposition of calcium oxalate crystals. Courtesy of Dr. David Pashley.

**Table 1** Shear bond strength of various bonding agents in the presence of BisBlock and Super Seal

| Bonding agent | Mean shear bond strength (standard deviation) (MPa) |               |                 |
|---------------|---|---------------|-----------------|
|               | Control   | With Bisblock | With Super Seal |
| One-Step      | 21.77 (0.49)  | 23.06 (3.80)  | 23.14 (1.59)    |
| Single Bond   | 22.64 (1.61)  | 22.38 (2.96)  | 11.34 (6.02)    |
| Solo Plus     | 20.04 (2.23)  | 10.60 (3.67)  | 7.30 (2.25)     |
| Prime Bond NT | 14.96 (5.44)  | 7.30 (2.87)   | 8.64 (5.52)     |
| Excite        | 17.99 (1.03)  | 7.38 (3.35)   | 3.82 (3.89)     |
| All-Bond 2    | 23.59 (2.95)  | 20.57 (3.19)  | 9.54 (4.71)     |

*Single Bond (3M ESPE, St. Paul, Minn.); Excite (Ivoclar Vivadent, Amherst, N.Y.); All-Bond 2 (Bisco)*

any dentin bonding agents used over it. With OptiBond Solo Plus (sds/Kerr, Orange, Calif.) and Prime & Bond NT (Dentsply), bond strength was much lower than with a control agent.<sup>8</sup> In their newsletter,<sup>9</sup> Clinical Research Associates, Inc. reported that “Super Seal reduced bond strengths significantly for 5 out of the 6 adhesives tested.” In contrast, BisBlock (Bisco, Schaumburg, Ill.) oxalate desensitizer uses a unique patented approach for sealing the dentinal tubules,<sup>10</sup> whereby the dentin is etched before application of the oxalate. Removal of calcium from the reactive surface creates a preferential deposition zone for the calcium oxalate crystals within the dentinal tubules, not on the dentinal surface (Figs. 1 to 3). When BisBlock is applied to the root surface, this deposition within the tubules prevents dislodgement caused by toothbrush abrasion.

It is of utmost importance to remember that many desensitizing agents affect the bond strength of the adhesives that we use every day. Specifically, oxalate desensitizers yield low bond strengths when used with low pH (highly acidic) adhesives. Table 1 shows that not all total-etch adhesives are compatible with oxalate desensitizers. For example, only One-Step (Bisco) is universal, working equally well with both BisBlock and Super Seal. ♦

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## QUESTION 3

What can I do for a patient who is taking bisphosphonates and who has an area of exposed bone in the oral cavity that will not heal?

### Background

**B**isphosphonates are synthetic analogues of inorganic pyrophosphate with a high affinity for calcium. They are rapidly cleared from the circulation, binding to bone mineral and thus selectively concentrating in bone. Bisphosphonates are potent inhibitors of osteoclastic activity, particularly when administered intravenously.<sup>1</sup> In addition, they have anti-angiogenic properties, reduce mineral loss in metastatic bone lesions and may be tumoricidal,<sup>2</sup> which makes them important agents in cancer therapy.<sup>1-3</sup>

Bisphosphonates constitute an important class of medications used to treat osteoporosis (for which they are often administered orally), Paget's disease of bone, primary lesions due to bone cancer, advanced cancer (specifically breast, lung and prostate) with metastasis to bone, and hypercalcemia due to malignancy (for all of which they are administered intravenously).<sup>1,3</sup> The complication of an oral area of bone that will not heal has been termed "bisphosphonate-associated osteonecrosis" (BON). The most common IV bisphosphonates associated with BON are pamidronate (e.g., Aredia, Novartis Pharmaceuticals, Dorval, Quebec) and zoledronic acid (e.g., Zometa, Novartis Pharmaceuticals). Recently, cases of delayed healing or absence of healing after dental extraction have been reported in patients with cancer who were receiving injectable bisphosphonates; spontaneous lesions in the jaw have also been reported.<sup>4,5</sup> BON has been reported among patients taking oral alendronate (e.g., Fosamax, Merck Frosst, Kirkland,

Quebec) to treat osteoporosis or osteopenia. The common link in all of the reported cases was the use of bisphosphonates for the treatment of cancer; some of the patients were also being treated with steroids.<sup>5</sup>

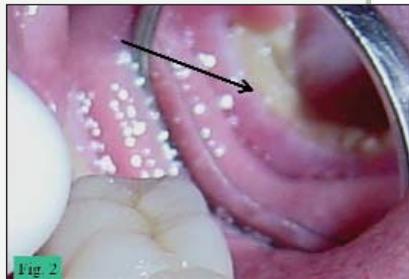
Although the exact mechanism leading to BON has not been confirmed, it is known that bisphosphonates potentially inhibit osteoclastic activity, increase mineralization of bone and reduce the vascularity of bone, all of which result in reduced repair and remodelling potential. The bone may become highly mineralized and dense and may be unable to meet the demands of remodelling associated with trauma, which ultimately leads to necrosis. Trauma associated with dental extractions, ill-fitting prosthodontic appliances, periodontal and dental disease, and systemic factors (e.g., oral infections, poor oral health and medical compromise) may increase the risk of BON. Spontaneous oral complications have been reported, and although the lesion may be asymptomatic, most common initial complaints can include intraoral pain and the presence of roughness because of exposed bone.

### Clinical Presentation

Patients usually present asymptotically, but there may be pain in the maxilla and/or the mandible; secondary infection may occur when the necrotic bone is exposed to the oral environment. The osteonecrosis is often progressive and may lead to extensive areas of bone exposure, dehiscence or sequestration. If secondary infection occurs, the patient may complain of severe pain, bad taste in the mouth, bad breath and paresthesia, which may indicate compression of a peripheral nerve. The history most commonly associated with this process is delay in or absence of healing after trauma. There are no radiographic manifestations in the early stages. The diagnosis of BON is based on a thorough medical, dental and pharmacological history, as well as a complete clinical examination. The exposed bone may become hydrated through exposure to saliva and may be elevated above the contours of the adjacent normal bone (Figs. 1 and 2).



**Figure 1:** Exposure of the bone on the lingual aspect of the mandible, with elevation of the bone contours above the height of the gingival tissue and no sign of clinical infection.



**Figure 2:** Exposed bone on the buccal aspect of the retromolar pad, with elevation of bony contours and no clinical signs of secondary infection.

## Management

Definitive guidelines for the diagnosis and management of BON have not yet been established, but current guidelines are based on those for osteonecrosis after radiation therapy. The ideal approach is preventive and consists of eliminating all potential sites of infection and trauma before bisphosphonate therapy is initiated. Thus, dental preventive measures should be in place before bisphosphonates are prescribed and should be reinforced at regular dental visits.

For patients who have been on IV bisphosphonate therapy for less than 3 months, a similar preventive strategy may be employed. However, if a patient has been receiving therapy for more than 3 months and dental treatment is required, the following approach is appropriate:

1. Routine dental care may be performed, with limited use of vasoconstrictors.
2. Grossly carious teeth should be treated endodontically. Extractions should be avoided if possible.
3. Periodontal procedures should be performed atraumatically.
4. Multiple dental extractions should be avoided; if needed, an atraumatic approach, such as sectioning multirooted teeth, should be undertaken.
5. Areas of BON with sharp edges of bone should be recontoured to reduce trauma to soft tissues (if this area is secondarily infected, antibiotics should be prescribed).
6. Prosthodontic appliances must have good fit and function.
7. Referral to providers experienced in the treatment of osteonecrosis and consultation with the patient's medical oncologist are suggested.
8. To date, there is no evidence to support discontinuation of bisphosphonate therapy to promote healing of necrotic bone.

9. While hyperbaric oxygen therapy is considered for postradiation osteonecrosis, its use for BON remains to be established.

## Conclusion

Dental professionals must be involved in the prevention of BON by providing excellent preventive and regular supportive care. ♦

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## QUESTION 4

## Why do some patients complain of a toothache long after a successful endodontic procedure?

### Background

Dentists are routinely asked to diagnose and treat pain of presumed dental origin. Orofacial pain is typically of dental origin and can usually be resolved by one or more dental treatments, including nonsurgical and surgical endodontic procedures. However, some patients continue to report pain in the teeth or adjacent areas, even when clinical and radiographic criteria for successful treatment have been met. Most of these patients have what is known as neuropathic pain. This condition presents a significant challenge to dentists, particularly endodontists, who are often asked to treat such patients.

### Epidemiological Studies

Only limited epidemiological studies are available on the prevalence of chronic neuropathic pain after an endodontic procedure. Marbach and others<sup>1</sup> conducted a survey of patients followed by clinical and radiographic examination of female patients who continued to report tooth pain more than 1 month after nonsurgical endodontic treatment and found that about 3% (8/256) of the female patients fulfilled

their criteria for continuous neuropathic pain. Campbell and others,<sup>2</sup> following a similar protocol with patients who had previously undergone surgical endodontic treatment, found that 5% (6/118) of the patients reported ongoing pain after surgery (average time of assessment 21 months after the procedure). In a recent study by Polycarpou and others,<sup>3</sup> patients from a tertiary referral centre were examined clinically and radiographically 12 to 59 months after undergoing nonsurgical or surgical endodontic treatment; 12% (21/175) of the patients had persistent pain in the absence of clinical or radiographic signs of dental disease.

### Neuropathic Pain in the Context of Dental Treatment

In studies by various authors,<sup>1-6</sup> most patients with continuous neuropathic pain related the onset of their pain to some form of dental treatment, a dental infection or dental trauma. In addition, patients who continued to seek invasive dental treatment did not experience any pain relief, and some patients had more pain after these procedures.

**Table 1** Medications used in the management of neuropathic pain

| Therapeutic Class                                  | Drugs   |
|--|---|
| <b>Antidepressants</b>                             |   |
| • Tricyclic antidepressants                        | Amitriptyline, desipramine, doxepin, imipramine, nortriptyline  |
| • Serotonin and norepinephrine reuptake inhibitors | Duloxetine, venlafaxine   |
| <b>Anticonvulsants</b>                             | Carbamazepine, gabapentin, lamotrigine, levetiracetam, oxcarbazepine, phenytoin, tiagabine, topiramate, valproic acid, zonisamide |
| <b>Benzodiazepines</b>                             | Clonazepam  |
| <b>Anti-arrhythmics</b>                            | Mexiletine  |
| <b>Analgesics</b>                                  | Acetaminophen, COX-2 inhibitors, NSAIDs, opioids, tramadol  |
| <b>NMDA antagonists</b>                            | Amantadine, dextromethorphan, ketamine, memantine   |
| <b>Topical formulations</b>                        | Capsaicin, clonidine, lidocaine   |
| <b>Others</b>                                      | Baclofen, tizanidine  |

NSAID = nonsteroidal anti-inflammatory drug, COX-2 = cyclooxygenase 2, NMDA = N-methyl-D-aspartate

## Deafferentation Pain

The pain that these patients experience may be due to deafferentation of the trigeminal nerve (cranial nerve V). Deafferentation is defined as the cutting or crushing of a peripheral nerve. The pain associated with deafferentation is similar to the pain described by amputees, who may experience unusual sensation or pain around the site of an amputation or peripheral to the site (known as phantom limb pain). Complex peripheral and central mechanisms are involved in the initiation and maintenance of neuropathic pain. The primary mechanism involves the release of chemicals from the peripheral tissues or primary afferent nerve endings as a result of tissue injury or inflammation. These chemicals can increase the excitability and decrease the activation threshold of peripheral nociceptors (a process known as peripheral sensitization), which in turn increases nociceptive input to the central nervous system. This bombardment of input induces spontaneous activity, expansion of receptive fields, lowering of activation thresholds and hyperexcitability of neurons in the central nervous system (central sensitization). An experimental animal model developed at the University of Toronto for assessing single-nerve injury after endodontic procedures (nerve amputation) has given us much insight into the mechanisms of neuropathic pain.<sup>7</sup>

## Diagnostic Considerations

Diagnosis should begin with a comprehensive history as well as clinical and radiographic examination. A differential diagnosis should be established to rule out pain of dental, soft tissue or pathological (peripheral or central) origin. Once the diagnosis of neuropathic pain is established, no further dental procedures should be performed unless specific findings of dental pathosis are identified. Otherwise, ineffective or inappropriate treatment may be rendered. The practitioner must then choose to initiate some form of treatment or refer the patient to a practitioner with a more comprehensive understanding of these neuropathic conditions.

## Management of the Problem

Current treatment modalities often require a multidisciplinary approach. Pharmacological management, often the treatment of choice, involves the use of peripheral and/or centrally acting medications (Table 1). Psychological counselling may also be considered. Practitioners who treat neuropathic pain include those with

backgrounds in oral medicine and orofacial pain, pain medicine and management, and behavioural medicine.

## Conclusions

All dental practitioners need to understand the concept of neuropathic pain and should be able to recognize the condition. However, the management of neuropathic pain requires a nonsurgical and pharmacological approach that may be beyond the training and experience of dental practitioners who are accustomed to treating acute pain. It is only with this awareness that appropriate and effective care can be delivered to patients with this type of pain. ♦

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