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 contribute to this section, please contact editor-in-chief Dr. John O'Keefe at jokeefe@cda-adc.ca.

QUESTION 1

What are the common causes of facial neuropathy?

Background

A neuropathy is a disease or abnormality of the nervous system. Facial neuropathy affects the facial nerves and can therefore be of great significance to the practising dentist. The symptoms include anesthesia, dysesthesia or paresthesia of the affected nerve. The condition may present as moderate to severe pain, burning, tingling, prickling or numbness.

The differential diagnosis for facial neuropathy usually includes local trauma, multiple sclerosis and diabetes mellitus. Other, less common diagnoses, including neoplasms, odontogenic lesions, amyloid disease, syphilis, osteomyelitis and AIDS, are not discussed in this article.

A good understanding of facial neuropathy is essential to avoid misdiagnosis and may even save the patient's life.

Local Trauma

One potential complication of many dental procedures is local trauma leading to nerve injury. Such trauma most commonly occurs during extraction of the lower third molar (rate of 1–4 cases per 1,000 extractions) or inferior alveolar nerve block injection (rate of 1 in 20,000 to 850,000 injections).[1,2] Injuries of this nature usually resolve spontaneously within 1–6 months.

Nerve injury during anesthesia is usually related to inferior alveolar nerve block but affects the lingual nerve more frequently than the inferior alveolar nerve.[3] The primary cause is usually neurotoxicity or mechanical injury.

Dental management of facial paresthesia or anesthesia resulting from nerve injury includes reassuring the patient and monitoring the affected area for 3 months. Monitoring involves the 2-point test, in which 2 sharp points are positioned lightly on the affected area, initially in close proximity and then at increasing distances until the patient can discriminate them as 2 separate points. A 2-point test on normal tongue or lip tissue would reveal 2-point discrimination at 3–5 mm, whereas a tongue or lip with paresthesia may exhibit 2-point discrimination at 7–20 mm or more.[4] Patients with dysesthesia or more debilitating paresthesias may need referral for further examination or additional treatment (e.g., low-dose antidepressants).[4]

Multiple Sclerosis

Multiple sclerosis (MS) is a degenerative disease of the central nervous system that causes demyelination of the brain and spinal cord. This chronic inflammatory disease affects primarily young adults in temperate climates.[5] The prevalence of MS in Canada, at 55–110 cases per 100,000, is one of the highest national rates in the world because of this country's temperate geographic location.[5]

The first symptom is often paresthesia of the trunk, face or extremities. Other symptoms include visual problems, locomotor problems, dizziness, and weakness or clumsiness of an appendage. The 3 most common facial symptoms of MS are trigeminal neuralgia, trigeminal sensory neuropathy and facial palsy.[5] Trigeminal neuralgia is the initial symptom in 1 of every 300 cases and eventually occurs in 1 of every 50 cases.[6] It is usually unilateral but when associated with MS, it can present bilaterally. Another symptom of MS is facial paralysis, which usually appears later in the disease process and can affect 25% of patients with this disease.[7] Patients with a suspected diagnosis of MS should be referred to an appropriate physician for definitive diagnosis (by magnetic resonance imaging) and possible treatment.

Many patients with confirmed MS are taking steroids, immunosuppressants, phenytoin or carbamazepine, or a combination of these drugs.[5] All of these medications have significant dental implications. Use of steroids may lead to adrenal atrophy, which in turn may cause adrenal crisis if the patient experiences anxiety at the dental

office. Patients who are taking immunosuppressants will probably need antibiotic prophylaxis and a complete blood count before any surgery.[5] Erythromycin can increase the toxic effects of phenytoin and carbamazepine and should therefore be avoided for patients with MS. In addition, nonsteroidal anti-inflammatory drugs should be used with caution in patients who are taking methotrexate, as they may amplify this drug's cytotoxicity to hazardous levels.[5]

Diabetes Mellitus

The most common known cause of neuropathy is diabetes. However, facial neuropathy is just one of many possible types of neuropathy affecting patients with this disease. Still, given the frequency of diabetes in the Canadian population (5% with confirmed diabetes, 3%–5% with undiagnosed diabetes),[8] most dentists are likely to encounter patients with facial neuropathy secondary to diabetes at some point during their years of practice.

Blood glucose control is the most important therapeutic objective in the management of diabetic neuropathy and is the only approach that has been proven to prevent or slow the progression of the condition.[9]

Conclusion

Dentists represent the main source of oral and maxillofacial care. As such, when a patient is affected by facial neuropathy, the dentist may be the first health care provider to encounter the problem. It is therefore important that dentists be able to provide treatment, referral or information pertinent to the condition. By knowing the possible causes and symptoms of facial neuropathies, dentists will be able to diagnose and manage these conditions effectively and efficiently.

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QUESTION 2

Is mineral trioxide aggregate a valid alternative to calcium hydroxide for promoting apexification in infected fully developed teeth with open apices?

Background

The success of root canal treatment depends on elimination of root canal contents, proper cleaning and shaping, followed by hermetic obturation of the root canal system. When treatment fails, endodontic retreatment, either with or without surgery, must be performed. Periapical surgery is unlikely to be successful unless the root canal system has been adequately debrided and sealed.

Endodontic retreatment of infected fully developed teeth with open apices (**Fig. 1**) usually presents a challenge. The lack of apical constriction makes complete debridement of the root canal and control of the obturation material almost impossible.[1] The creation of an apical barrier with hard tissue deposition (a procedure known as "apexification") is necessary to permit adequate filling.

Various techniques for apexification have been suggested, but the most common method involves the use of calcium hydroxide as a dressing material in the root canal system.[2] Although the mechanism by which calcium hydroxide induces the formation of a solid apical barrier is not fully understood, it has been suggested that this occurs as a result of its antibacterial properties (due to its high pH) and the presence of calcium and hydroxyl ions.[3] Calcium hydroxide inhibits periradicular osteoclastic activity and prevents the ingress of granulation tissue into the root canal.[3]

Despite its efficacy, calcium hydroxide has several disadvantages: variability of treatment time, the many appointments and radiographs needed to ensure apexification (resulting in difficulties with patient follow-up), the delay before final treatment and the possibility of increased risk of tooth fracture after extended use of calcium hydroxide.

Mineral Trioxide Aggregate

Recently, mineral trioxide aggregate (MTA) has been proposed for immediate closure of the apical opening without the need to wait for natural healing processes. Studies have demonstrated the formation of periradicular tissues (periodontal ligaments, bone and cementum) induced by MTA in endodontic procedures.[4] There are also several reports of superior biocompatibility of MTA with periodontal tissues,[5] its excellent sealing ability in the presence of moisture and its mechanical properties that render it effective as an apical sealing material.[6]

To date, MTA has been most successfully used to create an apical plug before final obturation with laterally condensed gutta-percha, thermoplasticized gutta-percha or composite resin.[1] MTA can also be considered as the sole obturation material, thus eliminating the gutta-percha obturation step and promoting an adequate seal before apexification by the intracanal delivery technique.

Procedure

After anesthesia, application of a rubber dam and preparation of adequate access, the root canal system is cleaned and shaped using intracanal instruments in combination with 2.5% sodium

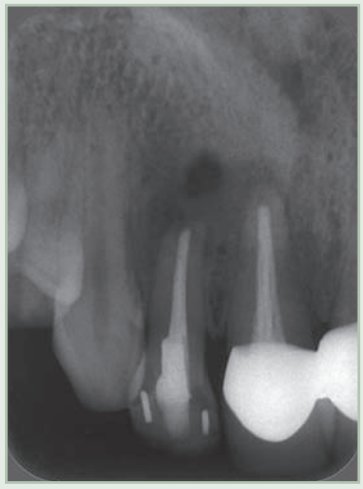

Figure 1: Pre-operative radiograph of an infected maxillary left lateral incisor with an open apex. Unsuccessful conventional root canal therapy had been followed by surgical treatment one year previously. Clinical examination revealed a sinus tract at the apical site of the tooth.

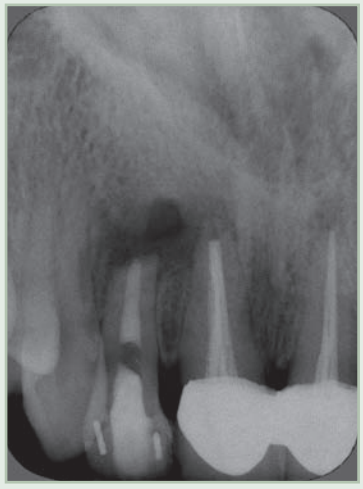

Figure 2: Periapical radiograph of retreated maxillary left lateral incisor filled with mineral trioxide aggregate.

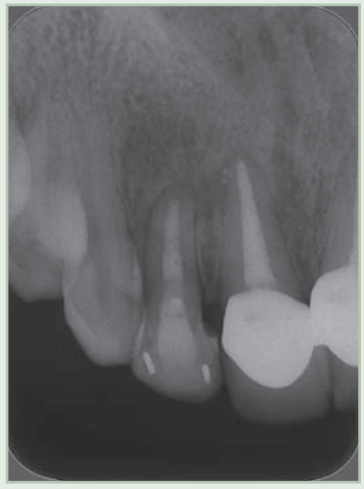

Figure 3: At one year, a follow-up radiograph of the maxillary left lateral incisor shows periapical healing with formation of new hard tissue at the site of the lesion.

hypochlorite (NaOCl) and 17% ethylenediaminetetraacetic acid (EDTA) irrigation. To limit bacterial infection, short-term treatment (1 week) with a calcium hydroxide dressing is recommended before application of MTA. After rinsing the calcium hydroxide from the root canal with NaOCl, large paper points are used to dry the root canal to the correct working length; care should be taken to avoid injuring the periapical tissue by inadvertent pressure.

MTA is mixed with the liquid provided to form a thick slurry, which is then placed in the canal using a small plugger and a gentle apical tamping technique to a level as close to the apex as possible. Placement of the material must be verified with radiographs. A moist cotton pellet is placed in the pulp chamber, which is then sealed with temporary restoration material for at least 3–4 h (**Fig. 2**). Once the MTA has set, the weakened root can be internally reinforced using composite resin restorative material, which will bond to the internal aspects of the root canal. Radiographic follow-up (**Fig. 3**) shows periodontal health and evidence of new hard tissue fomation in the apical area of the affected tooth.

MTA appears to be a valid option, not only for apexification, but also to treat failed infected root canal systems. Its advantages include reduced treatment time, good sealing ability and high biocompatibility.

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QUESTION 3

Why do dentists need to know about myofascial pain?

Background

When a patient presents with orofacial pain, the dentist must consider a variety of muscular disorders in the differential diagnosis (**Table 1**). One potential diagnosis is myofascial pain, which is characterized by regional dull, achy muscle pain and the presence of trigger areas (also called trigger points) within one or many craniofacial muscles, tendons or fascia. This pain may be referred to various facial areas, including the temporomandibular joint, one or more teeth, the mandible or the sinuses. In addition,

Table 1 Clinically important causes of facial pain

	Symptoms	Signs	Etiology
Muscle spasm[a]	Acute onset (not a chronic problem) Limited, painful mouth opening Feeling of tightness in the muscle	Palpable spasm, muscle tautness and acute tenderness Limited jaw mobility	Infection (e.g., pericoronitis) Direct trauma to the muscle Severe parafunction
Muscle co-contraction[b]	Limited mouth opening Pain with extended movement	Reduced voluntary mouth opening, but normal (though painful) assisted opening	Inflammatory joint disorder (e.g., osteoarthritis of the TMJ) Central excitation
Muscle soreness with delayed onset[c]	Episodic muscle stiffness Muscle soreness Pain on active muscle contraction (clenching) or function (chewing)	Muscle tenderness to palpation May or may not limit jaw opening (this movement may produce muscle co-contraction)	Local environmental change with release of bradykinin and substance P Fatigue and episodic use (e.g., eating a particularly tough food or episodic parafunction) Self-limiting (by definition)
Centrally mediated muscle pain[d]	Diffuse facial pain that increases with provocation or function Diffuse headache (tension type)	Generalized tenderness to palpation May have reduced serum 5-HT No identifiable peripheral pathology	Central nervous system excitation secondary to increased proprioceptive input, emotional stress, central sensitization from inflammation or neurogenic damage Etiology and distribution by sex (more common in females) similar to those of fibromyalgia
Myofascial pain[e]	Possible pain to the referred site	Reproducible tenderness in muscle Palpable taut band Pain referral on palpation Local anesthetic administered to trigger point resolves pain at local and referral sites	Central excitation results in hypersensitivity of nerve endings in a localized area of muscle, causing pain (afferent), taut band (efferent) and autonomic effects (temperature and blood flow) Palpation excites converging second-order neurons, resulting in referred pain

TMJ = temporomandibular joint, 5-HT = serotonin.
[a]Involuntary sustained contraction of a muscle.
[b]Protective contraction of antagonist muscles.
[c]Primary pain in response to prolonged contraction.
[d]Chronic deep pain that is not dependent on provocation but that may occur in response to provocation (particularly palpation), with lack of local tissue pathology.
[e]Hypersensitivity of localized bands of tissue within a muscle acting as a source of constant, deep pain.

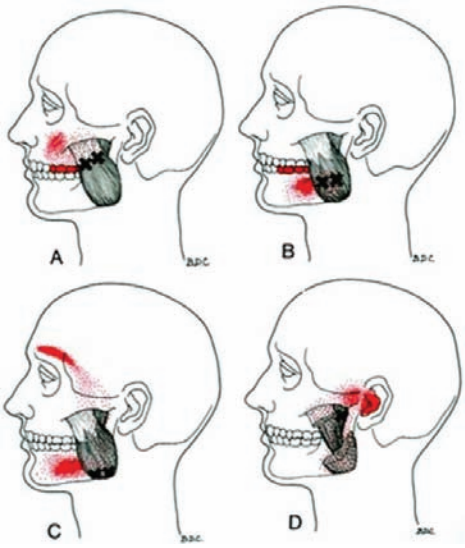

Figure 1: The **X**s indicate trigger areas in various parts of the masseter. Solid red shows essential referred pain zones, stippled areas are spillover pain zones.

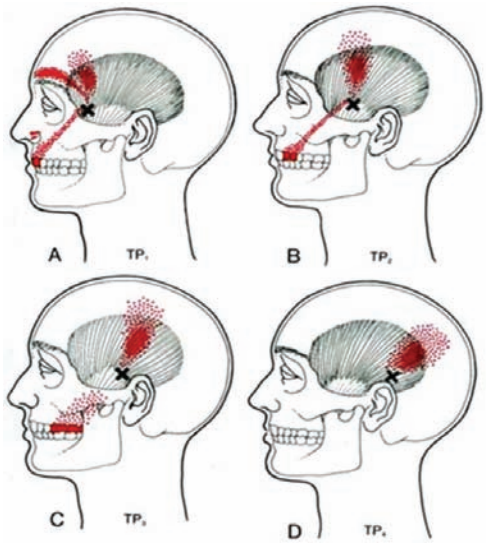

Figure 2: The **X**s indicate trigger areas in various parts of the temporalis muscle. Solid red shows essential referred pain zones, stippled areas are spillover pain zones.

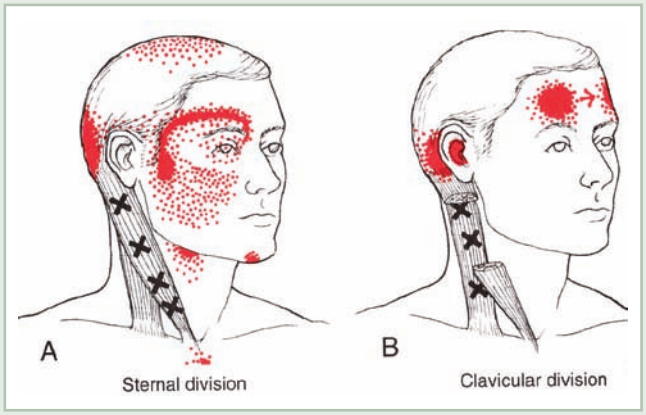

Figure 3: The **X**s indicate trigger areas in the sternal and clavicular divisions of the sternocleidomastoid muscle. Solid red shows essential referred pain zones, stippled areas are spillover pain zones.

referral of pain from trigger areas in the cervical musculature may produce symptoms of tension-type headache in the frontal or temporal skull.

The exact cause of myofascial pain remains unclear. It may be a response to acute trauma or chronic movements or positions causing anatomic or physiologic changes in the muscles and thus leading to formation of a trigger area. There may also be changes in the central nervous system, including the sympathetic nervous system. This form of muscle pain is controversial, as there are no laboratory tests or imaging techniques to diagnose the trigger points. In addition, the distribution of the referred pain rarely coincides directly with the distribution of a peripheral nerve. The diagnosis is usually based on symptoms and a clinical examination, although recent in vivo studies of human skeletal muscle near trigger areas have identified elevated levels of substance P, calcitonin-gene-related peptide, serotonin, norepinephrine, tumour necrosis factor α and interleukin 1β. Chairside diagnostic criteria for myofascial pain

include regional dull pain at rest, pain aggravated by function, a taut band or nodule that is noted in the muscle during palpation, and frequently a pattern of pain referral. Myofascial pain may be widespread (and diffuse) within the body or it may be limited to a single area (regional), for example, the superficial belly of the right masseter muscle.

The locations of trigger areas and their referred pain patterns are specific and reproducible. Trigger areas within the sternocleidomastoid, trapezius, masseter, temporalis and pterygoid muscles can all refer pain to the craniofacial region. Of significance for dentists is the fact that trigger areas within the masseter, temporalis, digastric, sternocleidomastoid and trapezius muscles may reproduce the patient's pain within one or more teeth (**Figs. 1–3**). In addition, the masseter, temporalis and lateral pterygoid trigger areas may refer pain to the temporomandibular joint area. Patients may report symptoms in the tongue, the pharynx, the hard palate and the sinuses.

Myofascial pain tends to be deep and more difficult to localize, whereas dental and cutaneous pain is generally easier to localize. Signs and symptoms suggestive of nonodontogenic pain include lack of an adequate local dental cause for the pain, recurrence of pain in spite of reasonable dental therapy or lack of lasting pain relief after local anesthetic block, or any combination of these.

Although myofascial pain appears to be distinct from fibromyalgia (a condition involving chronic widespread muscle pain), observation of patients in pain clinics over time has suggested that, in some cases, the localized pain of myofascial pain may develop into the more generalized pain seen in fibromyalgia. In addition, some scientific data support the idea that similar processes may be at work in the 2 conditions. Also, many of the treatments (exercise, stretching and antidepressants) are the same for both.

Principles of Management

The objectives of management for this condition are reducing pain and improving function. The choice of treatment is often empirical, based on the history and results of assessment in each individual case. Approaches to management may include the following:

- *Exercise:* Patients with myofascial pain often experience deconditioning through lack of use of the affected muscles. Exercise may therefore be an important component in the treatment of the condition.
- *Sleep:* Many patients with myofascial pain report poor sleep. Lack of restorative sleep usually leads to greater muscular aches and pains and changes in mood. Providing instructions for appropriate sleep hygiene can be extremely helpful.
- *Psychologic measures:* Methods such as relaxation techniques and coping skills are used to decrease activation of the central nervous system.
- *Pharmacologic management:* Nonsteroidal anti-inflammatory drugs (e.g., naprosyn), low-dose tricyclic antidepressants (e.g., nortriptyline) (i.e., at doses lower than those used to treat depression) and antispasmodic medications (e.g., tizanidine, baclofen) may be used. The efficacy of anticonvulsants in the treatment of chronic masticatory muscle pain and fibromyalgia has been demonstrated, but the appropriateness of using these medications to manage myofascial pain has not been established.
- *Dry needling:* The therapeutic effect of dry needling relies on mechanical disruption or direct stimulation of the trigger points.
- *Trigger-point injections:* Solutions such as 2% lidocaine or procaine may be injected at the site of the trigger point. Procaine is the least myotoxic of all injectable local anesthetics. Such injections are often combined with stretching or massage. Repeated injections into a particular muscle are not recommended if 2 or 3 previous attempts have been unsuccessful. Injectable steroids are usually not administered. In certain circumstances, injection of botulinum neurotoxin may produce more lasting relief, but should be considered only if there has been positive therapeutic response to local anesthetic injections beforehand. It should be noted that recent research has revealed that botulinum neurotoxin migrates centrally when it is peripherally administered and has been found to be no more effective than placebo (saline) injections in patients with chronic masticatory muscle pain.
- *Spray-and-stretch technique:* This technique combines application of a vapocoolant spray such as ethyl chloride with passive stretching of the muscle. The therapeutic goal is to reduce pain over the trigger points, restore the muscle to its normal length and improve the range of both active and passive motion.
- *Transcutaneous electrical nerve stimulation (also known as TENS):* This technique involves

the use of a device that provides electroanalgesia. The device consists of 1 or more electrical-signal generators, a battery and a set of electrodes. However, use of TENS for myofascial pain is controversial, with placebo-controlled studies failing to show statistically significant beneficial results.

- *Ischemic compression or release of trigger-point pressure:* This technique involves applying digital pressure to a trigger point to inactivate it. Despite little experimental evidence of efficacy, some patients report that acupuncture or acupressure of trigger points is helpful.
- *Mouth-opening exercises:* A comfortable range of mouth-opening exercises may be prescribed, along with deep massage of the masticatory and cervical muscles.
- *Patient education:* Patients can be given information about the causes of pain and its perpetuating factors, self-management of pain, behaviour modification and ways to avoid overloading the masticatory and cervical muscles.

Definitive diagnosis of the problem, followed by treatment appropriate to the diagnosis, are of paramount importance, especially for clinicians who dedicate part or all of their practice to the diagnosis and treatment of temporomandibular disorders and facial pain. Patients with facial pain of muscular origin presenting as pain within one or more teeth may undergo various dental procedures, including root canal therapy and extractions, before eventually presenting for treatment at a centre that specializes in temporomandibular joint disorders and other types of facial pain. Determining the *source* of the pain, as opposed to the *site* where the pain is perceived, must be appreciated and understood to avoid unnecessary treatment. The situation is analogous to patients with heart problems who never experience heart pain but do report arm, jaw or stomach pain. For further information on this fascinating type of muscle pain the reader is directed to the reference list.

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