

PRACTICE

Understanding the Patient with Epilepsy and Seizures in the Dental Practice

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ABSTRACT

Epilepsy, which is characterized by the risk of recurrent seizures, is a chronic disease that afflicts about 200,000 Canadians at any one time. Dentists with a thorough knowledge of seizure disorders and the medications used to treat them can provide necessary dental and oral health care to these patients. In this review, we summarize current knowledge of epilepsy, seizures and antiepileptic drugs and provide information on dental-related issues, as well as guidelines for the management of an acute seizure in the dental office.

MeSH Key Words: dental care for chronically ill; emergency treatment; epilepsy/physiopathology

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pilepsy is a chronic disease characterized by the risk of recurrent seizures; its prevalence in Canada is 5.6 per 1,000 people. In developing countries, this rate can be as high as 43 per 1,000 people. According to the World Health Organization, disability due to epilepsy accounts for about 1% of the global burden of disease, as measured by disability-adjusted life-years, ranking it just after some psychiatric problems such as alcohol dependence. The global burden of epilepsy is comparable to that of breast or lung cancer. Separate of the problems and the problems is comparable to that of breast or lung cancer.

Understanding epilepsy and seizures raises awareness of the disorder's impact on a patient's general medical and psychological health. Dental treatment of patients with epilepsy and seizures should be carried out by dentists who are knowledgeable about these disorders.

Seizures and Epilepsy

According to the International League Against Epilepsy, epilepsy is diagnosed when

a person has 2 or more unprovoked seizures.⁴ A seizure is classified as "partial" when the electrical discharge causing it occurs in a specific area of the brain or "generalized" when the discharge affects the entire brain cortex. When there is loss of awareness, seizures are termed complex (**Table 1**). The classification of epilepsy is similar. Epilepsy can be partial or generalized. Based on the cause, it can be symptomatic (caused by a developmental malformation), idiopathic (when a genetic condition is responsible) or cryptogenic (when the cause is unknown).

Diagnostic tools, such as electroencephalography (EEG) and magnetic resonance imaging (MRI), are required to classify epilepsy. EEG records waves generated by the brain cortex. These waves have characteristics that allow the differentiation of normal from abnormal electrical discharges and provide information about localization. EEG amplifies the waves and transfers them to a computer for interpretation.



Figure 1: Severe gingival hyperplasia due to chronic use of phenytoin. Photo courtesy of Dr. Thomas W. Mara.

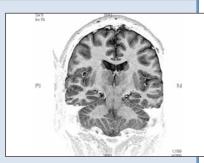


Figure 2: MRI of the brain reveals inversion recovery sequence showing atrophy of the right hippocampus (mesial temporal region).

Table 1 Simplified version of the classification of seizures according to the International League Against Epilepsy⁴

Partial seizures

Simple partial seizures (awareness not impaired)

- with minor signs (focal motor, versive, phonatory)
- with somatosensory or special-sensory symptoms (somatosensory, visual, auditory, olfactory, gustatory)
- with autonomic symptoms
- with psychic symptoms (déjà vu, illusions, hallucinations)

Complex partial seizures

- with simple partial onset followed by impairment of awareness
- · with impairment of awareness at onset

Partial seizures evolving to secondarily generalized seizures

- simple partial seizures evolving to generalized seizures
- complex partial seizures evolving to generalized seizures
- simple partial seizures evolving to complex partial and then to generalized seizures

Generalized seizures

Absence seizures

Myoclonic seizures

Clonic seizures

Tonic seizures

Tonic-clonic seizures

Atonic seizures

Unclassified seizures

Differential Diagnosis

Syncope, which is characterized by loss of consciousness without any premonitory signs and symptoms, can mimic a seizure. Other conditions included in the dif-

ferential diagnosis are migraine headaches, strokes or transient ischemic attacks and nonepileptic psychogenic events (or pseudoseizures), seen in association with such psychiatric conditions as conversion disorder, anxiety and depression.

Treatment of Epilepsy

Medical Treatment

Approximately 15 medications are available for the treatment of epilepsy in Canada (**Table 2**). The most common oral side effect of antiepileptic drugs seen in the dental office is gingival hyperplasia (**Fig. 1**). Although

gingival hyperplasia is seen almost exclusively with the use of phenytoin,^{5,6} some reports have also associated it with the use of carbamazepine. Gingival hyperplasia is characterized by unusual growth of the gingival subepithelial connective tissue and epithelium, for unknown reasons; it is reversed once the drug is discontinued.

Antiepileptic drugs can achieve seizure control in approximately 50% of patients; the rate can increase to 60% after 2 trials of medications (either a combination of 2 drugs or 2 monotherapies). After a third or fourth trial, seizure control is achieved in only an additional 5% of patients. Once therapy with medications has been determined to be hopeless, the patient is referred to an epilepsy program.

Presurgical Evaluation

In an epilepsy program, the objective is to find out whether the patient is a surgical candidate by using special tests, such as prolonged monitoring videoelectroencephalography (VEEG) and structural MRI. VEEG allows confirmation of epilepsy syndrome and location of the epileptogenic focus. The behavioural changes captured by video and the focal epileptiform abnormalities in the EEG are the most important pieces of information in the presurgical evaluation. MRI identifies abnormalities of possible epileptogenicity, such as tumours and arteriovenous malformations. Hippocampal or mesial temporal sclerosis is the most common epileptogenic brain abnormality in adults with medically refractory epilepsy (Fig. 2). In this condition, "scarring" of the mesial structures of the temporal lobe occurs by an unknown mechanism, apparently related to febrile seizures during childhood. In these cases, seizures are successfully controlled with surgical removal of the seizure generator.

When VEEG and MRI are inconclusive, other neuroimaging techniques are necessary. These include single photon emission computed tomography, which measures perfusion of the brain; positron emission tomography,

Table 2 Medications currently available for the treatment of epilepsy in Canada

Medication	Indications (seizure type)	Most common oral side effects and dental considerations
Phenobarbital	Partial and secondarily generalized	Drowsiness/sedation, osteopenia/osteomalacia
Carbamazepine	Partial and secondarily generalized	Xerostomia, stomatitis, gingival bleeding, rash, osteopenia/osteomalacia
Phenytoin	Partial and secondarily generalized	Gingival hyperplasia, gingival bleeding, osteopenia/osteomalacia
Valproate or valproic acid	Partial and generalized	Gingival bleeding, petechiae, decreased platelet aggregation
Primidone	Partial and generalized	Drowsiness/sedation
Lamotrigine	Partial and generalized	Rash
Topiramate	Partial and generalized	Mild cognitive side effects
Clobazam	Partial and generalized	Drowsiness/sedation
Oxcarbazepine	Partial and secondarily generalized	Unknown
Ethosuximide	Generalized	Drowsiness/sedation
Vigabatrin	Partial	Unknown
Lorazepam	Generalized	Drowsiness/sedation
Diazepam	Generalized	Drowsiness/sedation
Gabapentin	Partial	Drowsiness/sedation
Levetiracetam	Partial and generalized	Unknown

which measures the metabolism of certain substances, such as glucose or flumazenil; MRI spectroscopy, which measures the metabolic profile of certain brain substances; magnetoencephalography, which evaluates the location of magnetic fields generated by epileptiform discharges, coregistered in an MRI; and functional MRI, which allows localization of vital areas of the cerebral cortex, such as language, motor, visual and sensory areas.

Subdural or depth placement of electrodes through craniotomies are indicated when the above-mentioned tools are not successful in localizing the epileptogenic area.⁸

Surgical Treatment

Resections

Temporal lobectomy is perhaps the most common type of surgery for epilepsy. In the only randomized controlled trial of surgery versus medical treatment, the success rate was 64%.9 However, patients can experience a significant decline in verbal memory,10 which can be partly predicted through a detailed neuropsychologic evaluation.11

Disconnection Procedures

Multiple subpial transections consist of small incisions perpendicular to the surface of the brain cortex to disrupt the horizontal propagation of seizures.¹²

Section of the corpus callosum inhibits transmission between the hemispheres. This procedure is indicated in patients with drop attacks (bilateral loss of motor tone and posture control).¹³

Treating Dental Patients with Epilepsy General Situation

The medical literature contains little information on the influence of epilepsy in dental care. Most existing studies focus on phenytoin-induced gingival hyperplasia.

Patients living with epilepsy have special needs during dental treatment. In almost all aspects of oral health and dental status, the condition of patients with epilepsy is significantly worse than age-matched groups in the general (nonepileptic) population. Furthermore, patients who have poorly controlled epilepsy and experience frequent generalized tonic–clonic seizures exhibit worse oral health in comparison with patients who are better controlled or only have seizures that do not involve the masticatory apparatus.

The number of decayed and missing teeth, the degree of abrasion and periodontal indexes are significantly worse in patients with epilepsy. Those with epilepsy also have significantly fewer restored and replaced teeth than the general population. ^{14,15}

The fact that dental care is only partly reimbursed, if at all, in Canada and worldwide may partly explain the problem. The negative attitude of dentists toward patients with epilepsy can also be a barrier to obtaining

appropriate dental treatment. It has been suggested that dentists are likely to consider the treatment of patients with epilepsy as cumbersome and tend to offer treatment options that are quick and simple.¹⁴

Problems that a Dentist May Encounter Trauma

Generalized tonic–clonic seizures often cause minor oral injuries, such as tongue biting,¹⁶ but also frequently lead to tooth injuries¹⁷ and in some cases to maxillofacial trauma.¹⁸

Patients with epilepsy can be at increased risk of fracture because enzyme-inducing antiepileptic drugs (e.g., phenytoin, phenobarbital, carbamazepine) alter the metabolism and clearance of vitamin D and have been associated with osteopenia and osteomalacia.¹⁹ Of interest, increased fracture risk has also been associated with the use of benzodiazepines, antidepressants and antipsychotics, suggesting that underlying brain disease or adverse effects of the medication are responsible for falls and injuries.¹⁹

Fractures can have catastrophic effects on the lives of patients with epilepsy, and measures are available to minimize the risk of fractures, such as ensuring adequate calcium and vitamin D supplementation (a minimum of 1,000 mg and 400 IU daily, respectively) especially in patients taking phenobarbital, phenytoin or primidone.²⁰

Periodontal Problems

Gingival overgrowth as a complication of phenytoin use has been well studied. 21,22 About 50% of patients taking this medication will develop gingival hyperplasia within 12–24 months of initiation of treatment. Despite the existence of newer medications that are equally effective and have fewer side effects, phenytoin remains one of the most commonly used drugs. Evidence regarding best treatment for gingival hyperplasia is lacking. Some clinicians advocate the use of chlorhexidine, folic acid rinses or both, but excellent oral hygiene will probably prevent or significantly decrease the severity of the condition. In severe cases, surgical reduction is needed. 23

The newer antiepileptic drugs produce oral manifestations only infrequently. Xerostomia and stomatitis have been reported rarely as side effects of carbamazepine,²⁴ and rash that may involve the oral cavity has been associated with lamotrigine and can be exacerbated by the concomitant use of valproic acid.²⁵

Valproic acid can cause direct bone marrow suppression, which can impair wound healing and increase post-operative bleeding and infections. Decreased platelet count is the most common and best-recognized hematologic effect of valproic acid; the incidence varies from 5% to 40%. Clinically significant bleeding is uncommon because the thrombocytopenia is usually not severe. For elective surgery, laboratory evaluation — including bleeding time, fibrinogen level, prothrombin time, par-

tial thromboplastin time and von Willebrand factor level — is needed to assess the risk of peri- and postoperative bleeding. Bleeding as a potential side effect should be discussed with patients and their families in preparation for surgery.²⁶

Prosthodontic Problems

In a recent analysis of the prosthodontic status of patients with epilepsy,¹⁵ it was found that compared with age-matched controls, patients with epilepsy have a tendency to become edentulous earlier. It was also found that prosthodontic treatment is suboptimal, as significantly fewer teeth are replaced, despite the fact that epileptic patients tend to have more missing teeth. Based on these findings, the authors suggested a classification for patients with epilepsy according to dental risk factors and dental manageability and provided recommendations for dental treatment.

Specific guidelines were also provided, such as discouragement of incisal restorations, use of fixed rather than removable prostheses and inclusion of additional abutments if fixed partial dentures are to be used.¹⁵ In addition, the use of metal base for complete dentures and telescopic retention with denture bases made of metal or reinforced with metal for nearly edentulous patients was recommended for those with frequent partial seizures involving the masticatory apparatus, frequent generalized tonic–clonic seizures and other seizures associated with falls.

Dermatologic Problems

Rash is a common side effect of antiepileptic drugs. Although most drug-associated rash is benign, serious rashes, including Stevens–Johnson syndrome and toxic epidermal necrolysis do occur.²⁷ Between 5% and 7% of patients taking phenytoin and 5% to 17% of patients taking carbamazepine experience rash. The patients at highest risk for lamotrigine rash are children with a history of rash from another antiepileptic drug; 18.2% experience lamotrigine rash.²⁸

Drug Interactions

A number of drugs prescribed by dentists can jeopardize seizure control because they interact with antiepileptic drugs. For instance, metronidazole, antifungal agents (such as fluconazole) and antibiotics (such as erythromycin) may interfere with the metabolism of certain antiepileptic drugs.²⁹

The coadministration of fluconazole and phenytoin is associated with a clinically significant increase in phenytoin plasma concentration, and the dose of the latter may require adjustment to maintain safe therapeutic concentrations. Other anticonvulsants, such as vigabatrin, lamotrigine, levetiracetam, oxcarbazepine and gabapentin, are unlikely to interact with fluconazole.

Clarithromycin increases the plasma concentration of carbamazepine, and coadministration of these drugs should be monitored very carefully to avoid carbamazepine toxicity.³⁰

Valproic acid may be displaced from plasma proteins and metabolic pathways may be inhibited by high doses of aspirin; this interaction will free serum valproate concentrations resulting in subsequent toxicity.³¹

Seizure First Aid in the Dental Office

If a seizure occurs while a patient is in the dental chair

- 1. Clear all instruments away from the patient.
- 2. Place the dental chair in a supported, supine position as near to the floor as possible.
- 3. Place the patient on his or her side (to decrease the chance of aspiration of secretions or dental materials in the patient's mouth).
- 4. Do not restrain the patient.
- 5. Do not put your fingers in his or her mouth (you might be bitten).
- 6. Time the seizure (the duration of the event may seem longer than it actually is).
- 7. Call 911 if the seizure lasts longer than 3 minutes.
- 8. Call 911 if the patient becomes cyanotic from the onset.
- 9. Administer oxygen at a rate of 6–8 L/minute.
- 10. If the seizure lasts longer than 1 minute or for repeated seizures, administer a 10-mg dose of diazepam intramuscularly (IM) or intravenously (IV), or 2 mg of ativan, IV or IM, or 5 mg of midazolam, IM or IV.^{32,33}
- 11. Be aware of the possibility of compromised airway or uncontrollable seizure.

Once the seizure is over

- 1. Do not undertake further dental treatment that day.
- 2. Try to talk to the patient to evaluate the level of consciousness during the post-ictal phase.
- 3. Do not attempt to restrain the patient, as he or she might be confused.
- 4. Do not allow the patient to leave the office if his or her level of awareness is not fully restored.
- 5. Contact the patient's family, if he or she is alone.
- 6. Do a brief oral examination for sustained injuries.
- Depending on post-ictal state, discharge the patient home with a responsible person, to his or her family physician or to an emergency room for further assessment.

Conclusions

Advances in diagnostic technology, pharmacotherapy and understanding of neurologic processes allow dentists to understand and manage patients with epilepsy better. People with epilepsy can be safely treated in a general dental practice. A thorough medical history should be taken and updated at each visit. Seizure history must be taken into account when planning treatment. Dentists with a comprehension of seizure disorders can provide an invaluable service to their patients, providing not only oral health, but also maintaining and promoting the systemic health of these patients. *

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References

- 1. Tellez-Zenteno JF, Pondal-Sordo M, Matijevic S, Wiebe S. National and regional prevalence of self-reported epilepsy in Canada. *Epilepsia* 2004; 45(12):1623–9.
- 2. Burneo JG, Tellez-Zenteno J, Wiebe S. Understanding the burden of epilepsy in Latin America: a systematic review of its prevalence and incidence. *Epilepsy Res* 2005; 66(1):63–74.
- 3. Burneo JG, McLachlan RS. When should surgery be considered for the treatment of epilepsy? *CMAJ* 2005; 172(9):1175–7.
- 4. Proposal for revised classification of epilepsies and epileptic syndromes. Commission on Classification and Terminology of the International League Against Epilepsy. *Epilepsia* 1989; 30(4):389–99.
- 5. Scheinfeld N. Phenytoin in cutaneous medicine: its uses, mechanisms and side effects. *Dermatol Online J* 2003; 9(3):6.
- 6. Asconape JJ. Some common issues in the use of antiepileptic drugs. Semin Neurol 2002; 22(1):27–39.
- 7. Kwan P, Brodie MJ. Early identification of refractory epilepsy. N Engl J Med 2000; 342(5):314-9.
- 8. Noachtar S. Subdural electrodes in focal cortical dysplasia. *Epileptic Disord* 2003; 5(Suppl 2):S91–4.
- 9. Wiebe S, Blume WT, Girvin JP, Eliaziw M; Effectiveness and Efficiency of Surgery for Temporal Lobe Epilepsy Study Group. A randomized, controlled trial of surgery for temporal-lobe epilepsy. *N Engl J Med* 2001; 345(5):311–8.
- 10. McKhann GM 2nd, Bourgeois BF, Goodman RR. Epilepsy surgery: indications, approaches, and results. *Semin Neurol* 2002; 22(3):269–78.
- 11. Loring DW. Neuropsychological evaluation in epilepsy surgery. *Epilepsia* 1997; 38(Suppl 4):S18–23.
- 12. Morrell F, Whisler WW, Bleck TP. Multiple subpial transection: a new approach to the surgical treatment of focal epilepsy. *J Neurosurg* 1989; 70(2):231–9.
- 13. Devlin AM, Cross JH, Harkness W, Chong WK, Harding B, Vargha-Khadem F, and other. Clinical outcomes of hemispherectomy for epilepsy in childhood and adolescence. *Brain* 2003; 126(Pt 3):556–66.

- 14. Karolyhazy K, Kovacs E, Kivovics P, Fejerdy P, Aranyi Z. Dental status and oral health of patients with epilepsy: an epidemiologic study. *Epilepsia* 2003; 44(8):1103–8.
- 15. Karolyhazy K, Kivovics P, Fejerdy P, Aranyi Z. Prosthodontic status and recommended care of patients with epilepsy. *J Prosthet Dent* 2005; 93(2):177–82.
- 16. Pick L, Bauer J. [Dentistry and epilepsy]. *Nervenarzt* 2001; 72(12):946–9. [Article in German]
- 17. Buck D, Baker GA, Jacoby A, Smith D, Chadwick DW. Patients' experiences of injury as a result of epilepsy. *Epilepsia* 1997; 38(4):439–44.
- 18. Aragon CE, Burneo JG, Helman J. Occult maxillofacial trauma in epilepsy. *J Contemp Dent Pract* 2001; 2(4):26–32.
- 19. Mattson RH, Gidal BE. Fractures, epilepsy, and antiepileptic drugs. *Epilepsy Behav* 2004; 5(Suppl 2):S36–40.
- 20. Sato Y, Kondo I, Ishida S, Motooka H, Takayama K, Tomita Y, and others. Decreased bone mass and increased bone turnover with valproate therapy in adults with epilepsy. *Neurology* 2001; 57(3):445–9.
- 21. Angelopoulos AP. Diphenylhydantoin gingival hyperplasia. A clinicopathological review. 1. Incidence, clinical features and histopathology. *Dent J* 1975; 41(2):103–6.
- 22. Angelopoulos AP. A clinicopathological review. Diphenylhydantoin gingival hyperplasia: 2. Aetiology, pathogenesis, differential diagnosis and treatment. *Dent J* 1975; 41(5):275–7, 283.
- 23. Stoopler ET, Sollecito TP, Greenberg MS. Seizure disorders: update of medical and dental considerations. *Gen Dent* 2003; 51(4):361–6.
- 24. Ogunbodede EO, Adamolekun B, Akintomide AO. Oral health and dental treatment needs in Nigerian patients with epilepsy. *Epilepsia* 1998; 39(6):590–4.
- 25. Li LM, Russo M, O'Donoghu MF, Duncan JS, Sander JW. Allergic skin rash with lamotrigine and concomitant valproate therapy: evidence for an increased risk. *Arq Neuropsiquiatr* 1996; 54(1):47–9.
- 26. Archarya S, Bussel JB. Hematologic toxicity of sodium valproate. *Pediatr Hemat Oncol* 2000; 22(1):62–5.
- 27. Mockenhaupt M, Messenheimer J, Tennisj P, Schlingmann J. Risk of Stevens-Johnson syndrome and toxic epidermal necrolysis in new users of antiepileptics. *Neurology* 2005; 64(7):1134–8.
- 28. Hirsch LJ, Weintraub DB, Buchsbaum R, Spencer HT, Straka T, Hager M, and other. Predictors of lamotrigine-associated rash. *Epilepsia* 2006; 47(2):318–22.
- 29. Goulden KJ, Dooley JM, Camfield PR, Fraser AD. Clinical valproate toxicity induced by acetylsalicylic acid. *Neurology* 1987; 37(8):1392–4.
- 30. Patsalos PN, Frosher W, Pisani F, Van Rijn CM. The importance of drug interactions in epilepsy therapy. *Epilepsia* 2002; 43(4):365–85.
- 31. Miners JO. Drug interactions involving aspirin (acetylsalicylic acid) and salicylic acid. *Clin Pharmacokinet* 1989; 17(5):327–44.
- 32. Scott RA, Besag FM, Neville BG. Buccal midazolam and rectal diazepam for treatment of prolonged seizures in childhood and adolescence: a randomised trial. *Lancet* 1999; 353(9153):623–6.
- 33. Scott RC. Buccal midazolam as rescue therapy for seizures. *Lancet Neurol* 2005; 4(10):592–3.