Epilepsy and Oral Care

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Abstract: **Epilepsy is a common symptom of an underlying neurological disorder. The seizures can take a variety of forms. Both the condition and its medical management can affect oral health. Prevention of oral disease and carefully planned dental treatment are essential to the well-being of people with epilepsy.**

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Clinical Relevance: **Epilepsy and its drug management can impinge on oral health and the delivery of dental treatment.**

Epilepsy is a symptom of an underlying neurological disorder rather than a condition in its own right. The diverse nature of seizures has led to people being thought of as having 'one of the epilepsies' rather than epilepsy *per se*. An epileptic seizure is defined as:

'a transient paroxysm of excessive discharges of neurones in the cerebral cortex causing a stereotyped event, in which an individual's awareness of the surroundings may be impaired and their behaviour altered, that is discernible to the person experiencing it or to an observer'.[1]

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The clinical manifestations of a seizure reflect the function of the area of the cortex in which it arises.[1] Epileptic activity confined to one area is associated with specific symptoms and signs. Generally, a seizure has a sudden onset and a specific end-point. Seizures are usually brief, lasting from a few seconds to a few minutes, and are frequently followed by a post-ictal period of drowsiness and confusion. Epilepsy is usually considered to be active if a person has had a seizure within the last 2 years or is taking anti-epileptic medication.

AETIOLOGY

Brain damage due to injury, infection, birth trauma or a cerebrovascular accident accounts for 25% of cases (Table 1). Idiopathic epilepsy, in which there is no identifiable cause, accounts for the other 75%.

The aetiology is related to a person's genetically determined seizure threshold and its dynamic interaction with seizure precipitants or triggers such as:

- fever;
- infection;
- metabolic disturbance;
- hormonal disturbance;
- photosensitivity;
- sleep deprivation;
- hunger;
- stress.

An individual with a low seizure threshold is more likely to develop epilepsy than a person with a high seizure threshold when they are both exposed to the same seizure precipitant.[2]

Idiopathic epilepsy is the most common of the genetically determined epilepsies. If one parent has epilepsy, a child has a 4% chance of developing it. If both parents have the condition, the risk is increased to between 10 and 14% (compared with the general chance of

Age group aetiology

Young infants
- Hypoxia or birth asphyxia
- Perinatal intracranial trauma
- Metabolic disturbances
- Congenital brain malformations
- Infection

Children and adolescents
- Idiopathic
- Trauma
- Infection

Adults
- Idiopathic
- Head injury
- Alcohol abuse
- Brain tumours
- Cerebrovascular disease

***Table 1.** Common causes of epilepsy.*

Partial seizures (seizures beginning locally)
- A Simple partial seizures (consciousness not impaired)
- B Complex partial seizures (with impairment of consciousness)
  - 1. Beginning as simple partial seizures and progressing to impairment of consciousness
  - 2 With impairment of consciousness at the onset
- C Partial seizures secondarily generalized

Generalized seizures (bilaterally symmetrical and without focal onset)
- A Absence seizures
- B Myoclonic seizures
- C Clonic seizures
- D Tonic seizures
- E Tonic-clonic seizures
- F Atonic

Unclassified epileptic seizures
(inadequate or incomplete data)

Table 2. *International classification of epileptic seizures.*[12]

0.5%[2]). There are a number of inherited conditions which present as neurological or systemic illnesses (e.g. neurofibromatosis,[3] tuberous sclerosis[4]), and in some inborn errors of metabolism (e.g. phenylketonuria, porphyria[5]) epileptic seizures are a feature. Epilepsy may develop in some genetic syndromes (2–10% in Down's syndrome,[6] 15% in Fragile X syndrome[4] and up to 90% in Sturge–Weber syndrome[4]), and people with a learning disability also seem to be at higher risk.[4]

EPIDEMIOLOGY

The lifetime risk of developing epilepsy is 2–5%. The prevalence of active epilepsy between 4 and 10 people per 1000 of the population and the condition is more common in men than women. The incidence of epilepsy is relatively high in the first two decades of life, falling off in the 20s and increasing again, mainly as a result of cerebrovascular disease, after the age of 50 years.[7]

STIGMA

In ancient Greece, epilepsy was considered as 'a miraculous phenomenon', as 'only the gods could knock someone down, strip their reason, make their body thrash around uncontrollably, and afterwards bring them around without apparent ill-effect.'[1] Today, the stigma of epilepsy is reinforced by fear, superstition and misconception and, while people usually come to terms with their own attacks, they cannot always be sure of other people's reactions.[8,9] Although the stigma of epilepsy is often perceived rather than actual,[10] it can become a profound and lasting source of unease, self-doubt, disruption and prejudice in people's lives.[10,11] Consequently, people are not always prepared to disclose the fact that they have epilepsy, especially if they do not think that the information is relevant to the situation.[8]

DIAGNOSIS

Epilepsy is diagnosed clinically and relies on description of the seizure by the affected individual or, where loss of consciousness occurs, by an observer. A number of conditions involve alteration of consciousness or neurological symptoms that can be confused with epileptic seizures:

- syncope;
- hypoglycaemia;
- transient ischaemic attack;
- raised intracranial pressure;
- transient global amnesia;
- migraine;
- pseudoseizures;
- panic attacks;
- episodic dyscontrol (rage attacks);
- sleep disorders (e.g. narcolepsy).

The differential diagnosis is thus important.

Tests that may be carried out to determine the aetiology of epileptic seizures include magnetic resonance imaging scans, electroencephalography, cerebrospinal fluid examination, serum biochemistry and blood sugar assay.

CLASSIFICATION AND CHARACTERISTICS OF SEIZURES

The International Seizure Classification of the International League Against Epilepsy (Table 2)[12] is based on the clinical and electroencephalographic manifestations of seizures. Essentially it divides seizures into two main groups:

- partial seizures;
- generalized seizures.

Partial Seizures

Partial seizures originate from localized cortical areas and are subdivided into simple partial, complex partial and secondarily generalized seizures.

Simple Partial Seizures

Such seizures are characterized by abrupt onset, short duration and no loss of consciousness or amnesia. They are more common in later life and are invariably indicative of a structural intracerebral lesion. The symptoms depend on the area of the cortex involved – e.g. jerking or spasm of limbs in frontal lobe involvement and sensory symptoms if the parietal lobe is affected.

Complex Partial Seizures

These are the most common type of seizure. They are characterized by three components:

- an 'aura' or warning;
- an 'absence' or loss of consciousness; and
- 'automatism' or automatic behaviour. This can be oro-alimentary (lip smacking, chewing) mimetic (facial grimacing) gestural (scratching, rearranging objects) ambulatory (running, walking) or verbal.

Generalized Seizures

Generalized seizures are characterized by the simultaneous involvement of the whole cortex at the onset of the seizure. Consciousness is lost at the beginning of the event so there is no aura or warning. Such seizures include tonic-clonic, absence, myoclonic, tonic and atonic seizures.

Tonic-clonic seizures. Formerly known as 'grand-mal' fits, these may have a

Drug	Resumé
Carbamazepine	First-line: for partial or generalized tonic-clonic seizures. Tolerability generally good in children and younger adults, relatively less good in elderly people. Allergic reactions (rash) fairly common. Enzyme-inducing drug
Valproate	First-line: for generalized (both tonic-clonic and absence) and partial seizures. Weight gain often a problem. Allergic reactions uncommon. Not an enzyme-inducing drug
Lamotrigine	Recently promoted (monotherapy licence), wide spectrum of activity; may join first line. Allergic reactions (rash) fairly common and occasionally severe
Phenytoin	Formerly in first-line, now used less because of side effects. Spectrum of activity similar to that of carbamazepine. Narrow therapeutic window plus complex pharmacokinetics demand monitoring of drug concentration
Vigabatrin	Recent warning about visual field defects makes specialist review desirable (patients may require visual field testing)
Gabapentin	Not very effective as additional treatment in severe epilepsy, but may have a future as monotherapy
Clobazam	Has valuable special uses, particularly when seizures occur in clusters
Topiramate	For treating severe epilepsy
Tiagabine	Just launched in the UK. Its place in clinical practice is still to be established
Phenobarbitone	Formerly used widely in cases of refractory epilepsy; may still have a role when other treatments fail. Specialist opinion on withdrawal of drug is advisable
Primidone	Formerly used widely in cases of refractory epilepsy
Clonazepam	Formerly used widely in cases of refractory epilepsy; may still have a role when other treatments fail. Specialist opinion on withdrawal of drug is advisable
Ethosuximide	Alternative to valproate for petit-mal seizures only

Table 3. *Overview of current drug treatments for epilepsy (adapted from Feely[13]).*

prodromal phase of general malaise lasting several hours. At the onset the person becomes stiff (tonic phase); apnoea occurs and cyanosis may follow, the heart rate and blood pressure increase and the person collapses. The next phase consists of intermittent jerking movements (clonic phase), often involving all four limbs, followed by brief periods of muscle relaxation. The relaxation phases gradually increase in length and the clonic movements stop as the seizure ends. Recovery is slow, with a post-ictal period of drowsiness, confusion or sleep.

Absence seizures. Formerly known as 'petit-mal' fits, absence seizures occur most commonly in children and adolescents. The individual suddenly stops an activity and stares blankly. Consciousness is lost but the attacks, although frequent, are so short (only a few seconds) that they often pass unrecognized. Recovery is rapid and there is no post-ictal confusion.

MANAGEMENT

The management of epilepsy is primarily medical and a wide range of drugs is used for its control (Table 3).[13] Individual drug regimens can often only be established by trial and error. The usual practice is to start with a low dose of a single, first-line drug and gradually increase the dose until seizure control is achieved. If this is unsuccessful, a second first-line drug is introduced while the original drug is withdrawn gradually to prevent rebound seizures.[14] If the seizures continue, then either two first-line drugs are used together or a second-line drug is tried. An individual's drug regimen may be changed during establishment of seizure control and whenever the control becomes less stable.

Phenytoin, once the first choice in managing epilepsy in younger people, has fallen from favour because of its many side-effects, which include nausea, mental confusion, coarse facies, acne, hirsutism, erythema multiforme, hepatitis and gingival overgrowth. Also, phenytoin levels have to be monitored regularly as small changes in dosage can result in marked changes in plasma concentrations.[14] However, dental practitioners will encounter some patients who are still taking this drug.

Where there is a structural cause or if a specific site of the cerebral cortex can be identified as initiating seizures that cannot be controlled by drugs, surgery may be necessary. Some individuals may have a vagal nerve stimulator implanted to reduce the severity of their seizures.[15] However, such devices are unlikely to have any implications for dental care.

PROGNOSIS

Between 70% and 80% of people with one of the epilepsies will eventually become seizure free and approximately 50% will successfully withdraw their medication. However, 20–30% of people with epilepsy will go on to develop chronic epilepsy. The risk of recurrence depends on the cause of the seizure (a neurological deficit carries the highest risk of recurrence) and the length of time the person remains seizure free (the longer they are seizure free the less likely they are to have a recurrence).[1]

Epilepsy is associated with a mortality rate of 2–3 times that of the general population. This increase is mainly associated with the cause of the epilepsy (head injury, brain tumour, etc.); however, it is also linked to the type of seizure. People with myoclonic seizures have a mortality rate four times that of the general population whilst people with absence seizures are not at any increased risk.

Causes of death in epilepsy include:

- Aetiology
- Type of seizure
- What might trigger a seizure?
- Are any of the trigger factors associated with dental care e.g. stress, lights, infections?
- Is a preceding aura experienced?
- Does an aura always progress to a seizure?
- What to expect when the patient has a seizure
- Date of the last seizure
- Frequency of seizures
- Duration of seizures
- Any episodes of status epilepticus

Table 4. *Seizure history.*

- Status epilepticus – seizures occurring in quick succession without any period of recovery between one attack and the next.
- Sudden unexpected death – the cause is unknown but may include suffocation during seizure, deleterious action of anti-epileptic drugs, autonomic seizures affecting the heart and the release of endogenous opioids. It is responsible for an annual mortality rate from 1 in 200 to 1 in 1200 people with epilepsy.[16]
- Accidents during seizures.
- Bronchopneumonia – related to aspiration during a seizure.

CONSIDERATIONS FOR ORAL CARE

Most people who have seizure disorders are able to undergo routine dental care in the primary care setting of general dental practice.[7] Even so, the practitioner should check that the patient has taken his/her normal anti-epilepsy medication, is well, is not excessively tired and has eaten normally before embarking on any treatment. It is essential to take a thorough medical and seizure history (including the details shown in Table 4), as information about the seizures and their control helps in planning the appropriate dental treatment and place of care as well as helping to avoid seizure triggers. The drug history should be checked at each visit as frequent changes are indicative of poor control. In this situation it would be prudent to delay non-urgent dental care until seizure control has improved.

Oral Implications of Drug Therapy

A number of the drugs used in seizure control have implications for oral care or dental treatment.

Gingival overgrowth associated with phenytoin is the most widely known complication of anti-epileptic medication (Figure 1), with 50% of individuals being affected within 3 months of starting the drug.[17] Gingival overgrowth is greater in individuals with high plaque levels, although there is a genetic link that explains why some people, even with very poor oral hygiene, do not develop it.[18] The hyperplasia resolves spontaneously within 1–6 months of phenytoin withdrawal.[7]

Carbamazepine can also cause oral complications including ulceration, xerostomia, glossitis and stomatitis. Sodium valproate can decrease the platelet count and function resulting in clotting problems, albeit rarely.[3,14] This is probably insignificant for minor dental surgery such as straightforward extractions but a clotting screen would be advisable before performing major surgery on patients taking sodium valproate.

Any drug prescribed long-term as a syrup-based medicine has the potential to cause dental caries. Children, adults with a learning disability or elderly people are most likely to be prescribed this form of medication. Appropriate preventive measures (such as rigorous oral hygiene, use of fluoride and chlorhexidine varnishes, gels or pastes, regular dental reviews) should be enforced. Alternatively, the medical practitioner could be asked to prescribe a sugar-free medicine.

A number of drugs prescribed by dentists can jeopardize seizure control because they interact with anti-epilepsy agents. For instance, metronidazole, antifungal agents such as fluconazole and miconazole, aspirin and non-steroidal anti-inflammatory drugs inhibit the metabolism of phenytoin and increase its plasma concentration;[14]

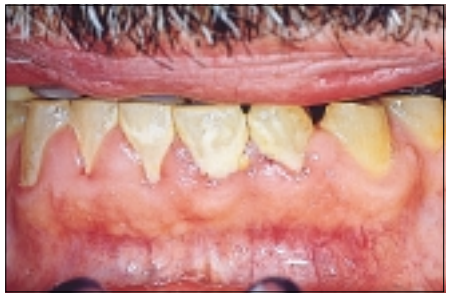

Figure 1. *Gingival overgrowth associated with phenytoin.*

erythromycin inhibits the metabolism of carbamazepine and sodium valproate. These drug combinations should be avoided.

Trauma

Falls associated with seizures can result in laceration of soft tissues, fractures of the facial skeleton, subluxation of the temporomandibular joints and devitalization, fractures, subluxation or avulsion of teeth.[3,19] Damaged teeth should be restored in the normal way; however, it has been suggested that it is not advisable to re-implant an avulsed tooth.[19] If an avulsed tooth cannot be found, a chest radiograph may be required to discover if the patient inhaled it during the seizure.[3]

Prevention of Dental Disease

Regular examination with careful treatment planning helps to avoid dental pain and oral infections that can adversely affect control of seizures.[20] A well maintained and regularly reinforced programme to prevent oral disease is essential.

People taking phenytoin need excellent plaque control to prevent gingival hyperplasia which, in extreme cases, can lead to delayed eruption and misalignment of teeth, compromised appearance and halitosis.[7] Gingivectomy is recommended to remove any hyperplastic tissue that interferes with appearance or function;[7] however, unless oral hygiene is exemplary, the hyperplasia will recur.[7] For patients with recurrent hyperplasia it is reasonable to request that the

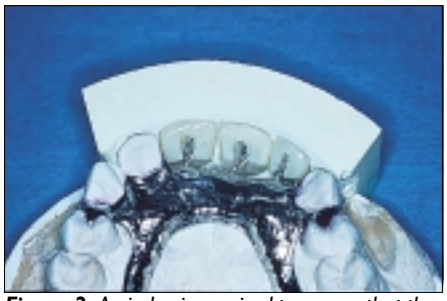

Figure 2. *An index is required to ensure that the denture teeth strengthened by metal posts are placed in the correct position.*

medical practitioner prescribes an alternative to phenytoin, although he or she may be reluctant to do so if the seizures are well controlled.

Restorative Dentistry

As a general rule, fixed prostheses are preferable in patients with epilepsy, as removable appliances are more likely to be dislodged during a seizure.[7] The individual's seizure history and dental history should be taken into account when providing any restorative treatment.

Although there is concern about inhalation of teeth and teeth fragments, few such events have been reported in the literature; nevertheless, it is prudent to use metal crowns and bridges where possible to limit the risk of fracturing amalgam and porcelain restorations.[7,21] The use of acrylic facings should be considered as acrylic is easier to repair than porcelain.[21] Modern composite materials and bonding agents offer a durable and relatively non-invasive form of treatment for fractured anterior teeth.

It is sensible to use a firm mouth prop throughout all restorative treatment, so that the mouth can be safely cleared of dental instruments if a patient has a seizure in the chair.

If an individual with restored anterior teeth (or with a Class II division 1 incisal relationship) experiences an aura or a recognizable prodromal phase before a seizure, he or she should carry a soft mouth guard to use at such times. The mouth guard should have palatal coverage and extend into the buccal sulci for purposes of retention.

Local Anaesthesia

There has been some confusion over the safety of the use of local anaesthetic agents in people with epilepsy. Although large doses of lignocaine given intravenously during the treatment of cardiac dysrhythmias can cause convulsions, there is no evidence that the small amount in dental local anaesthetic cartridges has a similar effect.[22] The mechanism of the fits caused by lignocaine overdose is quite different from the mechanism of seizures in the epilepsies,[22] and it is quite safe to use lignocaine with adrenaline as the local anaesthetic agent of choice for dental treatment.

Conscious Sedation

Inhalational sedation with nitrous oxide or intravenous sedation with a benzodiazepine are useful for reducing the risk of a stress-induced fit in patients who are anxious about dental care. It is better to delay a procedure if the patient is in a poorly controlled phase as, despite the known anti-epileptic effects of the benzodiazepines, seizures have been reported in patients undergoing intravenous sedation with midazolam.[23]

Care must be taken when sedating patients whose epilepsy is controlled by benzodiazepines (including clobazam and clonazepam) as they may have a high tolerance to midazolam or could experience an additive effect, leading to over-sedation.[24] The use of flumazenil, which reverses the effect of the benzodiazepines, is contraindicated because of the risk of reversing the anti-epileptic effects of the benzodiazepines taken on a regular basis.

General Anaesthesia

There are no particular problems associated with general anaesthesia, although it is important to maintain anticonvulsant therapy throughout the perioperative period. People with poorly controlled seizures or with additional medical problems are better treated on an in-patient basis than as day cases.

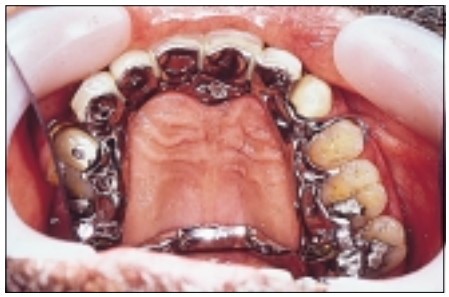

Figure 3. *Metal extended onto the backs of the anterior teeth of a partial denture for strength and safety.*

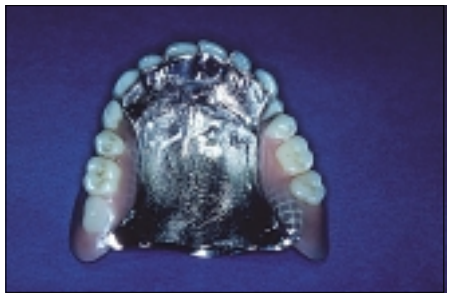

Figure 4. *Metal extended onto the backs of the anterior teeth of a complete denture for strength and safety.*

Prosthetic Treatment

It is not always possible to avoid the use of removable prostheses for people with epilepsy. In some instances, dentures can be avoided by accepting the concept of the shortened dental arch. However, if there are missing anterior teeth, a lack of posterior support causing temporomandibular discomfort or an edentulous arch, this is not feasible.

Wherever possible, partial dentures should have a metal framework, be tooth supported and be designed for maximum retention. The risk of loss or fracture of anterior teeth can be reduced by using metal posts to retain the denture (Figure 2) or extending the metal onto the palatal aspect (Figures 3, 4). Metal backings also reduce the risk of swallowing or inhaling any displaced denture teeth. An index must be used during denture construction to ensure that metal posts and backings are processed in the correct position (Figure 2).

If dentures cannot be tooth

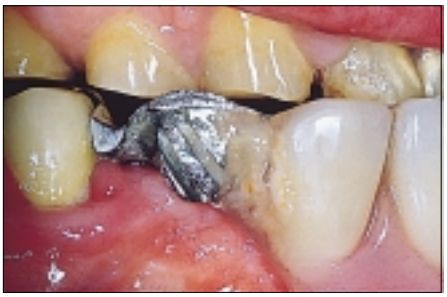

Figure 5. *The metal backing remains in place after loss of the denture tooth during a seizure.*

supported, a reinforced or metal denture base (Figure 5) or high-impact acrylic should be used.[25] Metal inserts such as wires and meshes often fail because stress concentrations that occur around the embedded materials have the net effect of weakening the denture polymer.[26] Carbon fibres and metal reinforcements should be extended over a wide area of the denture base, including the alveolar ridges, to reduce this effect (Figures 6, 7). However, in complete dentures it is advisable to avoid extending reinforcements to the palatal border (Figures 6, 7) as this makes any modification of the post dam area difficult. High-impact acrylic has the advantages of being aesthetic and easy to adjust. The disadvantage is that the flexural properties of these rubber-modified acrylic polymers are relatively poor and long-term failure due to fatigue can be a problem.[26]

If denture retention is compromised it may be sensible to advise the patient to use a denture fixative. Also, individuals who have an aura or a recognizable prodromal phase should be advised to remove their denture and put it in a safe place before a seizure.

Orthodontic Treatment

Orthodontic treatment is not contraindicated for people with epilepsy, even when seizure control is poor. Fixed appliances are preferable to removable appliances. If removable appliances are used the design must take into account the need for good retention and adequate strength.

MANAGEMENT OF A SEIZURE IN THE DENTAL SURGERY

Dealing with a patient undergoing an epileptic seizure should be part of the dental team's training in managing medical emergencies.

The first step is to protect the individual from further injury by clearing their mouth of all instruments, moving all equipment out of reach and reclining the dental chair to the supine position as near to the floor as possible.[7] It is not necessary to move the patient onto the floor, to restrain them or to place anything in their mouth. Avoid putting your fingers in their mouth as you might be bitten or, worse still, have your fingers broken if the patient clenches during a tonic-clonic seizure. Most fits are self-limiting and the individual will recover quite rapidly. Some people sleep deeply after a seizure and they should be placed in the recovery position and monitored until they wake. They are able to go home once they are alert but must be accompanied.

If a seizure does not terminate within 3 minutes, or if it stops and starts again, drug treatment should be considered. The administration of 10–20 mg of diazepam intramuscularly (i.m.) usually terminates status epilepticus;[27] an alternative is 10 mg midazolam i.m. into the front of the thigh.[28] Some people with epilepsy carry emergency midazolam to be placed in the buccal sulcus for rapid absorption through the oral mucosa. It is not necessary to administer oxygen during a short fit; however, if the fitting continues or restarts following drug administration, oxygen should be given and the emergency services called.

GENERAL PRINCIPLES OF ORAL HEALTHCARE

The following guidelines are suggested to achieve optimal oral health for people with epilepsy.

- Early contact with the dental team.
- Regular dental visits to establish trust and encourage disclosure about the pattern and type of seizures.
- Regular and rigorous preventive regime to avoid/minimize oral disease.

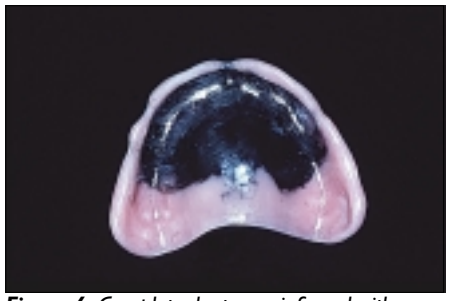

Figure 6. *Complete denture reinforced with carbon fibre, which is extended into the alveolar ridge area for strength but excluded from the post dam area for ease of modification.*

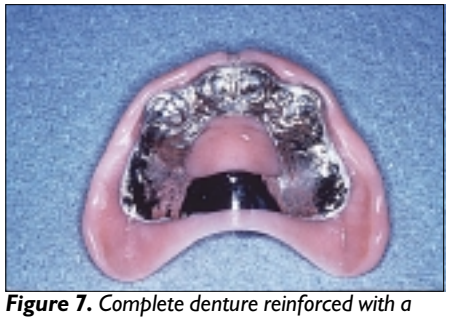

Figure 7. *Complete denture reinforced with a metal plate extended into the alveolar ridge area for strength but excluded from the post dam area for ease of modification.*

- Liaison with the physician to avoid caries or gingival overgrowth as a result of anti-epileptic medication.
- Careful treatment planning of restorative procedures.
- Training the dental team in seizure management.

CONCLUSION

Most people with epilepsy have well controlled seizures and can be treated safely in general dental practice. A careful medical and drug history should be taken and updated at each visit. The seizure history needs to be taken into account when treatment planning but should not mean that dental care is withheld.

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Useful Addresses

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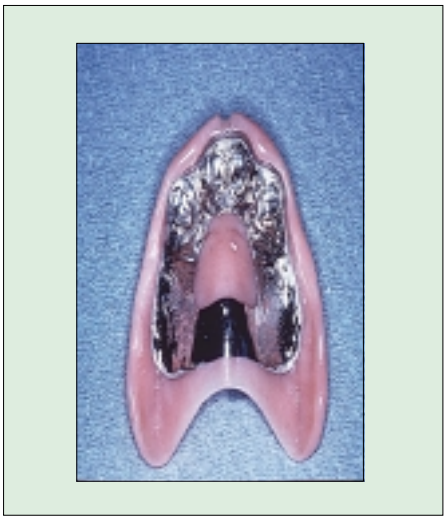
BOOK REVIEW

Adhesive Technology Theoretical and Practical Guidelines. By R. Hickel, K.H. Kunzelmann, P. Lambrechts *et al.* 3M ESPE AG, eds. 3M ESPE, 2001 (105pp., Free). ISBN 3-00-008488-6. (Obtainable by writing to 3M ESPE, 3M House, Morley Street, Loughborough, LE11 1EP, UK.)

It is not the policy of *Dental Update* to publish infomercials or advertorials among its papers or book reviews. The publication of this review does not infringe this policy, despite the fact that this book has been commissioned by a dental materials company, 3M ESPE, as this book presents a balanced and impartial review of the current status of adhesive dentistry. It covers a wide range of products and techniques in chapters written by clinicians in the forefront of contemporary dentistry in universities in Europe and scientists from the 3M ESPE company.

Chapter 1 covers the theoretical principles of adhesive bonding and presents the contemporary



classification for adhesive systems by a number of steps and types of interaction with dentine. The author of this chapter, Dr Frankenberger, also provides the reader with an excellent resumé of methods for testing adhesives, illustrated by a series of excellent diagrams.

The remainder of the book addresses the clinical applications of adhesives, with chapter 2 covering possible errors, such as over etching, underestimating the risk of allergy and misjudging the product's effect on the smear layer.

In chapter 3, we are presented with a classification of composite filler and resin systems, followed by the clinical stages of shade selection, moisture control, matrix selection, pulp protection, and the application of the materials and its polymerization.

While Chapters 5, 6 and 7 provide the results of clinical examinations of 3M ESPE materials – the only company-oriented parts of the book, Chapters 4 and 8 are essential reading for the dental team in respect of placement of adhesive restorations. Of particular value are a series of flow charts in Chapter 8 which provide step-by-step guides for a variety of adhesive clinical procedures.

Everyone concerned should be congratulated in producing an easily read text which is brimful of theoretical, relevant and clinical information.

This book would be good value at £25. For free, it is a give-away!

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