CJD and the Dentist

ANDREW SMITH AND JEREMY BAGG

Abstract: **The last 10 years have seen the emergence of a new disease termed variant CJD. This disease is thought to be initiated by abnormal prion proteins. This article reviews the different clinical manifestations of human prion diseases and provides some background information on the biological nature of this unique infectious agent. Prion proteins present a challenge to infection control because of their relative resistance to the conventional sterilization process. This article highlights the issues involved and discusses current guidelines for management of patients with human transmissible encephalopathies.**

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Clinical Relevance: **Prion diseases present a unique challenge to infection control protocols.**

The last 10 years have seen the emergence of a new infectious agent which reached epidemic proportions in cattle. The disease Bovine Spongiform Encephalopathy (BSE) has devastated the livestock industry in the UK. The causative agent of this epidemic is an abnormal protein termed prion protein. Unlike previous infectious agents, it appears that these abnormal prions do not depend on nucleic acid for their replication. Although it is unknown whether prion diseases, such as Creutzfeldt Jakob Disease (CJD), are due entirely to prion agents or whether other molecules may be an essential adjunct to produce disease, it is generally accepted that abnormal prion proteins are an important component.

The emergence of variant CJD (vCJD) in the mid 1990s and its probable link to the consumption of BSE-contaminated material has resulted in uncertainty about how many members of the UK population have been infected.[1] This in turn has led to widespread changes in the re-processing of surgical instruments in the UK. The key challenge to infection control and the dental treatment of patients suffering with prion diseases is the remarkable resistance of these agents to inactivation.

Patients suffering from prion diseases may require dental treatment and it is important to recall the GDC's statement on *Maintaining Standards* (Anon., General Dental Council, 2000) that it 'is unethical to refuse treatment of a patient solely on the basis that they have an infectious disease'.[2] Thus, CJD is an issue for all members of the dental profession and not just specialist centres. This article will provide a brief overview of the different forms of human prion diseases and the infection control implications for dentistry.

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PRION PROTEINS

In health, prion proteins exist as protease sensitive, cell surface proteins in many cells such as lymphocytes. This form of the protein is designated prion protein cellular (PrP^c) but its function is currently unknown. In humans, a gene located on chromosome 20, called the prion protein gene, codes for both the normal and abnormal forms of the protein. The normal prion protein is soluble and protease sensitive, whilst the abnormal prion protein (PrP^{sc}) is insoluble and resistant to proteinase. PrP^c is rich in α-helical structures, whereas PrP^{sc} seems to be composed mainly of a β-pleated sheet structure (Figure 1). PrP^{sc} is postulated to act as a conformational template that promotes the conversion of PrP^c to further PrP^{sc}.

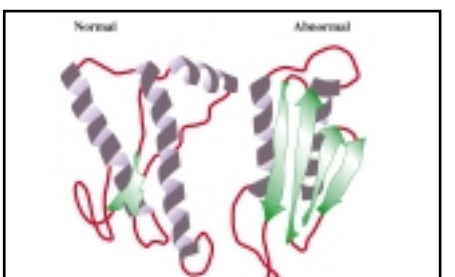

***Figure 1**. Schematic diagram of abnormal and normal forms of prion protein. The abnormal form of prion protein is richer in ß-pleated sheet structures.*

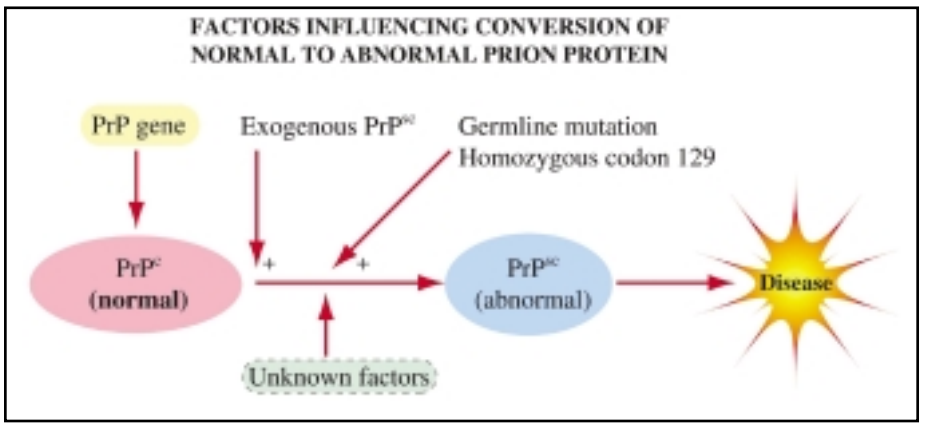

Figure 2. *Summary of events influencing prion replication.*

This triggers a chain reaction with the accumulation of further insoluble PrP^{sc} in neural cells, disrupting function and leading to vacuolization and cell death.

Prion replication, with recruitment of PrP^{c} into the aggregated PrP^{sc} isoform, may be initiated by a pathogenic mutation (resulting in a PrP^{c} predisposed to form PrP^{sc}) in inherited prion diseases, by exposure to a seed of PrP^{sc} in acquired disease, or as a result of the spontaneous conversion of PrP^{c} to PrP^{sc} (and subsequent formation of aggregated material) as a rare event in sporadic prion disease (Figure 2).

TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

CJD is part of a wide spectrum of diseases, affecting both humans and animals, that are termed transmissible spongiform encephalopathies (TSEs). Animal diseases include scrapie (sheep), BSE (cattle) and transmissible mink encephalopathy. All have incubation periods of months to years leading inexorably to death. None evokes an immune response and all share a common non-inflammatory pathologic process in the central nervous system. In all affected species, infectivity is greatest in brain tissue, is present in some peripheral tissues, but generally has been absent from body fluids, such as saliva.[3]

Kuru was the first human TSE shown to be transmissible. This fatal disease of cerebellar degeneration reached endemic proportions among the Fore ethnic group in a remote area of Papua New Guinea. The disease was spread by ritual cannibalism and had an incubation period of 4–40 years. The disease has gradually disappeared over the last 40 years since the practice ceased.

CJD

CJD occurs naturally as both a sporadic and a familial disease. Its epidemiological and clinical patterns are different from kuru, but it produces similar spongiform changes in the nervous system.

Sporadic CJD (sCJD)

This disease occurs world-wide, with an incidence of approximately one case per million population per year. There is no seasonal variation, no evidence of changing incidence over the years, and no convincing evidence for geographic clustering. Many series and case control studies have searched for risk factors such as diet, exposure to animals, occupational exposures and dental treatment. Some workers[4] have reported that surgical procedures were significantly associated with the development of sCJD. This group also found a significant association between risk of sCJD and residence or employment on a farm or market garden for longer than 10 years. No significant risk was associated with a history of major dental treatment.[4] However, another study[5] did not confirm the association of sCJD with a history of surgery, nor was there any history of occupational exposure to animals or leather. The documented evidence of a relative lack of communicability should reassure dental teams who care for patients with this disease.

Iatrogenic CJD

As indicated earlier, human –human transmission of a TSE was demonstrated among the Fore people with kuru. However, whether the original source was an index case of sCJD or some other event will probably never be known. Other cases of human–human transmission can be divided into two groups:

- Surgical transmission;
- Transmission by pituitary hormones.

Surgical Transmission

A number of surgical procedures have been associated with inadvertent transmission of CJD. The first suspected human transmission was reported in 1974, when rapidly progressive neurological disease

Feature	vCJD	sCJD
Mean age at onset (yrs)	28	60
Mean duration of disease (months)	14	5
Early signs	Neuropsychiatric symptoms	Dementia, myoclonus
EEG complexes (%)	0	94
Pathologic changes	Diffuse amyloid plaques	Sparse plaques in 10%

Table 1. *Key features of vCJD and sCJD.*

Disease	Tonsil	Spleen	L node
Variant CJD	10/10	11/11	9/9
Iatrogenic CJD	0/1	0/5	0/4
Sporadic CJD	0/18	0/21	0/10

***Table 2**. Tissue distribution of infectious agent.*

developed in a woman 18 months after a corneal transplant.[6] Contaminated neurosurgical instruments have also been suspected as modes of transmission in several cases, the most convincing being reported after two patients underwent neurosurgery, using electrodes that had previously been implanted into a patient with CJD. The electrodes had been 'decontaminated' with 70% alcohol and formaldehyde vapour, yet two years later these electrodes were retrieved and implanted into a primate which subsequently developed CJD.[7] Over the past decade, more than 80 cases of CJD have been recognized 1.6–17 years after neurosurgical placement of grafts of human cadaver dura mater.[8] Of concern is the use of human dura mater material in procedures outside the central nervous system (CNS), for example, periodontal regeneration techniques.[9]

Transmission by Pituitary Hormones

In 1985, CJD developed in four patients who had received human growth hormone (all aged under 40 years). Injection of the hormone, which was derived from pooled cadaveric human pituitary glands, had been discontinued 4–15 years before the onset of disease. In the UK, approximately 1% of recipients have been affected, with a mean incubation period of 12 years. Recombinant growth hormone, with which there is no associated risk, was licensed in 1985 and is now used exclusively.[10]

Familial CJD (fCJD)

Between 10–15% of individuals with CJD have a family history consistent with an autosomal dominant inheritance of the disease. More than 20 different mutations in the PrP gene have been described. On average, fCJD has an earlier age of onset and a more prolonged course than sCJD. It must be emphasized that fCJD is extremely rare and only close blood line relatives that carry the gene mutation are 'at-risk' from the disease.

Variant CJD (vCJD)

In 1996, the National CJD Surveillance Unit identified a small number of patients with a previously unrecognized but consistent disease pattern and reported that the most likely explanation was exposure to the agent responsible for BSE.[11] These patients were younger than those with classic forms of the disease (Table 1), with prominent early psychiatric and behavioural manifestations and persistent paresthesias and dysesthesias. Pathological examination showed prominent and diffuse PrP^{sc} plaques in brain tissue, similar to those found in kuru.

Subsequent laboratory experiments have provided compelling evidence that the causative agents of vCJD and BSE are the same.[12] Glycosylation patterns of PrP^{sc}, susceptibility studies in mice, and patterns of disease in brain tissue from vCJD patients and cattle with BSE are similar but distinct from the patterns associated with scrapie, sCJD and fCJD. There is also a specific genetic predisposition to the disease. All patients with vCJD analysed to date have been homozygous for methionine at codon 129.

In vCJD, unlike previous forms of human TSEs, post mortem studies have shown that abnormal prion is accumulated not only in the CNS, but also in lymphoreticular tissues, including the tonsils and appendix (Table 2). It is likely that infected humans who are developing vCJD will have potentially infective tissues in the pre-clinical phase of their illness. This has highlighted the theoretical risk of iatrogenic transmission of the infective agent of vCJD. Currently it is assumed that CNS tissue, material from the posterior chamber of the eye and lymphoreticular tissue are, in descending order, the tissues most likely to be infective. The potential infectivity of material transferred from one patient to another depends both on its source (for example, brain tissue), route of transfer (direct or oral inoculation) and where it is deposited (e.g. another patient's brain). Most of the data related to infectivity are derived from experiments in which material has been injected directly into the brain.

The number of cases of vCJD stays steady (Figure 3), though the total number remains low. Factors that are important in determining the probability of acquiring vCJD are likely to include infective dose, route of exposure and genetic susceptibility. At present, there

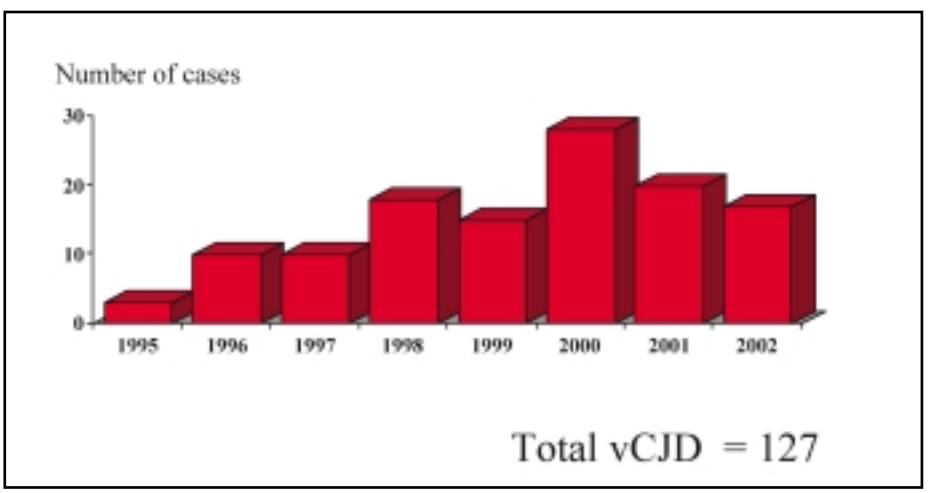

***Figure 3.** Epidemiology of variant CJD (figures correct up to 31/01/03).*

Definition of 'at-risk' = Asymptomatic but potentially at-risk of developing disease:

- recipients of hormone derived from human pituitary glands, e.g, growth hormone, gonadotrophin;
- recipients of human dura mater grafts;
- people with a close (blood line) family history of CJD.

***Table 3**. Definition of 'at-risk' patients.*[14]

is no evidence of occupationally acquired infection. Data from the various types of human TSEs suggest that incubation periods of prion diseases in humans, after peripheral or oral exposure, range from at least 4 years to 40 years, with a mean of about 10–15 years. Attempts by statistical modelling to predict the eventual scale of any vCJD epidemic only serve to emphasize the uncertainties, with estimates which have ranged from less than 100 to several million cases.

STERILIZATION AND DISINFECTION

A consistent experimental finding is that PrP^{sc} is very resistant to the usual techniques for inactivating infectious agents. Ionizing, ultraviolet and microwave radiation have little effect on prion proteins. Exposure of infectious material to steam heat at 134°C for 18 minutes in a vacuum autoclave may not achieve complete inactivation.[13] For some strains of prion, thermostability increased as the temperature was raised from 134°C to 138°C.[13] Concentrated bleach appears to achieve inactivation of all strains, but concentrated sodium hydroxide does not. A key feature of experimental work has been the recognition that the scrapie agent is more resistant to inactivation by autoclaving when infected tissue becomes dried onto glass or metal surfaces. Prior fixation of tissue in ethanol or formaldehyde has also been shown considerably to enhance the thermostability of the scrapie agent. These findings emphasize the importance of very thorough cleaning of equipment prior to sterilization, in order to reduce contamination with any residual prion protein to as low a level as possible.

MANAGING INFECTION CONTROL IN THE DENTAL TREATMENT SETTING

Patients Confirmed or Suspected of Suffering from CJD

There are a number of guidelines for the management of patients with CJD.[14–16] There is general agreement in the advice for dental treatment on patients confirmed as suffering from any type of CJD. In such cases, instruments used for dental treatment should be disposed of after single use. Apart from the use of disposable instruments, no other special precautions are necessary over the routine universal precautions adopted for all patients undergoing dental treatment. Although some workers have suggested the use of separate water lines and suction systems for the treatment of such patients,[17] there is no evidence that prions are likely to be transmitted via this route and these additional precautions are not routinely adopted.[18]

For patients with a suspected diagnosis of CJD, but which remains to be confirmed, instruments that have been used for their treatment should be quarantined until a decision has been made about the diagnosis. If the diagnosis is confirmed, the instruments should be destroyed.

Patients 'At-Risk' from CJD

At present, the situation for patients 'at-risk' from CJD (see Table 3) is slightly more problematic. The first issue is identification of the 'at-risk' patient. The current BDA medical history form contains questions designed to identify persons belonging to this group. It is clearly an emotive area for patients to be confronted at a dental appointment with the possibility (albeit extremely small) that they are 'at risk' of CJD. Clearly, great tact is required and special counselling skills are needed, which are unlikely to be available in a general practice environment. This is an issue that requires much thought prior to seeking to identify whether patients belong to an 'at-risk' category. If identified as 'at-risk', then the ACDP/SEAC infection control guidelines[14] for these patients recommend use of stringent decontamination processes (see Table 4) for treatment procedures (such as dentistry) on tissues other than brain, spinal cord or eye. Since these guidelines are extremely difficult to comply with in dental practice, this implies use of disposable dental instruments. Other than the use of disposable instruments, no other

Process	Method
Cleaning	Items should be cleaned a.s.a.p. after use to minimize drying of blood and other body fluids. All instruments should be cleaned thoroughly at least twice prior to disinfection. Do not mix routine instruments with those used in TSE-related work. Following cleaning of instruments the instrument washer should be run on an empty cycle. Any cleaning aids used, such as brushes, should be disposed of by incineration.
Sterilizing	
Chemical agents	20,000ppm available chlorine of sodium hypochlorite for 1 hour. 2M sodium hydroxide for 1 hour*
Physical processes	Porous load steam sterilizer 134–137°C for a single cycle of 18 minutes or 6 successive cycles of 3 minutes each*
(*but known not to be completely effective).	

***Table 4**. Stringent disinfection and decontamination procedures for instruments used in clinical procedures NOT involving brain, spinal cord or eye.*[14]

Observation	Workers
Very low levels of scrapie infectivity from gingivae of infected mice. Failed to transmit scrapie by dental burs used on infected animals	Adams & Edgar (1978)[19]
Infection of mice by scrapie agent via oral route is increased if gingival tissue is scarified	Carp (1982)[20]
Small clusters of sCJD cases possibly connected by dental procedures (3 cases UK & 3 cases Japan)	Will & Mathews (1982)[21] Arakawa *et al.* (1991)[22]
Epidemiological evidence seems to exclude any correlation between tooth extraction or dental surgery and human TSEs	van Duijn *et al.* (1998)[5] Collins *et al.* (1999)[4]
Gingival and pulpal tissues in hamster scrapie model have a high level of infectivity. Transmission of agent obtained by inoculation of tooth pulp	Ingrosso *et al.* (1999)[23]
No prion protein detected by western blotting in the dental pulp of 8 sCJD patients	Blanquet-Grossard *et al.* (2000)[24]

Table 5. *Dental tissues: information on distribution of infectious agents.*

special precautions are necessary over and above the routine universal precautions adopted for all patients. Some centres are identifying kits of instruments that are designated for use in the treatment of specific individual patients; these instruments are cleaned and sterilized according to the ACDP/SEAC protocol[14] but labelled and stored aseptically in a secure area until that patient re-attends for further treatment.

As an alternative, some centres have adopted the more recent WHO guidelines,[16] which do not recommend special precautions for the routine dental treatment of 'at-risk' patients. This removes the necessity for identifying such patients during medical history taking. The emphasis is then placed on high levels of infection control for each patient. Of interest is the WHO recommendation that endodontic instruments should not be re-used after use on such patients. In view of the difficulties in cleaning endodontic instruments, it seems prudent that this approach of single use is adopted for all patients undergoing root canal therapy.

Logically, the patients that represent the highest risk of cross-infection of abnormal prion proteins are those in the pre-clinical phase of vCJD. Since at this stage it is impossible to recognize asymptomatic patients with vCJD, this emphasizes the importance of universal precautions. In particular, the importance of maintaining a high standard for routine cleaning and sterilizing of reusable dental instruments cannot be over-emphasized.

PRION PROTEINS IN DENTAL TISSUES

Studies that have investigated the presence of prion proteins in oral tissues from human and animal models are outlined in Table 5. Of concern is the finding that prion protein can be detected in both gingival and pulpal tissue in the hamster scrapie model. However, owing to the differences in patterns of disease in animal models and the various strains of prion protein, it is difficult to extrapolate these findings directly to humans. Data concerning the distribution of prion protein in the human oral cavity are urgently required to allow risk assessments of the potential for cross-infection from common dental procedures.

CONCLUSION

Prion proteins are a challenge to infection control because of their relative resistance to conventional methods of decontamination. For most forms of human TSEs (sCJD, iCJD and fCJD) there appears little evidence that they pose a significant risk of cross-contamination during routine dental treatment. vCJD may pose more of a risk by virtue of the different tissue distribution of the abnormal prion proteins, which includes lymphoid tissue. However, we currently have no method of detecting patients in the pre-clinical phase of the disease. It seems rational in the face of a potential epidemic that our basic standard of infection control, namely universal precautions, should be rigorously enforced. The most important facet of this will probably be based on very thorough cleaning of re-usable instruments prior to autoclaving. In the interim, many decisions concerning diagnosis, treatment and management of patients with CJD are often based on incomplete knowledge. This information is rapidly evolving and it is vital that dentists keep abreast of current developments.

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BOOK REVIEW

Decision-making in Operative Dentistry. By P. Brunton. Quintessence Publishing Co. Ltd., New Malden, 2002 (100pp., £28.00). ISBN 1-85097-057-2.

Every dentist, after entering general practice, soon realizes that one of the hardest parts of their work entails deciding upon the correct course of treatment for their patients. Should a tooth be treated or monitored, what type of treatment is best for the long-term health of the tooth, what material to use, etc. Paul Brunton addresses these types of questions in his book *Decision-making in Operative Dentistry*. He has produced a clearly presented, well thought out textbook aimed at the general dental practitioner to help with this difficult aspect of dentistry.

The first two chapters deal with diagnosis, caries risk assessment and criteria for intervention. Apart from a few mistakes with the X-ray illustrations, the chapters are informative, giving the GDP a good understanding of available techniques, with some useful flow charts on findings and subsequent treatment modalities.

The next few chapters outline the newer ideas and techniques in caries treatment, especially minimal intervention techniques, with a good overview of current materials on the market. The days of putting a thick kalzinol liner for pulp protection are long gone.

The penultimate chapter, on whether to repair, replace or refurbish an existing restoration, is well worth reading and will certainly make the reader rethink the next time they are faced with a failing restoration.

The final chapter deals with non-carious tooth tissue loss. This is becoming an increasingly common problem and the reader is given a good introduction to this difficult and clinically demanding challenge.

Paul Brunton is successful in his remit to cover a large number of difficult topics. His book should stimulate the reader into delving more deeply into the topics that we, as general practitioners, are faced with on a daily basis.

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ABSTRACT

ARE FILM HOLDERS REALLY NECESSARY?
Effect of X-ray Beam Angulation and Intraradicular Contrast Medium on Radiographic Interpretation of Lower First Molar Root Canal Anatomy. H.J. Naoum, R.M. Love, N.P. Chandler and P. Herbison. *International Endodontic Journal* 2003; **36:** 12–19.

Radiographic guidelines for good clinical practice suggest that all periapical films should be taken using a long-cone parallel technique and a film-aiming device. Sometimes, however, advice as to best practice may not be substantiated by clinical relevance. The question 'why' may be a valid tool when considering new techniques or changes to established practices. Although the research was carried out *in vitro*, positioning extracted teeth in dried mandibles, the results are directly relevant to the clinical situation.

The authors took radiographs of the same tooth at 0° and 30° angulation. Three evaluators then assessed the films for clarity of six criteria, including canal morphology and number, apical anatomy and the presence of lateral canals. There was a statistically significant difference between the images, with greatly improved diagnosis in the 0° angled views.

Practitioners frequently report that root canal treatment is difficult because they are working 'blind'. It does make sense, therefore, to obtain the maximum possible information available from the pre-operative radiograph. This work provides the required 'evidence-base' for this practice.

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