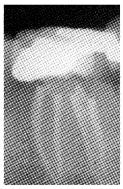
doi:10.1111/j.1365-2591.2008.01468.x



CLINICAL ARTICLE

Dens invaginatus. Part 1: classification, prevalence and aetiology

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Abstract

Alani A, Bishop K. Dens invaginatus. Part 1: classification, prevalence and aetiology. *International Endodontic Journal*, **41**, 1123–1136, 2008.

Aim To review and discuss the aetiology, prevalence and classification of this dental anomaly together with the morphology of an invagination and the most appropriate nomenclature.

Summary This review considers the different possible nomenclatures and concludes that dens invaginatus is the most appropriate description. The paper highlights the different reported prevalence figures and concludes that the problem is probably one of the most common of the dental developmental abnormalities with maxillary lateral incisors most commonly affected. The paper suggests that the classification system described by Oehlers (1957a) is probably the most clinically relevant and that the morphological features associated with this problem may increase the risk of pulpal pathology developing and complicate any possible endodontic treatment.

Key learning points

- The aetiology of dens invaginatus is still unknown, although there is some evidence that it may be genetic in origin.
- The problem is probably more prevalent than most clinicians are aware of and this is because of the diagnostic difficulties associated with the anomaly.
- The nature of the problem may increase the risk of pulp disease and complicate any root canal treatment.

Keywords: aetiology, classification, dens in dente, dens invaginatus, nomenclature, prevalence.

Received 21 January 2008; accepted 19 July 2008

Introduction

Dens invaginatus is a developmental anomaly resulting in a deepening or invagination of the enamel organ into the dental papilla prior to calcification of the dental tissues (Hülsmann 1997). A number of other terms have also been used to describe the condition.

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For example, according to Šutalo *et al.* (2004), in 1897 Busch first suggested the use of 'dens in dente' which implies the radiographic appearance of a tooth within a tooth. However, Hunter (1951) suggested the term 'dilated composite odontome' which infers an abnormal dilatation of the dental papilla whilst Colby (1956) recommended the use of 'gestant anomaly'.

The varied nomenclature probably reflects the lack of consensus on the formation, aetiology and classification of the condition (Gonçalves *et al.* 2002). Of the various terms, dens invaginatus would appear to be the most appropriate as it reflects the infolding of the outer portion (enamel) into the inner portion (dentine) with the formation of a pocket or dead space (Fig. 1a,b). Dens invaginatus also better represents the range of presentations than other descriptions that appear more applicable to specific variations fundamental to the condition. For example, the term 'dilated odontome' is not appropriate in minor invaginations where crown dilation may not occur (Šutalo et al. 2004).

Dens invaginatus is not an uncommon clinical finding in permanent teeth and probably occurs more often than other developmental anomalies. For example, Backman & Wahlin (2001) examined a group of 739 individuals and reported that 6.8% of subjects had evidence of dens invaginatus whilst peg-shaped lateral incisors occurred in 0.8%, gemination 0.3% and taurodontism 0.3%. In the same study the presence of dens invaginatus was comparable to hypodontia (7.4%) and more common than hyperdontia (1.9%) (Backman & Wahlin 2001).

Although dens invaginatus is common it may be easily overlooked because of absence of any significant clinical signs of the anomaly. This is unfortunate as the presence of an invagination is considered to increase the risk of caries, pulpal pathosis and periodontal inflammation. Furthermore, the nature of the problem can often mean that any necessary endodontic treatment may be complicated (Oehlers 1957a, Omnell *et al.* 1960, Beynon 1982, De Smit *et al.* 1984, Rotstein *et al.* 1987). This is reflected by a survey of general dental practitioners where 38.4% of the 307 respondents considered that if a tooth with dens invaginatus required root canal treatment then they would refer to a specialist endodontist (Hommez *et al.* 2003).

As such, early identification of a tooth affected by dens invaginatus is important. The aim of the present report is to review the prevalence, classification and aetiology of the anomaly; a second report will highlight the clinical and radiographic features together with management options.

Classification

The first documented attempt to classify dens invaginatus was by Hallet (1953) who suggested the existence of four types of invagination based on both clinical and radiographic criteria.

Other classifications have also been described involving a variety of criteria and standards (Ulmansky & Hermel 1964, Vincent-Townend 1974). For example, Schulze & Brand (1972) suggested an assessment based on twelve possible variations in clinical and radiographic appearance of the invagination. However, the system described by Oehlers (1957a) appears to be the most widely used, possibly because of its simple nomenclature and ease of application. This system categorizes invaginations into three classes as determined by how far they extend radiographically from the crown into the root.

Type I: The invagination is minimal and enamel-lined, it is confined within the crown of the tooth and does not extend beyond the level of the external amelo-cemental junction.

Type II: The invagination is enamel-lined and extends into the pulp chamber but remains within the root canal with no communication with the periodontal ligament.

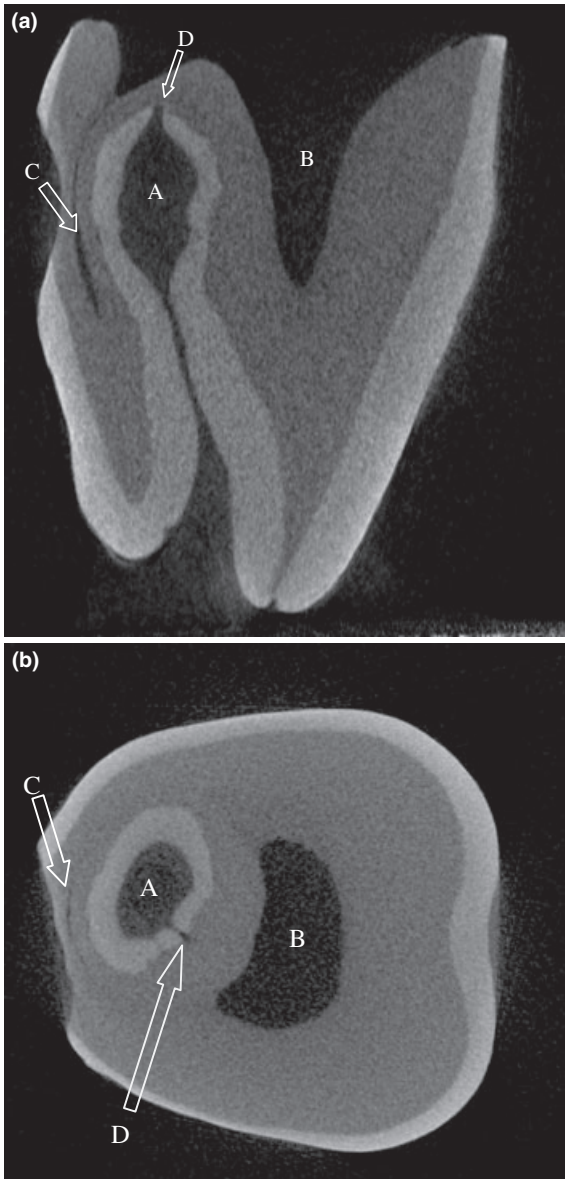

Figure 1 (a) Cross-sectional and (b) axial CT views of a crown of an extracted tooth affected by Oehlers' Type II invagination using a CT Scanner (Skyscan 1072; SkyScan, Kontich, Belgium). The infolding of the enamel and dentine is clearly visible (A), distinct from the main pulp (B) which has been divided to produce two distinct areas (B and C). Interruptions in the enamel layer are present in both planes (D). CT, computed tomography.

Type IIIA: The invagination extends through the root and communicates laterally with the periodontal ligament space through a pseudo-foramen. There is usually no communication with the pulp, which lies compressed within the root.

Type IIIB: The invagination extends through the root and communicates with the periodontal ligament at the apical foramen. There is usually no communication with the pulp.

In Type III lesions, any infection within the invagination can lead to an inflammatory response within the periodontal tissues giving rise to a 'peri-invagination periodontitis'.

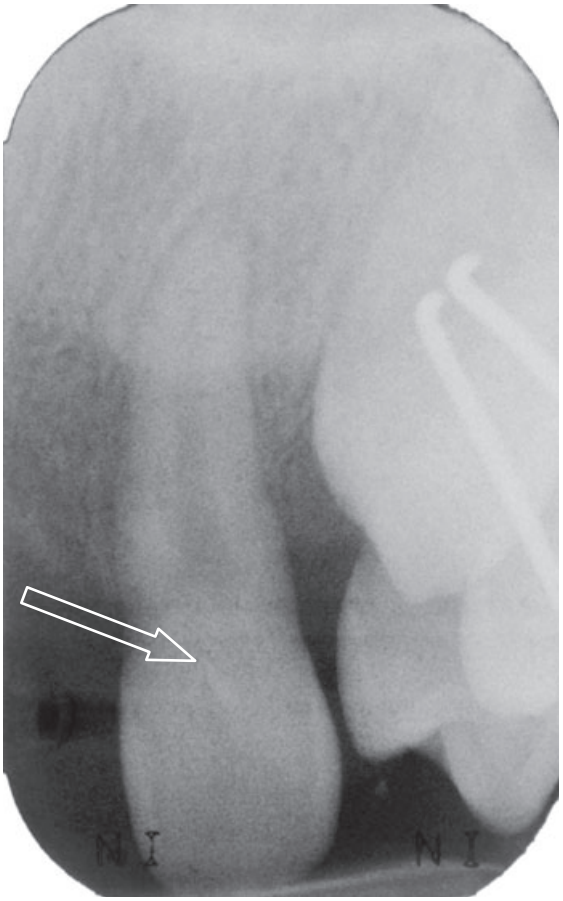

Figure 2 An intra-oral radiograph of maxillary lateral incisor illustrating Oehlers' Type I invagination. Note the deep fissuring (arrow) pointing towards the pulp.

Using Oehlers' Classification, the prevalence of each type of invagination was reported by Ridell *et al*. (2001) with Type I being the most common (79%) (Fig. 2) whilst Type II (15%) (Fig. 3) and III (5%) (Fig. 4) less frequently observed. Oehlers' system is based on a two dimensional radiographic image and as such may underestimate the true extent and complexity of the invagination. However, despite its limitations, Oehlers' classification makes a distinction between complete (Type III) and incomplete (Type I and II) invaginations which is important as the management of each is potentially different. As such, this system may be more valuable from a clinical perspective than more complex classifications.

The limitations associated with the use of conventional radiography in the classification and management of dens invaginatus may be overcome in the future with the increasing availability of computerized 3D imaging (Vannier *et al.* 1997, Sponchiado *et al.* 2006). Currently, such clinical techniques do not provide images of sufficient quality to fully evaluate the morphology of an invagination *in situ* although for extracted teeth sufficient detail can be obtained.

Prevalence and distribution

The reported prevalence of adult teeth affected with dens invaginatus is between 0.3% and 10% with the problem observed in 0.25% to 26.1% of individuals examined (Table 1).

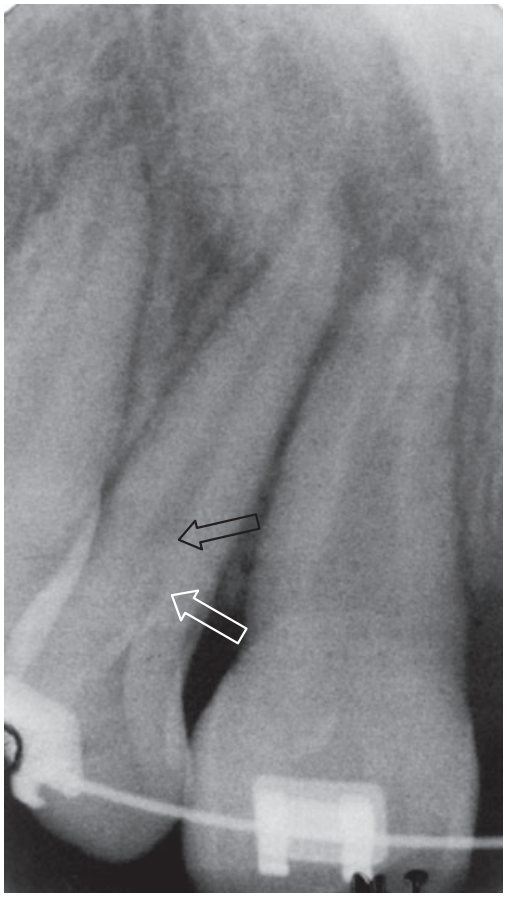

Figure 3 An intra-oral radiograph of maxillary lateral incisor illustrating Oehlers' Type II. Note the tear-shaped ribbon-like formation of the invagination cavity (White arrow) and the presence of blunting of the pulp canal horns because of encroachment by the invagination (Black arrow).

The wide variation in reported prevalence may be explained by the different cohorts studied, identification criteria used and diagnostic difficulties (Tagger 1977). For example, a number of studies failed to describe the parameters used to identify the presence of teeth affected with the problem (Ruprecht *et al.* 1986, Thongudomporn & Freer 1998) whilst others employed their own unique criteria (Parnell & Wilcox 1978, Gotoh *et al.* 1979). However, more recent studies (Ridell *et al.* 2001, Backman & Wahlin 2001) have utilized a recognized classification system (Hallet 1953, Oehlers 1957a).

The permanent maxillary lateral incisor appears to be the most frequently affected tooth (Hülsmann 1997) with posterior teeth less likely to be affected (Conklin 1978, Lee *et al.* 1988). This is supported by Hamasha & Al-Omari (2004) who reported that in 1660 subjects examined, 61 out of 14090 teeth had evidence of an invagination. Of the affected teeth, 90% were lateral incisors and only 6.5% were posterior teeth; interestingly, no mandibular teeth were reported to be affected by dens invaginatus. However, this latter observation is not supported by a number of case reports, which have identified dens invaginatus in mandibular teeth (Banner 1978, Altinbulak & Ergül 1993, Bramante *et al.* 1993, Tavano *et al.* 1994, Khabbaz *et al.* 1995, Hartup 1997, Er *et al.* 2007).

There is also some evidence that the problem may be symmetrical (Hamasha & Al-Omari 2004). However, conflicting opinions exist with Swanson & McCarthy (1947)

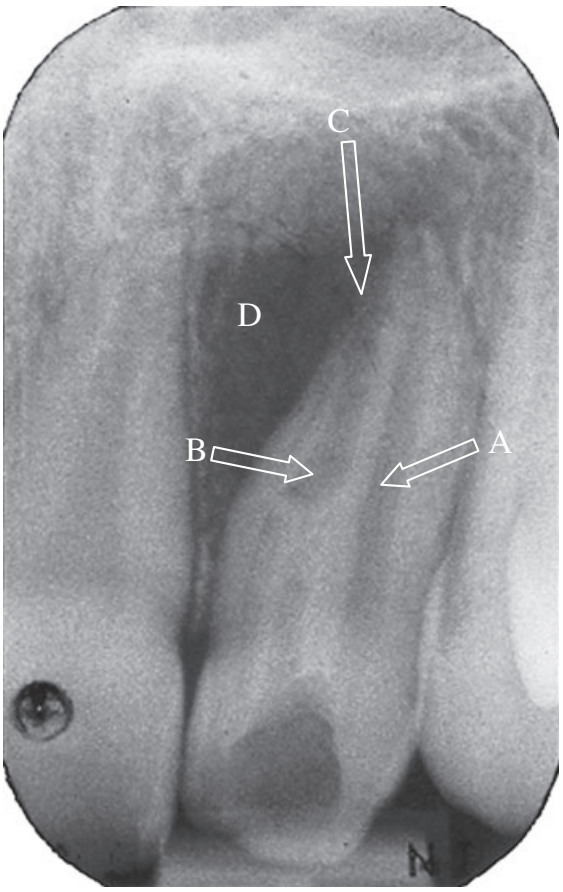

Figure 4 An intra-oral radiograph of a maxillary lateral incisor with Oehlers' Type IIIA. Note the the pulp canal (A) with an adjacent invagination (B) opening into the periodontal ligament (C) creating an apical radiolucency (D).

describing bilateral invagination as a rare occurrence whilst others (Atkinson 1943) considered symmetry a common finding. The latter view is supported by Grahnen *et al.* (1959) who in a study of 3020 lateral incisors, reported that dens invaginatus occurred bilaterally in 43% of patients examined.

There have also been case reports of dens invaginatus occurring in the primary dentition (Rabinowitch 1952, Holan 1998, Kupietzky 2000, Eden *et al.* 2002). Interestingly, Mann *et al.* (1990) in a study of skeletons identified the anomaly in the deciduous second molar of a 5-year-old fourteenth-century American-Indian child whose gender is not recorded. However, all the documented case reports are of males which, if a true reflection, contrasts to the permanent dentition where females appear to be more at risk (Thongudomporn & Freer 1998) or there is no gender difference reported (Hamasha & Al-Omari 2004). There appears to be no reports of deciduous lateral incisors being invaginated unlike their permanent successor, which has been reported as the most common tooth affected (Hülsmann 1997).

Aetiology

There is a lack of consensus on the aetiology of dens invaginatus. Rushton (1937) suggested that the cause was embryological with the stimulation and subsequent

Table 1 Prevalence studies on dens invaginatus

References	Sample	Frequency
Mühlreiter (1873)	500 Lateral maxillary incisors	2.8%
Atkinson (1943)	500 Lateral incisors	10% of teeth
Boyne (1952)	1000 Maxillary incisors	0.3% of teeth
Stephens (1953)	150 Full mouth surveys	8%
Shafer (1953)	2542 Full-mouth surveys	1.3% of patients (as cited by Hovland & Block 1977)
Hallet (1953)	586 Full-mouth surveys	6.6% of lateral incisors & 0.5% of maxillary central incisors.
Amos (1955)	1000 Full-mouth surveys 203 Full-mouth surveys	5.1% of patients 6.9% of students of dentistry
Grahnen *et al.* (1959)	3020 Right maxillary incisors	2.7% of patients
Ulmansky & Hermel (1964)	500 Full-mouth surveys	2% of patients
Poyton & Morgan (1966)	5000 Full-mouth surveys	0.25% of patients
Miyoshi *et al.* (1971)	Extracted maxillary lateral incisors	38.5% of teeth (as cited by Gotoh *et al.* 1979)
Fujiki *et al.* (1974)	2126 Lateral maxillary incisors	4.2% of teeth (as cited by Gotoh *et al.* 1979)
Thomas (1974)	1886 Full-mouth surveys	7.74% of patients
Gotoh *et al.* (1979)	766 Maxillary lateral incisors	9.66% of teeth
Ruprecht *et al.* (1986)	1581 Full-mouth surveys	1.7%of patients
Ruprecht *et al.* (1987)	300 Full-mouth surveys	10% of patients
Thongudomporn and Freer (1998)	111 Full-mouth surveys	26.1% patients
Backman & Wahlin (2001)	739 Full-mouth surveys	6.8% of patients
Hamasha & Al-Omari (2004)	1660	2.95% of patients and 0.65% of teeth
Ezoddini *et al.* (2007)	480 Dental panoramic tomograms	0.8%

proliferation and ingrowth of cells of the enamel organ into the dental papilla during development. In contrast, Kronfeld (1934) considered that the problem was the result of the retardation of a focal group of cells, with those surrounding continuing to proliferate normally.

However, Atkinson (1943) suggested that the problem was the result of external forces exerting an effect on the tooth germ during development. Such forces could be from adjacent tooth germs, e.g. the central incisor or canine which develop at least 6 months prior to the lateral incisor (Segura *et al.* 2002) whilst other external factors such as trauma (Gustafson and Sundberg 1950) and infection (Fischer 1936, Sprawson 1937) have also been suggested as a cause.

During tooth development the ectomesenchymal signalling systems that occur between the dental papilla and the internal enamel epithelium affect tooth morphogenesis (Ohazama *et al.* 2004). These signals have specific roles such as the regulation of growth and the folding of the enamel organ (Kettunen *et al.* 2000). The absence of certain molecules can result in abnormally shaped teeth as well as defects in the developing tooth germ (Dassule *et al.* 2000). For this reason the proposal that genetic factors may be the cause of dens invaginatus has some credibility (Grahnen *et al.* 1959, Casamassimo *et al.* 1978, Ireland *et al.* 1987, Hosey & Bedi 1996). Support for this possible cause also comes from a reported case of an individual lacking chromosome 7q32 who presented with dens invaginatus in addition to other dental abnormalities such as hypodontia (Pokala & Acs 1994). There is further support from a clinical study of 3020 Swedish children that reported 2.7% of patients with dens invaginatus, 43% of their parents and 32% of siblings also had evidence of the condition (Grahnen *et al.* 1959).

Additional support for a genetic influence is drawn from the fact that the invaginations appear to have a limited variation (Oehlers 1957a) and can occur in a number of teeth in the same individual (Altinbulak and Ergül 1993, Jimenez-Rubio *et al.* 1997, Lorena *et al.* 2003, Mupparapu *et al.* 2004) or in siblings (Hosey & Bedi 1996). In addition, there also appears to be an increased incidence in mongoloid groups and lower incidence in negroid groups (Oehlers 1957a) and clustering of other genetically determined dental anomalies has been observed (Peck *et al.* 1996, Shapira & Kuftinec 2001, Basdra *et al.* 2001, Segura *et al.* 2002, Leifert & Jonas 2003, Kansu & Avcu 2005). For example, Thongudomporn & Freer (1998) in a study of patients requiring orthodontic treatment reported that 26.1% of individuals had evidence of teeth with dens invaginatus. However, when considering the results from this study the selective nature of the cohort needs to be borne in mind.

Nature of the invagination

On eruption the invagination will contain remnants of the dental papilla or periodontal connective tissue (Kronfeld 1934). These elements become necrotic and become a nutrient-rich environment following bacterial contamination from the mouth. In mild forms, the invagination may be tear-shaped surrounded by calcified dental tissue whilst in more severe cases the lesion may give rise to a fissure that communicates with the periodontal ligament.

The invagination may also be associated with changes in the morphology of the root canal itself. One study that examined an extracted root filled invaginated tooth observed that the root canal was irregular in cross-section, with wave-like constrictions and dilatations (De Smit & Demaut 1982). There have also been reports of multiple root canals being present in association with the invagination (Walvekar & Behbehani 1997, de Sousa & Bramante 1998, Pai *et al.* 2004) (Fig. 5a–e).

Investigations into the histological, microscopical and radiographical nature of dens invaginatus have provided conflicting results. In some studies, the invaginated surface has been described as being uniform and regular with no communication with the pulp (Brabant & Klees 1956, Omnell *et al.* 1960, De Smit *et al.* 1984, Piatelli & Trisi 1993). In contrast, others have described interruptions in the invaginated surface which could potentially act as a portal for irritants to the pulp (Kronfeld 1934, Fischer 1936, Hoepfel 1936, Gustafson & Sundberg 1950, Hitchin and Mchugh 1954, Oehlers 1957a, Rushton 1958, De Smit *et al.* 1984) (Figs 1 and 6).

Kramer (1953) reported that defects in the structure of the enamel layer were restricted to the invagination, with the dentine intact but exposed (Fig. 6). He concluded that because of the absence of enamel in these areas bacterial contamination of the dentine tubules provide a direct portal for pulpal infection.

In addition, Morfis (1993) in an scanning electron microscope (SEM) microanalysis of an invagination described an alteration in the chemical structure of the enamel within the defect. The study reported the absence of any magnesium, but increased phosphate and calcium ions in comparison to the normal coronal enamel present. However, this is in contrast to the findings of Beynon (1982) who reported that the enamel and dentine surrounding an invagination was hypomineralized.

The structure of the dentine surrounding the invagination has also been reported as being irregular with connective tissue inclusions and communications towards the pulp (Atkinson 1943, Omnell *et al.* 1960, Vincent-Townend 1974, Beynon 1982, Piatelli & Trisi 1993).

The variation in findings of the nature of the invagination can possibly be explained by the different techniques used in each study, namely, histological (Piatelli & Trisi 1993, De Smit *et al.* 1984) chemical analyses (Morfis 1993), SEM (Bloch-Zupan *et al.* 1995, Stamfelj

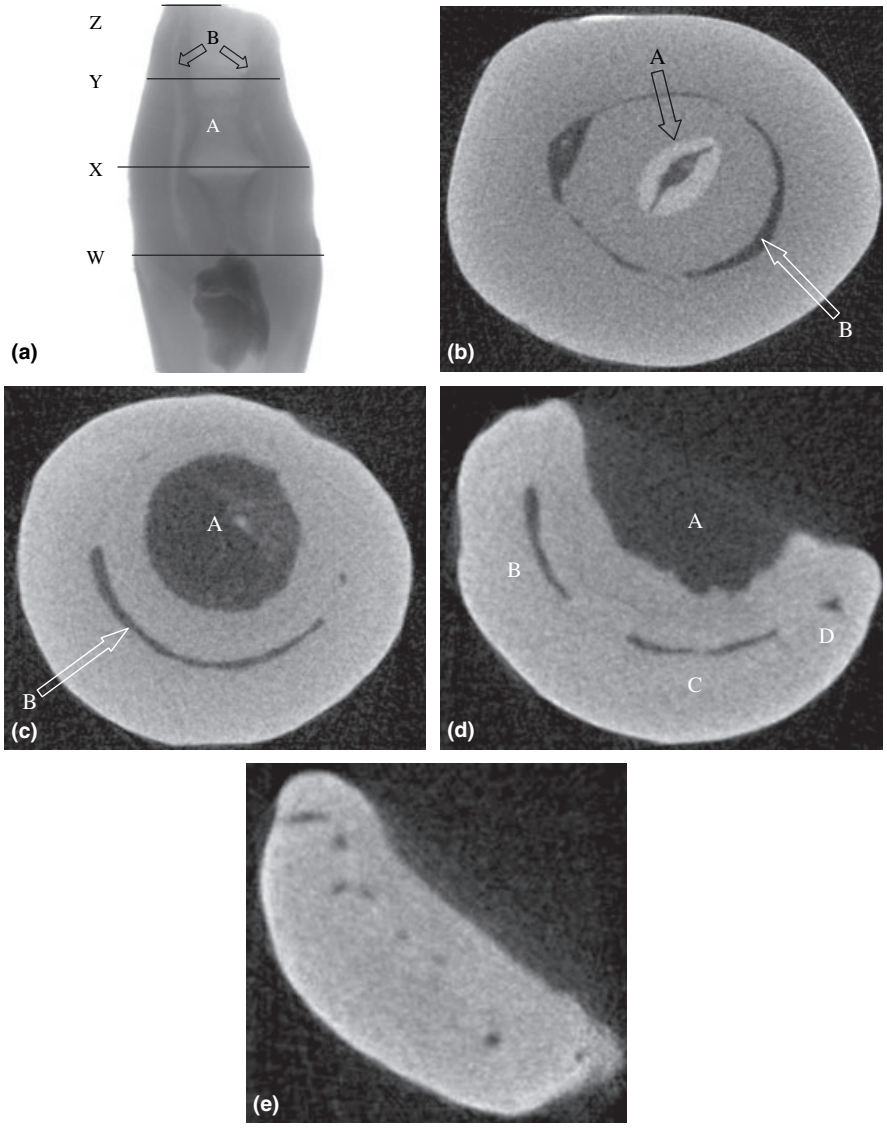

Figure 5 (A) Cross-sectional computed tomography scan of an extracted tooth with Oehlers' Type IIIB. Note the complex internal morphology of the invagination (A) and root canal system (B). (b) Axial slice of the tooth at point W. At this level the invagination is still lined by enamel (A) with the pulp almost obliterated (B). (c) Axial slice of the tooth at point X. The enamel lining has disappeared with the invagination now having a larger lumen (A) There are still some pulp remnants (B). (d) Axial slice of the tooth at point Y. The invagination has now merged with the periodontal ligament (A). Note that the pulp is now present in three different areas (B, C and D). (e) Axial slice of the tooth at point Z. The apex showing eight separate apical foramina.

et al. 2007) and microradiographic (Beynon 1982) and micro X-ray diffraction (Omnell *et al.* 1960) techniques employed. In addition, the number of teeth examined is usually small which makes definitive conclusions difficult. For example, in the study of Morfis (1993), only one tooth was examined and the histological study by De Smit *et al.* (1984) was performed on six teeth, with some of the conclusions drawn from only one of the teeth examined.

However, despite the limitations of these studies, the widely held view is that teeth affected with dens invaginatus are associated with an increased risk of developing pulpal

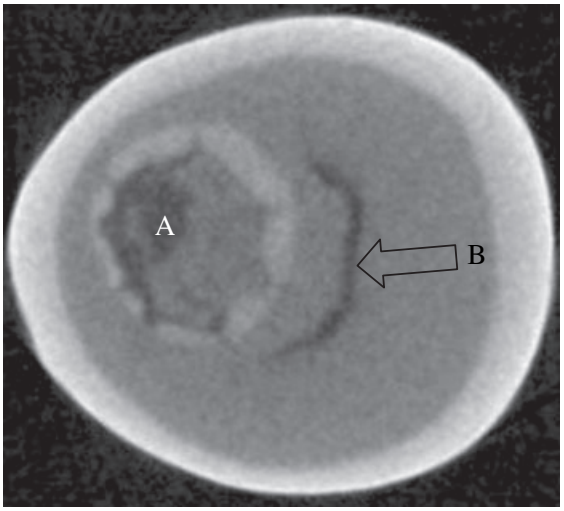

Figure 6 Axial computed tomography scan of an extracted tooth showing the invagination (a) with eight separate interruptions in the enamel layer (A). The pulp (B) is compressed by the presence of the invagination.

problems (Oehlers 1957a, Omnell *et al.* 1960, Beynon 1982, De Smit *et al.* 1984, Rotstein *et al.* 1987). This can occur without evidence of any obvious caries or history of trauma (Seow 2003). As such, those studies that have reported changes within the invagination which could increase the risk of bacterial contamination would appear to provide an explanation as to why these teeth are at more risk of developing pulpal pathosis.

However, interestingly there appears to be few studies that report the incidence of loss of vitality itself. In one study, the incidence of invaginated teeth with radiographic signs consistent with the presence of pulpal pathosis was reported as 11% of teeth (Ruprecht *et al.* 1987). However, figures based on radiographic criteria alone are likely to underestimate the true extent of the problem. Interestingly, similar results were reported by Ridell *et al.* (2001) who observed that 11.3% of teeth affected by dens invaginatus developed pulpal problems. However, in contrast to Ruprecht *et al.* (1987) the results from this second study may overestimate the incidence as all teeth had been treated prophylactically. As such, it is impossible to separate those teeth that had problems because of the active intervention from those that develop pulpal disease simply because of the presence of dens invaginatus. Other publications have referred to the possible incidence of loss of vitality, but their methods of presentation means that extrapolating accurate figures is difficult (Gotoh *et al.* 1979). The risk of pulpal complications associated with dens invaginatus is therefore probably related to the inherently poor anatomical features both on a macro and microscopic level that encourage bacterial contamination. For this reason, early diagnosis is important to prevent the need for possibly complex and difficult endodontic procedures at a later date.

Conclusion

It would appear that the exact aetiology of dens invaginatus is unknown although a genetic cause is probably the most likely factor. Equally, the prevalence of the problem is uncertain and the nature of the invagination itself is variable. The available evidence suggests that the condition is associated with an increased prevalence of pulp disease and that any necessary endodontic treatment may be difficult because of aberrant anatomy.

Clearly, there is a need for further scientific investigation of this condition. In the second paper of this series, the clinical implications of dens invaginatus are highlighted together with the diagnostic difficulties and possible treatment options.

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