

REVIEW

Taurodontism: a review of the condition and endodontic treatment challenges

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Abstract

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Taurodontism can be defined as a change in tooth shape caused by the failure of Hertwig's epithelial sheath diaphragm to invaginate at the proper horizontal level. An enlarged pulp chamber, apical displacement of the pulpal floor, and no constriction at the level of the cemento-enamel junction are the characteristic features. Although permanent molar teeth are most commonly affected, this change can also be seen in both the permanent and deciduous dentition, unilaterally or bilaterally, and in any combination of teeth or quadrants. Whilst it appears most frequently as an isolated anomaly, its association with several syndromes and abnormalities has also been reported. The

literature on taurodontism in the context of endodontics up to March 2007 was reviewed using PubMed, MEDLINE and Cumulative Index to Nursing & Allied Health Literature. Despite the clinical challenges in endodontic therapy, taurodontism has received little attention from clinicians. In performing root canal treatment on such teeth, one should appreciate the complexity of the root canal system, canal obliteration and configuration, and the potential for additional root canal systems. Careful exploration of the grooves between all orifices particularly with magnification, use of ultrasonic irrigation; and a modified filling technique are of particular use.

Keywords: endodontic treatment, enlarged pulp chamber, syndrome, taurodontism.

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Introduction

Dental morphological traits are of particular importance in the study of phylogenetic relationships and population affinities (Constant & Grine 2001). One of the most important abnormalities in tooth morphology is taurodontism. This abnormality is a developmental disturbance of a tooth that lacks constriction at the

level of the cemento-enamel junction (CEJ) and is characterized by vertically elongated pulp chambers, apical displacement of the pulpal floor, and bifurcation or trifurcation of the roots (Brkić & Filipović 1991, Hargreaves & Goodis 2002, Neville *et al.* 2002, Rao & Arathi 2006) (Fig. 1).

The term taurodontism comes from the Latin term *tauros*, which means 'bull' and the Greek term *odus*, which means 'tooth' or 'bull tooth' (Keith 1913, Terezhalmay *et al.* 2001). It was first described by Gorjanović-Kramberger (1908); however, the term taurodontism was first introduced by Sir Arthur Keith (Keith 1913) to describe molar teeth resembling those of ungulates, particularly bulls.

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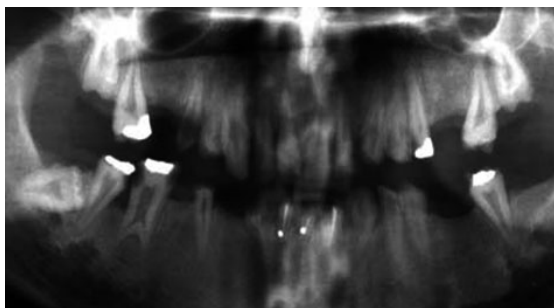


Figure 1 A taurodont tooth with enlarged pulp chamber due to the apical displacement of the furcation area (Courtesy of Dr. Tim Wright, University of North Carolina and <http://www.dent.unc.edu/research/defects/tds.cfm>).

The interest in these forms of molars first arose following the discovery of fossil remains of the Neanderthal race at Krapina in Croatia in 1899. Taurodontism is prominent amongst the Krapina Neanderthal specimens and the earliest example of taurodontism is that of a Krapina 70 000-year-old anthropological specimen (Barker 1976). Keith (1913) suggested that taurodontism is a distinctive characteristic of the Neanderthals (Fig. 2). He pointed out molars of the modern dentitions, which he called 'cynodont' (doglike teeth which have relatively small pulp chambers, set low in the crown with a constriction in outline form of the chambers at about the CEJ) could not have been



Figure 2 Maxillary molars of Pontnewydd 4 dated to Oxygen Isotope Stage 7 showing taurodontism characteristic of Neanderthals (Courtesy of the Natural History Museum, London).

evolved from such taurodont teeth. The controversy engendered by this hypothesis over the years has been vigorous (Barker 1976).

Because of the prevalence of taurodontism in modern dentitions (Shifman & Chanannel 1978, Ruprecht *et al.* 1987, MacDonald-Jankowski & Li 1993, Schalk-van der Weide *et al.* 1993, Darwazeh *et al.* 1998) and the critical need for its true diagnosis and management, this review addresses the aetiology, anatomic and radiographic features of taurodontism, its association with various syndromes and anomalies, as well as important considerations in the endodontic treatment of such teeth.

Search strategy

A literature search for relevant articles regarding endodontic management in taurodontism was performed using Ovid MEDLINE(R) In-Process & Other Non-Indexed Citations, Ovid MEDLINE(R) Daily, Ovid MEDLINE(R), and Ovid OLDMEDLINE(R), CINAHL – Cumulative Index to Nursing & Allied Health Literature, Evidence Based Medicine of Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, Database of Abstracts of Reviews of Effects, EMBASE, Health and Psychosocial Instruments, HealthSTAR/Ovid Healthstar, International Pharmaceutical Abstracts, and PubMed. Table 1 shows the keywords and combinations of the keywords used. The search was limited to holdings at the University of Toronto in English. After removing duplicates, 15 articles were retrieved and their reference lists were checked to identify any other articles relevant to the research question, which may have provided additional information. All of these were found in the original searches.

Aetiology

Taurodontism is caused by the failure of Hertwig's epithelial sheath diaphragm to invaginate at the proper horizontal level (Hamner *et al.* 1964, Terezhalmy *et al.* 2001). Interference in the epitheliomesenchymatose induction has also been proposed as a possible aetiology (Llamas & Jimenez-Planas 1993). Some reports suggest that taurodontism may be genetically transmitted (Fischer 1963, Witkop 1971, Goldstein & Gottlieb 1973), and could be associated with an increased number of X chromosomes (Gage 1978). However, other researchers have found no simple genetic association but have noticed a trend for X chromosomal

aneuploidy amongst patients with more severe forms of the trait (Jaspers & Witkop 1980).

Autosomal transmission of the trait has also been observed (Mangion 1962). These chromosomal abnormalities may disrupt the development of the tooth's form; however, a specific genetic abnormality cannot be ascribed to taurodontism (Neville *et al.* 2002). Blumberg *et al.* (1971) biometrically studied the trait, ascribed taurodontism to a polygenic system, and described the anomaly as a continuous trait without discrete modes of expression.

It is also proposed that taurodontism is a genetically determined trait and more advantageous than cynodontism in people with heavy masticatory habits [for example, the Neanderthals and Inuit (Eskimos), who prepared skins for protection from the cold by chewing] (Coon 1963) or in populations in which teeth were used as tools (Witkop 1976). Despite this theory, no evidence of taurodontism has been found in prehistoric American Indians, a group who must have also used their teeth extensively as tools (Sciulli 1977). Whilst genetic transmission can be demonstrated in most cases, other external factors can also damage developing dental structures in children and adolescents. Amongst these are infection (osteomyelitis) (Reichart & Quast 1975), disrupted developmental homeostasis (Witkop *et al.* 1988), high-dose chemotherapy (Greenberg & Glick 2003), and a history of bone marrow transplantation (Vaughan *et al.* 2005).

Diagnosis

The external features have been primarily used for the diagnosis of taurodontism. However, it should be noted that gross external characteristics are not sufficient to generate diagnosis (Mena 1971). In many cases, precise biometric methods are essential in diagnosis of taurodontism (Blumberg *et al.* 1971).

Tulensalo *et al.* (1989) examined a simple method of assessing taurodontism using orthopantomograms by measuring the distance between the baseline (connecting the mesial and distal points of the CEJ) and the highest point of the floor of the pulp chamber. They concluded that this technique is reliable in epidemiologic investigations for assessing taurodontism in a developing dentition.

Anatomic characteristics

In taurodontism, the pulp chamber is extremely large and elongated with much greater apicoocclusal height than normal (Yeh & Hsu 1999, Sert & Bayrl 2004) and, thus, extends apically below the CEJ (Keith 1913, Terezhalmay *et al.* 2001). The CEJ constriction is less marked than that of the normal tooth, giving the taurodont a rectangular shape. Also, the furcation is displaced apically, resulting in shorter roots whilst enlarging the body of the tooth (Keith 1913, Durr *et al.* 1980, Llamas & Jimenez-Planas 1993, Yeh & Hsu 1999, Terezhalmay *et al.* 2001, Sert & Bayrl 2004).

Clinical/radiographic characteristics

Clinically, a taurodont appears as a normal tooth. In fact, because the body and roots of a taurodont tooth lie below the alveolar margin, its distinguishing features cannot be recognized clinically (Terezhalmay *et al.* 2001, White & Pharoah 2004). Therefore, the diagnosis of taurodontism is usually a subjective determination made from diagnostic radiographs (Durr *et al.* 1980, Neville *et al.* 2002).

The radiographic characteristics of taurodont tooth are: extension of the rectangular pulp chamber into the elongated body of the tooth, shortened roots and root canals, location of furcation (near the root apices), despite a normal crown size (Terezhalmay *et al.* 2001,

Table 1 Search strategy

#	Search history	Results
1	(tauro or Taurodontism or taurodontic or taurodont\$ or bull tooth or cynodont or hypotaurodont or mesotaurodont or hypertaurodont or hypo-T or meso-T or hyper-T).mp. [mp = ti, ot, ab, sh, de, hw, kw, tn, dm, mf, it, rw, nm, ac, tx, ct]	995
2	(Endodontic\$ or root canal treatment or root canal therapy or pulp treatment or pulp therapy or pulpotomy or pulpectomy or pulp disease or pulp pathology\$ or root canal).mp. [mp = ti, ot, ab, sh, de, hw, kw, tn, dm, mf, it, rw, nm, ac, tx, ct]	40 962
3	1 and 2	31
4	Remove duplicates from 3	26
5	Limit 4 to English	25
6	Relevant articles remained after title/abstract screening	15

Table 2 Categorization indices for taurodontism

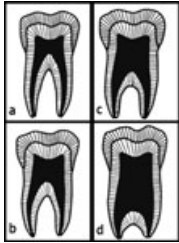
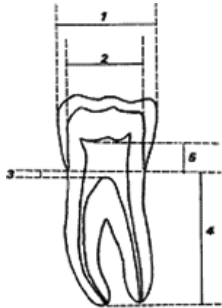
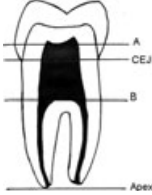
Author(s)/year	Criteria	Categories	Comments
Shaw 1928	<p>External morphological criteria (based on the relative amount of apical displacement of the pulp chamber floor)</p>  <p>a) cynodont b) hypotaurodont c) mesotaurodont d) hypertaurodont</p>	<p>Hypotaurodont: moderate enlargement of the pulp chamber at the expense of the roots</p> <p>Mesotaurodont: pulp is quite large and the roots short but still separate</p> <p>Hypertaurodont: prismatic or cylindrical forms where the pulp chamber nearly reaches the apex and then breaks up into 2 or 4 channels</p> <p>Single or pyramidal root (cuneiform): usually in the lower second molar where the pulp extends throughout the root without cervical constriction and exits via a single wide apical foramen</p>	<p>First quantitative study of taurodontism</p> <p>Using second molar as a standard tooth for determining the degree of taurodontism</p>
Keene 1966	<p>'Taurodont Index' (related the height of the pulp chamber to the length of the longest root)</p>	<p>Cynodont: index value of 0–24.9%</p> <p>Hypo-T: index value of 25–49.9%</p> <p>Meso-T: index value of 50–74.9%</p> <p>Hyper-T: index value of 75–100%</p>	<p>Relative method</p> <p>Disadvantages:</p> <ol style="list-style-type: none"> 1) Use of landmarks in biologic structures which undergo changes 2) Arbitrary selection and grading the index from 0 to 100 into 4 groups appears to be unrealistic
Blumberg <i>et al.</i> 1971	<p>Variable 1: mesiodistal diameter taken at contact points</p> <p>Variable 2: mesiodistal diameter taken at the level of the cemento-enamel junction</p> <p>Variable 3: perpendicular distance from baseline to highest point on pulp chamber floor</p> <p>Variable 4: perpendicular distance from baseline to apex of longest root</p> <p>Variable 5: perpendicular distance from baseline to lowest point on pulp chamber roof</p> 	<p>No categories provided as the authors believe that taurodontism is a continuous trait and therefore cannot be put into strict categories.</p>	<p>A biometric study</p> <p>A precise figure for each variable cannot be generally recommended because according to the race and type of molars, each variable may be different</p> <p>Taurodontism may be defined metrically</p>

Table 2 (continued)

Author(s)/year	Criteria	Categories	Comments
Shifman & Chanannel 1978	<p>Point A: lowest point at the occlusal end of the pulp chamber</p> <p>Point B: highest point at the apical end of the chamber (distance from A to B)/(distance from A to the apex of the longest root) ≥ 0.2</p> <p>Distance from B to CEJ ≥ 2.5 mm</p> 	<p>Hypo-T: 20–20.9%</p> <p>Meso-T: 30–39.9%</p> <p>Hyper-T: 40–75%</p>	<p>Advantage:</p> <p>overcome Keene's index problem by using radiographs of teeth not exhibiting reparative dentin or roots which varied morphologically</p> <p>Disadvantage:</p> <p>range of measurement of 'the distance from B to the CEJ' is small and thus subjected to error</p>

Hargreaves & Goodis 2002, White & Pharoah 2004). It should be noted that taurodontism may be masked by wear-induced secondary dentine deposition so caution should be employed in interpreting an expression of taurodontism in heavily worn molars (Constant & Grine 2001).

Categorization

Differences of opinion exist as to how much displacement and/or morphologic change constitutes taurodontism. Most authors do not provide an objective analysis of cases presented, preferring a subjective diagnosis. Another problem complicating accurate assessment of the incidence of taurodontism is the inclusion of premolar teeth by some investigators (Madeira *et al.* 1986, Llamas & Jimenez-Planas 1993, Tiku *et al.* 2003), whereas others have questioned this inclusion (Ruprecht *et al.* 1987). In classifying taurodont teeth, it is necessary to consider not only the size of the pulp chamber and roots, but also the position of the body of the tooth in relation to the alveolar margin (Mena 1971). Different categorization indices have been proposed in the literature and are listed in Table 2.

Epidemiology

Taurodontism was at first thought to be a primitive tooth form (Moorrees 1957). On the other hand, it is

found in such diverse groups as Inuit, Aleuts, Mongolians, Europeans, Scandinavians, African Americans, Chinese and white Americans amongst others (Moorrees 1957, Mjör 1972, Shifman & Buchner 1976, MacDonald-Jankowski & Li 1993, Goaz & White 1994, Darwazeh *et al.* 1998, Bäckman & Wahlin 2001, Tsesis *et al.* 2003). In the United States, most reports indicate a prevalence of 2.5–3.2% of the population (Neville *et al.* 2002).

Table 3 summarizes the reported prevalence for taurodontism from different studies. The wide range of variability of prevalence [from less than 0.1% (Pindborg 1970) to 48% (Sarr *et al.* 2000)] is most likely because of different diagnostic criteria and racial variations. Some investigators believe the alteration is more of a variation of normal rather than a definitive pathologic anomaly (Neville *et al.* 2002).

Except for a higher prevalence of taurodontism amongst females in a Chinese sample (MacDonald-Jankowski & Li 1993), no study has found a gender difference for this abnormality. Although permanent mandibular molars are most commonly affected (MacDonald-Jankowski & Li 1993, Rao & Arathi 2006), taurodontism can be seen in both the permanent and deciduous dentition [very low incidence (MacDonald-Jankowski & Li 1993, Goaz & White 1994, Darwazeh *et al.* 1998, Terezhalmay *et al.* 2001, Neville *et al.* 2002, Bhat *et al.* 2004, Rao & Arathi 2006)], unilaterally or bilaterally, and in any combination of teeth or quadrants (White & Pharoah 2004).

Table 3 Prevalence of taurodontism

Author(s)/Year	Tooth type	Most affected teeth	Least affected teeth	Population/location	Sample size	Total prevalence (%)	Method of study
Keene 1966	Molars	Mandibular 2nd molars (100%)	Other molars	Americans of European heritage	247 ^a	2.8 ^a (hypo-T)	Morphologic and biometric study
Blumberg et al. 1971	Mandibular molars	Mandibular 2nd molars (in Negroes)	Mandibular 1st molar	Negroes and white American patients	11 905 ^a	0.4 ^a (meso-T) 2.5 ^a	Biometric study
Shifman & Chanannel 1978	Molars	Mandibular 2nd molars (66%)	Maxillary 2nd and 3rd molars (0.1%)	Young adult Israeli patients	1200 ^a 10 204 ^b	5.6 ^a 1.5 ^b	Periapical and bitewing radiographs
Jorgenson et al. 1982	Deciduous and permanent molars	1st permanent molars	Deciduous molars	African American children	1074 ^a	4.37 ^a	Panoramic radiographs
Madeira et al. 1986	Premolars	Mandibular 1st premolars (0.42%)	Maxillary premolars (0%)	Mixed	4459 ^b	0.25 ^b	<i>In vitro</i> study (visual inspection and buccolingual radiographs)
Ruprecht et al. 1987	Molars	Not stated	Not stated	Saudi Arabian dental patients	1581 ^a 1647 ^b	11.3 ^a 43.2 ^b	Complete mouth and panoramic radiographs
Llomas & Jimenez-Planas 1993	Premolars	Maxillary premolars	Other premolars	Extracted premolars within a district area of Seville, Spain	379 ^b	0.7 ^b	<i>In vitro</i> study (anatomic and radiographic)
Schalk-van der Weide et al. 1993	Mandibular 1st molars	—	—	Dutch patients with oligodontia	117 ^a (with oligodontia) 91 ^a (normal)	28.9 ^a (with oligodontia) 9.9 ^a (normal)	Panoramic radiographs
MacDonald-Jankowski & Li 1993	1st and 2nd molars	Maxillary 2nd molars	Mandibular 1st molars	15–19 years old Chinese adults	196 ^a 1093 ^b	46.4 ^a 21.7 ^b	Panoramic radiographs
Darwazeh et al. 1998	All posterior teeth	Maxillary 2nd molars (31%)	Maxillary 2nd and mandibular 1st premolars (0%)	Jordanian adult dental patients	875 ^a 2636 ^b	8 ^a 4.4 ^b	Posterior periapical radiographs
Sarr et al. 2000	1st and 2nd molars	Maxillary 2nd molars	Mandibular 1st molars	150 consecutive 15–19 years old black Senegalese adults	150 ^a 1027 ^b	48 ^a 18.8 ^b	Panoramic radiographs

Bäckman & Wahlin 2001	All teeth	Maxillary and mandibular molars	-	Caucasian 7-year-olds Umea, northern Sweden in 1976	739 ^a	0.3 ^a	Biting and extraoral radiographs of the lateral side
Constant & Grine 2001	Mandibular molars	Mandibular 3rd molars	Mandibular 1st molars	Two recent population samples from southern Africa	28 ^a from Khoisan and 68 ^a from Zulu	Varies based on the definition	Lateral radiographs
Park <i>et al.</i> 2006	All teeth	Mandibular 1st molars	Not stated	Korean dental patients	1032 ^a	3.9 ^a	Clinical and radiographic examination

^aPatient.^bTeeth.

The degree of taurodontism increases from the first to the third molar (Mena 1971, Neville *et al.* 2002). Also, taurodontism is occasionally observed in mandibular premolars and even in maxillary premolars, mandibular canines, and incisors (Tennant 1966, Mena 1971, Osborn 1981).

Whilst taurodontism has been reported in premolar teeth (Tennant 1966, Mena 1971, Madeira *et al.* 1986, Llamas & Jimenez-Planas 1993, Tikunova *et al.* 2003), the true diagnosis of taurodontism in premolars cannot be ascribed *in situ* as necessary radiographs depict the tooth only in a mesiodistal orientation (Neville *et al.* 2002).

Conditions associated with taurodontism

Taurodontism appears most frequently as an isolated anomaly. However, its association with several syndromes and abnormalities has also been reported (Shifman & Buchner 1976, Genc *et al.* 1999, Yeh & Hsu 1999, Gedik & Cimen 2000). These conditions are summarized in Tables 4 and 5. Many of these disorders have oral manifestations, which can be detected on dental radiographs as alterations in the morphology or chemical composition of the teeth; thus, dentists may be the first to detect them (Witkop 1976).

Differential diagnosis

In certain metabolic conditions including pseudo-hypoparathyroidism, hypophosphatasia, and hypophosphatemic vitamin D-resistant and dependent rickets, the pulp chamber may be enlarged but the teeth are of relatively normal form (Witkop 1976, Terezhalmay *et al.* 2001, Chaussain-Miller *et al.* 2003). Another differential diagnosis is in the early stages of dentinogenesis imperfecta, where the appearance may resemble the large pulp chambers found in taurodontism (Hargreaves & Goodis 2002). Moreover, the developing molars may appear similar to taurodonts; however, an identification of wide apical foramina and incompletely formed roots helps in the differential diagnosis (White & Pharoah 2004).

Endodontic management

A taurodont tooth shows wide variation in the size and shape of the pulp chamber, varying degrees of obliteration and canal configuration, apically positioned canal orifices, and the potential for additional root canal systems. Therefore, root canal treatment becomes a

Table 4 Syndromes associated with taurodontism

Syndrome	Author(s)	Inheritance	Oral findings	Systemic findings	Prevalence of taurodontism
Down syndrome (Trisomy 21)	Jaspers 1981, Bell et al. 1989, Alpöz & Eronat 1997, Rajić & Mestrovic 1998	Additional 21 chromosome	Macroglossia Delayed eruption Absence of tooth germs	Small nose Short stature Mental retardation Muscular hypotonia	55% 36% 66% 56%
Klinefelter syndrome	Mednick 1973, Hata et al. 2001, Pinkham et al. 2005, Schulman et al. 2005	Additional X chromosome	Cleft soft palate Missing premolars Delayed development of the permanent tooth germs	Small testes Azoospermia Mental retardation Chromosome aberrations	75%
Lowe syndrome (Oculo-cerebro-renal syndrome)	Tsai & O'Donnell 1997	X-linked recessive	Severe bone loss Jaws underdevelopment Gross periodontal disease Permanent teeth Impaction Hypoplastic enamel	Cataracts Mental retardation Renal tubular dysfunction Curly hair Dense bone Skull sclerosis	Not stated
Tricho-dento-osseous syndrome (TDO)	Spangler et al. 1998, Islam et al. 2005	Autosomal dominant			Not stated
Tricho-onycho-dental syndrome (TOD)	Koshiba et al. 1978, Neville et al. 2002	Autosomal dominant	Dentin dysplasia Hypoplastic-hypomaturation enamel	Scanty hair Scanty eyebrows Thin dysplastic nails	Not stated
Williams syndrome	Axelsson 2005	Gene deletion at chromosome 7	Agenesis Wide mouth Smaller size of teeth Aberrant shape of teeth	Hoarse voice Cognitive profile Mental retardation Cardiovascular disease	1% for mandibular 1st molars and 41.7% for mandibular 2nd molars
Wolf-Hirschhorn syndrome	Breen 1998, Battaglia et al. 2001, Babich et al. 2004, Johnston & Franklin 2006	Partial deletion of the terminal portion of the short arm of chromosome 4	Microdontia Severe hypodontia Cleft lip and/or palate Taurodontic primary molars Delayed dental development	Microcephaly Growth retardation Muscle hypotrophy Feeding difficulties Learning difficulties	Not stated
Seckel syndrome	Seymen et al. 2002	Autosomal recessive	Microdontia Dental dysplasia Enamel hypoplasia	Microcephaly Small forehead Midfacial hypoplasia	Not stated
Smith-Magenis syndrome	Tomona et al. 2006	Gene deletion at chromosome 17	Tooth agenesis Root dilaceration	Posteriorly slanted ears Hoarse deep voice	87%
Mohr syndrome (Oral-facial-digital II syndrome)	Gorlin 1970, Goldstein & Gottlieb 1973, Goldstein & Medina 1974, Young et al. 2001	Autosomal recessive	Cleft palate Small tongue Notching of the upper lip	Cardiac and renal defects Polydactyly Brachydactyly Neuromuscular disturbance	Not stated

Prader-Labhart-Willi syndrome	Bassarelli <i>et al.</i> 1991	Gene deletion at chromosome 15	Enamel-dentine dysplasia	Obesity Hypotonia Oligophrenia Hypogonadism Small hands and feet Skeletal disorder Fibrous dysplasia Café-au-lait pigmentation Dwarfism Polydactyly Syndactyly	Not stated
McCune-Albright syndrome	Akintoye <i>et al.</i> 2003	Autosomal dominant	Oligodontia Malocclusion Tooth rotation Malocclusion Reduced crown size Supernumerary tooth Early eruption of teeth Shovel-shaped incisors		9%
Ellis van Creveld syndrome	Hattab <i>et al.</i> 1998, Hunter & Roberts 1998	Autosomal recessive	Anterior open bite Dental malocclusion Delayed tooth eruption Crowding of the dental arch		Not stated
Apert syndrome	Terezhalmay <i>et al.</i> 2001, Tosun & Sener 2006	Autosomal dominant	Hypodontia Micrognathia Conic-shaped incisors Agenesis of permanent teeth	Syndactyly Proptosis of eyes Mental retardation Skeletal deformities	Not stated
Lenz microphthalmia syndrome	Ersin <i>et al.</i> 2003	X-linked recessive	Hypodontia Wide spacing Delayed tooth eruption Screwdriver-shaped incisors	Microcephaly Microphthalmia Urogenital anomalies Developmental retardation Short stature Mental retardation Skeletal abnormalities Long palpebral fissures	Not stated
Kabuki syndrome	Petzold <i>et al.</i> 2003	Autosomal dominant			Not stated

Table 5 Other conditions associated with taurodontism

Abnormal condition	Author(s)	Prevalence of taurodontism
47,XXY karyotype (Additional X chromosome)	Varrela & Alvesalo 1988, Gardner & Girgis 1978	30%
47,XXX karyotype (Additional X chromosome)	Varrela & Alvesalo 1989	Direct relation with the number of X chromosomes
AIHHT	Gage 1978,	The relationship between AIHHT and taurodontism is controversial
Amelogenesis imperfecta (hypoplastic-hypomaturation with taurodontism)	Seow 1993, Winter 1996, Collins <i>et al.</i> 1999, Price <i>et al.</i> 1999, Aldred <i>et al.</i> 2002, Dong <i>et al.</i> 2005	(Winter 1996, Collins <i>et al.</i> 1999)
Oligodontia	Terezhalmay <i>et al.</i> 2001, Schalk-van der Weide <i>et al.</i> 1993, Lai & Seow 1989	29% (Schalk-van der Weide <i>et al.</i> 1993) 34% (Lai & Seow 1989)
Supernumerary teeth	Genc <i>et al.</i> 1999	Not stated
Triad of microdontia-taurodontia-dens invagination	Casamassimo <i>et al.</i> 1978, Galindo-Moreno <i>et al.</i> 2003	Not stated
Pulpal calcification	Darwazeh <i>et al.</i> 1998, Miller 1969	26% of taurodontic cases have pulpal calcification
Cleft lip or palate	Neville <i>et al.</i> 2002, Laatikainen & Ranta 1996	41%
Ectodermal dysplasia (e.g. Otodental dysplasia, Cranioectodermal dysplasia, and Rapp-Hodgkin syndrome)	Neville <i>et al.</i> 2002, Jaspers & Witkop 1980, Glavina <i>et al.</i> 2001, Levin <i>et al.</i> 1975, Chen <i>et al.</i> 1988, Zannolli <i>et al.</i> 2001	Not stated
Osteoporosis	Fuks <i>et al.</i> 1982	Not stated
Thalassaemia major	Hazza'a & Al-Jamal 2006	34%
Dwarfism	Neville <i>et al.</i> 2002, Terezhalmay <i>et al.</i> 2001, Gardner & Girgis 1977, Sauk & Delaney 1973, Stewart <i>et al.</i> 1971	Not stated
Dyskeratosis congenita	Terezhalmay <i>et al.</i> 2001	Not stated
Epidermolysis bullosa	Terezhalmay <i>et al.</i> 2001	Not stated

challenge (Hargreaves & Goodis 2002, Tsisis *et al.* 2003, Rao & Arathi 2006). Moreover, whilst the radiographic feature of a taurodont tooth is characteristic, pre-treatment radiographs produce little information about the root canal system (Yeh & Hsu 1999). Finally, the results of pulp testing contribute little information about the effect of a large pulp chamber on tooth sensitivity (Durr *et al.* 1980).

There are different views regarding access cavity design and preparation: Shifman & Buchner (1976) argued that access to the root canal orifices can easily be obtained as the floor of the pulp chamber cannot be affected by the formation of reactionary dentine as in normal teeth. In contrast, Durr *et al.* (1980) suggested that morphology could hamper the location of the orifices, thus creating difficulty in instrumentation and filling.

Each taurodont tooth may have extraordinary root canals in terms of shape and number. A complicated root canal treatment has been reported for a mandibular taurodont tooth with five canals, only three of which could be instrumented to the apex (Hayashi 1994). Therefore, careful exploration of the grooves between all orifices, especially with magnification (Tsisis *et al.* 2003), has been recommended to reveal additional orifices and canals (Yeh & Hsu 1999).

Because the pulp of a taurodont is usually voluminous, in order to ensure complete removal of the necrotic pulp, 2.5% sodium hypochlorite has been suggested initially as an irrigant to digest pulp tissue (Prakash *et al.* 2005). Moreover, as adequate instrumentation of the irregular root canal system cannot be anticipated, Wideman & Serene (1971) suggested that

additional efforts should be made by irrigating the canals with 2.5% sodium hypochlorite in order to dissolve as much necrotic material as possible. Application of final ultrasonic irrigation may ensure that no pulp tissue remains (Prakash *et al.* 2005).

Because of the complexity of the root canal anatomy and the proximity of the buccal orifices, complete filling of the root canal system in taurodontism is challenging. A modified filling technique has been proposed, which consists of combined lateral compaction in the apical region with vertical compaction of the elongated pulp chamber, using the system B device (EIE/Analytic Technology, San Diego, CA, USA) (Tsisis *et al.* 2003).

Another endodontic challenge related to taurodonts is intentional replantation. The extraction of a taurodont tooth is usually complicated because of a dilated apical third (Yeh & Hsu 1999). In contrast, it has also been hypothesized that because of its large body, little surface area of a taurodont tooth is embedded in the alveolus. This feature would make extraction less difficult as long as the roots are not widely divergent (Durr *et al.* 1980). Finally, it should be noted that in cases of hypertaurodont (where the pulp chamber nearly reaches the apex and then breaks up into two or four channels) vital pulpotomy instead of routine pulpectomy may be considered as the treatment of choice (Shifman & Buchner 1976, Neville *et al.* 2002).

For the prosthetic treatment of a taurodont tooth, it has been recommended that post-placement be avoided for tooth reconstruction (Tsisis *et al.* 2003). Because less surface area of the tooth is embedded in the alveolus, a taurodont tooth may not have as much stability as a cynodont when used as an abutment for either prosthetic or orthodontic purposes (Durr *et al.* 1980).

From a periodontal standpoint, taurodont teeth may, in specific cases, offer favourable prognosis. Where periodontal pocketing or gingival recession occurs, the chances of furcation involvement are considerably less than those in normal teeth because taurodont teeth have to demonstrate significant periodontal destruction before furcation involvement occurs (Shifman & Buchner 1976, Neville *et al.* 2002).

Conclusion

This review attempts to address the aetiology, anatomic and radiographic features of taurodontism, its association with some syndromes and anomalies, as well as important considerations in the endodontic treatment

of such teeth. Taurodont teeth show wide variations in the size and shape of pulp chambers, varying degrees of obliteration and canal complexity, low canal orifices, and the potential for additional root canal systems. On the basis of the evidence presented here, it can be seen that taurodontism has hitherto received insufficient attention from clinicians. In performing root canal treatment on these teeth, one should appreciate the complexity of the root canal system. Careful exploration of the grooves between all orifices, particularly with magnification; ultrasonic irrigation; and a modified filling technique are recommended. No long-term follow-up studies have been published regarding endodontically treated taurodont teeth.

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