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REVIEW

Pulp stones: a review

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Abstract

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Pulp stones are a frequent finding on bitewing and periapical radiographs but receive relatively little attention in textbooks. A review of the literature was therefore performed, initially using the PubMed database and beginning the search with 'pulp calcifications' and 'pulp stones'. Each term provided more than 400 references, many of which related to pulp calcification in general rather than pulp stones, and focussed largely on the problems these changes presented to clinicians. A manual search using references from this source was carried out. Contemporary textbooks in endodontology were also consulted, and an historic perspective gained from a number of older books and references. The factors involved in the development of the pulp stones are largely unknown. Further research may determine the reasons for their formation, but with current endodontic instruments and techniques this is unlikely to alter their relevance to clinicians.

Keywords: dental radiography, endodontics, pulp stones.

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Introduction

Pulp stones are discrete calcifications and are amongst changes that include more diffuse pulp calcifications such as dystrophic calcification. Stones may exist freely within the pulp tissue or be attached to or embedded in dentine (Johnson & Bevelander 1956). Two types of calcified bodies in the dental pulp have been described (Moss-Salentijn & Klyvert 1983): denticles possessing a central cavity filled with epithelial remnants surrounded peripherally by odontoblasts, and pulp stones being compact degenerative masses of calcified tissues. A single tooth may have from 1 to 12 or even more stones, with sizes varying from minute particles to large masses which occlude the pulp space (Johnson & Bevelander 1956). They are reported to occur more often in the coronal region but are also found in the radicular pulp (Arys *et al.* 1993). Pulp stones have been noted in patients with systemic or genetic diseases such as dentine dysplasia, dentinogenesis imperfecta and in certain syndromes such as Van der Woude syndrome (Kantaputra *et al.* 2002). Despite a number of microscopic and histochemical studies, the exact cause of such pulp calcifications remains largely unknown. Apart from the obvious endodontic problem of hindering access to root canals and their subsequent shaping (Ibarrola *et al.* 1997), it is not known whether they are of any other significance. Thus, this review will consider pulp calcification in general, and then focus on pulp stones.

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Age changes and pulpal calcifications

With age the pulp spaces of teeth decrease in size through the deposition of secondary and tertiary dentine. When tooth wear, caries or operative intervention is a feature this process becomes more evident. In most pulps, dystrophic calcification is found to be of a variable degree, and even in teeth without caries or restorations scattered calcification occurs, unrelated to

disease. A study of teeth obtained from individuals ranging from 15 to 75 years found not only a decrease in the size of the pulp chamber due to deposition of secondary dentine with increasing age, but also a progressive deposition of calcified masses that originated in the root pulp (Bernick & Nedelman 1975). This confirmed the earlier work that registered calcification in 90% of teeth from people more than 40 years, mainly involving apically located blood vessels (Bernick 1967a). A second report from the same study using the same material histologically demonstrated that the calcification process also involved the nerve tissue (Bernick 1967b). Initially, discrete isolated regions of calcification occur in the endoneurium and/or the perineurium. The calcifying process, however, soon becomes circumferential, forming a calcified ring around the nerve. The nerve fibre and its fasciculi are then impregnated, resulting in nerve obliteration (Bernick 1967b). Bernick & Nedelman (1975) reported an increase in the number of collagenous bundles in old coronal pulps that were associated with the connective tissue sheaths of blood vessels and nerves. At no age were thick collagen fibres seen independent of the connective tissue sheaths. Furthermore, the collagen bundles of vascular and neural sheaths of old pulps were the loci for calcification. As a result of calcification of the blood vessels and nerves in the pulp, their numbers decrease. The persistence of the connective tissue sheaths of nerves and blood vessels gives the pulp a histologically fibrotic appearance.

As part of the pulp ageing process there is also a considerable decrease in the number of cells (fibroblasts, odontoblasts and mesenchymal cells), with the cell density decreasing by half from 20 to 70 years (Ketterl 1983). At the same time, fibrous tissue accumulation occurs to the point where almost nothing exists except the fibrous tissue. This is termed fibrous degeneration or pulp atrophy. It is different from fibrous replacement (such as the replacement of infarcted heart muscle tissue) where the fibrous connective tissue contains viable fibroblasts (Morse 1991). Some authors also believe that fat deposits occur in the pulp with age, and that calcification commonly occurs within these deposits (Seltzer 1972), but this may be a tissue-processing artefact (Seltzer & Bender 1984).

Types and formation of pulp stones

Pulp stones can be structurally classified and based on location (Seltzer & Bender 1984). Structurally, there are true and false pulp stones; the distinction being morphological (Table 1). A third type, 'diffuse' or 'amorphous' pulp stones, is more irregular in shape than false pulp stones, occurring in close association with blood vessels (Mjör & Pindborg 1973). True pulp stones are made of dentine and lined by odontoblasts, whereas false pulp stones are formed from degenerating cells of the pulp that mineralize. Such mineralization occurs in stages; initially cell nests become enclosed by concentrically arranged fibres (i.e. an organic phase precedes mineralization) which then become impregnated with mineral salts. Calcified increments are then added (Johnson & Bevelander 1956).

Based on location, pulp stones can be embedded, adherent and free (Fig. 1). Embedded stones are formed in the pulp but with ongoing physiological dentine formation they become enclosed (sometimes fully) within the canal walls (Philippas 1961). They are found most frequently in the apical portion of the root, and the presence of odontoblasts and calcified tissue resembling dentine can occur on the peripheral aspect of these stones (Johnson & Bevelander 1956). Adherent pulp stones are simply less attached to dentine than embedded pulp stones; the difference between adherent and embedded can be subjective, but adherent stones are never fully enclosed by dentine. Adherent and embedded pulp stones can interfere with root canal treatment if they cause significant occlusion of canals or are located at a curve. They may also become

Table 1 Terminology

Pulp stone	True	Made of dentine and lined by odontoblasts.
	False	Formed from degenerating cells which mineralize.
	Free	Stone not related to pulp space wall, surrounded by soft tissue.
	Adherent	Stone attached to wall of pulp space, not fully enclosed by dentine.
	Embedded	Stone enclosed within canal wall, less attached than the above.
Denticle		An alternative term for pulp stone, more usually a calcification filled with epithelial remnants surrounded by odontoblasts.
Fibrodentine		Material produced by fibroblast-like cells against dentine prior to differentiation of a new generation of odontoblast-like cells.
Dystrophic calcification		Inappropriate biomineralization of the pulp in the absence of mineral imbalance.

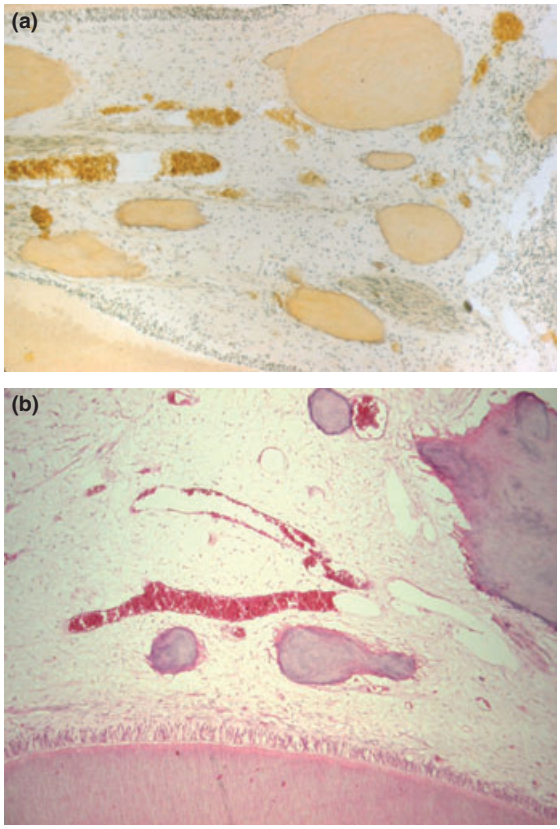

Figure 1 (a) Pulp stones of variable size within the dental pulp (reproduced with permission from Kardos & Kieser 2006). (b) Pulp stone (haematoxylin and eosin stain).

dislodged. Free pulp stones are found within the pulp tissue proper and are the most commonly seen type on radiographs (Fig. 2). They are very common and vary in size from 50 μm in diameter to several millimetres

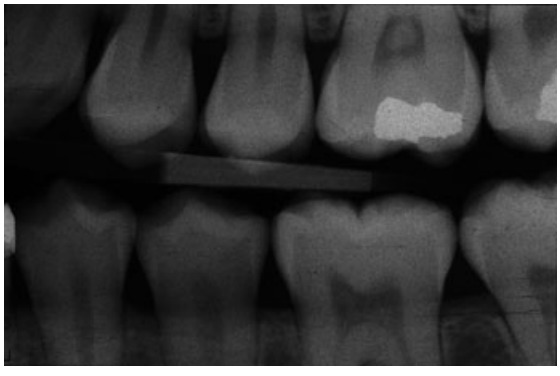

Figure 2 Bitewing radiograph of a 26-year-old female showing pulp stone in the maxillary left first molar.

when they may occlude the entire pulp chamber (Sayegh & Reed 1968). Stones can be further subdivided into those with distinct concentric laminations and those without distinct laminations. Laminated pulp stones are not usually associated with smaller pulp stones, whereas nonlaminated stones are rougher and may have smaller stones attached to their surfaces (Appleton & Williams 1973). This is in agreement with Pashley & Liewehr (2006) who histologically recognized two types of stones: those that are round or ovoid, with smooth surfaces and concentric laminations; and those that assume no particular shape, lack laminations and have rough surfaces.

The formation of pulp stones is still something of an enigma (Table 2). Studies show that a high frequency of cell islands, considered to be of epithelial origin, were observed together with pulp stone formation in teeth that had been subjected to experimental intrusion (Stenvik & Mjör 1970a, b, 1971). The experimental material comprised 25 teeth that had been intruded for 5–28 days with a 50–250 *g* force and then left *in situ* for 4–104 days after the force was removed. When present, more than one island was present for each of the teeth, with the cells having a similar appearance to those of Hertwig's epithelial root sheath (HERS). Radially arranged cells were found in the cell islands with matrix formation occurring adjacent or surrounding these cells. All of the pulp stones in their material had centrally entrapped cells. While this shows that the remains of HERS may induce pulp stone formation, the relationship to orthodontic treatment may be related to the stage of root formation, because root development was affected when root formation had not been completed at time of intrusion. The islands of epithelial-like cells were considered to be fragments of HERS which had been disrupted as a result of the intrusion. Sübay *et al.* (2001) investigated 40 teeth subjected to extrusive forces of approximately 75 *g* and extracted after 10 or 40 days. Their results showed no correlation between extrusion and pulp stone formation.

Other work on epithelia-induced pulp stone formation was carried out by Moss-Salentijn & Klyvert (1983). In their study, 85 maxillary and 90 mandibular newly erupted premolars from children aged 11 to 15 were extracted and investigated, primarily by buccolingual and mesiodistal radiographic projections. Selected material was histologically examined, and both true and false pulp stones identified. False pulp stones consisted of a mixture of tubular dentine (orthodentine) and atubular calcified tissue (fibrodentine) that surrounded one or several dense, irregularly

Table 2 Possible causative factors

Investigator	Possible causative factor	Methodology	Teeth (*n*)	Age of subjects (yr)
Rubach & Mitchell (1965)	Periodontal disease (not related to bone loss)	Histology	74	not stated
Sayegh & Reed (1968)	More calcification in carious teeth than noncarious, ages 10–34	Histology	591 (permanent and some primary teeth, numbers not stated)	6–63
Sundell *et al.* (1968)	Class *V* restorative procedures-weak association to post-operative interval	Serial sections	470	means 35.2 to 41.7
Holtgrave *et al.* (2001)	Fluoride prophylaxis, but duration not significant	Light microscopy	Primary molars; 24 experiments/17 controls	8–14
Edds *et al.* (2005)	Cardiac disease pilot study-increased incidence	Periapical radiographs, 55 patients	Not stated	20–55

calcified, basophilic cores. False pulp stones were found in coronal as well as in radicular pulp tissue and were often associated with dystrophic calcification. True pulp stones consisted of odontoblasts lining a shell of tubular dentine which surrounded a central cavity filled with cell remnants. In developing pulp stones the enclosed cell remnants resembled other isolated epithelial cell remnants. In teeth where root development was not complete, pulp stones nearest the apical end had a characteristic thimble shape with the open end facing apically. True, free pulp stones were abundant in teeth whose root development was not complete, whereas in teeth with complete root development most denticles were attached or embedded in the dentine wall. Where true pulp stones remained free in mature teeth, they were no longer thimble-shaped but surrounded fully by a dentine shell. Furthermore, the enclosed epithelial cells had undergone degeneration. These descriptions of true pulp stones are similar to those of Stenvik & Mjör (1970a,b, 1971). Moss-Salentijn & Klyvert (1983) suggested that true pulp stone formation may be limited in time to the period of root formation, and in location to the radicular pulp and furcation areas of multi-rooted teeth. They also felt that because formation of tubular dentine occurred in both true and false pulp stones, it reflected the ability of cells in the immature pulp tissue to undergo differentiation into odontoblasts for a limited period of time. These authors recognized that in older teeth, pulp stones consist entirely of atubular dentine or tubular dentine surrounded by fibrodentine. Thus, the response of the pulp tissue to an inductive stimulus may be age-dependent. On this basis, Moss-Salentijn & Klyvert (1983) questioned the true or false classification of pulp stones, preferring instead to make a distinction between calcified bodies in the pulp according to their mode of genesis. True pulp stones were called denticles, which form after an inductive interaction between epithelium and pulp tissue, whereas false pulp stones were simply called pulp stones, which form around foci of calcified components. Unfortunately, such a classification is difficult to apply because the mode of genesis may not always be clear, particularly in older teeth. Moreover, in older teeth denticles will often be surrounded by nontubular fibrodentine (resulting in a regular, laminated pattern) while pulp stones may still contain orthodentine (Moss-Salentijn & Hendricks-Klyvert 1988). In other words, the classification can still paint an erroneous picture; worse, the two terms have traditionally been interchangeable. Although it is relevant to state that the presence or absence of tubules should not be the sole classifying factor (Le May & Kaqueler 1991), the traditional morphologic classification in a broader sense is still preferable, with the realization that there will probably be a mixture of orthodentine and fibrodentine in both types of pulp stones.

Various mechanisms for inducing fibroblasts in the pulp to produce dentine or dentine-like tissue have been suggested (Weinreb & Michaeli 1984). Cultured pulp fibroblasts from human deciduous and supernumerary teeth formed crystals with an X-ray diffractometry pattern consistent with hydroxyapatite (Tsukamoto *et al.* 1992), showing that fibroblasts themselves could produce calcific changes. Moreover, with the addition of β-glycerophosphate to Eagle's culture medium, pulp

cells from third molar germs began to develop odontoblastic features including the formation and the subsequent hydroxyapatitic mineralization of type I collagen-rich matrix (Couble *et al.* 2000). When human dental pulp stem cells were transplanted into immunocompromised mice, they generated a dentine-like structure lined with human odontoblast-like cells that surrounded a pulp-like interstitial tissue (Gronthos *et al.* 2000). Other research using a rat model has shown mineralization by dental pulp-derived cells (Hayashi *et al.* 1993), as well as the potential for dental pulp-derived cells to differentiate into osteoblast-like cells (Ueno *et al.* 2001). Unidentified mesenchymal stem cells and pericytes are possible cell lines which might differentiate into odontoblast-like cells under suitable inductive signals.

Electron microscopy has shown that in the pulp tissue there can be many foci of calcification 1 μm or less in diameter, either as smooth-surfaced spherical clusters or closely packed layers around collagen fibres or as intracellular deposits (Appleton & Williams 1973). The smooth-surfaced spherical clusters often appeared to push the collagen fibres to one side, with the actual crystallites being closely packed and varying in size. Surrounding these spherical pulp stones was an electron dense narrow band, which suggested gradual deposition of mineral at the surface (i.e. no fibrous matrix). Some early foci, however, contained fine needle-like crystallites which were closely associated with the matrix collagen. These measured in the region of 50Å in width and 1,000Å in length. Following demineralization using EDTA it was apparent that these crystallites were embedded in an electron dense granular material coating small groups of collagen fibres. This is a similar appearance to that seen in early bone formation.

In the case of intracellular (fibroblast) deposition of crystallites, these appeared to be enclosed within the mitochondrial membrane. The crystallites were again needle-like, but measured approximately 25Å in width and 500Å in length. Both the crystallites surrounding the collagen fibres and the intracellular crystallites gave a similar diffraction pattern, typical of a hydroxyapatite-like material. Calcification that takes place within the mitochondria of some cells can be a reflection of a local metabolic change within the cells and may be related to minor circulatory disturbances in the pulp vessels (Saunders 1966). The initial action is likely to be the formation of amorphous calcium phosphate, which can be accumulated in the mitochondrial matrix (Wuthier *et al.* 1985). Variation in size and morphology of the crystallites composing the pulp stones may represent different forms of calcium phosphate (Schroeder 1965).

Large, free pulp stones, which appeared spherical in outline and had concentric laminations following demineralization, had an electron dense central zone surrounded by a less dense peripheral zone about 2–3 μm in width. The central zone consisted of closely packed crystallites approximately 50Å × 300Å associated with closely packed collagen fibres visible in the background. The collagen fibres in the matrix were coated with an electron-opaque material. Therefore, the large laminated pulp stones seemed to develop by the deposition of concentric layers of collagen fibres which then become mineralized (Appleton & Williams 1973).

Le May & Kaqueler (1993) used electron probe micro-analysis to investigate the mineral composition of human pulp stones. The stones were composed of two major elements: calcium and phosphorus. The average concentrations were 32.1% and 14.7%, respectively, resulting in a calcium/phosphorus weight ratio of 2.19, very close to the 2.15 of pure hydroxyapatite. Other elements included fluorine (0.88%), sodium (0.75%) and magnesium (0.51%). Potassium, chlorine, manganese, zinc and iron were present at trace concentrations.

Recent work has investigated the organic matrix component of human pulp stones (Ninomiya *et al.* 2001). Two free pulp stones from the centre of pulp cavities were demineralized, serially sectioned and subjected to immunohistochemical procedures using specific antibodies to type I collagen and noncollagenous proteins (osteopontin, osteonectin and osteocalcin). Type I collagen was evenly located throughout the pulp stones, showing that it is a major matrix component of free pulp stones. Given that the majority of the stones were fibrodentine, it is likely that nonodontoblastic pulp cells were responsible for the collagen. Furthermore, while osteonectin and osteocalcin were not detected, strong immunostaining of osteopontin in the peripheral area of the pulp stones suggested that it plays an integral part in the calcification front, and that it has come from less differentiated pulp cells. This is because osteocalcin usually expresses in mature osteoblasts, and generally osteopontin expression precedes that of osteocalcin (Sodek *et al.* 1995, Ueno *et al.* 2001). In respect of osteopontin, these immunohistochemical findings are similar to those from atherosclerotic plaques and urinary stones (Hirota *et al.* 1993, Kohri *et al.* 1993). Osteopontin produced

by macrophages also plays a significant role in the development of calcifying foci within the necrotic area of breast cancers (Hirota *et al.* 1995). These findings lend weight to the possibility of pulp stones being pathological to some degree.

Primary teeth and pulp stones

Few studies deal with pulp stones in the primary dentition. A radiographic study of pulp calcification in primary teeth reported that of the 120 teeth studied only 7 (1 central incisor and 6 second molars) had radiographic bodies within their pulp chambers regarded as pulp stones (Kumar *et al.* 1990). It was not stated if the stones were in maxillary or mandibular teeth. Yaacob & Hamid (1986) also examined 120 primary teeth from 95 patients aged between 3 and 11. The teeth included 6 central incisors, 16 lateral incisors, 20 canines, 38 first molars and 40 second molars. All teeth were serially sectioned, stained with haematoxylin and eosin and examined by light microscopy. Only 6.7% of the teeth had pulp stones, with 11.7% having some diffuse calcification. Given that the size of the pulp stones ranged from 0.05 to 3.3 mm, it is likely that the radiographic prevalence of pulp stones would be very low. Coupled with the absence of any true pulp stones, this gives credence to the view that the prevalence and complexity of pulpal calcification increases with age and has a large physiological component (Bernick & Nedelman 1975). In contrast, Arys *et al.* (1993) found that age did not have any influence on the occurrence of pulpal calcifications. Their material consisted of 42 primary molars, with less than one-third of their roots resorbed, from 42 healthy children of both sexes, aged from 5 to 13. There were 23 untreated carious teeth, 14 treated with amalgam restoration and 5 caries-free teeth. The teeth were examined by microradiography and light microscopy, and results revealing that pulp stones were present in 78% of the molars, with 95% of the material showing some form of pulpal calcification. There was a lower incidence of pulp stones in treated and carious molars, which might be related to tertiary dentine formation or the low number of caries-free teeth. Further, in contrast to the Yaacob & Hamid (1986) study, free or attached pulp stones were the most common type of calcification (as opposed to diffuse).

Saad (1997) looked at regressive changes in the pulps of retained primary molars with congenitally missing successor teeth. His material comprised 17 intact, caries-free primary molars that were histologically examined after extraction (ages of patients were not provided). Generally, the results showed a reduction in pulp size, declining vascularity and abnormal odontoblastic patterns, pulp degeneration and pulp stone formation. Pulp stones were free, attached or embedded and in both the coronal and root portions of the pulp. Some completely obliterated the coronal portion of the pulp. Pulp stone percentages were not given. Once again these changes appear to be of a physiological nature, confirming work on permanent teeth (Bernick & Nedelman 1975). Holtgrave *et al.* (2001) found a correlation between extensive pulp calcification and post-natally initiated fluoride prophylaxis, although the duration of tablet fluoridation (1–10 years) had no statistically significant influence on pulp calcification.

Other associations and prevalence (Table 3)

Sundell *et al.* (1968) examined the relationship between coronal pulp stone formation and experimental operative procedures. Their study was based on the microscopic examination of 470 serially sectioned teeth. In all teeth, Class *V* cavities were prepared under local anaesthesia using a variety of low- and high-speed cutting techniques, coolants and restorative materials. Teeth were divided into eight groups depending on the post-operative extraction time (0 to over 50 days). Their results revealed that pulp stone formation was most common perivascularly. However, in some specimens calcific nodules were found in the lumina of vessels. This might indicate that pericytes or even multi-potent stem cells derived from blood could be the source of hard-tissue forming cells in the dental pulp. Of various factors that underwent correlation analyses, the most promising relationship was between calcific nidi and the post-operative interval (correlation coefficient 0.2), rather than the traumatic potential of the restorative procedure. Although this is a very weak association and does not prove causation, if capillary thromboses or vascular wall damage as a result of operative procedures were to mineralize, then pulp stone formation could occur. A longer follow-up period with fewer initial variables might result in clearer associations.

Sayegh & Reed (1968) examined 591 teeth histologically from a group which included permanent and some primary teeth (numbers not stated). The incidence of calcification in carious teeth from these children and young adults (10–34 year-olds) was nearly five times than that in noncarious teeth (36

Table 3 Prevalence in some large surveys

Investigator	Methodology	Sample (*n*)	Age of subjects (yr)	Prevalence %
Yaacob & Hamid (1986)	Histology	120 teeth, 95 patients	3–11	6.7%
Kumar *et al.* (1990)	Radiographs (extracted teeth)	120 primary teeth	not stated	6% central incisor 25% second molar
Arys *et al.* (1993)	Microradiography and light microscopy	42 teeth from 42 children	5–13	78% of molars
Saad (1997)	Histology	17 primary molars (congenitally missing successors)	–	–
Tamse *et al.* (1982)	Full mouth periapical surveys (including bitewings)	150 males, 150 females	20–40	20.7%
Hillmann & Geurtsen (1997)	Histology	332 permanent teeth	10–72	3% 10–30 yrs 13% others
Baghdady *et al.* (1988)	Radiography, 6228 premolars and molars	2880 from males, 3348 from females	12–13	19.2%
Hamasha & Darwazeh (1998)	Radiography	814 patient records, 73 teeth	18–60	22.4%
Ranjitkar *et al.* (2002)	Bitewings	217 subjects, 3296 teeth	17–35	10.1%
Chandler *et al.* (2003)	Bitewings	121 subjects, 445 first molars	18–25	4%

vs. 8% respectively). This difference was not present in older adults (over 45), which supports the theory that pulp calcification is, under normal conditions, a physiological process. Under pathological conditions, however (e.g. caries), the process may speed up and this correlates to the concept of an organism's gradual dysfunction with increasing age. Thus, it can be difficult to draw a line between a physiological and a pathological process in old age. The influence of caries on pulp stone formation may actually be related to properties of dentine such as the number and dimensions of tubules, and the progression rate and activity of the disease. These would influence the rate of bacterial and/or bacterial toxin penetration. Examining the other major oral disease Rubach & Mitchell (1965) attempted to correlate periodontal condition with pulp stone formation and concluded that neither pulp stones nor diffuse calcifications were related to bone loss.

Other suggested correlations to pulp stones have been plethoric as opposed to anaemic personalities (Kirk 1893), metabolic imbalance or dysfunction (Norman & Johnstone 1921) and orthodontic treatment and traumatic occlusion (Shroff 1955). While Stafne & Szabo (1933) found no definite relationship between pulp stones and cholelithiasis, renal lithiasis, gout, hypercementosis, migraine or torus linguae/palatinus, they did find a stronger (albeit only suggestive) correlation with the presence of arteriosclerosis, osteitis deformans and acromegaly. Although this is a dated study, case reports exist where (generalized) pulp stones are found in the dentitions of individuals with various conditions. These include tumoral calcinosis (Burkes *et al.* 1991), dentine dysplasia type II (Diamond 1989, Dean *et al.* 1997), Saethre-Chotzen syndrome (Goho 1998), elfin facies syndrome (Kelly & Barr 1975), familial expansile osteolysis (Mitchell *et al.* 1990), Ehlers Danlos syndrome type I (Hollister 1978, Pope *et al.* 1992), osteogenesis imperfecta type I (Lukinmaa *et al.* 1987, Levin *et al.* 1988) and otodental syndrome (Sedano *et al.* 2001). Unusual cases of idiopathic generalized pulp stone formation have been reported (Weiss 1927, Hitchin 1936, Siskos & Georgopoulou 1990), although sometimes a genetic predisposition has been noted (Rao *et al.* 1970, VanDenBerghe *et al.* 1999). A pilot study of correlations of pulp stones with cardiovascular disease demonstrated that patients with cardiovascular disease have an increased incidence of pulp stones (Edds *et al.* 2005).

Many prevalence studies have identified pulp stones using radiographic criteria. The true prevalence is likely to be higher than figures from these studies, because pulp stones with a diameter smaller than 200 μm cannot be seen on radiographs (Moss-Salentijn & Klyvert 1983). Furthermore, in histological observations the limited number of sections through each tooth may result in underreporting (Willman 1934).

Tamse *et al.* (1982) examined the full-mouth radiographic surveys (which included bitewing and periapical radiographs) of 150 male and 150 female patients aged 20 to 40, viewing 1380 mandibular premolars and molars (679 in females and 701 in males). Medical histories were noncontributory. Their results showed that 20.7% of the teeth had pulp stones, as defined by the criterion of a definite radiopaque mass being visible

within the pulp chamber. A far higher prevalence of pulp stones was observed in molars compared with premolars; 45.2% and 6.1%, respectively, for males and 65.9% and 5.8%, respectively, for females. In females, pulp stones were found in 24.7% of teeth compared to 16.9% in males. This was a significant difference ($P < 0.0001$) and confirmed the study of Stafne & Szabo (1933). However, no significant difference was found between the existence of pulp stones and the condition of the crown of the tooth (intact, carious or restored). It was also reported that no significant difference was found between periapical and bitewing radiographs in disclosing pulp stones.

Baghdady *et al.* (1988) studied 515 healthy 12–13-year-olds from a middle-class district in Baghdad. A total of 6228 maxillary and mandibular premolars and molars were radiographically evaluated; 2880 in males and 3348 in female patients. Results showed that 19.2% of the teeth contained pulp stones; 18.8% in the female group and 19.8% in the male. This difference was not statistically significant. The mandibular teeth had a significantly higher number of pulp stones than the maxillary teeth, 20.02% vs. 18.21% respectively ($P < 0.03$). The mandibular (53.4%) and maxillary (48.9%) first molars had far higher percentages of pulp stones than second molars and premolars. No significant difference was found between intact teeth and carious teeth in the number of pulp stones.

Hamasha & Darwazeh (1998) examined patient records of 814 Jordanian adults (59.2% male and 40.8% female), aged 18–69, with a total of 4573 teeth. Pulp stones (in one or more teeth) were present on radiographs in 51.4% of the patients; 60% in males and 40% in females. There was no significant difference between the genders. Pulp stones were present in 22.4% of the teeth studied, with the first molar affected 42% of the time and the second molar 32% of the time. Incisors and canines (particularly in the mandible) were the least affected (5.5%). The study noted a high incidence of pulp stones associated with conditions such as dilacerations, impactions, taurodontism and enamel pearls. In another study by the same authors investigating the prevalence of taurodontism in Jordanian adults (8% of patients and 4.4% of teeth studied), 26.7% of the taurodont teeth had pulp stones or calcifications (Darwazeh *et al.* 1998).

Ranjitkar *et al.* (2002) examined the prevalence of pulp stones in an Australian population. The study sample included 3296 teeth identified in bitewing radiographs of 217 undergraduate dental students, aged between 17 and 35. The sample comprised 56.7% males and 43.3% females. Pulp stones were found in 46.1% of the subjects and 10.1% of the teeth examined. There was a significant difference between occurrences in molars (19.7%) as opposed to premolars (0.4%), and first molars (27.5%) as opposed to second molars (11.9%). In the case of the first molars, the occurrence was significantly higher in maxillary teeth (34.4%) as opposed to mandibular (20.3%). Maxillary right first molars that were restored and/or carious had significantly higher occurrences of pulp stones (41.7%) as opposed to those that were unrestored and intact (28.8%). A similar trend was noticed for all molar teeth, although the only other group that had a difference of statistical significance ($P < 0.05$) was the maxillary left second molars (12.1% vs. 25.0%, respectively).

Chandler *et al.* (2003) studied coronal pulp dimensions in 445 human first molars teeth using bitewing radiographs of 121 young adults. Pulp stones were present in 9.9% of individuals and 4% of the molar teeth examined. Willman (1934) examined 164 teeth from patients of different ages; some form of calcification was found histologically in 87.2% of these, whereas pulp stones were visible radiographically in only 14% of the specimens. Hill (1934) examined histologically 132 teeth from patients of various ages and found that the frequency of calcification (primarily pulp stones) was 66% in the 10- to 30-year-old group, 80% in the 30- to 50-year-old group and 90% in the 50- to 70-year-old group. In the histological study by Sayegh & Reed (1968), the 45- to 63-year-old group had an incidence of pulpal calcification of 90%, irrespective of whether caries was present.

Nitzan *et al.* (1986) studied histological sections of 52 impacted, uninjured canines that were extracted from patients ranging in age from 11 to 76. There were 19 teeth in the 11- to 24-year group, 17 in the 25- to 39-year group and 14 in the 40- to 76-year group. Pulp stones (all false) were present in 56% of the pulps, with an equal distribution among the age groups.

Hillmann & Geurtsen (1997) histologically examined calcifications in 332 permanent teeth. The teeth were either caries-free or had only minute carious defects or restorations. Most of the teeth were molars, although some single-rooted teeth were also included; teeth were erupted or nonerupted. Three age groups were involved: 10–30, 31–51 and 52–72 years. When combining pulp stones and diffuse calcifications, a statistically significant increase occurred over the age groups; 14.9%, 44.4% and 65.1% respectively. However, when pulp stones alone were investigated, the

percentages were around 3% (10–30 years) and 13% for both the other groups. These percentages are clearly in contrast to those of other studies.

In summary, it appears that pulp stone prevalence can be close to 100%, particularly if associated with carious or restored first molars. The prevalence may increase with age, where the cumulative effect of restorative procedures upon pulp stone creation may be seen; alternatively, physiological factors that lead to pulp stone formation may also manifest (Bernick & Nedelman 1975). Meanwhile, continuous secondary and tertiary dentine deposition may envelop existing pulp stones and mask their true prevalence.

Clinical implications

Given the association between pulp stones and nerve tissue, both in terms of pulp stone formation and nerve fibre entrapment, it has been suggested that some pain of an idiopathic nature may be caused by pulp stones (Seltzer & Bender 1984). Case reports and letters to editors about such pain appear in the literature (Norman & Johnstone 1921, Abdel Wahab & Kennedy 1986, Ataman *et al.* 1987). Along similar lines, pulp stones have been compared to kidney and gall bladder stones (Martin 2002), but a much higher incidence of unexplained dental pain would be expected, given the high prevalence of pulp stones and pulp calcifications. Pulp-related pain with no apparent cause is relatively common, and pulp stones may be a finding, but this does not imply causality. Pulp stones have been described as symptoms of changes in the pulp tissue, rather than their cause (Moss-Salentijn & Hendricks-Klyvert 1988). The presence of pulp stones or diffuse calcifications does not affect the threshold of electric pulp testing (Moody *et al.* 1989). In the absence of any additional signs or symptoms, pulp stones should not be interpreted as a disorder requiring endodontic therapy.

Textbooks discuss the clinical relevance of pulp stones in terms of their effect upon root canal treatment. Their large size in the pulp chamber may block access to canal orifices and alter the internal anatomy. Attached stones may deflect or engage the tip of exploring instruments, preventing their easy passage down the canal (Pashley *et al.* 2002). Sometimes a large pulp stone can be dissected out of an access cavity using burs, but ultrasonic instrumentation with the use of special tips makes their removal far easier (Stamos *et al.* 1985, Pitt Ford *et al.* 2002). Within narrow canals ultrasonics should ideally be coupled with the dissolving action of sodium hypochlorite to produce a synergistic effect (Cunningham & Balekjian 1980). Should a stone be attached to the canal wall and a file can be passed alongside the stone, it may be removed by careful instrumentation (Pitt Ford & Mitchell 2004). Generally speaking however, pulp stones present little clinical difficulty during root canal treatment when magnification, good access and appropriate instruments are employed.

Conclusions

It would appear that pulp stones are primarily a physiological manifestation (as are most other pulpal calcifications) and may increase in number and/or size due to local or systemic pathology. The aetiological factors involved in their formation are still not fully apparent. Their primary clinical relevance remains in the area of endodontic treatment, much in the way that secondary and tertiary dentine formations also influence root canal treatment. While further investigation may shed more light on their formation, it seems unlikely to alter their significance to the endodontist and the general dentist.

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