# Common Dental Anomalies in Pediatric Dental Practice

# 7

This chapter contains examples of common dental anomalies, as encountered in pediatric dental practice, which were not treated in Chap. 6. The radiographic images taken with different radiographic techniques and their radiographic differential diagnoses are presented. The aim of this chapter is to familiarize the reader with the different results of the techniques explained in the previous chapters. Obviously not all types of anomalies were captured in this textbook chapter, but at least the reader will get a better feel about how the images turn out and what information can be retrieved from them.

Names of distinguished colleagues who supplied the images for this chapter are mentioned with the radiographs. If there is no name mentioned with the radiographs, the radiographs were taken by the author of this book or collected from the different university clinics he has worked in (Ghent University in Belgium, University of Washington in Seattle, USA, and University of Western Australia in Perth, Australia).

## 7.1 Amelogenesis Imperfecta

There are three types of amelogenesis imperfecta (AI): hypoplastic type (type 1 in which the enamel matrix is inadequately formed, causing pitted, grooved, and thin enamel); hypomaturation type (type 2, in which the form appears normal, but the enamel is soft and vulnerable, and brownish-yellow in color); and hypocalcified type (type 3, in which the enamel is normal in quantity but poorly calcified, which gives it an opaque appearance and makes it weak). Figure 7.1 is an illustration of a case of amelogenesis imperfecta of the hypomaturation type.


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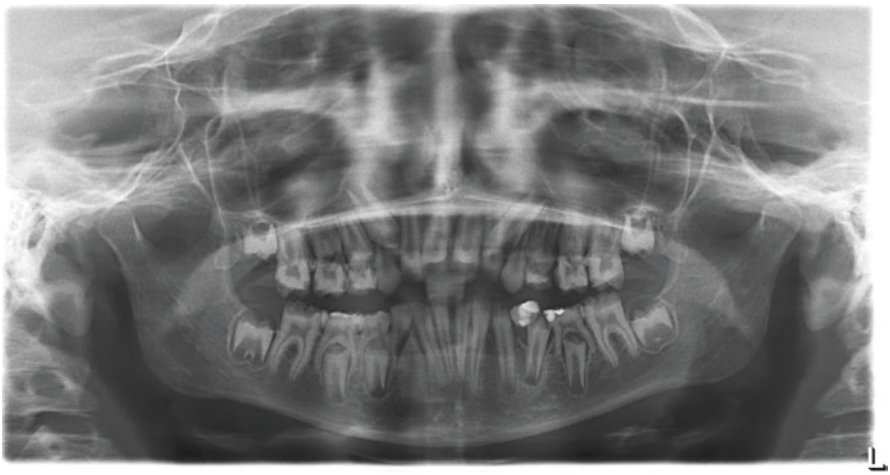

**Fig. 7.1** This panoramic radiograph was taken in a 9-year-old patient with amelogenesis imperfecta type 2 (hypomaturation type). Notice the missing, chipped off, enamel on occlusal surfaces of the molars (courtesy of Dr. Bieke kreps, Belgium)

*Non-syndromic amelogenesis imperfecta (AI) belongs to a group of rare disorders that affect tooth enamel formation in either a qualitative or a quantitative fashion. Recent studies on genome sequencing have revealed more information about its mechanism of inheritance and phenotype appearance. In hypoplastic AI, the enamel is thin and the interproximal contacts are often missing (spacing between teeth) and the patients also have a tendency to develop an anterior open bite. In hypocalcification AI, the enamel is extremely soft, but has the normal thickness, as opposed to the hypoplastic form. However, due to its hypocalcified nature, the enamel is quickly worn and lost after eruption, leaving the remaining enamel to be rough and discolored. In hypomaturation AI, the enamel appears brown or yellowish due to a defective mechanism during maturation of the enamel matrix, though the enamel has the normal thickness. This type also leads to quick wear and chipping off of the enamel. Because of the phenotypical similarities between these three types, a more simple and broader classification system was developed by Prasad* et al. *in 2016. They suggested only hypoplastic AI and hypomineralized AI to be used. The hypomineralized AI holds both the hypomaturation and hypocalcification AI types. Based on the recent literature, there are more than 17 genes involved in non-syndromic AI. Both autosomal dominant and recessive mutations in these genes (mutations in enamel matrix proteins or protease-encoding genes) can result in any of the abovementioned phenotypes. For further detailed information, the interested reader is referred to the literature.*

## 7.2 Dentinogenesis Imperfecta

Dentinogenesis imperfecta (DI) is a hereditary developmental anomaly of dentin, in the absence of any systemic disease. Osteogenesis imperfecta may be associated with similar dentinal aberrant anatomy and appearance. It is then called osteogenesis imperfecta with opalescent teeth (DI type 1). Three classification systems (Shields versus Witkop versus Levin) are used, but discussing this is beyond the scope of this chapter and book. Dentinogenesis imperfecta can be divided into three types: type 1 is associated with osteogenesis imperfecta and teeth appear opalescent, type 2 is isolated opalescent teeth, and type 3 is isolated opalescent teeth with wide pulp chambers and canals. What is specific to dentinogenesis imperfecta is that all teeth in both dentitions are affected. That is the greatest differential with the dentinal dysplasia types that will be discussed and illustrated in Sect. 7.3. Since the dentin is affected, the adherence between enamel and dentin is compromised and patients will suffer from chipping enamel and hence sensitive teeth, which subsequently wear down quickly too. The teeth have a blue to brown color when they erupt. Pulp chambers may get obliterated, but sometimes they don't and seem to be expanded, which renders the radiographic appearance typical and is called "shell teeth." Usually the shape of the crowns of the teeth is bulbous and sometimes called tulip shaped. Figure 7.2 is an illustration of an adult patient with dentinogenesis imperfecta and figure 7.3 is a child with dentinogenesis imperfecta.

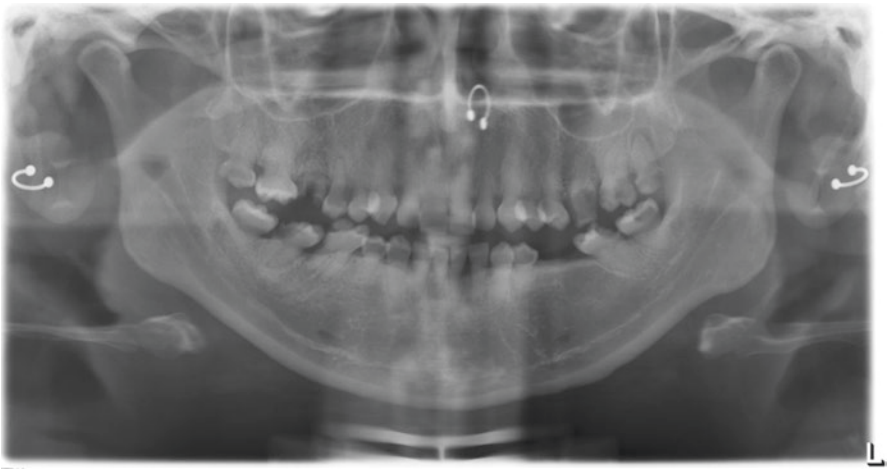

**Fig. 7.2** This panoramic radiograph of a 20-year-old woman with dentinogenesis imperfecta type 2 shows generalized pulpal obliterations and bulbous crowns of the premolars in particular. Unfortunately she has a serious caries problem, with periapical inflammations, which makes treatment more complicated

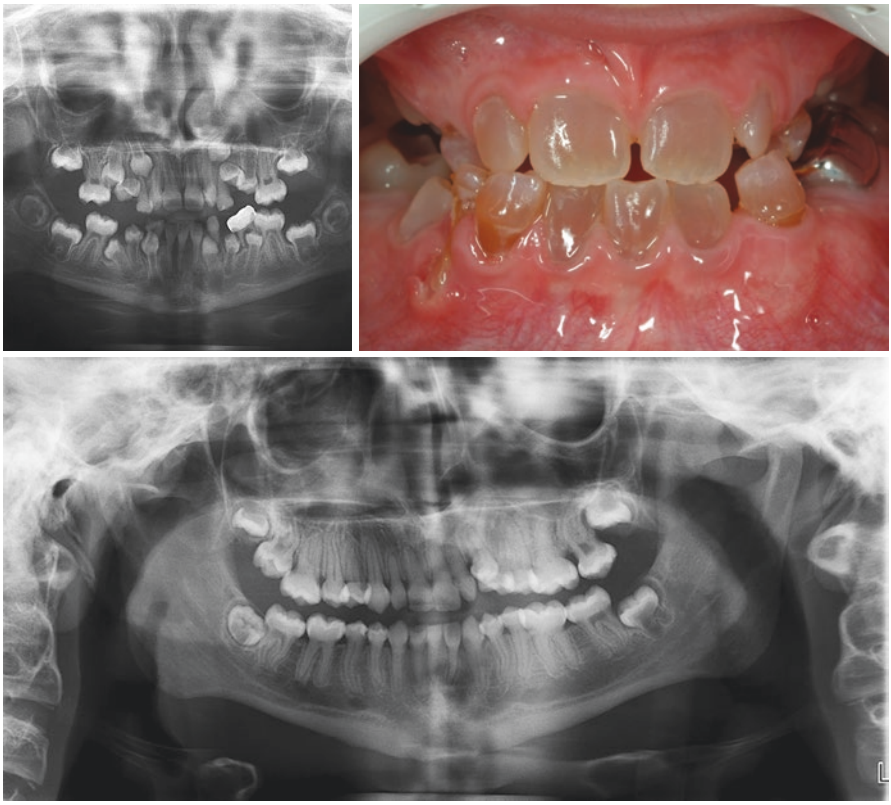

**Fig. 7.3** The panoramic in the upper left-hand-side corner is taken 5 years before the panoramic radiograph at the bottom. One can appreciate the clinical opalescent appearance of the teeth in the upper right-hand-side corner image. Besides the bulbous crowns of the teeth, it can be observed that the pulp systems in nearly all teeth have obliterated. This is a case of dentinogenesis imperfecta type 2 (courtesy of Dr. Marc Jeannin, Belgium)

*Dentinogenesis imperfecta (DI) is a rare hereditary disorder, primarily characterized by defective dentine formation, which subsequently results in early enamel loss. The latter is usually normal in its histological appearance though and is not affected by the DI mutations. However, one study reported the enamel to be hypoplastic (Bloch-Zupan* et al. *2016). The estimated incidence is 1:8000 and 1:45,455 live births. The non-syndromic types of DI are associated with mutations in the dentine sialophosphoprotein gene DSPP (chromosome 4, 4q22.1). The latter plays a paramount role in the mineralization and maturation of dentine and especially in the non-collagenous component of the dentine. DI type II affects both the primary and the permanent dentition, but the former is usually more severely affected. The teeth usually have an opalescent hue, ranging from amber brown to grayish-blue. The teeth show typical bulbous crowns with a cervical constriction, leading to a narrow root,*

*which contains an obliterated pulp canal. Histologically the dentine appears as dysplastic with reduced mineralization, irregular dentine tubules, and in some cases interglobular dentine. The obliterated pulp system does not mean that there is no access for bacteria, as the dentine tubules will allow for bacterial spreading and eventually for periapical inflammation. It is possible that DI type 1 is often not diagnosed accurately as it has a great variety in appearance and expression and therefore osteogenesis imperfecta might be underdiagnosed as well, as this condition also shows a myriad of expressions (bruising, prolonged bleeding, spraining, fractures, hearing impairment, joint hypermobility, and blue sclerae).*

## 7.3 Dentinal Dysplasia and Regional Odontodysplasia

*Dentinal dysplasia* (DD) should not be confused with dentinogenesis (or sometimes called odontogenesis) imperfecta (see Sect. 7.2). The first differential diagnosis is that DD does not affect the entire dentition. In DD the roots are short and conical, hence the name rootless teeth, and the pulp chambers are obliterated by multiple nodules of poorly organized dentine. Usually patients loose these teeth early after eruption. There are two types of DD: type 1 involves radicular dentin (rootless teeth) and type 2 involves coronal dentin and is easily mistaken for dentinogenesis imperfecta. DD type 2 teeth are similar to dentinogenesis imperfecta teeth, but the coronal pulp chambers contain denticles accompanied by thistle tube appearance of the pulps in the permanent teeth. However the pulps will be totally obliterated in deciduous teeth, which clinically resemble dentinogenesis imperfecta (amber colored and translucent).

*Regional odontodysplasia* is a localized condition affecting only a few teeth in a quadrant. These teeth have aberrant enamel, dentine, and pulp and therefore are called ghost teeth, because of their radiolucent appearance on radiographs. They usually fail to erupt, but if they do they appear yellowish and rough. Regional odontodysplasia can occur both in the deciduous and permanent dentition and maxillary teeth are supposedly more often involved.

*Segmental odontomaxillary dysplasia* (formerly known as hemimaxillofacial dysplasia) is another type of condition, which can be mistaken for fibrous dysplasia (see Chap. 8) or regional odontodysplasia. It is a rare disorder (only 62 well-documented cases have been published) of unknown etiology, and it causes hemimaxillofacial dysplasia with expansion of the maxilla in the primary or mixed dentition. The enlargement is caused by fibrous tissue of the gingiva and expansion of the alveolar crest. Teeth in this area are usually delayed in eruption and appear hypoplastic with varying degrees of aberrant pulp and dentin. Therefore there is an easy confusion with regional odontodysplasia. The latter, however, is not associated with expansion of the maxilla. Figures 7.4 through 7.8 are all illustrations of patients with either dentinal dysplasia or regional odontodysplasia.

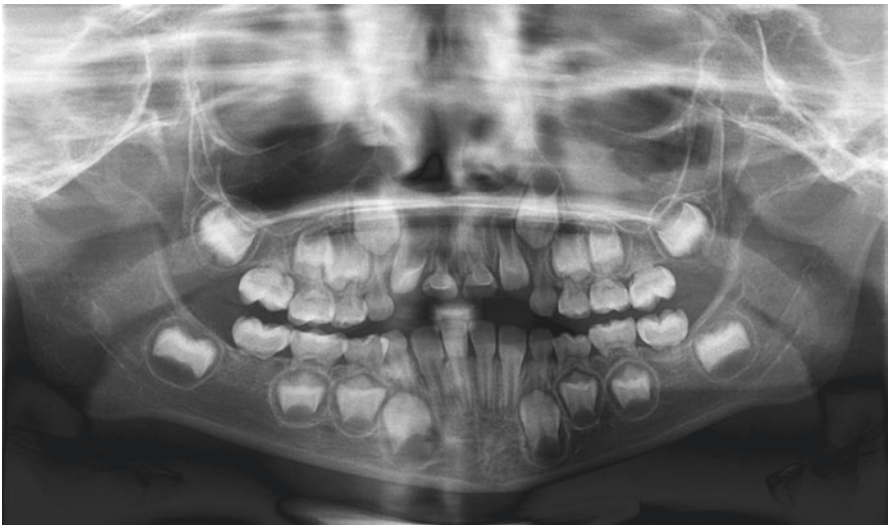

**Fig. 7.4** This panoramic radiograph of a 7-year-old boy shows lack of root formation on both deciduous and permanent molars. Also his maxillary permanent central incisors appear to have aberrant anatomy and radiographic appearance. This is a case of dentinal dysplasia

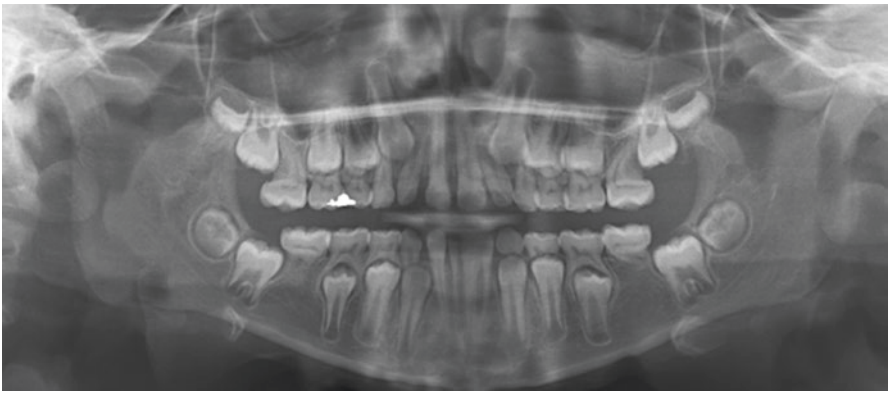

**Fig. 7.5** This panoramic radiograph of a 10-year-old with dentinal dysplasia shows the lack of roots on the first permanent mandibular molars and the partially missing roots on the first permanent maxillary molars (courtesy of Dr. James Howard, USA)

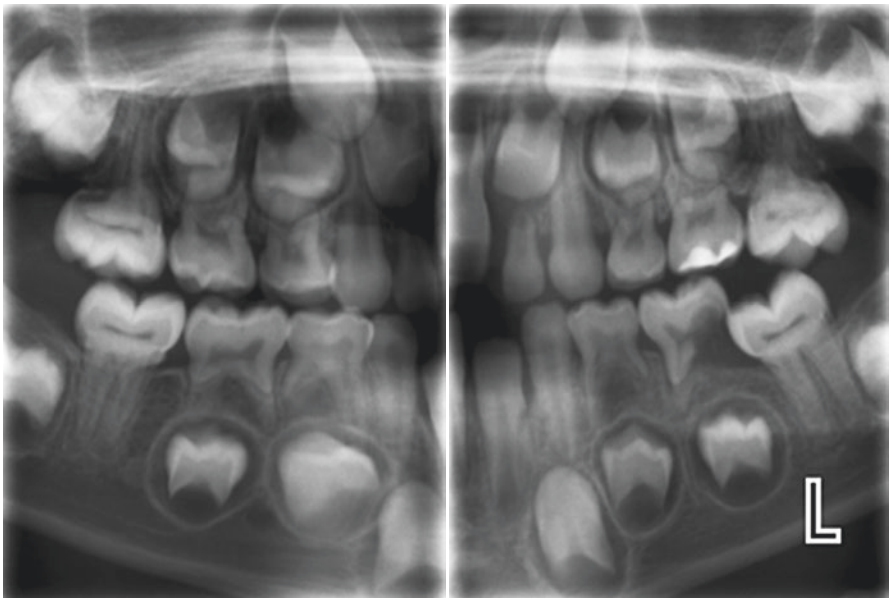


**Fig. 7.6** These extraoral bitewing radiographs taken on a 7-year-old boy with dentinal dysplasia show clearly the thistle-shaped pulp chambers in the first permanent molars, which also appear to have poorly developed roots (courtesy of Dr. Elise Sarvas, USA)

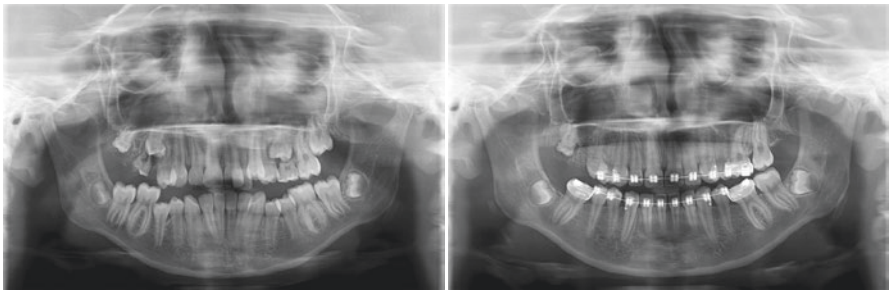

**Fig. 7.7** The panoramic radiograph on the left-hand side was taken to evaluate the clinical appearance of the first permanent maxillary right-hand-side molar. Dental development and radiographic appearance of the second permanent right-hand-side molar appeared to be similar with aberrant enamel and dentin, which confirmed the diagnosis of regional odontodysplasia. It was decided to extract the first permanent right-hand-side maxillary molar and to wait for spontaneous eruption of the second premolar. Follow-up panoramic radiograph during the orthodontic treatment shows the delayed development and lack of spontaneous eruption of the second maxillary molar (courtesy of Dr. Anouk Eloot, Belgium)

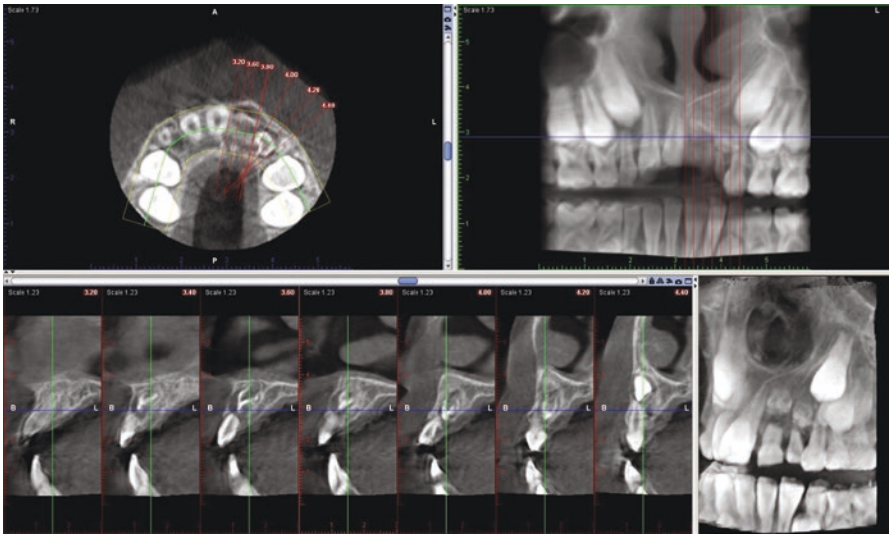

**Fig. 7.8** This is a small-field-of-view cone beam computed tomography of a 9-year-old girl with regional odontodysplasia of the permanent left-hand-side maxillary incisors. Notice the ghostlike appearance of both teeth due to poor and aberrant development of enamel and dentine, as well as the fact that the teeth are not erupted and never will

- *Dentine dysplasia (DD) type 1 has been linked to three pathogenicity genes (VPS4B, SSUH2, SMOC2) and it is believed that it is a genetically heterogeneous disease, which needs to be distinguished from other dentine disorders. DD type 1 teeth appear clinically as normal teeth, but show hypermobility due to the short conical roots. Radiographically the latter is confirmed as well as the pulpal obliteration changes. Even in noncarious DD type 1 teeth, periapical inflammation is noticed. In contrast DD type 2 and dentinogenesis imperfecta appear as amber translucent teeth, short and thin roots with obliterated pulps. DD type 2, coronal DD, is more rare than DD type 1 and its prevalence is not really known. DD type 2 is inherited autosomal dominantly. Histologically the dentine shows entrapped cells within lacunae, suggestive of osteodentine, and also a breakup between dentine and predentine can be found, as well as irregular arrangement of the dentine tubules.*
- *Regional odontodysplasia, unlike general odontodysplasia, is thought to be linked to viral infection, use of certain inappropriate medication during pregnancy, trauma, nutritional deficiency, infection, and metabolic abnormalities.*

- *The etiology of segmental odontomaxillary dysplasia is unknown, but according to some researchers it should be linked to a nonhereditary localized developmental anomaly of the first branchial arch that occurs* in utero. *Other reports link it to endocrine abnormalities, and even bacterial or viral infections. Some patients have been reported with local facial or cutaneous abnormalities on the same side as the segmental odontomaxillary dysplasia: hypertrichosis, hyperpigmentation, hypopigmentation, erythema, rough erythema, ectopic eyelashes, increased number of melanocytic nevi, Becker nevus, facial depression, commissural lip pits, and unilateral lip clefting. With regard to the affected teeth, reports have described the following: smaller or larger teeth than normal, over-retained primary teeth, congenitally missing teeth, delayed eruption of especially premolars, increased spacing between teeth, and primary molars in particular can show abnormal coronal and/or root morphology, and atrophic pulp chambers. Also multiple white papules have been described overlying the alveolar crest, mimicking the dental lamina cysts of the newborn. Erythema with blanchable patch on the gingiva has also been described. The alveolar bone shows aberrant patterns as well: ill-defined sclerosis or coarse bone trabeculation, which may affect the maxillary sinus. The latter changes often cause confusion with fibrous dysplasia (Chap. 8); however fibrous dysplasia is not accompanied by the typical dental abnormalities described above.*

## 7.4 Fusion, Gemination, and Concrescence

*Fusion* implies two tooth germs fused into one tooth, whereas *gemination* assumes a single tooth bud that has separated into two teeth. In the first case one will have one tooth too few albeit it is a wider tooth, whereas in the second there will be a supernumerary tooth in the arch. The latter is usually fused at the root though, making it a challenge to treat if indicated. The prevalence of fusion in the primary dentition is reported to be about 0.4–0.9%, whereas in the permanent dentition it is around 0.2%. Bilateral fusion of permanent teeth is even more rare and is estimated at 0.05%. *Concrescence* is another phenomenon where teeth are fused at root level, but by formation of root cementum and not dentin. Examples of fusion and gemination are shown in figures 7.9 through 7.12.

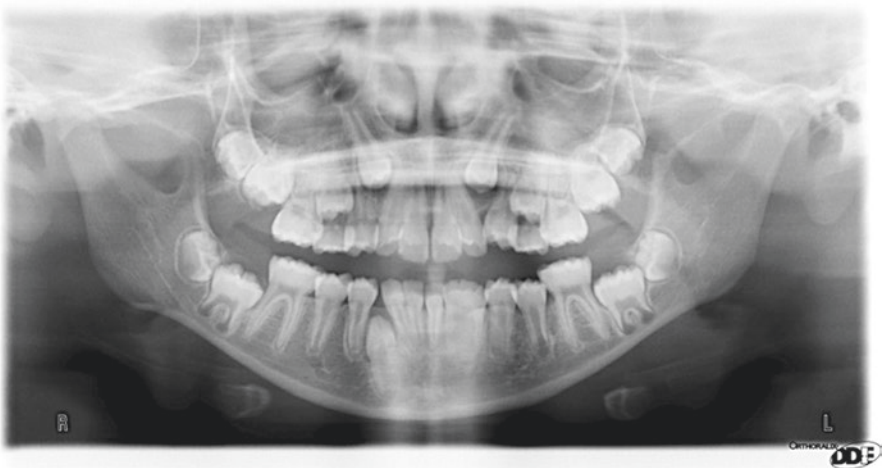


**Fig. 7.9** This panoramic radiograph shows a fusion of left-hand-side mandibular canine and lateral incisor. Counting the number of teeth in the third quadrant shows only seven teeth, including the third molar. Also notice the impacted mandibular canines as well as the right-hand-side mandibular canine

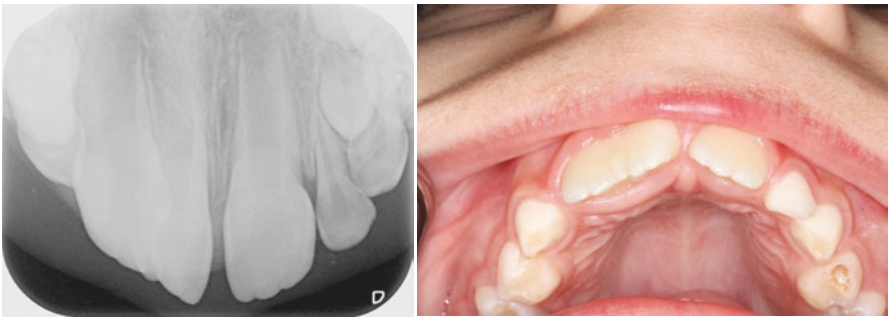

**Fig. 7.10** This periapical radiograph was taken to verify the true nature of the clinical finding of an unusual wide permanent maxillary right-hand-side central incisor. The crowns of the lateral and central incisors have fused, while the roots and pulp canals appear to be separate. Therefore the final tooth count is one too few (courtesy of Dr. Wouter Van den Steen, Belgium)

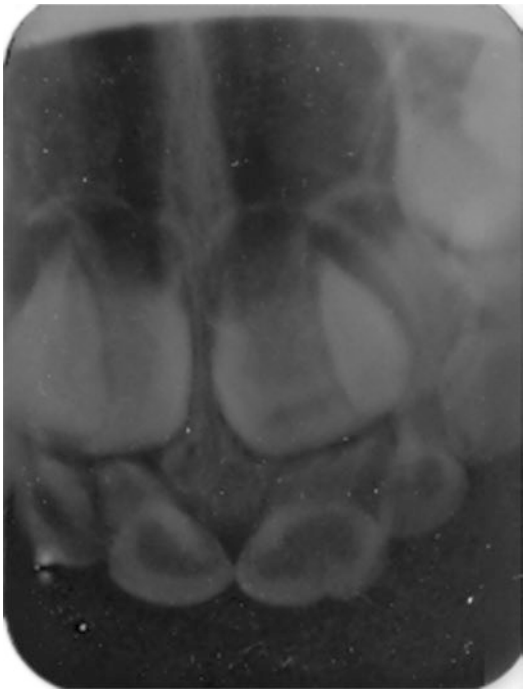

**Fig. 7.11** This periapical radiograph was taken because of the unusual large deciduous left-hand-side maxillary central incisor. The presence of the lateral deciduous incisor implies that the central incisor is the result of a gemination; hence the count of teeth in the second quadrant equals five, which is normal in this 3-year-old child

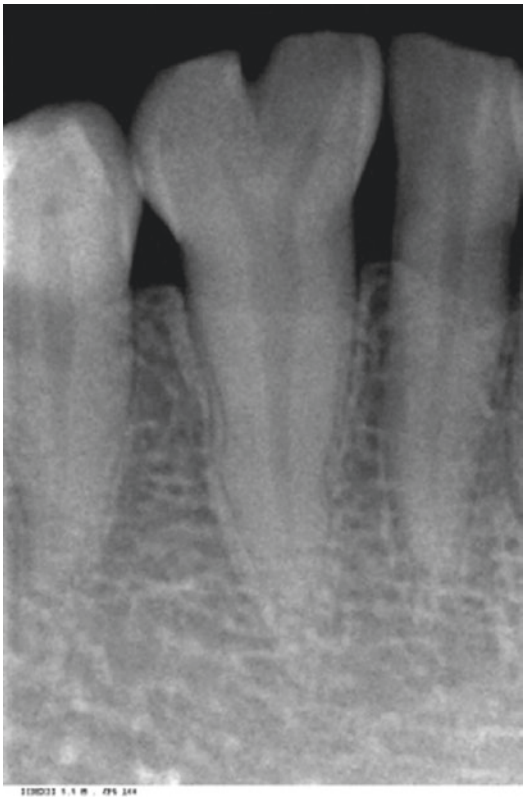

**Fig. 7.12** This periapical radiographs was taken to verify if this was a fusion or gemination or even a supernumerary tooth, as clinically there were two crowns visible. The radiograph shows the single root and the bifid pulp chamber. This was a gemination (courtesy of Dr. Amelie Julie Lambregts, Belgium)

## 7.5 Odontomas

*Odontomas* are the most common types of odontogenic tumors and are considered hamartomas (developmental anomalies), consisting of enamel, dentin, and pulpal tissue. A compound odontoma is composed of toothlike particles, whereas a complex odontoma is a conglomerate mass of enamel and dentin. We will leave it open for discussion if a supernumerary tooth (e.g., mesiodens or fourth molar) is a compound odontoma or not. The etiology of odontomas is unknown. Some have reported links with trauma, infection, genetic mutations, odontoblastic hyperactivity, and even alterations of dental development control gene.

In general they are asymptomatic, but in some cases they can lead to swelling, pain, suppuration, or bony expansion. Eruption of inverted odontomas into the nasal cavity has been described. Radiographically they are typically surrounded by a radiolucent rim, which again is surrounded by a radiopaque rim. The treatment is enucleation as they do not recur. Figures 7.13 through 7.17 show examples of odontomas.

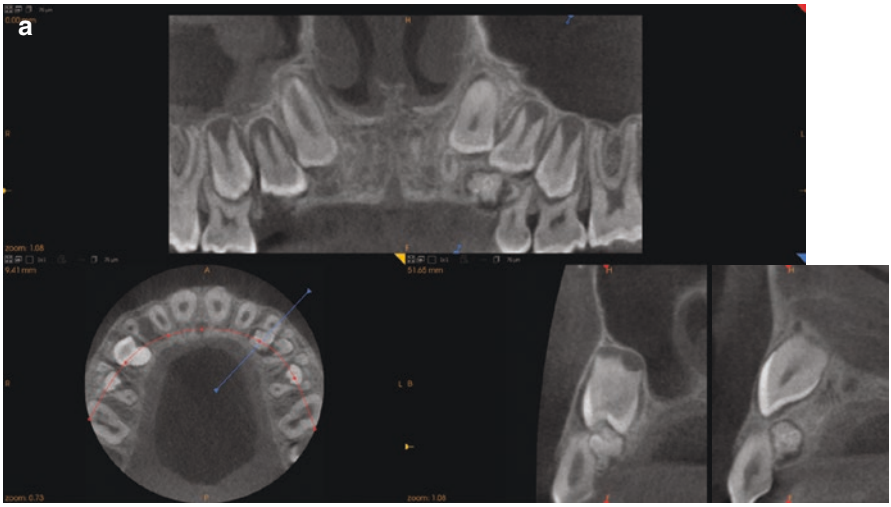


**Fig. 7.13** This small-field-of-view cone beam computed tomography was taken to verify what was obstructing the maxillary left-hand-side premolars. From the images one can appreciate a complex odontoma at the occlusal surface of the left-hand-side first premolar and palatal of the deciduous canine

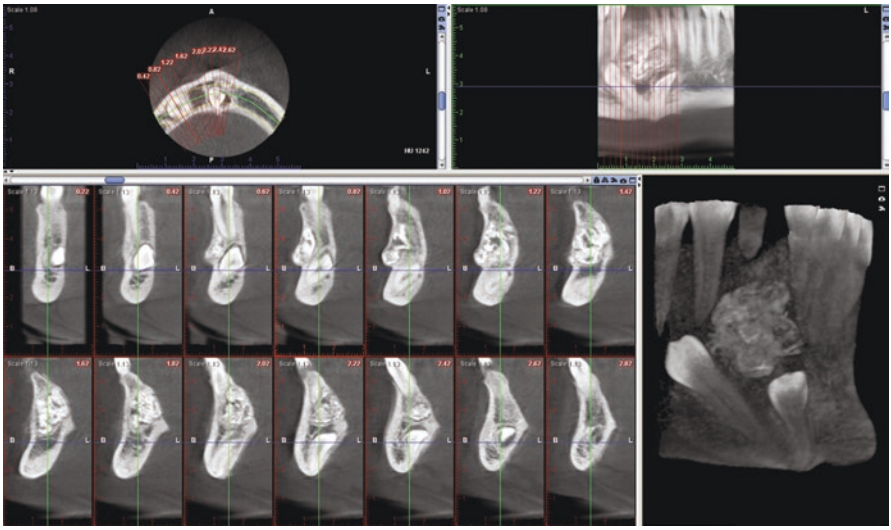

**Fig. 7.14** This is a small-field-of-view cone beam computed tomography of a 17-year-old male, who was missing his right-hand-side mandibular lateral incisor and canine, while his deciduous mandibular canine was still present. The scan shows a large irregular mixed radiopaque-radiolucent mass and two impacted permanent teeth. The mass is a complex odontoma, which has also caused expansion of the buccal mandibular cortical plate

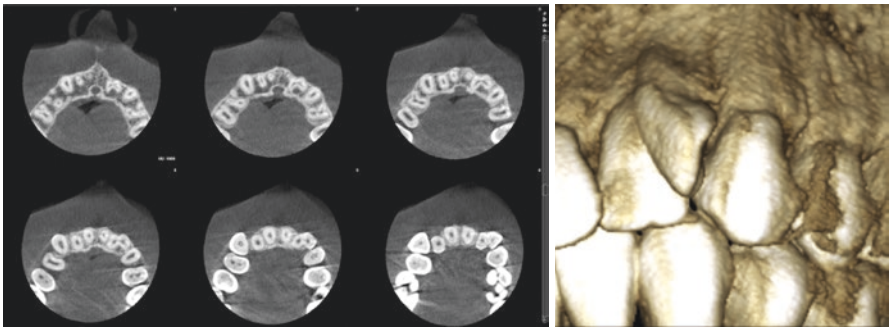

**Fig. 7.15** This small-field-of-view cone beam computed tomography of the left-hand-side permanent maxillary lateral incisor, which clinically appeared to have a double crown or to be a dens evaginatus (see Sect. 7.6), shows that the tooth is actually a gemination with fused pulp systems

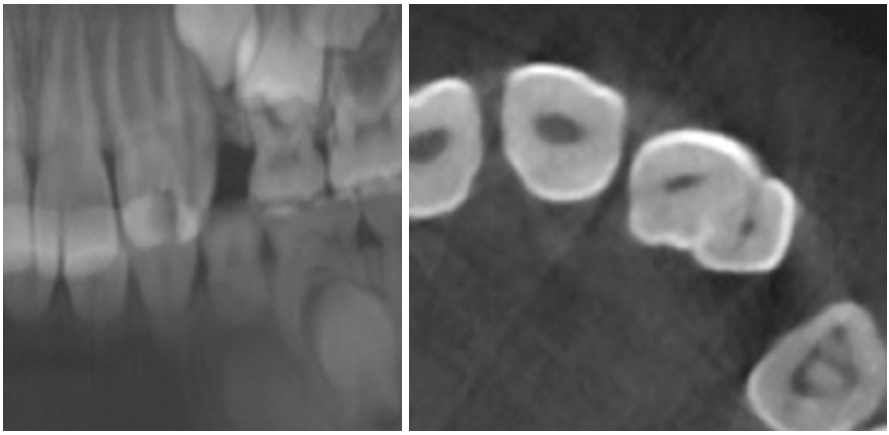

**Fig. 7.16** These cropped cone beam computed tomography images illustrate a fusion of the left-hand-side maxillary permanent lateral incisor with a supernumerary tooth or a compound odontoma. The splitting of the pulp and root is clearly visible (courtesy Dr. Annelore De Grauwe, Belgium)

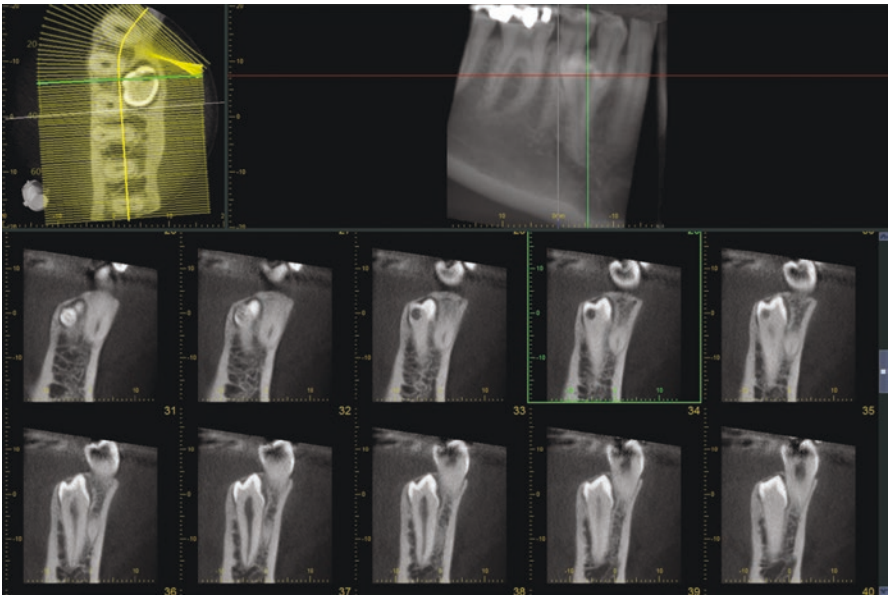

**Fig. 7.17** This is a small-field-of-view cone beam computed tomography of a 17-year-old patient soon to undergo orthodontic treatment. From the CBCT one can observe that the supernumerary mandibular premolar is positioned lingually and that it shows preeruptive intracoronal resorption (also see Chap. 6)

## 7.6 Dens Invaginatus and Dens Evaginatus

A *dens evaginatus* (Figs. 7.18–7.20) is a cusp-like elevation of enamel located in the central groove or lingual ridge of the buccal cusp of a premolar or permanent molar. It occurs mostly in mandibular premolars and consists of enamel, dentin, and pulp. The latter means that attrition may cause pulpal exposure and hence periapical infection can develop. One can also find it in shovel-shaped incisors.

*Dens invaginatus* (dens in dente) (Figs. 7.21 and 7.22) is a deep enamel-lined invagination in the crown and/or root of a tooth. Maxillary permanent lateral incisors are the most common teeth involved, followed by central incisors, premolars, canines, and molars. The extent of the invagination will determine the caries risk and the treatment options.

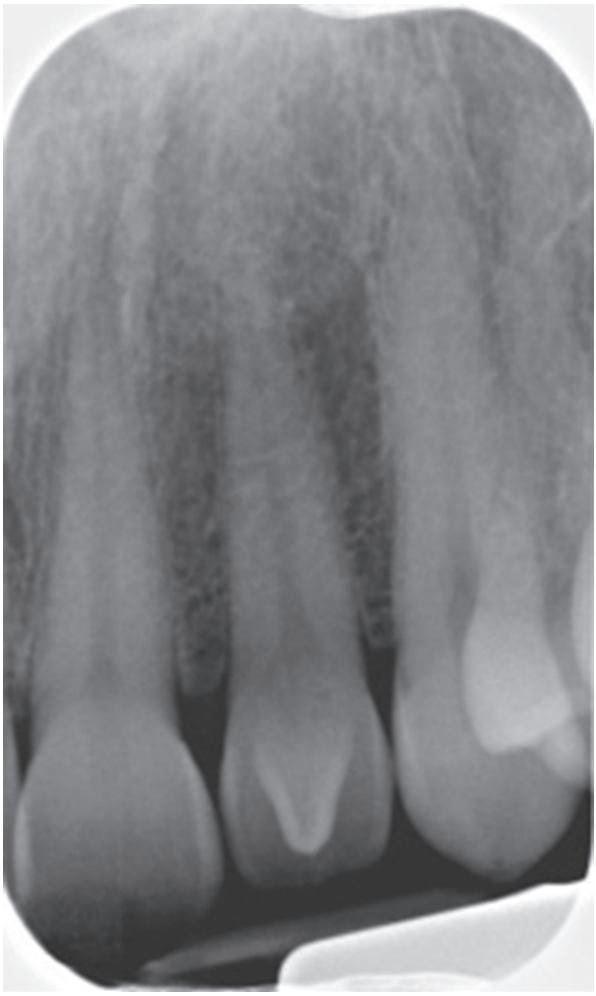

**Fig. 7.18** The periapical radiograph was taken to evaluate the extent of pulpal tissue into the extra palatal cusp of this maxillary left-hand-side permanent lateral incisor. It can be appreciated that there is no pulpal extension into the extra cusp

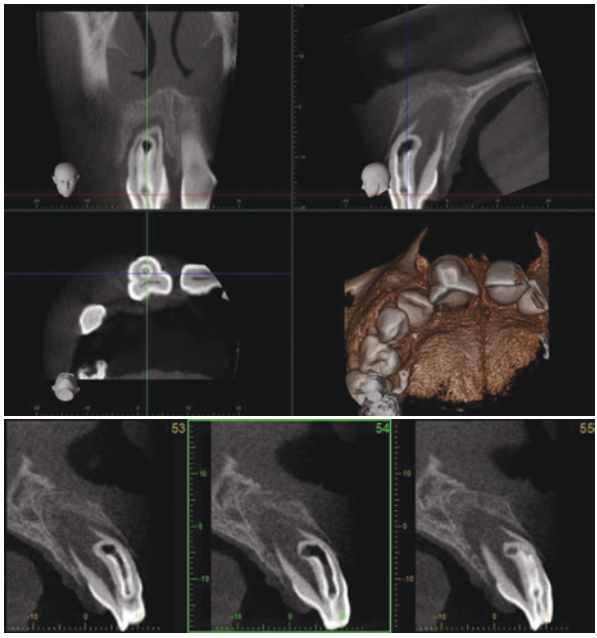


**Fig. 7.19** This small-field cone beam computed tomography was taken to evaluate the unusual dens evaginatus on the right-hand-side maxillary permanent central incisor in this 9-year-old boy. The scan shows the complexity of the pulpal anatomy and a periapical infection that has developed since the eruption of the tooth probably

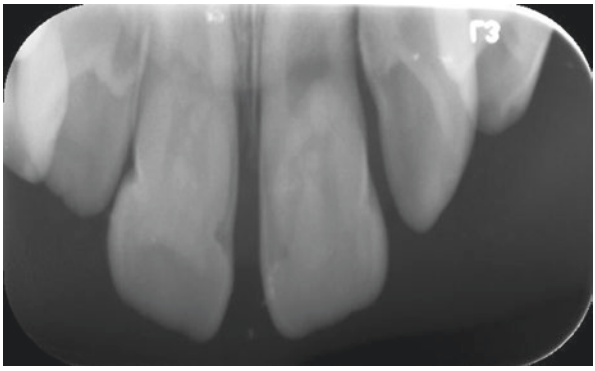

**Fig. 7.20** This occlusal radiograph, taken with the wrong-size photostimulable phosphor plate (size 1 should have been size 2), shows two maxillary central incisors with both a dens in dente. The complex nature of the pulp system does not really require cone beam computed tomography, but a proper occlusal or periapical radiograph in this 7-year-old would be better, as one would want to assess the periapical tissues better

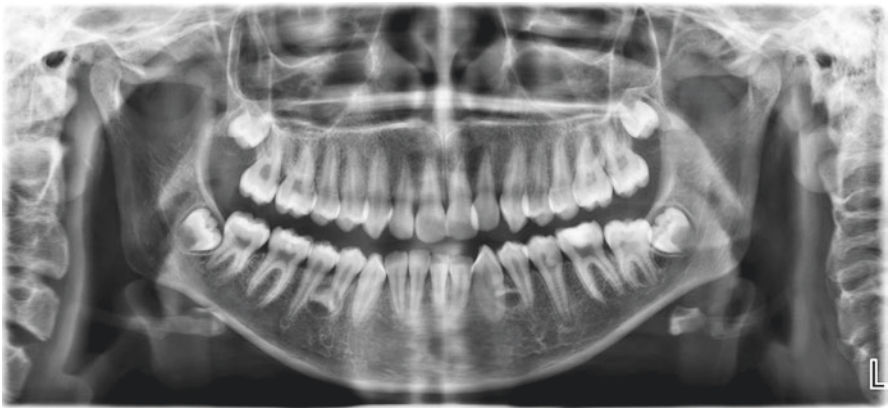

**Fig. 7.21** This panoramic radiograph shows besides two supernumerary premolars in the mandible a dens in dente on the mandibular left-hand-side second premolar. Also notice the small radiolucent areas mesial of the tooth buds of the mandibular third molars, which could indicate two more supernumerary tooth buds. Follow-up is required

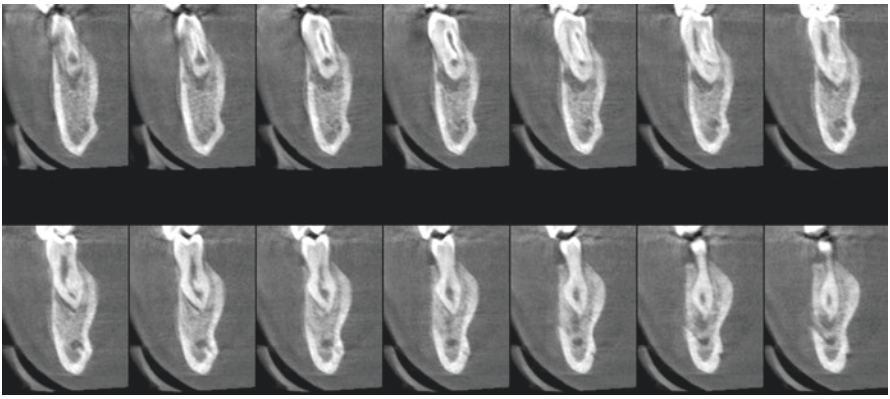

**Fig. 7.22** This is a small-field-of-view cone beam computed tomography scan of the first left-hand-side mandibular premolar which shows a dens invaginatus. It can be appreciated that there is already a periapical infection present and that the endodontic treatment will be complicated due to the complexity of the pulp system

*An attempt to classify dens invaginatus was first proposed by Hallett (1953). The first three types in Hallett's classification refer to crown invaginations. However, all crown invaginations were designated as type 1 in a classification system put forward by Oehlers (1957). Other classifications have also been described by Ulmansky and Hermel (1964), Schulze and Brand (1972), Vincent-Townend (1974), Parnell and Wilcox (1978), and Hicks and Flaitz (1985). The broad classification put forward by Schulze and Brand (1972) includes 12 variations, starting at the incisal edge of teeth, and therefore classifies malformations with respect to morphology of the invagination as well as the anatomic crown form and dysmorphic root configurations. However, the system described by Oehlers (1957) appears to be the most widely used, possibly because of its simple nomenclature and ease of application. The invagination varies in size and shape from a small dip in the enamel to a severe form, which looks like tooth within a tooth. The invagination frequently communicates with the oral cavity, allowing the entry of irritants and microorganisms either directly into pulpal tissues or into an area that is separated from pulpal tissues by only a thin layer of enamel and dentin. This continuous ingress of irritants and the subsequent inflammation usually lead to necrosis of the adjacent pulp tissue and then to periapical or periodontal abscesses. In many cases dens invaginatus and also dens evaginatus pose serious clinical and therapeutic problems. Endodontic treatment seems extremely difficult in a lot of cases and therefore a good long-term vision and plan are essential, which necessitates well-orchestrated collaboration between several dental specialties: pediatric dentist—orthodontist—periodontist—endodontist and perhaps even oral surgeon in some cases. Cone beam CT is the ideal imaging modality as it will clearly show the complexity of the pulpal system and its relationship with the periodontal surroundings of the tooth.*

## 7.7 Taurodontism

Taurodontism Fig. 7.23 is an enlargement of a multiradicular tooth's body and pulp chamber with consequent apical displacement of the pulpal floor and bifurcation of the roots of the tooth. Taurodontism can be associated with amelogenesis imperfecta, ectodermal dysplasia, and Down syndrome, to name a few.

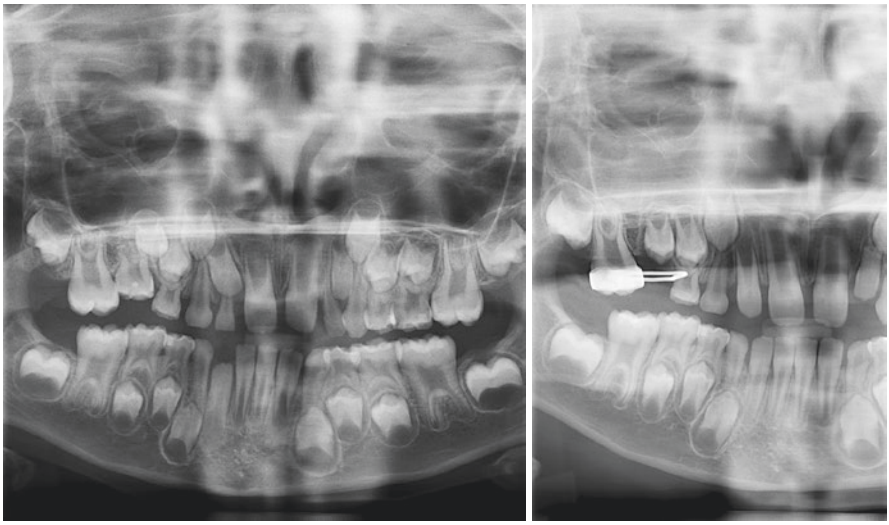

**Fig. 7.23** The collimated panoramic radiograph on the left-hand side shows infraocclusion of the right-hand-side maxillary second deciduous molar, which was the reason for this radiograph to be taken. One can also appreciate the presence of taurodont-type permanent first molars in the four quadrants. The partial panoramic radiograph (not cropped) on the right-hand side is taken a year later (courtesy of Dr. Marc Jeannin, Belgium)

*Taurodontism was first introduced in 1913 by Sir Arthur Keith, but it had been described in 1908 by Gorjanovic-Kramberger. The word originates from the Latin word Taurus, which means bul, and the Greek word odos, which means tooth. Shaw in 1928 tried to make classifications in taurodontism, by describing the level of apical displacement of the pulp chamber (hypo-, meso-, and hyper-taurodontism). The prevalence of taurodontism lies between 0.1 and 48%, depending on the population studied and the criteria used to determine the anatomical aberration. There does not seem to be a gender difference. The most commonly affected teeth are the mandibular first molars, but it has also been described in primary molars. It can appear unilateral or bilateral and it can be present in more than two quadrants as well. The etiology is unknown, but it has been proposed that failure of the Hertwig's epithelial root sheath to invaginate at the proper height is responsible. Other causes that have been put forward are interference in the epithelia-mesenchymatose induction, genetic transmission, increased number of X chromosomes, polygenetics, heavy masticatory habits, infection, disrupted developmental homeostasis, high-dose chemotherapy, and bone marrow transplantation. One should keep in mind that the association with other dental anomalies and/or syndromes may indicate a same genetic origin.*

## 7.8 Cementoblastoma, Hypercementosis

A *cementoblastoma* (Fig. 7.24) is also called a *true cementoma* and is an odontogenic neoplasm of cementoblasts. The majority of these rare neoplasms occur in mandibular molars or premolars and are diagnosed before the age of 20. Very seldom a primary molar is affected. Radiographically they are seen as a densely mineralized mass attached at the apex of the affected tooth.

They can be confused with *hypercementosis*, (Fig. 7.25) which is not a neoplastic deposition of excessive cementum, as it is continuous with the normal radicular cementum around the root. The apices of the teeth appear a little more blunted on radiographs, as the cementum accumulates at the apical third of the root. The biggest differential with the cementoblastoma is that with hypercementosis the periodontal ligament space and the lamina dura remain intact and it occurs more in adulthood than in childhood.

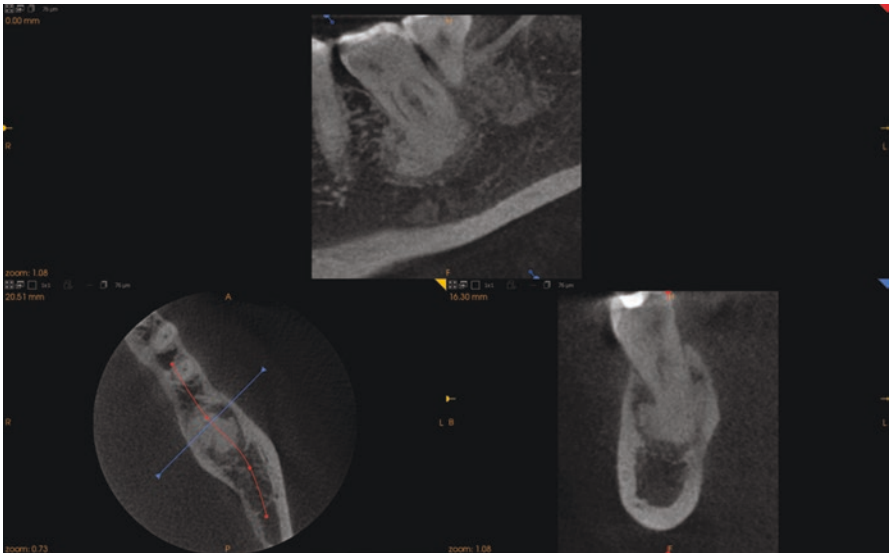

**Fig. 7.24** This small-field-of-view cone beam computed tomography scan of the first left-hand-side mandibular permanent molar with a cementoblastoma shows a uniform radiopaque mass attached to the mesial root. The lamina dura and the periodontal ligament space have disappeared and the mass extends towards buccal and lingual cortical plates of the mandible

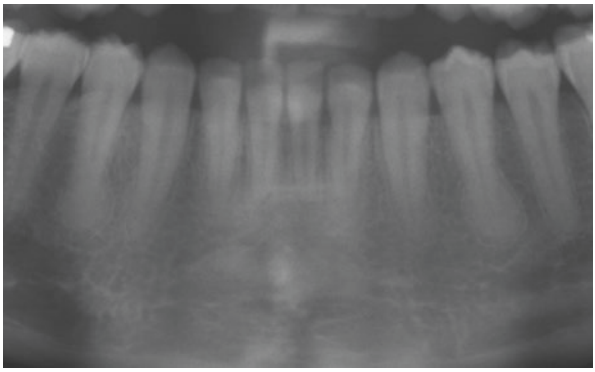

**Fig. 7.25** This is a cropped panoramic radiograph which shows hypercementosis on the second right-hand-side mandibular premolar and the first left-hand-side mandibular premolar. One can appreciate the presence of a lamina dura and a periodontal ligament space around the hypercementosis area in the apical third of the respective root. This is a radiograph of a 25-year-old woman. Nevertheless the author considered it was useful to show an example, since hypercementosis is not common in children and one needs to be able to differentiate from cementoblastoma or true cementoma

## 7.9 Eruption Issues Requiring Imaging

There can be several reasons why teeth are delayed in eruption or are simply not erupting. The latter can involve anatomical restrictions (e.g., no space to erupt), pathology (e.g., dentigerous cyst), or trauma. Delayed eruption can be related to the patient's growth in general or due to therapy (e.g., chemotherapy), which in many cases also causes hypoplasia of teeth. Figures 7.26 and 7.27 are illustrations of common eruption issues with mandibular third molars. Both cases deal with the proximity and location of the inferior alveolar nerve which has to be identified prior to extraction of the respective tooth.

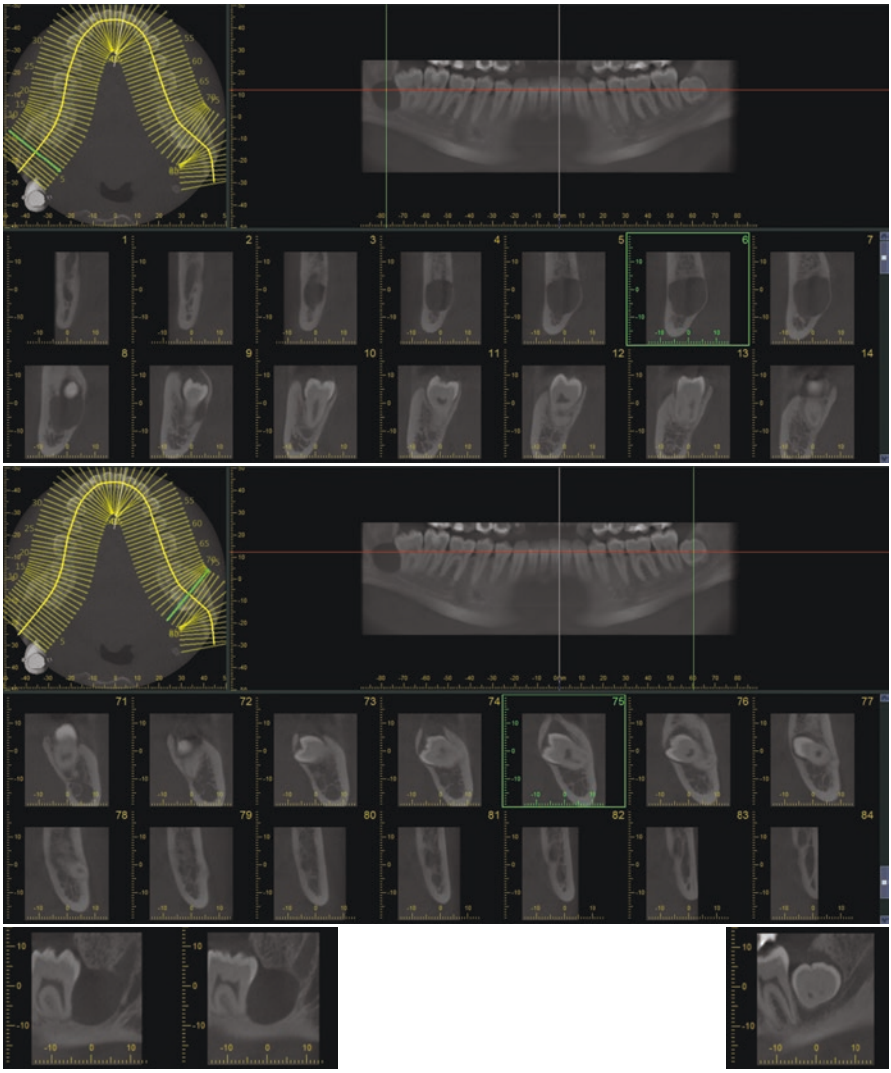

**Fig. 7.26** This is a case of an 18-year-old male who required mandibular third molar extractions. In the fourth quadrant one can appreciate a round, uniform radiolucent, well-defined lesion, adjacent to the distal root of the third molar, which appears to have displaced the inferior alveolar nerve inferiorly and which also appears to be expanding the lingual cortical plate of the mandible and eroding the buccal plate of the mandible. Based on its radiographic appearance, this can be either a dentigerous cyst, an odontogenic keratocyst, or an ameloblastoma. Histopathology determined it as a dentigerous cyst. The third molar in the third quadrant appeared to be horizontal with its crown facing the lingual aspect of the mandible and its apices appeared to almost perforate the buccal cortical plate of the mandible. The inferior alveolar nerve can be identified as running against the inferior surface of the crown of the tooth. The latter information was important for the oral surgeon who performed the exodontia

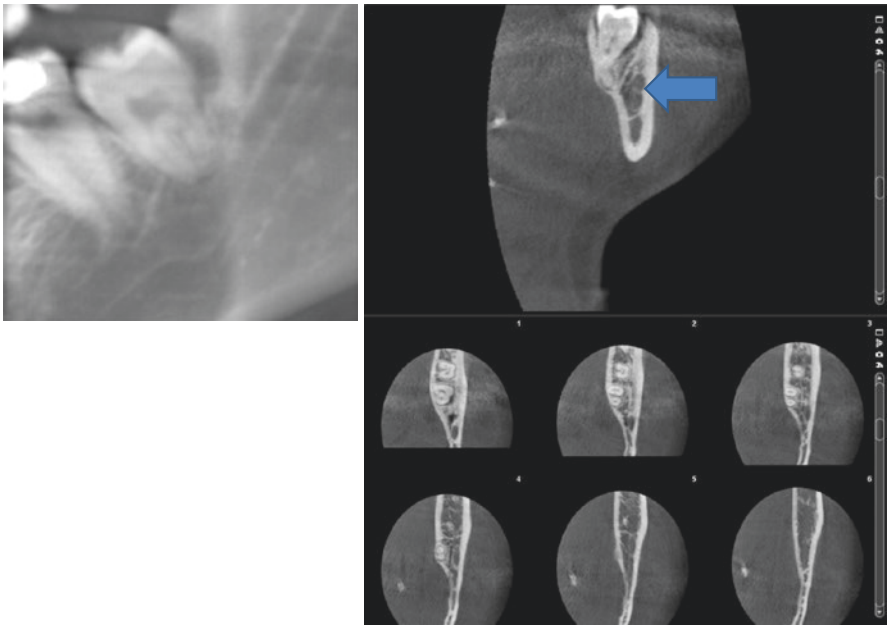

**Fig. 7.27** This 18-year-old girl was planned for surgical exodontia of the third molar in the third quadrant. From the cropped panoramic radiograph, one can see that the apices of the third molar appear to deviate the inferior alveolar nerve. Therefore a cone beam computed tomography was ordered, which showed that the mandibular canal ran buccal to the apices of the tooth, instead of what is mentioned in anatomy books. This information was important to the oral surgeon who performed the extraction

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