Failure of Tooth Eruption: Diagnosis and Management

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Abstract

Tooth eruption disorders are diverse in their etiologies and can be difficult to diagnose. Management of tooth eruption disorders is predicated largely on establishing a correct diagnosis and will depend on the clinical phenotype (e.g., what teeth are affected, severity of the condition, patient age, and health status). The etiologies of abnormalities in tooth eruption include inadequate space, presence of obstructions such as cysts, ankyloses, and hereditary conditions, to name just a few. Treatment approaches will depend on the age of the patient, number of teeth involved, diagnosis, treatment cost, and other factors. The goal of this chapter is to provide a foundation for the diagnosis of tooth eruption disorders and review some of the available treatment options.

Introduction

The clinical management of tooth eruption disorders presents a significant challenge, largely because the diagnosis is so complex and the primary mechanism of eruption itself is poorly understood. The etiologies of tooth eruption disorders are diverse and include environmental stresses such as trauma and a variety of genetic conditions such as primary failure of eruption (PFE, OMIM #125350) and cleidocranial dysplasia (OMIM #119600). A search for tooth and eruption on OMIM reveals 119 conditions listed as having tooth eruption issues that range from natal teeth (OMIM #187050) to various forms of amelogenesis imperfecta (OMIM #s130900, 204690, 613211). Individuals with different forms of osteogenesis imperfecta (see Chap. 7) are at increased risk for developing dentigerous cysts around developing teeth that can obstruct normal tooth eruption. Gingival overgrowth, such as occurs in a variety of conditions (e.g., gingival fibromatosis with hypertrichosis, OMIM #135400), has disturbed tooth eruption. It is helpful to begin with a clear understanding of what is known about tooth eruption, including normal and abnormal events,

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to provide the necessary foundation to diagnose and manage eruption disorders. The diversity of these conditions and their etiologies make the diagnosis and subsequent treatment difficult.

In the permanent human dentition, the normal process of eruption can be divided into major clinical stages – preemergent and postemergent. The preemergent eruption stage is the most important in the initiation of the eruption process; combined resorption and eruption process facilitates eruption of the permanent tooth [1]. Resorption of alveolar bone and primary tooth roots overlying the crown of the erupting permanent tooth facilitates the resultant eruption of the permanent tooth; this coordinated process moves the tooth into the pathway cleared by resorption. It is the uncoupling of these two processes, eruption and resorption, in naturally occurring human conditions (i.e., primary failure of eruption or osteopetrosis) that illustrates that the two processes are actually separate. For instance, in osteopetrosis (OMIM #259700), a syndromic condition, teeth fail to erupt due to the absence of an eruptive pathway resulting from a defective metabolic process of the bone [2]. In this case the resorption process is faulty. In PFE, a nonsyndromic disorder, the opposite scenario exists; the resorptive pathway is cleared, but the tooth fails to erupt [3, 4]. Postemergent tooth eruption disorders also occur. Ankylosis is a relatively prevalent condition in the primary dentition (prevalence: about 7–8 % of children have one or more affected teeth) and results from a loss of normal periodontal ligament and the bone attaching directly to the tooth root. Ankylosis occurs more commonly in the siblings of children that have ankyloses, is more common in the mandibular dentition, and more commonly affects teeth that do not have a permanent successor (often a primary molar with no secondary dentition premolar) [39]. Ankylosed primary teeth also are commonly associated with other dental anomalies in a high percentage of cases including tooth agenesis, microdont lateral incisors, and palatally displaced permanent canine teeth [40].

It is useful to begin our exploration of eruption anomalies by contrasting the molecular events surrounding eruption and, by extension, these two diametrically opposed scenarios. Molecular studies have revealed that eruption is, in fact, a tightly coordinated process, regulated by a series of signaling events between the dental follicle and the alveolar bone [5]. As indicated above, in osteopetrosis, the resorptive process is faulty due to an osteoclast defect. This is in contrast to a complete failure of the primary eruption mechanism that is not associated with defective osteoclasts [3, 7]. In PFE, a genetic alteration in the parathyroid hormone receptor 1 (*PTH1R*) gene [4, 8] further confirms the molecular basis of tooth eruption; a mutation in the *PTH1R* gene results in a striking failure of eruption that is hereditary. This finding is significant as nonsyndromic eruption disturbances are difficult to distinguish from one another (i.e., ankylosis versus PFE or a mechanical obstruction of eruption) [6]. This finding is significant as nonsyndromic eruption disturbances are difficult to distinguish from one another (i.e., ankylosis versus delayed eruption) [6].

Theories Associated with Preemergent Tooth Eruption

The normal eruption of permanent teeth is highly varied and multifactorial. In order to properly categorize eruption disorders, it is critical to have a thorough understanding of normal eruption. Previous studies using implants and cephalometric radiography have shown that as the developing tooth bud forms, it remains in the same location in the bone [9], and it is from this point that eruption begins. The stages of eruption are determined by whether or not the tooth has emerged into the oral cavity.

While preemergent tooth eruption is defined by the initial eruptive movements that occur at the beginning of root formation and alveolar bone resorption in the eruption pathway, postemergent eruption is marked by the relatively rapid eruption after the tooth has entered the oral cavity. In preemergent eruption, the developing

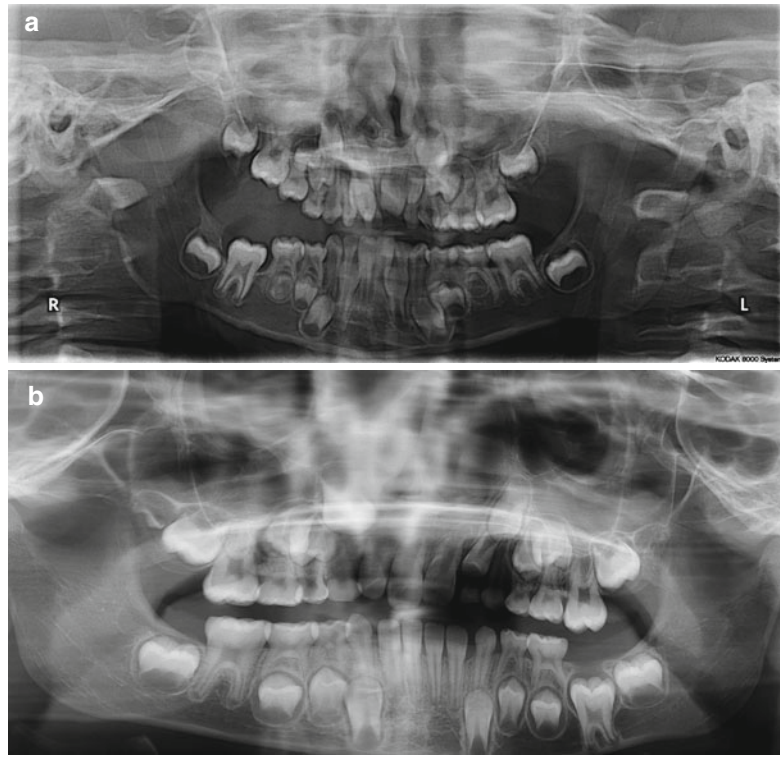


Fig. 2.1 (**a**) Type I primary failure of eruption in a developing dentition (7.5-year-old child). The right posterior segment shows a progressive eruption failure in the upper and lower arches with an eruption pathway that is clear. The left posterior segment does not appear affected. (**b**) Failure of eruption due to a bony pathway that is not clear. The alveolar bone can still be observed coronal to the erupting first molar in the lower left quadrant. This scenario often represents an idiopathic eruption failure due to some other pathology (i.e., not PFE or syndromic cause)

tooth moves occlusal and away from the point where root development is occurring. The precise mechanism of preemergent tooth eruption remains widely debated, but historical studies offer many lines of evidence for various theories [10–14]. Canonical theories in the recent literature include the "bone remodeling theory," the "hydrostatic theory," and the "follicle theory," which we will consider in detail below. Other historical theories that have been proposed include the "fibroblastic contraction theory" and those theories involving collagen maturation, localized variations in blood pressure or flow, alterations in the extracellular ground substances of the periodontal ligament, and the "root elongation theory." Briefly, the "fibroblastic contraction theory" hypothesizes that contraction of the fibroblast is responsible for the occlusal eruption of a tooth. Evidence in favor of this theory is the observation that fibroblasts move incisally along the erupting tooth [15] and that the contraction of fibroblasts generates significant force [16] to bring about eruption of the tooth. However, experiments in a rat model using lathyrogens (amino acid derivatives that cause defective fibril formation when applied to the PDL) to weaken the periodontal ligaments did not show a significantly different rate of eruption when compared to untreated rats [17]. The lack of PDL organization in the unerupted tooth does not support the eruptive theory of preemergent collagen maturation.

In the "root formation theory" [18, 19], it is hypothesized that lengthening of the root causes pressure on the apical portion of the alveolar bone leading to obvious propulsion of the tooth into the oral cavity. This theory suggests that the pressure of a developing tooth root on the underlying bone causes osteogenesis apically and bone resorption coronal to the erupting tooth. This argument is weakened by the fact the teeth with root apices surgically removed erupt normally [20] and that when eruption of a tooth is prevented by surgically ligating a premolar tooth bud to the lower border of the mandible in a dog model, the eruption path was still cleared [12]. The human equivalent of this phenomenon can be observed in accidental ligation of a developing permanent tooth to the adjacent mandibular bone

in the case of mandibular fractures *or* cases of PFE (Fig. 2.1a) where it is obvious that an eruption pathway has cleared but the tooth has failed to erupt to the occlusal plane. This is in direct contrast to a scenario where the eruption pathway is not cleared and eruption is essentially mechanically obstructed (Fig. 2.1b).

The remaining theories possibly offer the strongest explanation of tooth eruption, particularly in light of recent molecular biological advances [4, 5, 8]. However, these theories have fueled a debate of whether the bone "pushes" the teeth or the tooth "pulls" the bone with it during the eruption process. The "bone remodeling theory," originally proposed by Ten Cate [11] and endorsed by Wise et al. [14, 21], asserts that bone growth within the area apical to the developing tooth "pushes" the tooth during the eruption process. The consideration is whether this bone growth is causal and indeed represents the "motive force" described by Wise [14] or whether the bone growth occurs as a response to the occlusal movement of the developing tooth. Significant evidence exists in animal models to support this theory; experiments in rats reveal that the amount and duration of bone growth occurring at the apical base of the tooth is necessary and sufficient to propel the tooth into the oral cavity [14]. However, the fact remains that tooth eruption in humans occurs over a protracted time period with limited accessibility for study and the above studies in rodents may not parallel the human response; hence the complete understanding of events leading to eruption in humans remains elusive. The theory of preemergent eruption that most closely fits this model is the "dental follicle theory," which relates to the physiologic coupling of the resorptive eruption path formation and root development processes and contends that the dental follicle is necessary for eruption [12, 13]. This provides the most compelling explanation of the mechanism underlying tooth eruption. Moreover, it is this theory that aligns best with the "bone remodeling theory" and the association of the *PTH1R* gene with PFE. The follicle theory stems from classical studies in dogs where removal of the dental follicle prevented eruption [12, 13]. The dental follicle has since been shown to provide the environment and chemoattractants for monocytes to differentiate into osteoclasts [5]; this facilitates the bone resorption necessary for normal tooth eruption. Specifically, stellate reticulum cells found in the dental follicle are observed to secrete parathyroid hormone-related peptide (PTHrP), which induces overexpression of colony-stimulating factor-1 (CSF1) and receptor activator of NF-kappaB ligand (RANKL) responsible for osteoclastogenesis [24, 25]. A concomitant overexpression of BMP2 that leads to osteogenesis is occurring in the apical end of the dental follicle [24] in a chronological and spatial fashion [21].

The complete explanation of the physiologic coupling of the eruption and resorption processes associated with preemergent tooth eruption is not yet fully understood, but we know that the molecular crosstalk surrounding the erupting tooth is somehow activated upon completion of the crown. We can therefore postulate that the rate-limiting factor of preemergent eruption is the resorptive pathway formed by osteoclast cells. Accordingly, a tooth embedded in the bone has the potential to begin to erupt after root formation is completed, as long as the eruptive pathway is mechanically cleared at the appropriate developmental stage. This natural phenomenon of a "clear pathway" forms the basis of the diagnostic rubric for eruption disorders discussed in detail below.

Theories Associated with Postemergent Tooth Eruption

Although the dominant theories of tooth eruption appear to correlate with the preemergent stage, the postemergent stage of eruption is central to some theories. Postemergent tooth eruption is defined as the eruption stage of a developing tooth after it has broken through the gingiva into the oral cavity. This stage continues until the tooth reaches the level of the occlusal plane and is in complete function and the overall growth of the jaws has completed. Postemergent eruption is further broken into four phases, the pre-functional spurt (rapid phase), the juvenile occlusal equilibrium (slower phase), the pubertal or adolescent eruptive spurt, and the adult occlusal equilibrium. After the gingival barrier is broken, the postemergent

spurt results in rapid eruption until the tooth reaches the level of functional occlusion. As the tooth continues to erupt during its postemergent stage, the theory of "collagen cross-linking, contraction, and maturation" introduced above becomes more viable, due to the fact that the PDL indeed becomes more organized after the tooth comes into functional occlusion. This theory contends that increased organization in collagen cross-linking creates a propulsive thrust to facilitate eruption. Even though the tooth is subjected to occlusal forces, the actual eruption rate is increased.

The "hydrostatic pressure theory" occurs during postemergent eruption and is based on the ability of the extracellular matrix apical to the developing tooth to swell considerably (30–50 %) facilitating occlusal migration of the tooth [22, 23]. This theory asserts that increases in the periapical tissue fluid pressure (especially vasculature) push the tooth occlusally [11]. Moreover, human studies of premolar eruption following a local injection of vasodilators resulted in tooth eruption [23]. The argument against this theory is that a short-lived exposure to pharmacologic agents such as vasodilators would not be sufficient to sustain the long-term physiologic activity necessary for tooth eruption [14].

Postemergent Eruption and the Equilibrium Theory

After the functional plane is reached by the tooth, it undergoes the juvenile occlusal equilibrium, in which the eruption of the tooth is balanced in response to the vertical growth of the mandibular ramus. As the mandible grows vertically away from the maxilla, the teeth have more room to erupt occlusally in order to maintain occlusal contact with the opposing arch. This model of tooth eruption reinforces the idea that postemergent tooth eruption, after reaching functional occlusion, is controlled by forces impeding eruption, as opposed to encouraging forces. These balancing forces of masticatory function and the soft tissue pressures from the lips, cheeks, and tongue are the rate-limiting factors of post-functional occlusal eruption [1]. However, studies have shown that lasting eruptive movement occurs while the teeth are not in contact, which supports the idea that most of the eruptive control is based on the light and continuous force of the soft tissues. While the mechanism itself is not fully understood, when this process of vertical growth and occlusal tooth eruption is not adequately matched, eruption problems arise, as seen with issues of ankylosis and other eruption disorders which can result in areas of posterior open bites and over-closed jaw relationships.

The last phase of postemergent eruption is called the adult occlusal equilibrium. In this continuous phase, teeth will continue to erupt at an extremely slow rate throughout adult life. It has been demonstrated that if a tooth is lost at any age, the opposing tooth has the ability to erupt more rapidly, demonstrating that the eruption mechanism remains active throughout life and is capable of producing significant tooth during any stage in the life cycle. Finally, both pre- and postemergent eruption stages play a significant role in clinical eruption disorders and form the basis of our diagnostic approach reviewed below.

Diagnosis of Tooth Eruption Problems

While rodent models and molecular advances lend some support to the various theories of eruption, the details of the entire process of tooth eruption, including the micro- and macro-environment, remain poorly understood. Nonetheless, the biological facts surrounding the proposed theories provide the basis for understanding and diagnosing clinical disorders of eruption. Accordingly, adopting a diagnostic system that uses biologically rather than clinically based categories would provide a more effective means of accurately distinguishing eruption disorders [26]. Such categories should include those based on (1) a biological dysfunction such as PFE or eruption failure secondary to a genetic syndrome [16] and/or (2) a physical obstruction such as mechanical failure, cysts, and lateral tongue pressure, for instance. Impacted teeth may potentially belong to either of the above categories depending upon the location of the impacted tooth (i.e., palatal canine impaction versus buccal canine impaction). While the

occurrence of palatally impacted canines is hypothesized to be both multifactorial and genetic in origin [27–29], teeth can also become impacted secondary to an obstruction of the eruption pathway, such as crowded dental arches.

It is for this reason that a diagnostic rubric to distinguish eruption disorders must ask the necessary question "is the eruptive pathway clear?" [30]. The answer to this creates the foundation for determining whether the eruption failure is due to an obstruction or not. The diagnostic rubric shown in Fig. 2.2a is based on studies that examined characteristics of eruption disorders; the accompanying case study (Fig. 2.2b, c) nicely illustrates how this tool can be utilized for a clinical diagnosis. The combination of objective

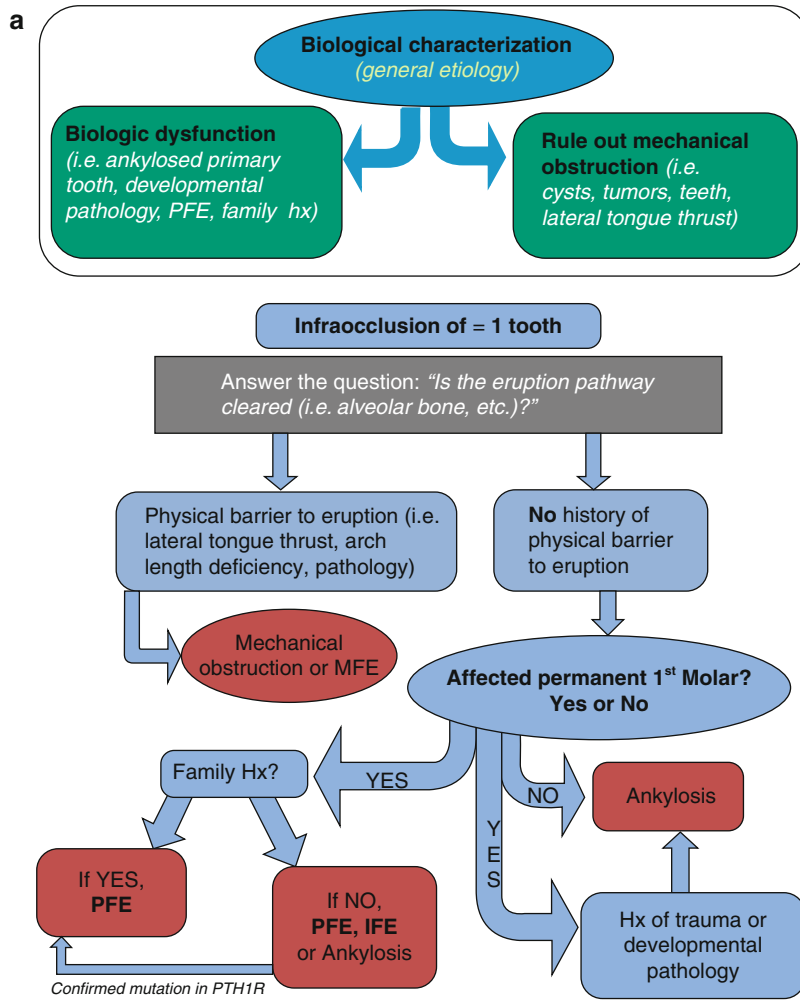
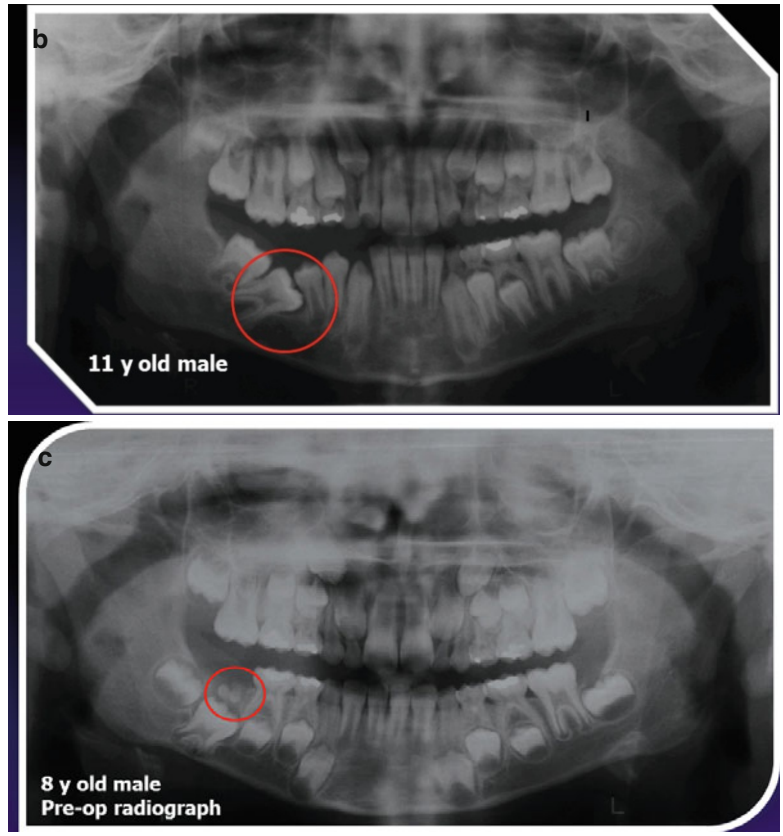


Fig. 2.2 (**a**) Diagnostic rubric for nonsyndromic eruption disorders based on a retrospective study of PFE subjects who carry a mutation in the *PTH1R* gene and those who do not. The flowchart provides a decision tree to allow a more systematic diagnosis of eruption disorders. Although there is still some uncertainty, initially sorting based on biological versus mechanical factors provides a sound basis to triage clinical scenarios (**b, c**). In the clinical scenario shown here, the natural history of this patient was extremely important. A differential diagnosis of the initial panoramic radiograph taken at the information gathering visit could be PFE or MFE. It was evident after acquiring historical radiographs (3 years earlier) that the cause of the eruption failure was an odontoma that was not removed before the 6 year molar was ready to erupt. Shown encircled is the unerupted 6 year molar in the 8 year old patient radiograph (2.2c) and the same molar at 11 years old that is now permanently impacted. Removal of the adjacent second premolar allowed eruption of the impacted molar and correction of the subsequent malocclusion. Reprinted from American Journal of Orthodontics and Dentofacial Orthopedics. Aug 144 (2)194–202

Fig. 2.2 (continued)



genetic information and clinical data from affected persons can be used to establish a genotype-phenotype correlation for PFE and, by extension, an objective diagnosis, i.e., determined by associating clinical (phenotypic) features with genetic (genotypic) analysis. Eruption disorders from a cohort of 64 patients were analyzed phenotypically and genetically in order to categorize them into clinical groups: (1) those definitively diagnosed with PFE through genetic analysis, (2) those that showed a mutation in *PTH1R* ($n=11$; genetic PFE cohort), (3) patients diagnosed with PFE based on clinical records alone ($n=47$; clinical PFE cohort), and (4) patients diagnosed with ankylosis based on clinical criteria ($N=6$; clinical ankylosis cohort). Those in the ankylosis cohort had a confirmed history of trauma or were treated with extraction of the affected tooth or teeth and had successful orthodontic treatment of the remaining teeth. All other subjects were diagnosed with PFE based on history of unsuccessful orthodontic treatment or genetic analysis. For those PFE patients who underwent genetic (mutational) analysis, a mutation or polymorphism in the *PTH1R* gene was identified in 11 patients, and an unclassified nonfunctional single nucleotide polymorphism in *PTH1R* was identified in the remaining [30]. Based on the findings of the above study, collectively all PFE subjects (genetic cohort) had at least one affected first permanent molar; the affected teeth in each dental quadrant were adjacent to one another and had a supracrestal presentation (i.e., completely cleared eruption pathway, with no alveolar bone occlusal to the affected tooth). These criteria represent the hallmark features of PFE versus a mechanical obstruction since it is based on cases of PFE genetic cohort that were categorized based on objective genetic confirmation. Other classifications of PFE include type I versus type II PFE [7, 26]. Type I is marked by a progressive open bite from the anterior to the posterior of the dental arches, while type II presents similarly but with greater although inadequate eruption of a second molar (Fig. 2.3). In either case, we speculate that

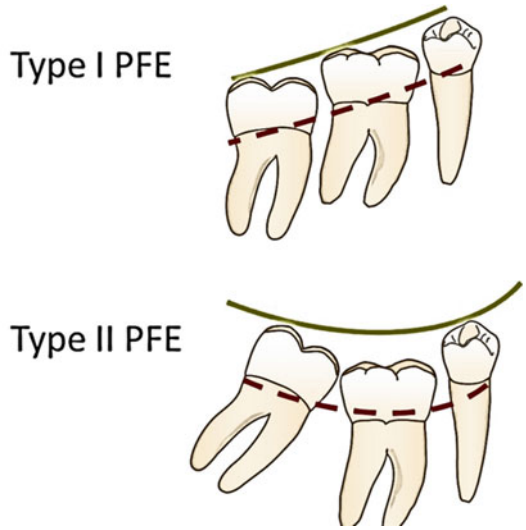


Fig. 2.3 Graphic representation of primary failure of eruption in type I and type II showing the progressive worsening of the posterior lateral open bite, to a lesser extent in type II PFE. Ankylosis occurs at about a 6.6% prevalence with mandibular primary molars more commonly affected than maxillary primary molars [38].

the eruption defect, which we now know is genetically controlled, is expressed at the same developmental time for all affected teeth but the predominant "molar" phenotype that we observe may be the result of a coordinated series of molecular events that act in a *temporally* and *spatially* specific manner such that posterior rather than anterior alveolar bone is affected. The exact reason for the variation in eruption potential between the first and second molars in type II is unknown but may be related to this same temporal and spatial specificity of expression.

Despite the more definitive criteria established through the eruption disorder rubric, difficulty still exists for those clinical situations that present with isolated ankylosis since it may initially appear indistinguishable from PFE. Ankylosis, or the fusion of a tooth to the bone in the absence of a periodontal ligament, can be thought of as a mechanical eruption failure, primarily because it can occur secondary to trauma and the fusion to the bone provides a mechanical barrier to eruption [31]. It is true that ankylosis can also occur secondarily from orthodontic forces applied to a tooth with a defective eruption mechanism as in PFE [3]. The diagnosis of ankylosis can at times be made radiographically by the absence of a periodontal ligament space [32] and based on the absence of physiologic mobility and the sharp solid sound on percussion of the tooth [31]. However, the determination of an absent periodontal ligament space can be often misinterpreted on a radiograph (e.g., if ankylosis occurs in facial/lingual root surfaces, the PDL loss will not be visible on a 2D radiograph), making the diagnosis of ankylosis somewhat subjective [33]. In these instances, ankylosis can be difficult to distinguish from PFE. This fact has been exemplified in two siblings previously diagnosed with ankylosis that were re-diagnosed as PFE following identification of a mutation in the *PTH1R* gene [4] (Figs. 2.4a and 2.5). The two siblings diagnosed with ankylosis, later determined to be PFE, also have an affected mother (not shown) and brother (Fig. 2.6a–c) who harbor a mutation in *PTH1R*. In both cases treatment with a continuous archwire failed to correct the posterior open bite (Fig. 2.4c). It is therefore quite reasonable that many other cases previously diagnosed as ankylosis are in fact PFE since the clinical presentation of PFE due to a genetic defect shows great clinical variation and is similar to ankylosis [4, 8]. Hence, the recent identification of a gene associated with PFE not only contributes to our understanding of the specific biological mechanism underlying the eruption process, but it provides greater clarity to the various terminologies used to describe eruption failure.

In some clinical situations however, the diagnosis of ankylosis is rather straightforward and not confused with eruption failure – specifically, ankylosis associated with retained deciduous teeth. Despite the apparent distinction between ankylosis and PFE, the actual biologic differences remain elusive; ankylosis is indeed similar to PFE in that a familial tendency has been reported and an overall prevalence of 8.9%. This percentage increases with age in children [39]. As discussed earlier, ankylosis of primary molars occurs most frequently with agenesis of second premolars, which are the most common congenitally missing teeth second only to third molars [33, 34]. It is not uncommon to see that resorp-

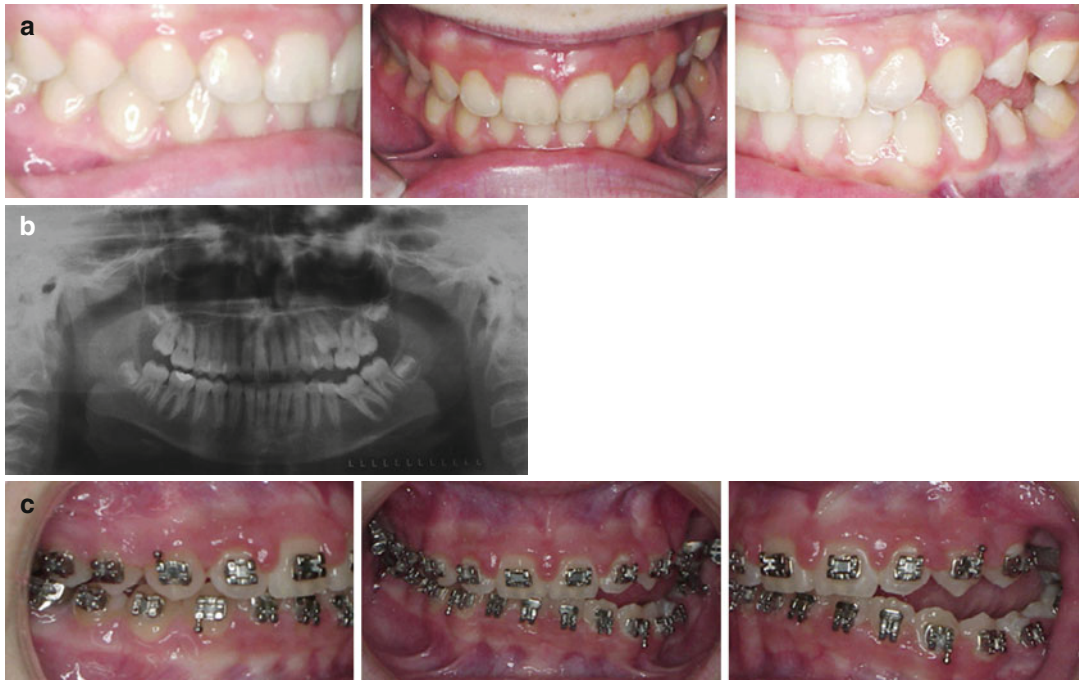


Fig. 2.4 (**a**) Clinical photographs of an 11-year 5-month-old patient who presented for orthodontic treatment and was subsequently diagnosed with ankylosis in the lower left posterior quadrant. (**b**) Pretreatment panoramic radiograph also reveals a blocked-out maxillary left second premolar due to the mesial tipping of the first molar – this was most likely due to the early exfoliation of the second primary molar. This patient did not have history of prior trauma, nor remarkable health history. (**c**) Subsequent treatment with a continuous archwire resulted in worsening of the lateral posterior open bite exemplifying the inability of teeth affected with PFE to respond to orthodontic forces. Several years following treatment, it was determined that the patient harbored a mutation in the *PTH1R* gene similar to her mother and two siblings

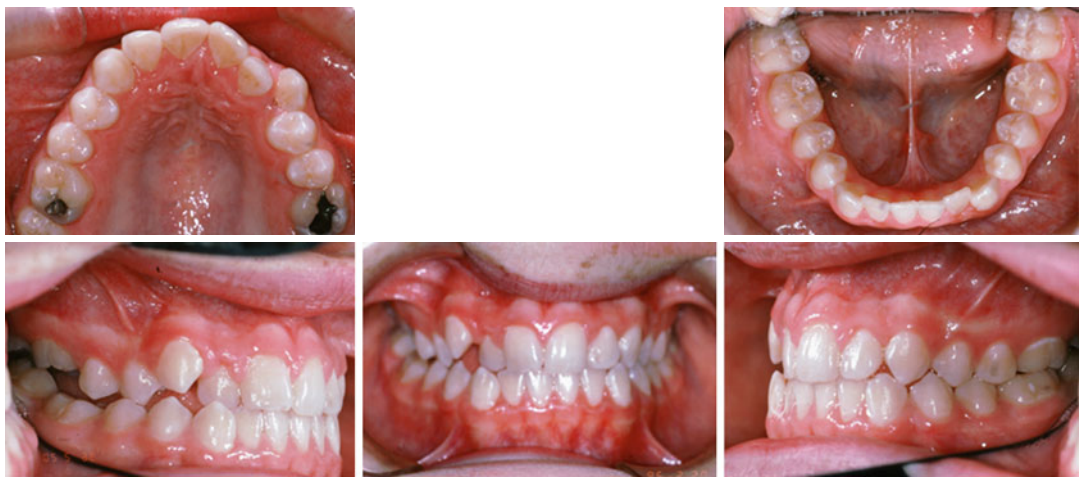


Fig. 2.5 Clinical photographs of a 16-year 5-month-old patient diagnosed with ankylosis of the lower right first molar using "bone sounding methods." This patient did not have history of prior trauma nor remarkable health history. Similar to his siblings, he was later diagnosed with PFE based on the presence of a mutation in the *PTH1R* gene

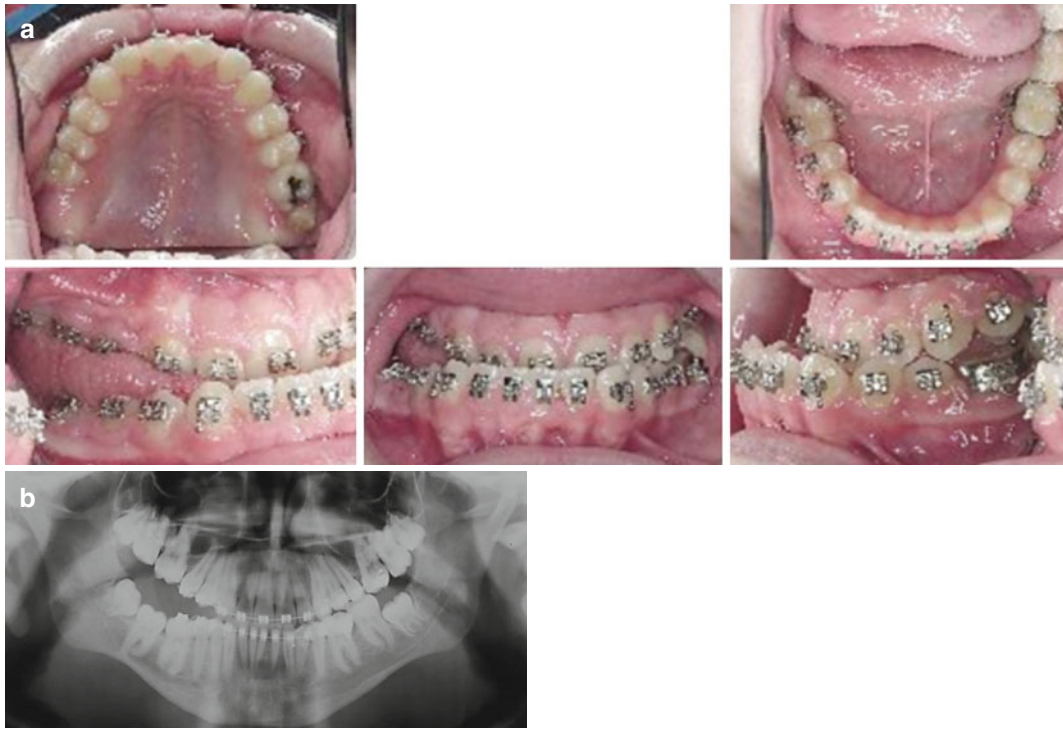


Fig. 2.6 (**a**) Clinical photographs of a 17-year 4-month-old patient undergoing orthodontic treatment primarily for his "underbite." This patient presented with a skeletal Class III malocclusion, severe anterior crossbite, and unilateral posterior crossbite on the right. Treatment with a continuous archwire did not correct the vertical posterior open bite (PFE). (**b**) Panoramic film illustrating eruption failure with a progressive worsening from anterior to posterior. Orthognathic surgical treatment (maxillary advancement) corrected his Class III malocclusion but not his posterior open bite due to PFE. This patient is the sibling of the patient in Figs. 2.4 and 2.5 also harboring a mutation in the *PTH1R* gene

tion of the primary roots may not occur or may be significantly delayed due to the absence of its permanent successor (Fig. 2.7a–c). If the primary tooth ankyloses in a young child [37], it may be overgrown by the surrounding dentition that continues to erupt and the area has further alveolar growth. Teeth that ankylose at a very young age can be completely overgrown by the surrounding dentition and bone creating a complicated surgical problem. If the second primary molar becomes ankylosed, the first permanent molar can tip over the primary molar's occlusal surface causing tipping and space loss. In this instance the primary molar can be built up with a stainless steel crown or by bonding resin to the primary molar occlusal surface to maintain an appropriate contact height with the first permanent molar. In many instances the primary molar that has a permanent successor will undergo normal root resorption and exfoliation requiring no special treatment.

Orthodontic and Surgical Tooth Eruption Therapy

The location of an impacted canine is closely related to the etiology. For instance, a buccally impacted canine is most often a result of crowded dental arches while a palatally impacted canine is often more closely related to a defect in the primary eruption mechanism [36]. Therefore, an approach to manage canines that are buccally impacted may often include extraction of the adjacent first premolars to create space and allow them to erupt into the arch unimpeded. In complete contrast to the

blocked-out buccal canine, the palatally impacted canine is more likely to occur with certain features including congenitally absent first premolars, small lateral incisors, enamel hypoplasia, and hypodivergent facial profile [29, 36]. It is essentially always necessary to surgically expose palatally impacted canine teeth and ligate with a bonded pad and chain using orthodontic traction (see Fig. 2.8a–c).

Cleidocranial dysplasia (OMIM #119600) is inherited as an autosomal dominate trait and is caused by mutations in the *RUNX2* gene that is an important signaling protein for normal bone formation and tooth eruption. Affected individuals have short stature, delayed closure of the cranial fontanelles, frontal bossing, supernumerary teeth, and abnormal eruption of the permanent dentition. The phenotype and severity are variable, and in cases of new mutations where there is no family history, it can be difficult to diagnose in children. Treatment will frequently require a team approach involving oral surgery to manage supernumerary teeth and help expose unerupted permanent teeth so they can be orthodontically brought into occlusion (Fig. 2.9a–h). The orthodontist and surgeon should evaluate the affected

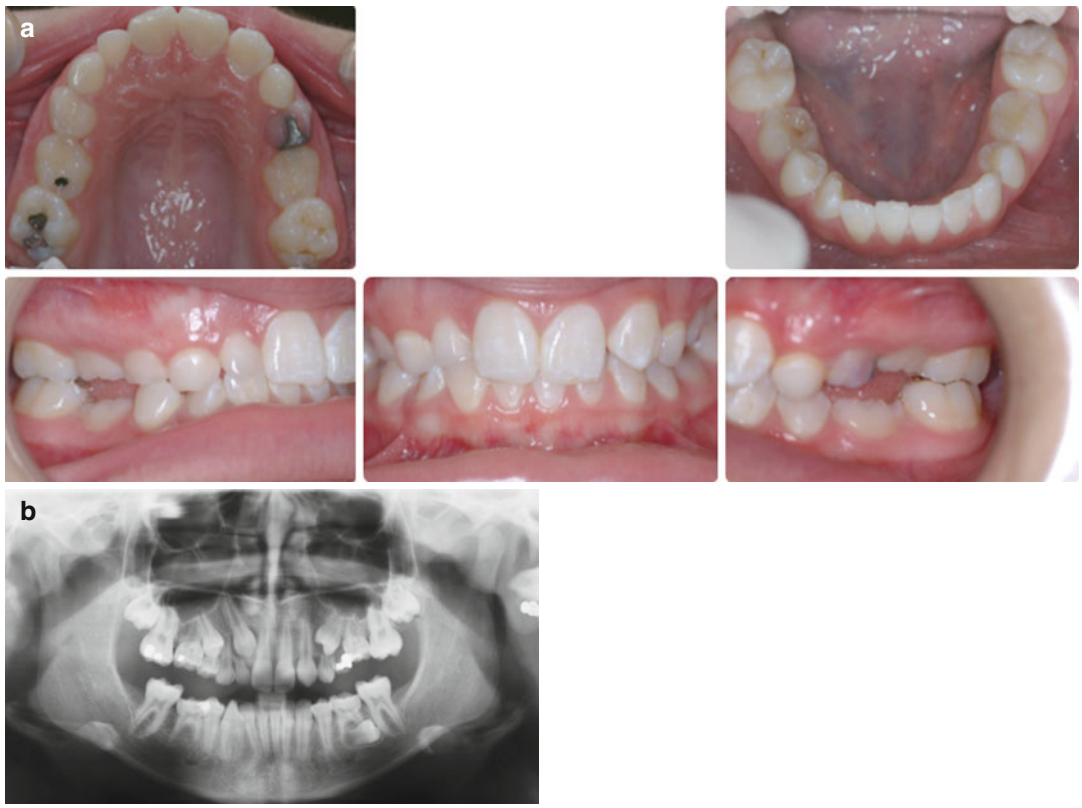


Fig. 2.7 (**a**) Example of ankylosis due to congenitally missing teeth (hypodontia). Initial clinical photographs of a 12-year 6-month-old patient with no history of prior trauma or a familial history of eruption disorders. (**b**) Panoramic radiograph illustrates the ankylosis of primary second molars and maxillary canines associated with congenitally missing maxillary second premolars and laterals as well as an ectopic LL5. The ankylosis is radiographically confirmed due to the infraocclusion of the select teeth, while the adjacent teeth display normal eruption. (**c**, **d**) Posttreatment clinical records including photographs and panoramic radiograph of the same patient after the extraction of primary canines, primary second molars, and ectopic mandibular left second premolar. The treatment plan included canine substitution to replace maxillary laterals and maintaining spaces for future implants or other prosthetics. After growth is completed, the patient will have the option of either implant/crowns, fixed bridges, or removable partial dentures

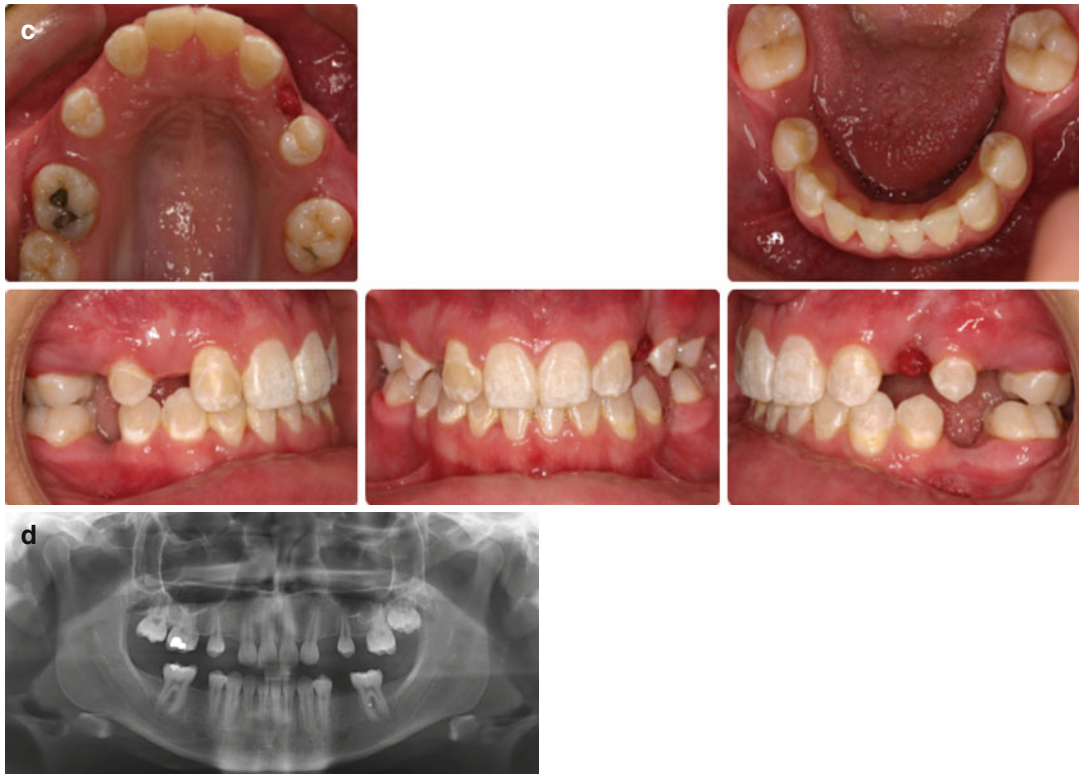


Fig. 2.7 (continued)

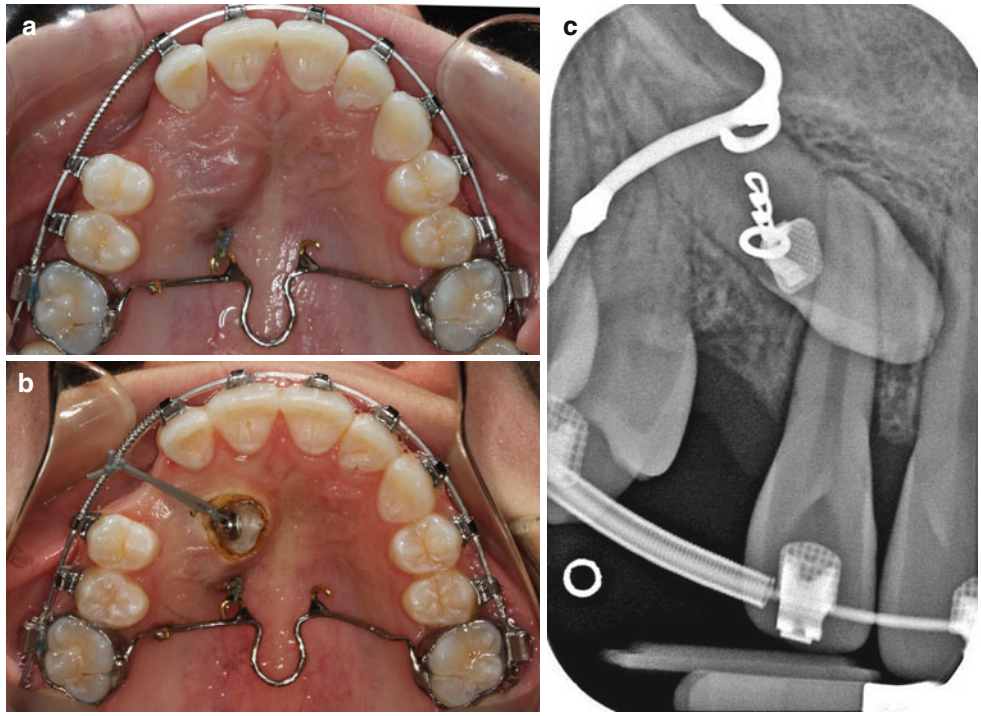


Fig. 2.8 (**a**) Occlusal photograph of an edentulous area where a palatally impacted canine failed to erupt. The patient has the contralateral canine that has erupted into the arch normally. (**b**) The canine was surgically exposed and bonded during the exposure surgery with a linked chain. Subsequent recovering of canine with soft tissue flap resulted in the need to perform soft tissue laser surgery to re-expose the canine. (**c**) A periapical radiograph reveals the canine that is still impacted but ligated to the bonded pad and chain. This clinical scenario will result in a successful result of the canine into the arch

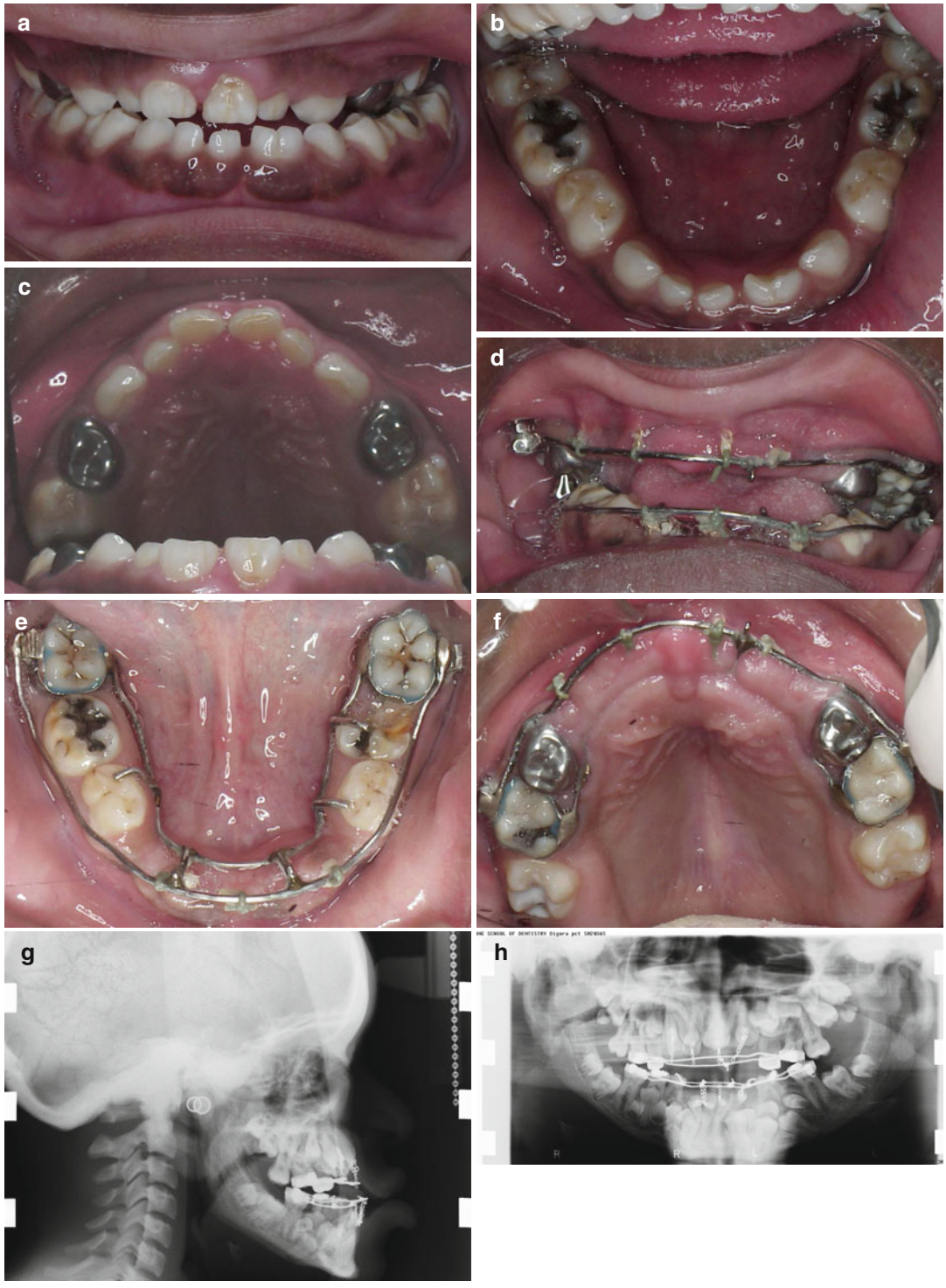


Fig. 2.9 Preoperative photos of a patient with CCD in retained primary dentition before (**a**–**c**) and after surgical exposures and ligation to a heavy mil arch bar to place traction on the teeth and bring them into occlusion (**d**–**f**). Radiographic evaluation of the same patient with cephalometric and panoramic films reveals the extent of the unerupted permanent teeth and impaction (**g**–**h**)

individual to determine what supernumerary teeth are best extracted and when is the optimal time to begin treatment. Some patients will benefit from craniofacial surgery to address the frontal bossing and craniofacial anomalies.

Conclusion

Whether the propulsive force of eruption is created by the bone "pushing" the tooth, or the tooth "pulling" the bone with it, the role of genes critical to the bone remodeling process is evident. Nonetheless, several gaps remain in our understanding of tooth eruption process. Future studies to evaluate additional candidate genes and investigate the role of environmental factors, such as trauma or orthodontic forces, will be essential to completely understand the normal eruption process. Indeed, as suggested by Berkovitz [10], a multifactorial theory (i.e., a combination of environmental factors and the canonical eruption theories) may largely explain the normal process of eruption, but the complex interplay of regulatory factors and environmental cues that contribute to this mechanism is still poorly understood. It is possible that each of the eruption theories above contributes to some portion of the whole process of tooth eruption. For instance, as the Hertwig epithelial root sheath (HERS) moves apically followed by its eventual disintegration and the formation of cementum during root formation (i.e., root elongation theory), it may signal the dental follicle and stellate reticulum cells to secrete mediators of bone remodeling (i.e., dental follicle theory). Mediators secreted from the dental follicle, such as VEGF, also cause angiogenesis and a concomitant increase in the apical tissue pressure propelling the tooth occlusally through the bone (i.e., hydrostatic pressure theory). It is likely that the biological mechanisms above represent portions of the cascade of events that facilitate normal eruption. An alteration of any part of these coordinated signaling events will lead to eruption failure.

From a clinical perspective, the ultimate goal is to understand the normal process of eruption in order to manage those cases of primary and permanent tooth eruption disorders. Primary teeth that fail to erupt fully or that have erupted but are secondarily submerged as the surrounding alveolar bone continues to develop around it are more likely to be ankylosed than permanent teeth with the same fate. However, isolated ankylosis of permanent first molars can be managed by extraction of the offending ankylosed tooth allowing for normal eruption of the second and third molars. A failure of the second and third molars to erupt fully would be pathognomonic for PFE. A hallmark of PFE is the response of affected teeth to orthodontic force; orthodontic force *will not* result in eruption of the affected tooth but will in fact lead to ankylosis of the affected teeth or intrusion of the adjacent teeth. Finally, another critical clue to diagnosing and managing cases of eruption failure is that the genetic association with *PTH1R* confirms the importance of determining a good family history. The American Society of Human Genetics suggested that taking a family history represents the gold standard in the diagnosis of and management of medical (and by extension, dental) disorders [35]. This judicious combination of clinical, biological, and genetic factors will change the way we have practiced in the past but will lead to the successful diagnosis and treatment of nearly all clinical disorders in the not so distant future.

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