

FIGURE 5-6 Facial asymmetry in an 11-year-old boy whose masseter muscle was largely missing on the left side. The muscle is an important part of the total soft tissue matrix; in its absence growth of the mandible in the affected area also is deficient. A, Age 4. B, Age 11. C, Age 17 after surgery to advance the mandible more on the left than right side. The soft tissue deficiency from the missing musculature on the left side still is evident.



FIGURE 5-7 Facial asymmetry in a 6-year-old girl with torticollis. Excessive muscle contraction can restrict growth in a way analogous to scarring after an injury. Despite surgical release of the contracted neck muscles at age 1, moderate facial asymmetry developed in this case, and a second surgical release of the muscles was performed at age 7. Note that the asymmetry affects the entire side of the face, not just the mandible.

the condylar cartilage is a prominent aspect; however, because the body of the mandible also is affected (Figure 5-10), hemimandibular hypertrophy now is considered a more accurate descriptive term. The excessive growth may stop spontaneously, but in severe cases removal of the affected condyle and reconstruction of the area is necessary.

Disturbances of Dental Development

Disturbances of dental development may accompany major congenital defects but are most significant as contributors to isolated Class I malocclusion. Significant disturbances include:

Congenitally Missing Teeth

Congenital absence of teeth results from disturbances during the initial stages of formation of a tooth—initiation and proliferation. *Anodontia*, the total absence of teeth, is the extreme form. The term *oligodontia* refers to congenital absence of many but not all teeth, whereas the rarely used term *hypodontia* implies the absence of only a few teeth. Since the primary tooth buds give rise to the permanent tooth buds, there will be no permanent tooth if its primary predecessor was missing. It is possible, however, for the primary teeth to be present and for some or all the permanent teeth to be absent.

Anodontia or oligodontia, the absence of all or most of the permanent teeth, is usually associated with a systemic

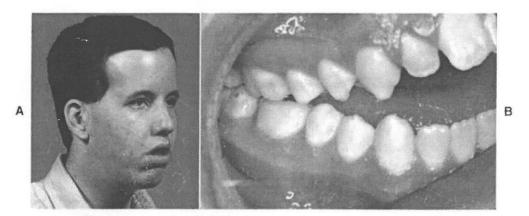


FIGURE 5-8 A, Lengthening of the lower face typically occurs in patients with muscle weakness syndromes, as in this 15-year-old boy with muscular dystrophy. B, Anterior open bite, as in this patient, usually (but not always) accompanies excessive face height in patients with muscular weakness.

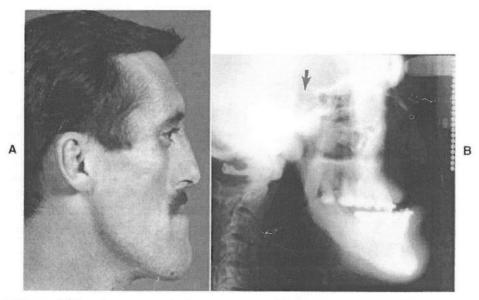


FIGURE 5-9 Profile view and cephalometric radiograph of a 32-year-old man with acromegaly, which was diagnosed 3 years previously after he went to his dentist because his lower jaw was coming forward. After irradiation of the anterior pituitary area, elevated growth hormone levels dropped and mandibular growth ceased. Note the enlargement of sella turcica and loss of definition of its bony outline in the cephalometric radiograph (arrow), reflecting the secretory tumor in that location. (From Proffit WR, White RP, Sarver DM. Contemporary Treatment of Dentofacial Deformity. St. Louis: Mosby; 2003.)

abnormality, ectodermal dysplasia. Individuals with ectodermal dysplasia have thin, sparse hair and an absence of sweat glands in addition to their characteristically missing teeth (Figure 5-11). Occasionally, oligodontia occurs in a patient with no apparent systemic problem or congenital syndrome. In these children, it appears as if there is a random pattern to the missing teeth.

Anodontia and oligodontia are rare, but hypodontia is a relatively common finding. A recent review concludes

that a polygenic multifactorial model of etiology is the best explanation of etiology. As a general rule, if only one or a few teeth are missing, the absent tooth will be the most distal tooth of any given type. If a molar tooth is congenitally missing, it is almost always the third molar; if an incisor is missing, it is nearly always the lateral; if a premolar is missing, it almost always is the second rather than the first. Rarely is a canine the only missing tooth.



FIGURE 5-10 A, Facial asymmetry in this 21-year-old woman developed gradually in her late teens, after orthodontic treatment for dental crowding during which there was no sign of jaw asymmetry, due to excessive growth of the mandible on the right side. B, The dental occlusion shows an open bite on the affected right side, reflecting the vertical component of the excessive growth. C, Note the grossly enlarged mandibular condyle on the right side. Why this type of excessive but histologically normal growth occurs, and why it is seen predominantly in females, is unknown.



FIGURE 5-11 A, A child with ectodermal dysplasia, in addition to the characteristic thin and light-colored hair, is likely to have an overclosed appearance because of lack of development of the alveolar processes. B, Panoramic radiograph of the same boy, showing the multiple missing teeth. When this many teeth are congenitally missing, ectodermal dysplasia is the most likely cause.

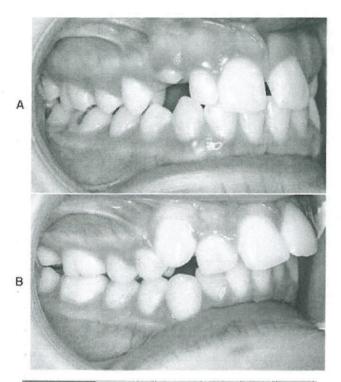


FIGURE 5-12 Disproportionately small (A) or large (B) maxillary lateral incisors are relatively common. This creates a tooth-size discrepancy that makes normal alignment and occlusion almost impossible. It is easier to build up small laterals than reduce the size of large ones, because dentin is likely to be exposed interproximally after more than 1-2 mm in width reduction.

Malformed and Supernumerary Teeth

Abnormalities in tooth size and shape result from disturbances during the morphodifferentiation stage of development, perhaps with some carryover from the histodifferentiation stage. The most common abnormality is a variation in size, particularly of maxillary lateral incisors (Figure 5-12) and second premolars. About 5% of the total population have a significant "tooth size discrepancy" because of disproportionate sizes of the upper and lower teeth. Unless the teeth are matched for size, normal occlusion is impossible. As might be expected, the most variable teeth, the maxillary lateral incisors, are the major culprits. The diagnosis of tooth size discrepancy, discussed in Chapter 6, is based on comparison of the widths of teeth to published tables of expected tooth sizes.

Occasionally, tooth buds may fuse or geminate (partially split) during their development. Fusion results in teeth with separate pulp chambers joined at the dentin, whereas gemination results in teeth with a common pulp chamber. The differentiation between gemination and fusion can be diffi-

cult and is usually confirmed by counting the number of teeth in an area. If the other central and both lateral incisors are present, a bifurcated central incisor is the result of either gemination or, less probably, fusion with a supernumerary incisor. On the other hand, if the lateral incisor on the affected side is missing, the problem probably is fusion of the central and lateral incisor buds. Normal occlusion, of course, is all but impossible in the presence of geminated, fused or otherwise malformed teeth.

Supernumerary or extra teeth also result from disturbances during the initiation and proliferation stages of dental development. The most common supernumerary tooth appears in the maxillary midline and is called a mesiodens. Supernumerary lateral incisors also occur; extra premolars occasionally appear; a few patients have fourth as well as third molars. The presence of an extra tooth obviously has great potential to disrupt normal occlusal development (Figure 5-13), and early intervention to remove it is usually required to obtain reasonable alignment and occlusal relationships. Multiple supernumerary teeth are most often seen in the congenital syndrome of cleidocranial dysplasia (see Figure 3-23), which is characterized by missing clavicles (collar bones), many supernumerary and unerupted teeth, and failure of the succedaneous teeth to erupt (see further discussion following).

Interference With Eruption

For a permanent tooth to erupt, the overlying bone as well as the primary tooth roots must resorb, and the tooth must make its way through the gingiva. Supernumerary teeth, sclerotic bone, and heavy fibrous gingiva can obstruct eruption.

All of these interferences are present in cleidocranial dysplasia. The multiple supernumerary teeth contribute an element of mechanical interference. More seriously, children with this condition have a defect in bone resorption, and the gingiva is quite heavy and fibrous. If the eruption path can be cleared, the permanent teeth will erupt (see Figure 3-23). To accomplish this, it is necessary not only to extract any supernumerary teeth that may be in the way but also to remove the bone overlying the permanent teeth and reflect the gingiva so that the teeth can break through into the mouth.

In patients with less severe interferences with eruption, delayed eruption of some permanent teeth contributes to malocclusion only when other teeth drift to improper positions in the arch. In 5% to 10% of U.S. children, at least one primary molar becomes ankylosed (fused to the bone) before it finally resorbs and exfoliates. Although this delays eruption of its permanent successor, there is usually no lasting effect, but a primary molar that becomes ankylosed at a young age can become totally submerged. In that case, the primary molar is unlikely to exfoliate, the permanent premolar is severely delayed, and drift of other permanent teeth into the space of the delayed tooth can create a significant malocclusion.

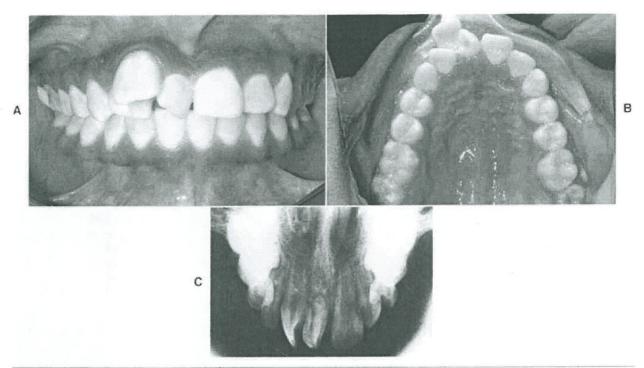


FIGURE 5-13 The maxillary midline is the most common location for a supernumerary tooth, which can be of almost any shape. The supernumerary may block the eruption of one or both the central incisors, or as in this girl, may separate them widely and also displace the lateral incisors.

Ectopic Eruption

Occasionally, malposition of a permanent tooth bud can lead to eruption in the wrong place. This condition is called *ectopic eruption* and is most likely to occur in the eruption of maxillary first molars. If the eruption path of the maxillary first molar carries it too far mesially at an early stage, the permanent molar is unable to erupt, and the root of the second primary molar may be damaged (Figure 5-14). The mesial position of the permanent molar means that the arch will be crowded unless the child receives treatment.

Ectopic eruption of other teeth is rare but can result in transposition of teeth or bizarre eruption positions. Mandibular second premolars sometimes erupt distally, and can end up beneath the permanent molars or even in the ramus (Figure 5-15).¹² A poor eruption direction of other teeth, especially maxillary canines, usually is due to the eruption path being altered by a lack of space.

Early Loss of Primary Teeth

When a unit within the dental arch is lost, the arch tends to contract and the space to close. At one time, this space closure was attributed entirely to mesial drift of posterior teeth, which in turn was confidently ascribed to forces from occlusion. Although a mesially-directed force when posterior teeth are brought together, ¹³ it probably is not a major factor in closure of spaces within the dental arches.



first molar apparently results from mesial position or inclination of the tooth bud. This causes the eruptive path of the first molar to contact the root of the primary second molar, as in this 8-year-old boy. The result is a delay in eruption of the first molar and root resorption of the second primary molar.

The contemporary view is that mesial drift is a phenomenon of the permanent molars only. The major reason these teeth move mesially when a space opens up is their mesial inclination, so that they erupt mesially as well as occlusally. Experimental data suggest that, rather than causing mesial

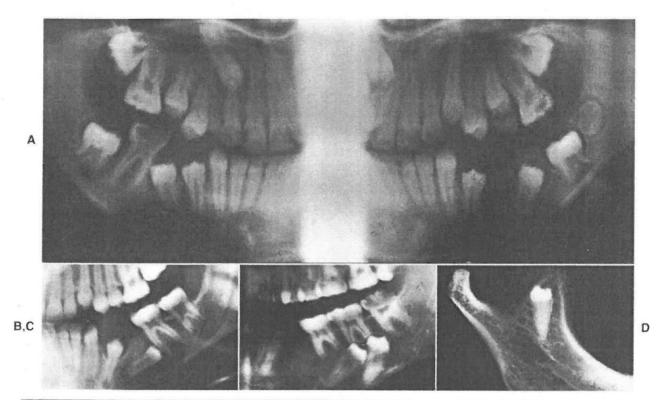


FIGURE 5-15 A, Mandibular second premolars tend to erupt tipped distally, and are prone to horizontal impaction, especially if the first molar is lost prematurely, but orthodontic correction is possible. B, If the first molar is lost prematurely and the unerupted second premolars are tipped distally, the second premolar can migrate back against the second molar, and may erupt in tight contact with it. C, Rarely, the premolars migrate distally beneath the permanent molars, and D, extreme migration into the mandibular ramus, even to the point that a premolar is found at the top of the coronoid process, is possible. (D, Courtesy Dr. K. Mitchell.)

drift, forces from occlusion actually retard it.¹⁴ In other words, a permanent molar is likely to drift mesially more rapidly in the absence of occlusal contacts than if they are present.

Mesial drift of the permanent first molar after a primary second molar is lost prematurely (Figure 5-16) can significantly contribute to the development of crowding in the posterior part of the dental arch. This has been a significant cause of crowding and malalignment of premolars in the past. For this reason, maintenance of the space after a primary second molar has been lost is indicated (see Chapter 11)

When a primary first molar or canine is lost prematurely, there is also a tendency for the space to close. This occurs primarily by distal drift of incisors, not by mesial drift of posterior teeth (Figure 5-16, *B*). The impetus for distal drift appears to have two sources: force from active contraction of transseptal fibers in the gingiva, and pressures from the lips and cheeks. ¹⁵ The pull from transseptal fibers probably is the more consistent contributor to this space closure tendency, whereas lip pressure adds a variable component (see the following section on equilibrium). If a primary canine or first molar is lost prematurely on only one side, the permanent

teeth drift distally only on that side, leading to an asymmetry in the occlusion as well as a tendency toward crowding.

From this description, it is apparent that early loss of primary teeth can cause crowding and malalignment within the dental arches. Is this a major cause of Class I crowding problems? The impact of fluoridation and other caries-preventive treatment on the prevalence of malocclusion indicates that it is not. Although fluoridation greatly reduced caries and early loss of primary teeth in typical U.S. communities, there was little or no impact on the prevalence of malocclusion. Even without fluoridation, in other words, most crowding problems are not caused by early loss of primary teeth.

Traumatic Displacement of Teeth

Almost all children fall and hit their teeth during their formative years. Occasionally, the impact is intense enough to knock out or severely displace a primary or permanent tooth. Dental trauma can lead to the development of malocclusion in three ways: (1) damage to permanent tooth buds from an injury to primary teeth, (2) drift of permanent teeth after premature loss of primary teeth, and (3) direct injury to permanent teeth.

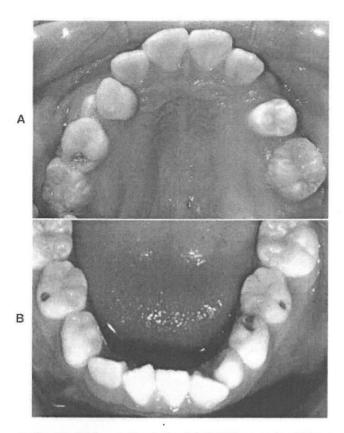
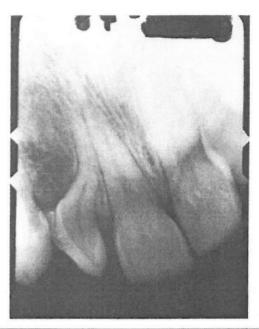


Figure 5-16 A, In this child's maxillary arch, early loss of the left second primary molar led to marked mesial drift of the permanent first molar. Note the space closure on the patient's left side (the right side of this mirror image photo), where almost no room for the second premolar remains. B, In this child's mandibular arch, early loss of the left primary canine led to a shift of the permanent incisors lingually and to the left.

Trauma to a primary tooth can displace the permanent tooth bud underlying it. There are two possible results. First, if the trauma occurs while the crown of the permanent tooth is forming, enamel formation will be disturbed and there will be a defect in the crown of the permanent tooth.

Second, if the trauma occurs after the crown is complete, the crown may be displaced relative to the root. Root formation may stop, leaving a permanently shortened root. More frequently, root formation continues, but the remaining portion of the root then forms at an angle to the traumatically displaced crown (Figure 5-17). This distortion of root form is called *dilaceration*, defined as a distorted root form. Dilaceration may result from mechanical interference with eruption (as from an ankylosed primary tooth that does not resorb), but its usual cause, particularly in permanent incisor teeth, is trauma to primary teeth that also displaced the permanent buds.

If distortion of root position is severe enough, it is almost impossible for the crown to assume its proper position—that



of this lateral incisor resulted from trauma at an earlier age that displaced the crown relative to the forming root.

might require the root to extend out through the alveolar bone. For this reason, it may be necessary to extract a severely dilacerated tooth. Traumatically displaced permanent teeth in children should be repositioned as early as possible (see Chapter 12). Immediately after the accident, an intact tooth usually can be moved back to its original position rapidly and easily. After healing (which takes 2 to 3 weeks), it is difficult to reposition the tooth, and ankylosis may develop that makes it impossible.

GENETIC INFLUENCES

A strong influence of heredity on facial features is obvious at a glance—it is easy to recognize familial tendencies in the tilt of the nose, the shape of the jaw, and the look of the smile. Certain types of malocclusion run in families. The Hapsburg jaw, the prognathic mandible of this European royal family, is the best known example (Figure 5-18), but dentists routinely see repeated instances of similar malocclusions in parents and their offspring. The pertinent question for the etiology of malocclusion is not whether there are inherited influences on the jaws and teeth, because obviously there are, but whether different types of malocclusion can be directly caused by inherited characteristics.

For much of the 20th century, thoughts about how malocclusion could be produced by inherited characteristics focused on two major possibilities. The first would be an inherited disproportion between the size of the teeth and the