Journal section: Periodontology
Publication Types: Review

doi:10.4317/medoral.16.e100
http://dx.doi.org/doi:10.4317/medoral.16.e100

Altered passive eruption (APE): A little -known clinical situation

Francisco Alpiste-Illueca

Professor of Periodontics, Valencia University Medical and Dental School. Valencia, Spain

Correspondence:
Clínica Odontológica
Gascó Oliag 1
46010 – Valencia (Spain)
francisco.alpiste@uv.es

Received: 28-02-2010
Accepted: 31-05-2010

Alpiste-Illueca F. Altered passive eruption (APE): A little -known clinical situation. Med Oral Patol Oral Cir Bucal. 2011 Jan 1;16 (1):e100-4.
http://www.medicinaoral.com/medoralfree01/v16i1/medoralv16i1p100.pdf

Article Number: 16906 http://www.medicinaoral.com/

eMail: medicina@medicinaoral.com
Indexed in:
Science Citation Index Expanded
Journal Citation Reports
Index Medicus, MEDLINE, PubMed
Scopus, Embase and Emcare
Indice Médico Español

Abstract

Gummy smile constitutes a relatively frequent aesthetic alteration characterized by excessive exhibition of the gums during smiling movements of the upper lip. It is the result of an inadequate relation between the lower edge of the upper lip, the positioning of the anterosuperior teeth, the location of the upper jaw, and the gingival margin position with respect to the dental crown.

Altered Passive Eruption (APE) is a clinical situation produced by excessive gum overlapping over the enamel limits, resulting in a short clinical crown appearance, that gives the sensation of hidden teeth. The term itself suggests the causal mechanism, i.e., failure in the passive phase of dental eruption, though there is no scientific evidence to support this. While there are some authors who consider APE to be a risk situation for periodontal health, its clearest clinical implication refers to oral esthetics.

APE is a factor that frequently contributes to the presence of a gummy or gingival smile, and it can easily be corrected by periodontal surgery. Nevertheless, it is essential to establish a correct differential diagnosis and good treatment plan. A literature review is presented of the dental eruption process, etiological hypotheses of APE, its morphologic classification, and its clinical relevance.

Key words: *Tooth eruption, altered passive eruption (APE), dentogingival junction (DGJ), gummy smile.*

Concept of Altered Passive Eruption

The dentogingival junction (DGJ) is habitually located close to the cementoenamel junction, the gingival margin slightly covering the limits of the dental crown (1). Different physiological situations do not reveal this morphological configuration, however, and the gingival margin tends to occupy a much more incisal position – thus giving rise to short clinical crowns. This variation in habitual morphology involving a more coronal periodontium has been referred to as altered passive eruption (APE) (2), or delayed passive eruption (3).

Tooth eruption

The term "altered passive eruption" refers to the supposed causal mechanism underlying this morphological variant. It should be remembered that tooth eruption comprises two phases: an active eruption phase which causes the tooth to emerge into the oral cavity, and a passive eruption phase involving apical migration of the soft tissues covering the crown of the tooth. The phenomena observed in both phases are reviewed below.

Active eruption phase

According to Steedle et al. (4), 6 stages – three prefunc-

tional and three postfunctional – can be cited in active eruption of the human tooth, as classified below:

-A follicular growth phase in which the tooth grows symmetrically within the crypt without undergoing displacement. This period lasts until the crown has calcified and 2-4 mm of root have formed.

-A pre-emergence eruptive outbreak beginning with displacement of the tooth in the occlusal direction, with a considerable increase when the cuspid of the crown approaches the immediate vicinity of the gums.

-A post-emergence eruptive outbreak at the moment when the tooth breaks through the gums and begins to emerge into the oral cavity. Maximum eruption velocity is reached in this stage. As the opposing cuspids approach each other, eruption gradually slows.

-Juvenile occlusal equilibrium. Once the tooth has reached occlusion with its antagonist, eruption movement ceases for some years.

-A puberal eruptive outbreak, characterized by rapid somatic growth during puberty and which at facial level is characterized mainly by an increase in lower facial height (5). Such bone growth is accompanied by a new active tooth eruption period that lasts at least 2-3 years, and concludes when the face reaches maturity. A relative equilibrium or balance is then restored at around 18 years of age.

-Adult occlusal equilibrium. Eruption does not cease abruptly on reaching physical maturity; rather, the potential is maintained throughout life, with small increments in lower facial height and tooth eruption.

Tooth eruptive movement is believed to involve a set of opposing forces (4). On one hand, the forces that promote eruption of the tooth originate from root formation, the increase in hydrostatic pressure at periapical level, the mechanisms of selective bone reabsorption and deposition around the tooth, and the contraction capacity of the periodontal ligament with its cells and fibers. In turn, the forces opposing those commented above (and thus tooth eruption) act in different ways in each of the different eruption stages.

Prior to gingival emergence of the tooth, eruptive displacement is regulated by reabsorption of the tissues standing in the way of tooth eruption (i.e., bone, root of the deciduous tooth and gums). The tooth follicle has been cited as the main element responsible for the mediation of growth factors that control this entire reabsorption process (6).

Once the tooth emerges through the gums, forces generated by occlusion constitute the most important factor regulating eruptive movement. It can be seen in animal and human studies that eruption restarts when teeth loose occlusal contact with their antagonist. continuous orthodontic forces applied on erupting teeth are able not only to stop eruption, but can also cause dental intrusion. Lee and Proffit (7) studied this eruption phase in humans and detected the existence of a circadian rhythm of eruption whereby significant tooth eruption occurs during the resting (sleeping) hours at night. Significant intrusion is likewise observed at around dinnertime and at breakfast, with slight intrusion during the rest of the day. The authors attributed this eruptive rhythm to the action of functional-type intraoral pressures (of the tongue, cheeks, lips and occlusal contact) generated by swallowing and speech, or the adoption of a lying or standing position. However, this rhythm also seems similar to that described for growth hormone (GH) concentrations in blood. In children, Risinger et al. (8) found the blood GH peak concentration to coincide with the greatest increase in tooth eruption during the day. In effect, the relationship between alterations in GH secretion and tooth eruption anomalies has already been reported in the past (2, 9).

Following the juvenile occlusal equilibrium stage, the puberal growth phase begins, and tooth eruption again activates. At the level of the face, the bony growth of the jaw causes the latter to move away from the base of the skull, increasing in size and modifying its morphology thanks to the reabsorption mechanisms and selective appositioning of bone. The direction in which the jaw is developed follows the general pattern of facial growth. Björk et al. (5) demonstrated an interrelation between facial growth and tooth eruption, whereby rotation of the lower jaw during its growth requires compensating adaptation mediated by eruption of the anterior and/or posterior teeth.

On reaching physical maturity in the adult, an occlusal equilibrium or balance is again established, though the teeth retain their capacity to erupt. Some studies have shown that in this period, eruption is regulated not only by occlusal contact but also by the periodontal ligament and supracrestal fiber complex (10, 11). It is known that forced extrusion of the tooth with orthodontic treatment generates tension in the supracrestal fibers that stimulates the osteoblasts to form new bone at crestal level – extruding both the tooth and the alveolar bone. Anthropometric studies have shown that eruption in adult individuals can compensate incisal attrition or wear of the teeth (12). In cases of intense occlusal wear, vertical tooth movement can compensate the existing attrition by up to 60%, without modifying facial height. However, the behavior of these latter two factors appears to depend on the age of the individual; accordingly, in young subjects, teeth without occlusal contact can exhibit uninterrupted eruption, while in adults supraeruption is limited. Studies in monkey teeth with a healthy periodontium show that when the antagonist tooth is extracted in adult individuals, limited supraeruption takes place in the tooth without occlusal contact, while in younger individuals the tooth supraerupts in a non-interrupted way. In relation to this, Compagnon et al. (13)

demonstrated in humans that in adult individuals with a healthy periodontium, the absence of a tooth causes limited eruption of the antagonist during the first years after extraction, with simultaneous eruption of tooth and periodontal tissues. In contrast, when the tooth is affected by periodontitis, supraeruption can cause it to exceed the line of the occlusal plane, and in this case the tooth is not followed by periodontal tissues, and thus shows its root.

It therefore can be concluded that the active eruption phase is a complex phenomenon. In the young individual it appears to be associated to general somatic development, as suggested by some authors who point to GH as the possible factor responsible (8). In such subjects, facial (and particularly maxillary) bone growth is compensated by tooth eruption – occlusal contact being the fundamental underlying mechanism. In contrast, adults, who have lost their capacity for bone growth, still preserve a considerable tooth eruptive potential.

Passive eruption phase

The term passive eruption is attributed to Gottlieb and Orban in 1933, and implies apical migration of the dentogingival junction (DGJ). Classically, the passive phase has been divided into four stages according to the location of the DGJ with respect to the cementoenamel line (14): (a) the DGJ is located on the enamel; (b) the epithelial attachment is located on the enamel and also on the root cement surface; (c) the epithelial attachment is entirely located on the cement; and (d) both the epithelial attachment and gingival margin lie apical to the cementoenamel junction. At present, only the first stage is considered to be physiological, while the remaining three are a consequence of pathological periodontal destruction processes.

Thus, from the current perspective, the active phase of eruption is defined by emerging motion of the tooth in the occlusal direction until the tooth reaches the occlusal plane of its antagonist. This vertical motion causes the gums to displace along with the crown. With the passive eruption phase, the gums migrate in the apical direction, with gradual exposure of the crown of the tooth and final stable localization of the DGJ at cervical level (Fig. 1) .

Fig. 1. Active and passive phases of dental eruption.

Altered passive eruption (APE)

Goldman and Cohen (2) defined APE as the situation in which "the gingival margin in the adult is located incisal to the cervical convexity of the crown and removed from the cementoenamel junction of the tooth". In the literature, the condition is also referred to as "retarded passive eruption" or "delayed passive eruption" (3). In any case, this clinical situation is attributed to failure in concluding the passive eruption phase.

Coslet et al. (15) morphologically classified APE into two types according to the location of the mucogingival junction with respect to the bone crest, and contemplating two subtypes in reference to the position of the bone crest with respect to the cementoenamel line. In type 1, and in addition to excessive gingival margin overlap on the crown, the dimension of the keratinized gum is considerable – the mucogingival junction being located more apically than the bone crest. In comparison, in type 2 the keratinized gingival band is narrow, the mucogingival junction coinciding at the level of the cementoenamel line. Both types are in turn classified into subtypes A and B. In subtype A, the distance between the bone crest and cementoenamel junction is 1.5 - 2 mm (which allows a normal dimension of connective fiber attachment in the root cement), while in subtype B the bone crest lies very close to, or even at the same level as the cementoenamel line (Fig. 2) . These authors found subtype B to be very frequent in children and adolescents still in the active eruption phase.

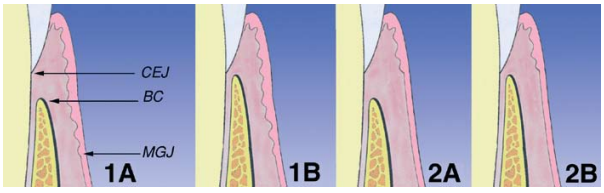

Fig. 2. Morphological classification of altered passive eruption (APE) according to Coslet (15).

Pathogenesis of APE

Although the literature points to failure or a delay in conclusion of the passive eruption phase as the mechanism underlying APE, few studies have evaluated the possible causes of such phase interruption. A number of factors have been proposed, such as interocclusal interference on the part of soft tissues during the eruptive phase, the presence of thick and fibrotic gums that tend to migrate more slowly during the passive phase than fine gingival tissue, and even a certain hereditary tendency in families with individuals presenting APE.

According to some authors, a bone crest close to the cementoenamel junction (Coslet subtype B) could impede gingival migration during the passive phase of eruption (16), thus establishing a distinction between altered passive eruption and altered active eruption. Based on this

consideration, the hypothesis has been proposed that two mechanisms intervene in APE, giving rise to two different morphological patterns at DGJ level (Fig. 2):

APE type 1. This type would be determined by exclusive failure of passive eruption, giving rise to excessive gingival overlap on the anatomical crown of the tooth, while in contrast the distance from the bone crest to the cementoenamel junction would be normal.

APE type 2. This type would be determined by primary failure of the active eruption phase, as a result of which the tooth would not emerge sufficiently from the alveolar bone, thereby leaving the cementoenamel junction very close to the bone crest. This situation would in turn prevent apical migration of the gums during the passive eruption phase.

However, even if this pathogenic hypothesis of APE were accepted, the literature fails to clarify the circumstances causing arrested tooth eruption and conditioning DGJ morphology. Many authors have investigated the causes and mechanisms that may lead to tooth eruption failure (17), though few studies have related such mechanisms to the morphology adopted by the coronal periodontium.

Thus, in the literature there are mechanical etiological factors that can modify the mechanism of eruption like root ankylosis, mechanical blocks to eruption such as the presence of supernumerary teeth, odontogenic tumors, cysts, interposed soft tissues between teeth, and deformities of the crown or root of teeth. In turn, certain endocrine alterations such as hypopituitarism, in addition to causing delayed dental eruption, give rise to incomplete formation of the root apex of the permanent teeth. Hypogonadism also produces delayed dental eruption. In Barbería's (9) clinical trial involving 50 children, some with hormonal diseases, the author reported that children who had a shortage of GH also presented a delay in dentition and bone age. As early as 1968, Goldman (2) reported that the association between hypothyroidism and the presence of APE is not infrequent. At present we have a clearer view of the possible relevance in the eruption process of certain hormones such as GH and insulin-like growth factors I and II (18), as well as of the thyroid hormones and epidermal growth factor (19).

Piattelli et al. (17) used the term "primary failure of eruption" in application to those cases with no apparent underlying cause, suggesting as etiological mechanisms an alteration of the metabolism or of the blood flow in the periodontal ligament. Proffit suggests this mechanism to explain certain open posterior bites. Primary failure of eruption in turn could be associated to other general alterations such as osteoporosis, sternocleidal dysplasia, achondroplasia, Gardner syndrome, chondroectodermal dysplasia and Down's syndrome.

Clinical relevance of APE

The prevalence of APE in the adult population has been little studied to date, possibly because of the lack of clear diagnostic criteria. Based on a series of 1025 patients with a mean age of 24.2 ± 6.2 years, Volchansky and Cleaton-Jones (3) recorded a 12.1% incidence of APE.

The literature discusses whether APE is a genuine risk to periodontal health, and points to difficulties in oral hygiene and a narrow connective tissue attachment as possible causes (15, 16). Coslet indicated that APE 2A, 1B and especially 2B are risk situations for health, particularly before dental treatment - identifying a narrow band of gum and the absence of connective insertion to the root as risk factors. Volchansky and Cleaton-Jones (3) reported a statistically significant relationship between the presence of APE and acute necrotizing ulcerative gingivitis, arguing that a deep gingival sulcus creates the necessary anaerobic conditions for the development of this infection.

Other authors indicate that an excess of gum on the tooth impedes oral hygiene and can cause disease especially in individuals who already have a high predisposition to periodontitis. Dello Russo (20) considers APE to be a clear risk situation for the periodontium in teeth that are going to be restored with total crowns or class II or III restorations. The author gives three reasons for this: the presence of a short clinical crown forces the clinician to make intrasulcular margin restorations; the difficulty of hygiene in this zone; and the absence of connective attachment to the radicular cement that can pose problems for the periodontal defenses.

The greatest clinical relevance of APE may be its esthetic consequences. In effect, when APE affects the upper anterior teeth, it usually alters dentofacial harmony – the patient in person often taking the initiative to consult the dental professional because of the short and hidden appearance of his or her teeth.

In relation to smile esthetics, a very important consideration is the relation of the gingival margins to the edge of the upper lip (21-23). An analysis of the esthetic alterations produced by APE in the anterosuperior sextant reveals the influence of three factors:

(a) The square appearance of the crowns. The gums positioned coronally over the tooth produce a square clinical crown silhouette, when the actual anatomical shape may be ovoid or elliptic, and thus esthetically much more attractive.

(b) On smiling, the gums are exposed by the upper lip. When such gum exposure exceeds 2-3 mm, it can produce a poor esthetic effect known as gummy smile (22).

(c) Flattened gingival festooning.

In APE, these three factors determine the so-called gummy smile, where in addition to producing excessive gingival exposure, the smile is globally lacking in expressivity (Fig. 3) .

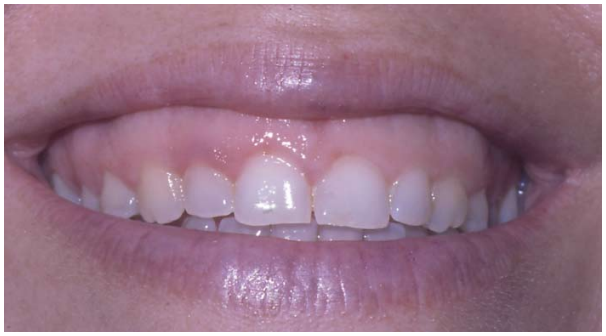

Fig. 3. Gummy smile associated to altered passive eruption (APE).

The presence of a gingival or gummy smile and of teeth with a short appearance are typical of APE, although they can also be manifestations of other entities involving a different etiology and treatment. Robbins (24) contemplates 5 clinical situations that can result in gingival smile: short upper lip, hypermobile upper lip, dentoalveolar upper extrusion, excessive upper jaw growth, and APE.

Reflections on APE

The prevalence of APE in the adult population has been little studied to date, probably because of a lack of clear diagnostic criteria. In the literature, the definition of APE is not very precise: "location of the gingival margin on the anatomical crown excessively occlusal and far from the cementoenamel junction in the adult individual" (15, 16). In order to establish a precise diagnosis, three factors should be evaluated: a) quantitative estimation of the gum overlap; b) clinical signs of EPA; and c) specification of the age from which we could affirm that there has been a failure to conclude eruption.

It is believed that during childhood and puberty, excess gum overlapping of teeth is frequent, because eruption has not yet been completed, and that eruption of the anterior and posterior teeth respectively finishes at the end of adolescence and in the third decade of life. Few literature references can be found describing the magnitude of gum overlapping that characterizes EPA. In my doctoral thesis, I found that gum overlapping of 20% of the anatomical crown length of a tooth to be the percentage most associated with EPA in upper anterior teeth (25).

It is interesting that in these literature reviews, none of the authors refer to the mechanism proposed by Björk (5), suggesting that dental eruption is an important mechanism for compensating maxillary bone growth, thus harmonizing facial development. From this point of view, one might think that the spatial relationship between both basal bones and the distance between them, both determined by the type of craniofacial bone growth pattern, could in part determine the amount of tooth eruption. Intermaxillary distance should be understood as the space that teeth have to erupt and contact with their antagonists in occlusion. This hypothesis contemplates an interrelation between bone facial growth and eruption of the teeth, which finally determines the facial appearance.

References

References with links to Crossref - DOI

1. Ainamo J, Löe H. Anatomical characteristics of gingiva. A clinical and microscopic study of the free and attached gingiva. J Periodontol. 1966;37:5-13.
2. Goldman HM, Cohen DW. Periodontal Therapy, de 4 St. Louis, C.V. Mosby Company 1968.
3. Volchansky A, Cleaton-Jones PE. Delayed passive eruption. A predisposing factor to Vincent´s infection? J Dent Asso S Africa 1974;29:291-294.
4. Steedle JR, Proffit WR. The pattern and control of eruptive tooth movements. Am J Orthod. 1985;87:56-66.
5. Björk A, Skieller V. Facial development and tooth eruption. An implant study at the age of puberty. Am J Orthod. 1972;62:339-83.
6. Fisher DA, Lakshmanan J. Metabolism and effects of epidermal growth factor and related growth factors in mammals. Endocr Rev. 1990;11:418-42.
7. Lee CF, Proffit WR. The daily rhythm of tooth eruption. Am J Orthod Dentofacial Orthop. 1995;107:38-47.
8. Risinger RK, Trentini CJ, Paterson RL, Proffit WR. The rhythms of human premolar eruption: a study using continuous observation. J Am Dent Assoc. 1996;127:1515-21.
9. Barbería Leache E, Marañes Pallardo JP, Mourelle Martínez MR, Moreno González JP. Tooth eruption in children with growth deficit. J Int Assoc Dent Child. 1988;19:29-35.
10. Ingber JS. Forced eruption. I. A method of treating isolated one and two wall infrabony osseous defects-rationale and case report. J Periodontol. 1974;45:199-206.
11. Simon JH, Lythgoe JB, Torabinejad M. Clinical and histologic evaluation of extruded endodontically treated teeth in dogs. Oral Surg Oral Med Oral Pathol. 1980;50:361-71.
12. Murphy T. Gradients of dentine exposure in human molar tooth attrition. Am J Phys Anthropol. 1959;17:179-86.
13. Compagnon D, Woda A. Supraeruption of the unopposed maxillary first molar. J Prosthet Dent. 1991;66:29-34.
14. Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. J Periodontol. 1961;32:12-35.
15. Coslet GJ, Vanarsdall R, Weisgold A. Diagnosis and classification of delayed passive eruption of the dentogingival junction in the adult. Alpha Omegan. 1977;10:24-8.
16. Evian CI, Cutler SA, Rosenberg ES, Shah RK. Altered passive eruption: the undiagnosed entity. J Am Dent Assoc. 1993;124:107-10.
17. Piattelli A, Eleuterio A. Primary failure of eruption. Acta Stomatol Belg. 1991;88:127-30.
18. Blom S, Holmstrup P, Dabelsteen E. The effect of insulin-like growth factor-I and human growth hormone on periodontal ligament fibroblast morphology, growth pattern, DNA synthesis, and receptor binding. J Periodontol. 1992;63:960-8.
19. Wise GE, Lin F. The molecular biology of initiation of tooth eruption. J Dent Res. 1995;74:303-6.
20. Dello Russo NM. Placement of crown margins in patients with altered passive eruption. Int J Periodontics Restorative Dent. 1984;4:58-65.
21. Garber DA, Salama MA. The aesthetic smile: diagnosis and treatment. Periodontol 2000. 1996;11:18-28.
22. Vig RG, Brundo GC. The kinetics of anterior tooth display. J Prosthet Dent. 1978;39:502-4.
23. Kois JC. Altering gingival levels: the restorative connection. I. Biologic variables. J Esthet Dent. 1994;6:3-9.
24. Robbins JW. Differential diagnosis and treatment of excess gingival display. Pract Periodontics Aesthet Dent. 1999;11:265-72.
25. Alpiste FM. Estudio de las peculiaridades morfológicas en la erupcion pasiva alterada: la relación dentogingival y dentoalveolar y el patrón facial. Doctoral Thesis. Universidad de Valencia. Facultad de Medicina y Odontología de Valencia. 1999.