Altered passive eruption (APE) and active secondary eruption (ASE): differential diagnosis and management

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Abstract

The process of tooth eruption consists of two phases, active and passive. While the distinction is unequivocal, the mechanism is ambiguous. This article compares and contrasts altered passive eruption (APE) and active secondary eruption (ASE). Although these phases present with similar clinical manifestations, each has its own etiology, physiogenesis, and pathogenesis. Furthermore, the differential diagnosis between the two, and that between other mirror imaging conditions, is essential for arriving at a definite diagnosis and correct treatment strategies, which may be similar but have different consequences. This article concludes with two case studies that show the management of APE and ASE, highlighting the treatment similarities and differences that are dependent on the specific etiology.

Introduction

Excessive maxillary gingival display, colloquially referred to as a “gummy smile,” detracts from pleasing anterior pink esthetics.\(^1\) People who have this esthetic aberration often suffer from social stigma, ridicule, and sarcasm, and are erroneously judged by others in terms of their intellect, trustworthiness, friendliness, and self-confidence.\(^2\) The degree of maxillary gingival exposure in patients with a gummy smile has attracted much attention in the dental literature. The accepted norm among clinicians for anterior pink esthetics is 3 mm; a measurement that is endorsed by innumerable texts and surveys on the subject.\(^3\)\(^-\)\(^6\) However, it should be remembered that opinions are subjective. Surveys of populations in different countries with disparate esthetic values and differing education levels, social mores, and religious taboos will have different outcomes. So while surveys can serve as a general guide, there are instances, for example, where discerning patients may regard any amount of gingival exposure or any minor gingival zenith disparity as detrimental to their smile and general appearance. Therefore, in order to avoid treatment failure, it is essential to take heed of patients’ wishes, irrespective of the clinical and laboratory prowess.\(^7\)

The clinical appearance of excessive gingival display is usually concurrent with short clinical crowns relating to dental or gingival factors. Dental causes include acute trauma, rampant decay, or attrition due to tooth surface loss (TSL), while gingival causes include gingival hypertrophy due to systemic illness or medication, or a coronal location of the gingival margin due to erratic eruption patterns. In addition, numerous mirroring conditions, unrelated to dental and/ or gingival factors, also manifest as a wide band of maxillary gingiva. As well as detracting from anterior pink esthetics, excessive maxillary gingival display can impact on oral health and the longevity of the dentition. The correction of these anomalies is often not straightforward, and its success frequently depends on correct diagnosis and subsequent appropriate treatment. Treatment may involve surgical, restorative, prosthetic or orthodontic treatment, musculature taming, or a combination of these modalities.

Definitions

Before embarking on this discussion, it is important to avoid ambiguity by defining some basic dentogingival anatomical terminology (Fig 1).

The cervicoincisal height of the crowns of natural teeth is classified into three categories: clinical length, anatomical length, and biological length. Clinical crown length is the visible height measured from the incisal edge to the most coronal aspect of the free gingival margin (FGM). The anatomical crown length, which may or may not be clinically visible, is the distance from the incisal edge to the cementoenamel junction (CEJ). Lastly, the biological crown length, which is invisible and determined either through radiography, tactile bone sounding, or by raising a mucogingival flap, is the distance from the incisal edge to the midfacial alveolar bone crest.
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(clinical crown length + sulcus depth + biologic width).

Other terminologies include the visible width of keratinized gingiva, which is measured from the coronal aspect of the FGM to the mucogingival junction; and the invisible biologic width, which is the linear measurement from the epithelial attachment to the alveolar bone crest, consisting of approximately 1 mm of connective tissue and 1 mm of epithelial attachment, and which is a prerequisite for periodontal integrity and health.

Physiogenesis of tooth eruption

The physiogenesis of tooth eruption consists of two distinct phases: active primary eruption, from when the tooth emerges into the oral cavity until it reaches its antagonist counterpart. This is followed by passive eruption, which results in apical migration of the gingiva to fully expose the clinical crown. Although the active phase (active primary eruption) predominates during the juvenile and puberty phases of development, it can nevertheless be triggered throughout life as active secondary eruption (ASE) when a tooth is unopposed by its antagonist. Situations causing interocclusal clearance include missing or extracted teeth, loss of tooth substrate following trauma, caries, TSL (tooth wear), periodontal disease or intentional space creation to stimulate this process, eg, the Dahl concept. Passive eruption is histologically divided into four stages:

![Dentogingival terminology diagram]

Fig 1 Dentogingival terminology.
1. The epithelium attachment of the den- 
togingival complex (DGC) is entirely 
located on enamel, coronal to the CEJ.
2. The epithelium attachment is partly 
on enamel and partly on cementum.
3. The epithelium attachment is entirely 
on cementum, with its coronal apex at 
the CEJ.
4. The epithelium attachment and FGM 
are apical to the CEJ (gingival reces-
sion).

The prevalence of altered passive erup-
tion (APE) is around 12% in the popu-
lation. Stages 1 to 3 are regarded as 
normal physiological processes, while 
the last stage, characterized by expo-
sure of cementum, is considered path-
ological and is caused by sequelae to 
periodontal disease or Type 3 ASE (ex-
plained below).

Etiology

Altered passive eruption (APE)

In normal circumstances, the DGC is lo-
eated near the CEJ, with the FGM slightly 
concealing the anatomical crown. How-
ever, in APE (also known as delayed 
passive eruption or retarded passive erup-
tion), the FGM is located more incis-
ally or coronally over the enamel, result-
ing in short clinical crown lengths and a 
so-called gummy smile.

The excessive gingival coverage 
of the anatomical crown seen in APE 
is caused by retardation of the pas-
sive eruption phase of tooth eruption. 
There are two morphological types 
of APE (Type 1 and 2), each with two fur-
ther subdivisions (Subtype A and B)14 
(Fig 2). In both types, the FGM is in a 
more coronal position.

The distinguishing feature of Type 1 
is a wide band of keratinized attached 
 gingiva with a grossly apical location 
of the mucogingival junction in relation 
to the alveolar crest. In subtype 1A, the 
distance from the CEJ to the bone crest 
is within the norm of 1.5 to 2 mm, while 
in subtype 1B, the CEJ is almost coinci-
dent with the alveolar crest.

In Type 2, the keratinized gingiva is 
narrower and the mucogingival junction 
closer to the CEJ, which could be at-
tributed to a failure of active or passive 
eruption. In subtype 2A, the distance 
between the CEJ and the alveolar bone 
is normal (accommodating the normal biologic width), while in subtype 2B, the 
CEJ almost approximates the alveolar 
crest, allowing little space for the epithe-
lium and connective tissue attachments.

Types 1B and 2B are common in ado-
lescence but rare in adulthood, ie, they 
are a transitional phase to the second-
ary dentition. In addition, Type 1B is the 
most commonly encountered, and has 
been termed altered active eruption, 
which is a failure in the active eruption 
phase. This interruption, or diapause, in

![Fig 2 Coslet’s altered passive eruption (APE) classification.](image-url)
the tooth eruption process is attributed to a variety of causes, including genetic predisposition, unfavorable environmental and systemic factors such as occlusal interferences, root ankylosis, incomplete root formation, space limitations, thick periodontal biotypes, cardiofacial spacial relationship of the jaws, and metabolic disruption (hormones and growth factors). Whether a single factor or multiple factors are responsible remains an enigma, since physiogenesis of tooth eruption is still unresolved, and further research is required for elucidating this complex phenomenon.

Active secondary eruption (ASE)

The occlusion of the dentition is maintained in a state of equilibrium due to intermittent stimulation by antagonist teeth during mastication. However, without this periodic stimulation, the eruption process is once again activated. ASE occurs when interocclusal space is created, either by a variety of unwanted causes such as missing antagonist teeth, TSL, dental caries, acute traumas, chronic periodontitis, mesial tilting of opposing teeth, or intentional orthodontic space creation.

Three types of ASE are described (Fig 3). In Type 1, the tooth suffers TSL and over-erupts to meet its antagonist counterpart in order to maintain occlusal vertical dimension (OVD) or face height, also known as dentoalveolar compensation. This is achieved by periodontal growth and the concomitant migration of the alveolar housing and periodontal ligament toward the occlusal plane, resulting in a short clinical crown...
length and disparate gingival zeniths.\textsuperscript{20}

It is important to realize that dentoalveolar compensation is a reaction to an event (interocclusal space and lack of intermittent stimulation), while the action of compensation is accomplished by triggering ASE with periodontal growth.

Type 2 is also characterized by periodontal growth, but is initiated by a missing antagonist tooth or antagonist tooth wear. In this scenario, the tooth overerupts coronal to the occlusal plane, maintaining its original crown length but with disparity of the gingival zeniths, as the tooth is more coronal compared with adjacent teeth.

Lastly, Type 3 (also known as extrusion) involves unilateral overeruption of the tooth in a coronal direction, beyond the occlusal plane, leaving the FGM in its original location, and therefore exposing the dentin and cementum of the root surface, resulting in a long clinical crown length. If the tooth is unopposed, it will continue erupting until reaching the antagonist alveolar ridge.\textsuperscript{21} This type of movement is distinguished from that occurring in stage 4 of APE (gingival recession), where the tooth maintains its occlusal position while the FGM recedes apically. In addition, depending on tooth configuration in the arch, ASE can also be accompanied by mesial drifting and tilting caused by missing teeth, tooth wear, or loss/fraction of dental restorations/prostheses.

As mentioned earlier, ASE can also be intentionally activated for correcting certain hard and soft tissue anomalies by means of orthodontic extrusion, moving the DGC in a coronal direction to line up erratic gingival zeniths, leveling arches, creating a favorable crown/root ratio, increasing bone volume prior to implant placement, and raising the OVD for compensating TSL using the Dahl principle.

**Fig 4** Excessive maxillary gingival display not caused by APE or ASE.

**Differential diagnosis and mirroring conditions**

Differential diagnosis between APE, ASE, and other mirroring conditions involves four diagnostic stages.

The first diagnostic stage is visual assessment, determining the degree of maxillary gingival exposure and the length of the teeth in the maxillary anterior sextant during smile dynamics (repose and laughter). If excessive gingival exposure is evident, but the teeth have an average width/length (w/l) ratio in the region of 78\%,\textsuperscript{22} with the length of the central incisors approximately 10.5 mm, and there are no signs of tooth wear, then the patient does not have APE or ASE\textsuperscript{23} (Fig 4). In these circumstances, the wide band of maxillary gingival display could be attributed to other mirroring conditions such as Angle Class II, division II;
Angle Class III; a short upper lip (average female lip is 21 mm, and male lip is 23 mm); hypertonicity of the orofacial muscles; dentoalveolar extrusion; protrusion of the pre-maxilla; increased vertical dimension of the maxilla; canting of the incisal plane and/or maxilla; or a combination of these anomalies. If any of these are suspected, crown lengthening is futile, as creating long clinical crown lengths will deteriorate white esthetics and further compound the already compromised pink esthetics. Therefore, other treatment options should be considered, depending on the etiology, including orthodontics, orthognathic surgery, local muscle relaxants, lip elongations with rhinoplasties, lip muscle detachments, myotomies, surgical lip repositioning, or a combination of these modalities. Hence, differential diagnosis is quintessential for informing patients of available therapies, and most importantly, for ensuring realistic outcomes and expectations.

In the presence of excessive maxillary gingival display and reduced clinical crown length, the differential diagnosis is either APE or ASE. APE is clinically diagnosed as short clinical crowns with 19% or more coverage of the anatomical crown by the overlying gingiva, which is flattened and festooned, with the interproximal papilla base wider than its coronal apex height. On the other hand, short clinical crowns with apparent tooth wear are diagnosed as ASE, usually by attrition at the incisal edges. The wear at the incisal edges is classified according to the Smith and Knight Tooth Wear Index (TWI) for attrition as follows: 0 = intact incisal edge; 1 = non-visualization of the enamel lobes; 2 = the dentin is seen by transparency; 3 = exposed dentin.

This is a simplistic index, since to date there is no single internationally recognized index of TSL for clinical and research applications that encompasses etiology, morphology, prevalence, terminology, pathogenesis, monitoring, or management of this insidious condition.

The second diagnostic stage is the location of the CEJ by periodontal probing. If TSL is evident, with the CEJ situated within the sulcus near the FGM, and the measurement from the CEJ to the incisal edge (anatomical crown length) is < 10.5 mm, the differential diagnosis is ASE. In the absence of tooth wear, reduced clinical crown length, and the CEJ not situated within the sulcus or near the FGM, the diagnosis is APE (see Fig 47).

The third diagnostic stage is bone sounding for ascertaining the type of APE. If the periodontal probe measurement from the FGM to the alveolar crest is > 3 mm, with an apical location of the mucogingival junction, then the diagnosis is APE Type 1A, and with a normal location of the mucogingival junction, the diagnosis is Type 2A. However, when the measurement from the FGM to the alveolar crest approximates 3 mm or less, and the mucogingival junction is apical or normal, the diagnosis is Type 1B and Type 2B, respectively (Fig 2).

The fourth diagnostic stage is radiographic evaluation, either by parallel profile radiography (PPRx) or cone beam computed tomography (CBCT), confirming the thickening of both the crestal alveolar bone and the connective tissue attachment, as well as the location of the CEJ. Typically, APE Types 1B and 2B
are diagnosed when there are smaller biologic widths (ie, a smaller distance from the CEJ to the crestal bone, leaving little space for the connective tissue and epithelial attachments), while in Types 1A and 2A, the relationship of the CEJ and alveolar crest is normal, around 1.5 to 2 mm.

Finally, the sulcus depth can be within normal parameters (1 mm), and hence its depth is an unreliable diagnostic indication of APE, and in the presence of short clinical crowns, bone sounding is a better diagnostic assessment tool (Table 1).

### Table 1  Differential diagnosis of APE and ASE

<table>
<thead>
<tr>
<th></th>
<th>APE</th>
<th>ASE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short clinical crown length</td>
<td>Yes</td>
<td>Yes (Type 1)</td>
</tr>
<tr>
<td>Long clinical crown length</td>
<td>No</td>
<td>Yes (Type 3)</td>
</tr>
<tr>
<td>Tooth wear</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Missing antagonist tooth/teeth</td>
<td>No</td>
<td>Yes (Types 2 or 3)</td>
</tr>
<tr>
<td>CEJ location to FGM</td>
<td>Apical to FGM</td>
<td>Normal parameters to FGM (Types 1 and 2) or Coronal to FGM (Type 3)</td>
</tr>
<tr>
<td>Alveolar crest location to CEJ/occlusal plane</td>
<td>Nearer CEJ (Types 1B and 2B) or Normal parameters to CEJ (Types 1A and 2A)</td>
<td>Nearer occlusal plane (Types 1 and 2) or Normal parameters to occlusal plane (Type 3)</td>
</tr>
<tr>
<td>Keratinized gingival width</td>
<td>Wide (Types 1A and 1B) or Normal parameters (Types 2A and 2B)</td>
<td>Wide (Types 1 and 2) or Normal parameters (Type 3)</td>
</tr>
<tr>
<td>Mucogingival location</td>
<td>Apical (Types 1A and 1B) or Normal parameters (Types 2A and 2B)</td>
<td>Normal parameters</td>
</tr>
</tbody>
</table>

APE - altered passive eruption  
ASE - active secondary eruption

**Management of APE and ASE**

The implications of APE to oral health are possible risk to periodontal pathosis, including chronic inflammation, acute necrotizing ulcerative gingivitis or gingival hyperplasia. However, the salient issues are either compromised pink esthetics, which prompts patients to seek professional help, or when restorative treatment is contemplated on the affected tooth/teeth. If uncorrected, and the restorative margins are placed supra- or equigingivally, the esthetic anomalies
include short wide crowns with a large w/l ratio, exposure and/or staining of the restoration finish line, persistence of excessive gingival display or gummy smile, and festooned flattened gingiva. All of these compromise the dentofacial esthetic composition or attractiveness of the smile. Conversely, subgingival margins can violate the biologic width, causing periodontal insult, again resulting in unsightly and unhealthy pink esthetics.

The clinical implications of ASE can be short or long capriciously shaped clinical crowns, depending on the type of active eruption; unsightly uneven incisal edges; erratic gingival zenith margins; and dentin exposure at the incisal edges and/or root surfaces, causing sensitivity and discoloration, combined with accelerated tooth substrate loss with possible fracture and/or endodontic involvement.

Following precise diagnosis, a management strategy is planned. This strategy may simply involve restorative corrections, or it may necessitate perioplastic surgery, orthodontics, orthognathic surgery, systemic or local muscle relaxants, or a combination of these disciplines.

If the diagnosis is limited to APE, there is only one option: periodontal plastic surgery for crown lengthening, either gingivectomy alone or gingivectomy with osseous resection, depending on the relationship of the alveolar crest to the CEJ, and the width of keratinized gingiva.

The outcomes of these procedures are predictable, with a high degree of patient satisfaction. The significance of bone sounding is in determining which crown-lengthening procedure is required. With Types 1A and 2A, and assuming adequate keratinized tissue, gingivectomy alone suffices, leaving 3 mm from the FGM to the alveolar crest for maintaining the biologic width and gingival sulcus. Conversely, in Types 1B and 2B, gingivectomy plus osseous recession by ostectomy and osteoplasty are indicated for removing crestal bone and creating the necessary 3-mm space from the FGM (2 mm for biologic width, and 1 mm for sulcus). However, depending on the location of the mucogingival junction, the flap design and repositioning may require modification for ensuring an adequate band of keratinized gingiva for periodontal health. The incisions and tissue resection (soft and hard) are limited to the facial aspect, and involve reestablishing the correct gingival scallops for mimicking underlying osseous architecture. In addition, gingivectomy incisions are confined within the facial line angles for creating the correct gingival scallop, without resecting the interproximal papillae, to avoid creating unwanted gingival embrasures causing so-called “black triangles.” The soft tissue healing process is erratic, at times with postsurgical recession or a coronal rebound of the FGM. Furthermore, the healing period is variable, from a few weeks to several years, depending on surgical protocols, as well as patient constitutional and systemic factors. For patients with high lip lines, minor gingival zenith aberrations after healing can be corrected with judicial incisions with scalpel blades or diode lasers.

Correcting asymmetrical gingival margins for ASE is also accomplished with perioplastic surgery, or alternatively by orthodontic intrusion, usually in combination with restorative or prosthetic modalities for replacing the lost enam-
el and dentin, depending on whether the etiology is TSL or unopposed antagonist(s). While for APE, excess tissue removal establishes correct gingival zeniths and exposes the clinical crown to its proper w/l ratio, a similar protocol for ASE results in elective exposure of the tooth root surface, which may require desensitizing agents or restorative coverage for protecting the vulnerable exposed dentin. The second option for correcting ASE is orthodontic intrusion for reestablishing correct gingival zenith heights, plus either resin-based composite or porcelain restorations for replacing TSL. Furthermore, the etiological causes should be addressed, missing teeth replaced, counseling given for mitigating TSL, and nightguards provided to curb and minimize occlusal parafunctional activities. Finally, it is prudent to foresee the consequences of extractions for preventing ASE, and patient counseling is essential to limit future unnecessary and costly treatment.

The following two case studies illustrate the correction of APE and ASE using a combination of perioplastic surgery and restorative/prosthetic approaches for the restitution of pink and white anterior esthetics. They highlight the similarities and differences in treatment modalities for short clinical crowns with different etiologies.

**Case study 1: APE**

A 24-year-old female was referred to the dental hospital with a request for improving her anterior dental esthetics. Counseling revealed that an accident 5 years before had caused the fracture of her left central incisor, necessitating a full-coverage crown. The right central incisor escaped major trauma but incurred minor incisal edge damage, which was repaired with a resin-based composite restoration. Although unable to fully articulate her esthetic anomalies, she was generally unhappy with the appearance of her smile, and described her anterior teeth as “short and fat.” Esthetic analysis revealed a lack of tooth display during the habitual lip position (Fig 5); a reversed smile line during repose smiling and laughter; a small median diastema; eschewed dental midline; and incisal plane canting to the left; an anomaly generally regarded as one of the most displeasing features of a smile (Fig 6). Further scrutiny established short clinical crown lengths of the canines and central incisors, lack of dominance of the central incisors, and a defective bulbous crown on the left central incisor causing bruising of the gingival margin. In addition, the left lateral incisor sagged coronal to the incisal plane, while the left canine displayed a distofacial rotation, both of which disrupted the pleasing distal incisal embrasure progression (Fig 7). The gingival zeniths of the maxillary sextant were erratic, caused by the short clinical crown lengths of the canines and central incisors. This resulted in excessive gingival exposure, especially on the left. In addition, a wide band of keratinized attached gingiva was evident apical to the anterior teeth, and the canine gingival zeniths were coronal to that of the central incisors, creating a gingival esthetic line (GAL) Class IV on both sides.

The provisional diagnosis was APE Type 1A on the central incisors and left
lateral incisor, and Type 1B on the canines. Bone sounding established that surgical crown lengthening would be necessary, involving gingivectomy alone for teeth 11, 21, and 22, and gingivectomy with osseous recession for teeth 13 and 23 to create space for the biological width. A diagnostic wax-up (Fig 8) was utilized for simulating the correct w/l ratio of the central incisors to 80% (8.5 mm ÷ 10.5 mm = 0.8), increasing their length from 8 to 10.5 mm to allow greater tooth exposure during the habitual lip position, which is regarded as a prerequisite for a youthful appearance.50

Following prophylaxis, the offending crown on the left central incisor was removed, revealing circumferential inflammation of the gingival margins (Figs 9

Fig 5 Dentofacial preoperative view of the habitual lip position showing lack of anterior maxillary tooth display.

Fig 6 Dentofacial preoperative view during smiling showing a reversed smile line, median diastema, slanted dental midline, and incisal plane canting to the left.

Fig 7 Dental preoperative view showing lack of dominance of the central incisors, short clinical crowns of the central incisors and canines, and disruptive distoincisal embrasure progression on the left side of the maxillary sextant.

Fig 8 Diagnostic wax-up of the central incisors for increasing the length of the porcelain laminate veneer (PLV) on tooth 11, and the all-ceramic crown on tooth 21, with a corresponding silicone index for an intraoral mock-up and temporization.
A chairside, correctly fitting acrylic temporary restoration was fabricated for promoting gingival health.

The subsequent stage was esthetic crown lengthening of the canines and left lateral incisor for creating a GAL Class I on both the right and left sides of the maxillary sextant (Figs 11 and 12). After bone sounding under local anesthesia, bleeding points were placed for guiding the ensuing surgical procedures (Fig 13). The initial step was carrying out a gingivectomy by incising...
Fig 14  Gingivectomy on teeth 22 and 23 prior to discarding the excess gingival tissue.

Fig 15  Gingivectomy on tooth 13 to increase the clinical crown length for establishing the correct w/l ratio of 80%.

Fig 16  Papillae preservation incisions followed by flap elevation to expose osseous bone crest approximating the CEJ, consistent with APE Type 1B.

Fig 17  Interrupted sutures (5-0 nonresorbable) securing the flap around the left canine following ostectomy and osteoplasty.

Fig 18  Ten-day healing prior to suture removal.

Fig 19  Five-week healing showing establishment of GAL Class I on the right and left sides (compare with Fig 7).
excess tissue to the correct gingival zenith heights using a No. 15c scalpel blade, guided by the bleeding points placed beforehand (Figs 14 and 15). A full-thickness mucoperiosteal flap was raised around the left canine to expose the osseous crest, which was adjacent to the CEJ, confirming the provisional diagnosis of APE Type 1B (Fig 16). An ostectomy and osteoplasty were performed around the left canine, creating a 3-mm space for the biologic width and sulcus from the gingivectomy margin to the midfacial osseous crest. A similar procedure was performed on the right canine, but using a flapless approach. Since the left lateral incisor was an APE Type 1A, surgery was limited to a gingivectomy without osseous contouring. The crown lengthening was completed by suturing the flap around the left canine with 5-0 nonresorbable interrupted sutures (Fig 17). Uneventful healing is shown in Figures 18 and 19, after
10 days and 5 weeks, respectively. At a later date, crown lengthening, limited to gingivectomy, was carried out for the central incisors for establishing a more apical position of the gingival zeniths.

A further 2 months was allowed for the tissues to mature and gingival zeniths to stabilize before proceeding with tooth preparations for the central incisors. During this healing phase, the patient bleached her teeth to improve the shade prior to delivery of the definitive restorations. The preparation for the full-coverage all-ceramic crown on the left central incisor was refined, and the acrylic temporary crown relined until gingival health was evident (Figs 20 and 21). On the right central incisor, the porcelain laminate veneer (PLV) preparation was minimal, confined to the enamel layer, and defining the cervical, interproximal, and palatal finish lines (Fig 22). Following the fabrication of the feldspathic PLV and all-ceramic IPS e.max (Ivoclar Vivadent) crown in the dental laboratory, the latter was bonded to the tooth substrate using an adhesive protocol. The teeth were isolated with gingival retraction cord, a wooden wedge, and polytetrafluoroethylene (PTFE) tape during the cementation procedure (Fig 23). A resin-based cement (Variolink Veneer, Ivoclar Vivadent) with a dentin bonding agent (OptiBond XTR, Kerr) adhered the ceramic restorations with a hermetic seal at the margins. Finally, judicial esthetic contouring was carried out on the left lateral incisor and canine to harmonize the distal incisal embrasure progression. The postoperative results show integration of the indirect ceramic restorations with the soft tissue, dominance of the central incisors, increased crown lengths of the canines and central incisors with the correct w/l ratios, elimination of the incisal cant on the left, and GAL Class I on both sides (Figs 24 to 27). The dentofacial view shows acceptable maxillary gingival exposure during a relaxed smile, a smile line coincident with the curvature of the lower lip, and increased tooth exposure during the habitual lip position (Figs 28 and 29).

**Fig 24** Postoperative view in centric occlusion showing increased clinical crown lengths of the maxillary canines and central incisors, together with increased anterior overbite of the latter, and impeccable gingival health (compare with Fig 7).

**Fig 25** Postoperative anterior view showing GAL Class I on the right and left maxillary sextant. Notice the esthetic contouring by enameloplasty on the left lateral incisor and canine to establish correct distal progression of the incisal embrasures.
Case study 2: ASE

A 45-year-old woman attended the specialty clinic of the dental hospital complaining of a “crooked smile” and gaps between her teeth. The dentofacial composition revealed a substantial median maxillary diastema, TSL, a reversed smile line, lack of dominance of the central incisors, and excessive gingival display apical to the canines (Fig 30). Intraoral examination findings included short clinical crown length of the canines, considerable TSL on the facial surfaces of the maxillary anterior teeth due to abrasion, and serrated incisal edges caused by

Fig 26  Postoperative right lateral view showing GAL Class I and correct w/l ratios of the right canine and central incisor following esthetic crown lengthening.

Fig 27  Postoperative left lateral view showing GAL Class I and correct w/l ratios of the left canine and central incisor. Notice the esthetic contouring by enamelo-plasty on the left lateral incisor and canine to establish correct distal progression of the incisal embrasures.

Fig 28  Dentofacial postoperative view during a relaxed smile showing coincidence of the incisal plane with the curvature of the lower lip, acceptable maxillary gingival exposure, perpendicular dental midline, and elimination of the cant on the left (compare with Fig 6).

Fig 29  Dentofacial postoperative view showing maxillary incisor display during the habitual lip position (compare with Fig 5).
attrition. In addition, cervical decay was evident on the right canine, as well as a defective composite veneer on the right lateral incisor (Fig 31). The gingival zeniths of the canines on both sides were coronal to that of the central incisors, consistent with a GAL Class IV gingival scalloping of the maxillary sextant. The occlusal view confirmed tooth wear with dentin exposure, scale 3, according to the Smith and Knight TWI classification (Fig 32). Other dental issues such as an anterior mandibular diastema, defective fixed partial dentures, and missing mandibular teeth also required addressing at a later date.

The diagnosis for the short clinical crowns of the canines was ASE Type I, caused by attrition and subsequent dentoalveolar compensation moving the gingival zeniths (and dentogingival complex) closer to the occlusal plane. The treatment plan for the maxillary sextant was, firstly, esthetic crown lengthening to increase the clinical crown lengths of the canines and thereby reduce gingival exposure during smiling. Secondly, prepless direct composite restorations to establish correct w/l ratios of the central incisors, eliminate the median diastema, replace the defective filling on the right lateral incisor, remove the cervical decay on the right canine, and replace the facial surface enamel loss caused by abrasion.

After prophylaxis, a diagnostic wax-up with a silicone index was fabricated and used as a framework for guiding the esthetic crown lengthening and resin-based composite restorations (Fig 33). To achieve correct proportion for the left canine, the existing clinical crown length of 8 mm (Fig 34) required increasing to
Fig 33  Diagnostic wax-up and silicone index for guiding crown lengthening and composite fillings.

Fig 34  Existing clinical crown length of left canine of 8 mm.

Fig 35  Proposed clinical crown length of left canine of 10.5 mm.

Fig 36  Bone sounding of 2.5 mm from the FGM to the midfacial osseous crest.

Fig 37  Gingivectomy using a No. 15c scalpel blade.

Fig 38  The 2.5 mm of incised gingiva is removed with a periodontal curette.
would require ostectomy for creating a space for the new biologic width. The initial step was a gingivectomy to remove a thickness flap was elevated following vertical and horizontal ostectomy and osteoplasty were carried out using end-cutting and cylindrical burs, respectively, under copious irrigation with sterile saline. Figure 40 shows the new biologic crown length of the canine of approximately 13.5 mm. A similar procedure was performed on the right canine, and the flaps were sutured. Uneventful healing after 10 days shows a more cervical location of the gingival zeniths around both canines (Fig 41). Note how the increased clinical crown lengths of the canines are at the expense of root exposure. A further 4.5 months was allowed for stabilization of the gingival zeniths; in the interim period, the patient opted for home bleaching prior to the restorative phase (Fig 42).

The restorative stage commenced by using a transparent composite shade (Empress Direct, Ivoclar Vivadent) for building up the incisal edges of the central incisors, aided by the previously fabricated silicone index from the diagnostic wax-up (Fig 43). All composite restorations were performed according...
**Fig 42** After 4.5 months of healing and post-bleaching (compare with Fig 30).

**Fig 43** Incisal edge build-up of the central incisors with a transparent shade resin-based composite using the silicone index of the diagnostic wax-up shown in Figure 33.

**Fig 44** Postoperative result showing the restitution of pink and white anterior dental esthetics by esthetic crown lengthening and prepless composite restorations, respectively (compare with Fig 31).

**Fig 45** Postoperative result showing GAL Class I on the right and left sides of the maxillary sextant (compare with Fig 31).

**Fig 46** Postoperative dentofacial view showing acceptable maxillary gingival exposure and a smile line coincident with curvature of the lower lip (compare with Fig 30).
to strict prepless and adhesive protocols, relying on micromechanical and chemical adhesion of a dentin bonding agent (OptiBond XTR). The postoperative result shows the correct w/l ratio of the central incisors, median diastema closure, and replacement of the lost enamel and dentin due to the previously mentioned TSL (Figs 44 and 45). The dentofacial view during repose smiling shows reduced gingival exposure around the maxillary canines, GAL Class I on the right and left sides of the maxillary sextant (Fig 45), dominance of the central incisors, and a smile line parallel to the curvature of the lower lip (Fig 46). Finally, the patient was counseled about diet and oral hygiene procedures, and provided with nightguards to mitigate tooth wear and protect the composite restorations.

Discussion

In the two case studies discussed here, the esthetic anomaly was short clinical crowns with compromised pink esthetics, and while the treatments in both cases were similar and involved perioplastic surgery, the etiology – and hence future management – was different. In the first case (APE), the crown lengthening itself was a fait accompli, while in the second case (ASE), the patient required dietary advice, nightguards, and future periodic reviews for monitoring parafunctional activity for the cessation and mitigation of TSL.

One of the defining features for the differential diagnosis of both APE and ASE is the location of the CEJ. In the first case (APE), the location of the CEJ was apical to the FGM, while in the second case (ASE), the CEJ approximated the CEJ (Fig 47). Therefore, achieving the correct clinical crown lengths for the ASE patient necessitated exposing the root surface, which in itself could cause future problems that would require desensitizing agents and/or protecting the exposed dentin with restorative materials. In addition, the ASE case showed TSL at the incisal edges of the canine.
while in the APE case, the canines were pristine and unworn. Furthermore, the keratinized band of tissue in the APE patient was wide, which is consistent with APE Type 1, while in the ASE case study, this width was within the norm due to dentoalveolar compensation. Finally, since soft tissue healing is erratic, periodic monitoring is essential for both cases to ensure long-term periodontal health and the maintenance of both pink and white esthetics.

Conclusion

This discussion has focused on short clinical crowns concurrent with excessive maxillary gingival display due to various soft and hard tissue anomalies. Apart from numerous mirroring conditions, the dental and gingival causes of short clinical crowns are APE or ASE, and differential diagnosis of the two is essential for arriving at correct management strategies of these conditions, which compromise anterior dental esthetics. The two case studies presented in this article show striking clinical similarities, but with different etiologies and diagnoses. Although the initial treatment may be similar, the long-term management differs, and careful counseling and monitoring is essential for ongoing oral health, function, and esthetics.  

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References

11. Compagnon D, Woda A. Supraeruption of the unop-


