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REVIEW ARTICLE

Complications and treatment errors involving periodontal tissues related to orthodontic therapy

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1 | INTRODUCTION

The interplay between the application of orthodontic forces to stimulate tooth movement and the reaction of the affected periodontal tissues is complex. While most of the time possible damage to the periodontal tissues is transient with no long-term detrimental effects on periodontal health, in some instances major undesirable consequences have been reported. In other situations, orthodontic therapy (OT) can be of benefit in solving problems caused by periodontitis or by mucogingival conditions. Corresponding to the new classification on periodontal diseases and conditions,[1] this article is structured into three major sections:

- OT in relation to periodontal health,
- OT in relation to periodontitis, and
- OT in relation to mucogingival conditions.

In each chapter we have tried to illustrate typical scenarios encountered in the clinic that may be accompanied by complications and possibly even be induced by treatment errors, together with recommendations on how they can be avoided or how they can be rescued, based on the currently available evidence and on expert opinion.

While clinical periodontal health and also the end points of successful periodontal therapy to establish periodontal health on a reduced periodontium are clearly defined,[2,3] these may differ from how clinical success of OT is defined. Orthodontic treatment is considered successful when the treatment goal is achieved and the result remains stable. In general, these treatment goals may be defined as achievement of oral health, esthetics, occlusion, function, and stability, although the treatment goals may vary among patients.[4] Chung et al concluded that the examiners of the American Board of Orthodontics continue to see consistent problems, including lack of attention to finishing details, inappropriate treatment objectives, excessive proclination of mandibular incisors, excessive expansion of mandibular intercanine width, closing skeletal open bites with extrusion of anterior teeth leading to excessive gingival display, and failure to recognize the importance of controlling the eruption or extrusion of molars during treatment.[5] It is obvious that all of these problems may have implications for the status of the periodontal tissues involved.

2 | OT IN RELATION TO PERIODONTAL HEALTH

Most of the time, OT is performed in children and adolescents with healthy periodontal conditions. Gingival and periodontal health have been described in detail and defined clinically in a recent World Workshop on the classification of periodontal and peri-implant diseases and conditions.[2,6]

Orthodontic therapy moves teeth from one position in the jaw, through bone and the surrounding soft tissues, to another position, in response to forces applied by specific appliances. This movement of teeth is accompanied by tissue remodeling, which modifies the morphology of the periodontal tissues. The periodontal ligament, as a dynamic structure, makes this possible. The whole periodontal attachment apparatus, including the alveolar bone, shows biological responses and changes, including a modification of the local vascularization. Even though there are many innovative mechanical devices for tooth movement, therapists are still not completely successful in preventing trauma to the periodontium, resulting in undesirable side effects. This may be due to lack of complete understanding of cellular complexities.[7] In general, most adverse effects

are believed to be transient and not associated with any lasting detrimental effect on the surrounding periodontal tissues.[8–12]

2.1 | Plaque/Biofilm

It is well established that orthodontic fixed appliances make maintenance of proper oral hygiene more difficult, resulting in increased accumulation of plaque and subsequent mild inflammation of the gingival tissues, thus acting as a local risk factor for the development of gingivitis.[2,13] With regard to facilitation of oral hygiene, a recent randomized clinical trial (RCT) found no differences when comparing clear aligners, self-ligated brackets, and traditional fixed orthodontic appliances.[14] However, there was a significant change in biofilm composition after the placement of orthodontic appliances[15] including an increase in the percentage of potentially pathogenic gram-negative bacteria,[16] and a significant impact of bracket design was found.[17] Such changes in microbial parameters were only partially normalized 3 months following the removal of fixed orthodontic appliances.[10] Placement of orthodontic fixed appliances was shown to be associated with a qualitative change of the subgingival microbiota, with increased prevalence of subgingival *Aggregatibacter actinomycetemcomitans* and *Tannerella forsythia* persisting for up to 6 months after appliance removal[18,19]; however, another review reported transient increases in the levels of subgingival pathogens with a return to pretreatment levels several months later.[20] Studies on the effect of orthodontic appliances/therapy on the composition of the oral microbiome using next-generation sequencing of the bacterial 16S rRNA gene confirmed significant changes[21]; however, this effect was transient[22] and only minor compositional changes were found after completion of treatment.[23]

2.2 | Gingival inflammation and overgrowth/enlargement

The biofilm-retentive characteristics of orthodontic appliances result in the development of gingivitis as a natural consequence of incomplete removal of plaque. The presence of gingivitis is characterized by increased pocket probing depth, increased bleeding on probing, increased crevicular fluid volume, as well as elevated gingival and bleeding indices,[10,24] although mostly found to be transient after the first 3–6 months post-treatment.[8–10]

Abnormal inflammatory overgrowth of gingival tissues is a complication that may occur after orthodontic appliance insertion, as verified by histological examination.[25] Several studies found a positive association among duration of orthodontic treatment, gingival inflammation, and gingival enlargement in subjects undergoing fixed OT.[26–28] The exact mechanism for the development of overgrowth is not yet completely understood but it probably involves increased production by fibroblasts of amorphous ground substance with a high level of glycosaminoglycans. Increased expression of type I collagen mRNA and upregulation of keratinocyte growth factor receptor may play an important role in excessive proliferation of epithelial cells.[29] The tissue enlargement in hyperplastic gingivitis is generally mild and occurs in about 10% of patients undergoing treatment with a fixed othodontic appliance.[17] An example of a patient with generalized gingival enlargement is shown in Figure 1. Slight hyperplastic changes of the interdental papillae in patients with good oral hygiene disappear following removal of orthodontic appliances, but notably the results of one study[30] showed that even 2 years after debonding not all periodontal parameters had returned to normal, indicating that some changes are only partially reversible. As further changes occurred between 3 months and 2 years after debonding, the authors suggested not to carry out a gingivectomy or gingivoplasty As further changes occurred between 3 months and 2 years after debonding, the authors suggested not to carry out a gingivectomy or gingivoplasty procedure already at 3 months after completion of orthodontic therapy.

2.3 | Periodontal alterations

2.3.1 | Clinical attachment level

A systematic review investigated periodontal clinical attachment changes in periodontally healthy patients undergoing OT. The average clinical attachment loss after values were pooled was 0.11 mm (nine studies; 335 patients; 95% CI = 0.12 mm gain to 0.34 mm loss; $P = .338$), but it should be noted that the studies demonstrated high heterogeneity and the quality of evidence was overall low.[18]

These findings are confirmed by another systematic review and its recent update, in which minimal changes were reported in clinical attachment levels (three studies; 43 patients; Mean effect (ME) = 0.248 mm; 95% CI [−0.055 to 0.551]; $P = .109$).[31,32]

2.3.2 | Probing pocket depth

A systematic review reported 0.23 mm of increased probing pocket depth (PPD, two studies; 95% CI = 0.5–0.3 mm; $P = .0001$) compared

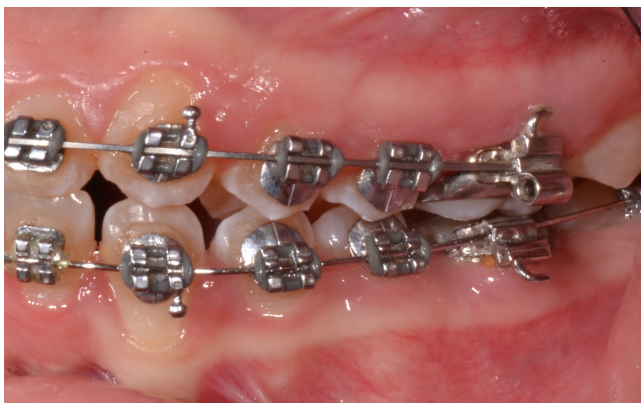

FIGURE 1 Inflammatory gingival overgrowth in the premolar and molar areas as a result of insufficient and challenging plaque control after placement of orthodontic devices.

with no orthodontic treatment.[13] A more recent systematic review and its update observed a statistically significant reduction in PPD (mm) (five studies; 63 patients; ME = 0.325 mm; 95% CI = 0.123–0.526; $P < 0.001$), although this can be considered as being not clinically relevant (as PPD reduction was <0.5 mm).[31,32]

2.3.3 | Bone level

The same systematic review[13] demonstrated that OT was associated with 0.13 mm of alveolar bone loss (three studies; 95% CI = 0.07–0.20 mm; $P = .0001$) when compared with no treatment. More recently, Martin et al[31] reported in their systematic review also a slight loss in radiographic bone level (one study; 122 patients; ME = −0.400 mm; 95% CI = −0.579 to −0.221 mm; $P < .001$) in patients without periodontitis undergoing OT. These results were based on the analysis of conventional radiographs.

With the introduction of three-dimensional computerized technology (cone beam computed tomography [CBCT]), marginal bone level changes following OT could also be evaluated on the vestibular and oral tooth surfaces. Lund et al[33] examined patients before and after orthodontic treatment following premolar extraction. In anterior jaw regions in which retraction of teeth causes remodeling of the alveolar bone, 84% of lingual surfaces of mandibular central incisors exhibited a bone-height decrease of >2 mm. Fewer than 1% of proximal surfaces exhibited changes of >2 mm. Castro et al used CBCT to evaluate the distance between the cemento-enamel junction (CEJ) and the alveolar bone crest before and after nonextraction orthodontic treatment; they found that this distance increased in 822 (57%) of the 1440 surfaces after orthodontic treatment.[34] The buccal surfaces of the lower central incisors had the greatest frequency of increased distance (75%). Jager et al performed scans using the i-CAT Next Generation technology and reported a significant decrease in periodontal bone height (dehiscence: −0.82 ± 1.47 mm) and bone thickness (−0.56 ± 0.7 and −0.69 ± 0.9 mm at 5 mm and 10 mm apical to the CEJ, respectively) after orthodontic treatment.[35] Changes in alveolar bone height and cortical bone thickness around the mandibular incisors after orthodontic treatment were also observed by Garlock et al.[36] In patients who did not undergo tooth extraction, the average facial and lingual vertical bone loss was 1.16 ± 2.26 and 1.33 ± 2.50 mm, respectively.

2.4 | Root resorption

Loss of periodontal attachment in the course of OT can also occur from external apical root resorption, also referred to as orthodontically induced inflammatory root resorption.[37] The sterile inflammation induced by the orthodontic load brings about resorption of the superficial root cementum, or the inflammation can become more severe with eventual resorption of the underlying dentin and loss of overall root length. The underlying biological events are not completely understood. A classification of external root resorption, proposed by Feller,[38] is displayed in Table 1.

Depending on the severity of orthodontically induced apical external root resorption data on the incidence/prevalence vary among studies and range from 2.9% for severe root resorption to 98.1% for mild root resorption.[39] In other publications values range from 14.8%[40] up to 46%.[34,41,42]

Apical external root resorption is considered to be an invariable iatrogenic side effect/complication of orthodontic treatment[38] and is associated with several treatment-related risk factors, such as extraction treatment, long treatment duration, heavy continuous treatment force, and large distance of tooth movement with apical displacement especially of the maxillary incisors.[37,39,40,43–48] Even though genetics may account for the variation in external apical root resorption there is insufficient evidence for specific genetic risk factors.[47] More recently, CBCT was shown to be a reliable tool for examining orthodontically induced external root resorption.[49] An example of a patient with severe root resorption is shown in Figure 2.

As some degree of apical external root resorption is a frequent and unavoidable complication of OT, during treatment planning, the patient or parent should be informed about this risk. Strategies to minimize external root resorption should include limitation of prolonged treatment, the use of light intermittent forces, and biannual radiographic monitoring in order to detect any possible root resorption at the earliest stage.[48,50] If any apical external root resorption is detected, active treatment should be suspended for 2–3 months, with the aim of preventing further resorption and to allow some healing with cellular cementum. If further resorption is detected after active treatment has been resumed, the orthodontic treatment plan should be modified.[38,51]

In summary, OT can be safely performed in periodontally healthy patients and has no detrimental effects on periodontal tissues. However, it is well established that orthodontically induced inflammatory gingival overgrowth and/or apical external root resorption are common complications that need to be recognized and adequately addressed to prevent more severe damage. Most importantly, periodontal health and adequate oral hygiene have to be assured prior to OT and constantly monitored throughout treatment. Failure to do so has to be viewed as treatment error.

3 | OT IN RELATION TO PERIODONTITIS

There is an increase in the number of adult patients with various stages of periodontitis who seek orthodontic treatment for esthetic

TABLE 1 Classification of the degree of external root resorption.

Mild	Apical root resorption <2 mm of the original root length
Moderate	Apical root resorption >2 mm but <1/3 of original root length
Severe	Root resorption >4 mm or 1/3 of original root length

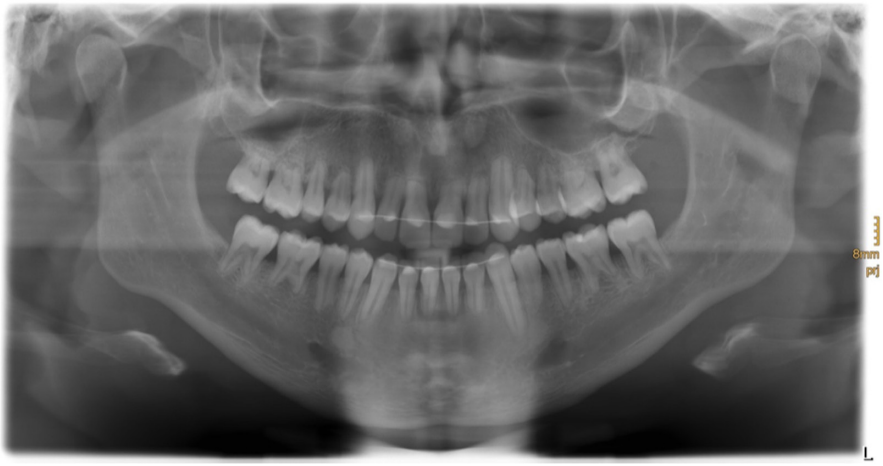

FIGURE 2 Radiographic image of a 28-year-old healthy male patient presenting with severe apical root resorption after orthodontic treatment.

and functional concerns.[52] The reduction of periodontal support can be associated with elongation, labial flaring, extrusion, rotation, spacing, and drifting of teeth.[53] Maxillary incisors are reported to be particularly susceptible to pathological migration.[54] In fact, this clinical scenario is recognized as one of the specific phenotypic variations of patients with stage IV periodontitis amenable to orthodontic correction.[32]

There is consensus that the periodontal inflammation must be controlled before orthodontic tooth movement and remain controlled during OT. This is based on evidence derived from well-controlled experimental animal studies demonstrating that orthodontic forces on teeth which have a reduced, but healthy, periodontium did not cause additional attachment loss,[55] whereas, if associated with plaque-induced inflamed periodontal tissues and intrabony defects, orthodontic movement of teeth was found to increase loss of connective tissue attachment.[56]

Thus, an interdisciplinary approach is required to control the periodontal infection and to realign the migrated teeth.[57–60] However, according to recent reviews, there has been minimal clinical investigation of the complex interaction between orthodontic and periodontal treatment and there are a lack of well controlled studies of patients with periodontal and orthodontic treatment needs to determine whether orthodontic treatment may improve or aggravate periodontal conditions in patients with periodontitis.[31,61–63]

Very recently, a European Workshop has addressed these challenges in a European Federation of Periodontology (EFP) S3 level clinical practice guideline on the treatment of stage IV periodontitis.[32]

In periodontal clinical practice, different scenarios can be encountered:

- Successfully treated periodontitis patient in need of orthodontic treatment
- Patient with undiagnosed, untreated periodontitis undergoing orthodontic treatment
- Patient with untreated periodontitis in need of periodontal and orthodontic treatment
- Patient with undetected incipient periodontitis undergoing orthodontic treatment
- Periodontally healthy but periodontitis-susceptible patient undergoing orthodontic treatment

3.1 | Successfully treated periodontitis patient in need of orthodontic treatment

There is consensus regarding what a clinical case of a successfully treated periodontitis patient with stable periodontal conditions should look like. In the World Workshop on the 2018 Classification, a case of clinical health on a reduced periodontium has been defined[2] and, in line with this, a European S3-Level Clinical Guideline Workshop has determined an end point of active periodontal therapy.[3] At the same time, there is consensus that these patients—even when treated successfully—always remain at risk of recurrent periodontitis and therefore should be enrolled in a supportive periodontal care program.[3]

Earlier clinical studies confirmed that in patients with good plaque control, teeth with a reduced (but healthy) periodontium can undergo successful tooth movement without compromising the periodontal support.[64,65] In a recent systematic review with meta-analysis[31] it was shown that the periodontal outcomes of OT in patients with treated periodontitis are similar to those obtained in patients with a healthy periodontium. Based on these findings, the new S3 level clinical practice guideline[32] gave the following evidence-based recommendation (R7.1):

> "In successfully treated stage IV periodontitis patients in need of orthodontic therapy (OT), we suggest undertaking OT based on evidence that: a) it does not significantly affect periodontal outcomes (probing pocket depth - PPD and clinical attachment levels - CAL); b) it does not significantly affect gingival inflammation (bleeding on probing—BOP) and gingival recession; c) it does not lead to a significant increase in root resorption."

Furthermore, based on expert opinion and data from preclinical studies, the following expert consensus-based recommendation (R7.2) regarding the appropriate time point for starting OT was provided:

> "In successfully treated stage IV periodontitis patients in need of orthodontic therapy, we recommend starting OT once the endpoints of periodontal therapy have been achieved (no sites with PPD = 5 mm and BOP and no sites with PPD ≥6 mm)."[3]

Therefore, in patients with a history of treated periodontitis, healthy periodontal conditions should be assured and documented at the start of OT.[66] A deviation from this may be looked upon as a treatment error.

A clinical example of a patient with treated periodontitis, for whom the successful outcome was not assessed by a re-evaluation and OT was immediately initiated is shown in Figure 3.

3.2 | Patient with undiagnosed, untreated periodontitis undergoing orthodontic treatment

Based on the information given above, the scenario of a patient with undiagnosed, untreated periodontitis undergoing orthodontic treatment has to be considered as a treatment error. It can, and must, be avoided by periodontal screening, followed by a careful periodontal examination and diagnosis in patients of all age groups, before orthodontic treatment is initiated.

3.3 | Patient with untreated periodontitis in need of periodontal and orthodontic treatment

The orthodontic treatment of the typical sequela of advanced periodontal attachment loss (pathological tooth migration) usually involves intrusive, retrusive, and alignment tooth movements, which may potentially cause adverse effects (further periodontal attachment or bone loss, increased gingival inflammation, or increased root resorption) or secondary effects (undesired esthetic outcomes, such as gingival recession and loss of interdental papilla) on the affected teeth. A recent systematic review with meta-analysis[63] on periodontal–orthodontic treatment of teeth with pathological flaring, drifting, and elongation in patients with severe periodontitis concluded that orthodontic treatment might be associated with small improvements of periodontal parameters and negligible adverse effects. These analyses were hampered by the fact that the periodontal status after periodontal therapy and before initiation of OT was not reported in most of the studies, and the authors emphasized that their conclusions were based on limited evidence of moderate quality because of the absence of adequate studies and therefore should be viewed with caution. Nevertheless, in the new S3 level clinical practice guideline[32] the following evidence-based recommendation (R7.3) was stated:

> "In stage IV periodontitis patients with pathological tooth migration, we suggest undertaking orthodontic therapy once the endpoints of periodontal therapy have been reached, based on the evidence that this therapy: a) does not significantly affect periodontal outcomes [CAL, PPD, and radiographic bone levels (RBL)]; b) seems to reduce gingival inflammation (BOP); c) does not significantly alter gingival margin levels; d) seems to improve inter-dental papilla height; e) does not significantly affect root resorption and seems to reduce tooth mobility."

There is ongoing debate regarding how long after active periodontal therapy the clinician has to wait before OT can be initiated:[66] immediately,[67] 1–2 weeks,[57,68–70] 2–6 months,[67,71] or 8–12 months.[72,73] Taking into consideration the healing dynamics of the periodontium following the different modalities of periodontal therapy,[24,74,75] Pini Prato and Chambrone[66] proposed a periodontal–orthodontic treatment algorithm, where orthodontic therapy should start 3–6 months after non-surgical therapy, 6–9 months after surgical periodontal treatment, and 12 months after regenerative periodontal procedures (Figure 4).

Patients with advanced or severe attachment loss as a result of periodontitis often present with intrabony defects and pathological tooth migration.[76] There is an evidence-based clinical practice guideline on how to manage patients with stage I–III periodontitis,[3] including regenerative procedures for intrabony defects.[77] A comprehensive combined periodontal–orthodontic treatment would include steps 1 and 2 of periodontal therapy to control the periodontal infection, followed by step 3 including regenerative periodontal surgery to reconstruct the defects, and subsequent OT to realign the migrated teeth. So far, clinicians have had to rely mainly on case reports and on prospective, as well as retrospective, clinical case series. In particular, the optimal time interval between regenerative periodontal surgery and the initiation of OT has been a matter of ongoing debate. It may be safe to wait until the end point of regenerative therapy has been reached (up to 12 months) and not to interfere with periodontal wound-healing.[66] Case reports and series with long-term follow-up periods have reported favorable periodontal outcomes using such a delayed approach.[72,73,78,79] The question then arises of whether a deviation from these recommendations would have to be considered as a treatment error. This is an intriguing question, as other reports have suggested that orthodontic tooth movement may be initiated almost immediately or up to 3 months after regenerative surgery.[80–83] No adverse effects were reported, and some authors speculated that early tooth movement could even stimulate periodontal wound healing. More recently, a large retrospective case series of patients with stage IV periodontitis, in whom OT was started 3 months after regenerative surgery, showed substantial improvements after 12 months that could be maintained up to 4 years.[84] As many patients affected by such a condition are interested in seeking orthodontic treatment because of the esthetic and functional changes caused by pathologic tooth migration,[52] this question of early versus late initiation

FIGURE 3 Clinical situation of a 22-year-old healthy male patient with a diagnosis (retrospective, based on documentation available) of localized periodontitis stage IV (tooth #12, 25), grade B. The patient was worried about his elongated upper right lateral incisor from an esthetic point of view. A, Flaring of the maxillary front, and severe elongation and spacing of tooth #12. B, Occlusal view of tooth #12, with large spaces (>2 mm) between it and the neighboring teeth. C, Elongation and rotation of tooth #12. D, Panoramic radiograph, taken before orthodontic treatment, showing very limited accuracy and detail for adequate periodontal evaluation. E, As reported by the patient, nonsurgical periodontal therapy was given followed immediately by orthodontic intrusion movement. F, Directly after debonding, frontal view. G, Occlusal view, with crowns perfectly aligned. H, Tooth #12 appears to be intruded perfectly. I, Evidence of severe vertical bone loss on the mesial and distal aspects, as well as apical root resorption #12. K, Panoramic radiograph showing limited accuracy and detail for adequate periodontal evaluation. L, Three years after debonding, the patient was referred to our periodontal clinic; he presented with an increasing gingival recession type 3 (RT3) on tooth #12 (this was the patient's main concern). Periodontal examination revealed now evident signs of recurrent periodontitis and a hopeless tooth #12 with a periodontal abscess and progressive attachment loss of 12 mm. BoP, bleeding on probing; CAL, clinical attachment loss; Pus, suppuration; PPD, periodontal probing depth. After nonsurgical periodontal therapy, the severely damaged tooth could be treated successfully with a periodontal regenerative technique, using a connective tissue graft (M, N) in combination with a bone substitute (Photographs and case courtesy of Dr. Tobias Waller). O, Clinical situation 3 years after periodontal regenerative surgery with shallow pockets of ≤4 mm on all maxillary incisors, well maintained in a stringent recall program.

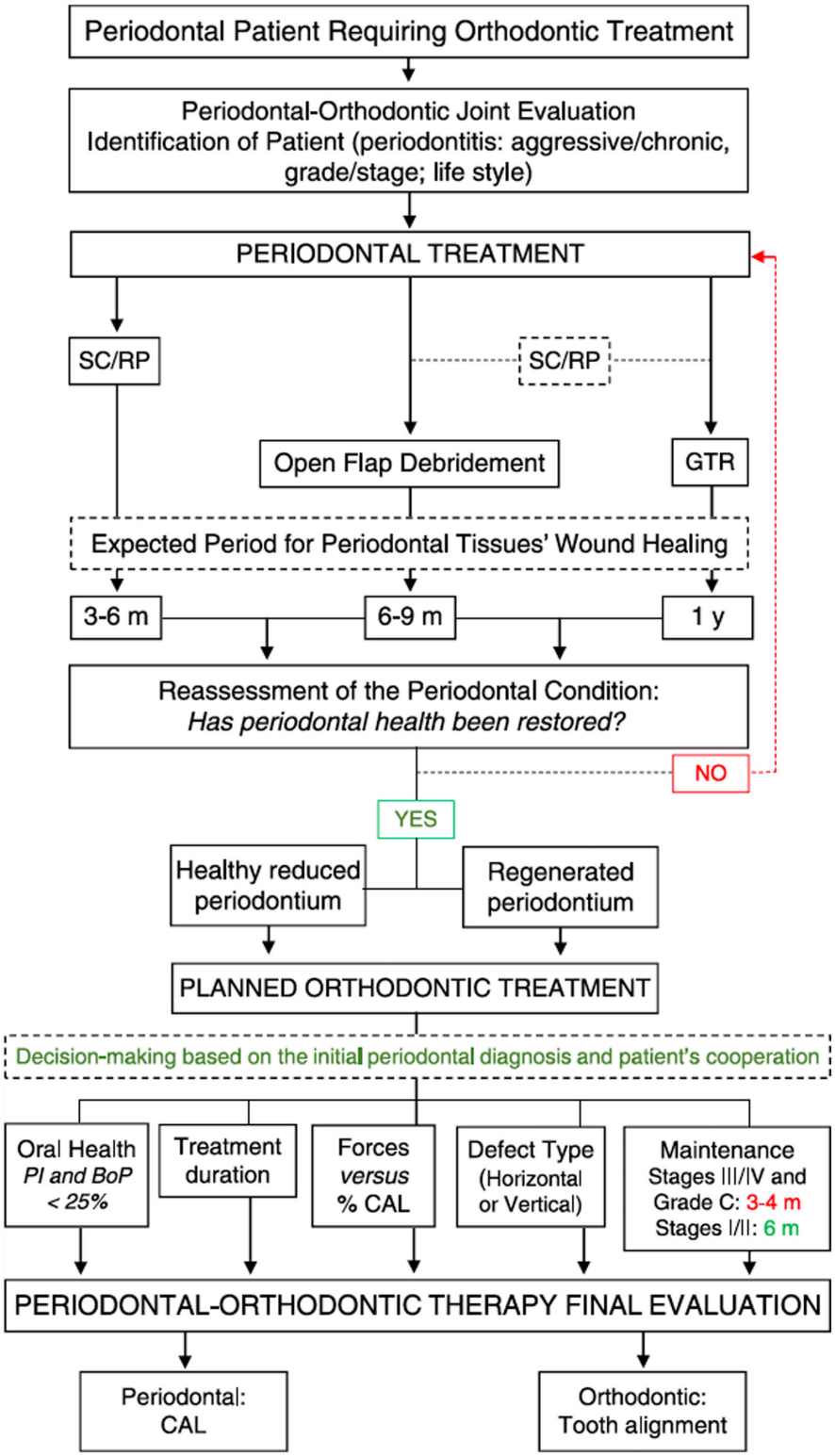

FIGURE 4 Periodontal–orthodontic treatment algorithm proposed by Pini-Prato and Chambrone.[66] BoP, bleeding on probing; CAL, clinical attachment loss; PI, Plaque Index; SC/RP, Scaling and root planing.

of OT is of high clinical relevance. The results of a recently published multicenter randomized trial could shed new light on this challenging treatment decision.[85] After 12 months, significant periodontal improvements of similar magnitude were accomplished by regenerative treatment of intrabony defects following early (after 4 weeks) and late (after 6 months) initiation of orthodontic tooth movement in patients with a high level of oral hygiene and infection control (Figure 5). In view of these findings, early OT after periodontal regenerative surgery of intrabony defects may not be viewed as treatment error. Based on systematic reviews,[63,86] the new EFP S3 level clinical practice guideline[32] gave the following evidence-based recommendation (R7.5):

FIGURE 5 Twenty-five-year-old patient with the diagnosis periodontitis stage IV with pathologic tooth migration (spacing and flaring). A, Clinical situation after initial periodontal therapy, showing flaring teeth #11, 12, 21 and 22 together with severe attachment loss, labial displacement and elongation'. B, Regenerative surgical procedure for an intrabony two-walled defect, 6 mm deep, followed up at 1 week and 2 weeks. C, Clinical situation 12 months after regenerative surgery/11 months after starting active orthodontic therapy and after 24 months. (Photographs from Jepsen et al[85]).

> "In stage IV periodontitis patients where intra-bony defects have been treated following the recommendations of the clinical practice guideline using the appropriate regenerative interventions: 1. We recommend undertaking OT based on the evidence that the combined treatment significantly improves periodontal outcomes (increased CAL gain, PD reduction and RBL gain) and significantly reduces gingival inflammation (BOP). 2. We suggest not to wait for a prolonged healing period after the regenerative intervention, before initiating OT, since there is evidence that a short (1 month) and a prolonged (6 months) period between periodontal/regenerative and OT result in comparable outcomes."

It was also emphasized that the combination of such complex periodontal and orthodontic therapies requires the coordinated efforts of different oral care providers, namely specialists or dentists with advanced training and skills in periodontal and orthodontic therapies.

3.4 | Patient with undetected incipient periodontitis undergoing orthodontic treatment

Discriminating between a case of gingivitis[2] or of incipient (stage I) periodontitis[87] is not easy and in such a scenario sometimes orthodontic treatment could have been initiated based on an incorrect periodontal assessment/diagnosis. The situation may turn into a treatment error (supervised neglect) if the clinician continues to ignore (to fail to detect) signs of periodontal inflammation because of lack of adequate monitoring.

A clinical example of this scenario is illustrated in Figure 6.

In retrospect and based on the radiographs available prior to orthodontic tooth movement (Figure 6E), indicating slight bone loss in the molar region, the patient could have been identified as having

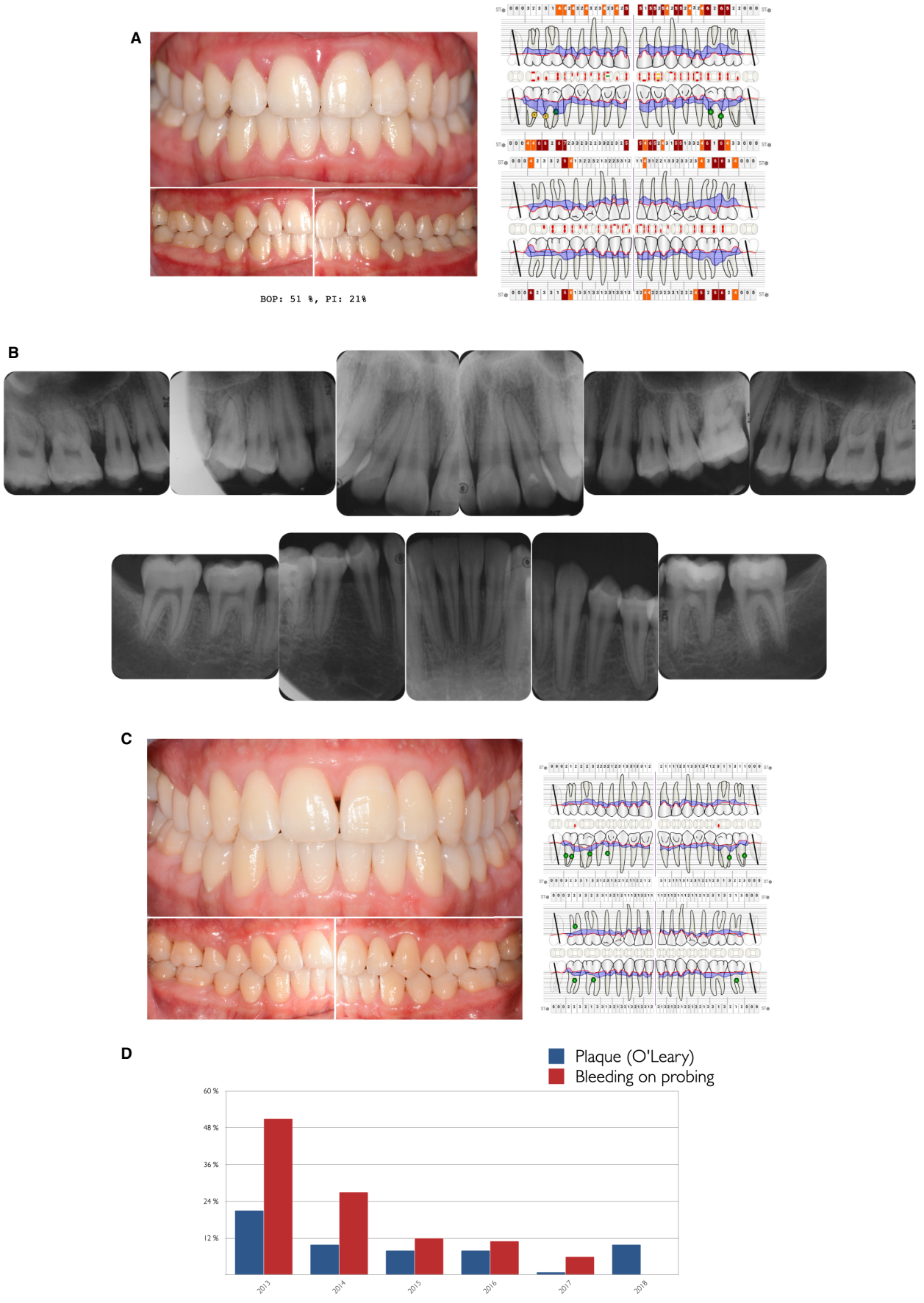

FIGURE 6 (Continues)

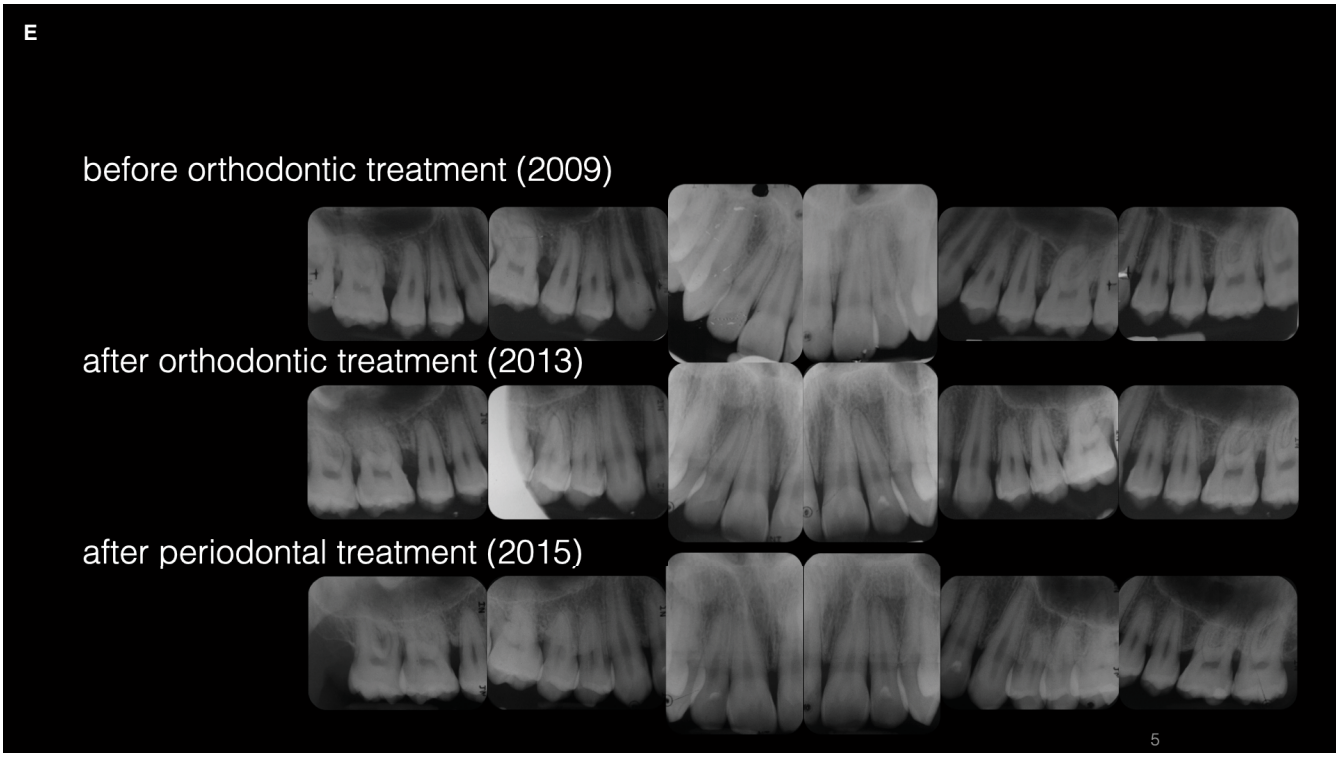

FIGURE 6 A, Clinical situation of a 17-year-old patient complaining about his upper right lateral incisor, which had Grade II mobility, bleeding of his gums, and food impaction. His orthodontic therapy had just reached completion, and his primary concern was losing all his teeth. He presented with a good level of oral hygiene, with a plaque index (PI) of 21% but a high full-mouth bleeding on probing (BOP) score of 51%. The diagnosis was periodontitis stage III, grade C (Photographs—Courtesy of Ph. Skora). B, Radiographs of the patient showing distinct bone loss on all first molars. C, Clinical situation, 5 years later, of the now 22-year-old patient after comprehensive periodontal treatment including steps 1–3 of therapy and a strict maintenance program. D, Over the course of active periodontal treatment and maintenance the patient could be kept stable with plaque and bleeding on probing scores decreasing to below 10%. E, Comparison of radiographs taken before and after orthodontic treatment and following comprehensive periodontal therapy. Distinct bone loss on the maxillary right first molar was already visible before tooth movement, and bone loss was present on all first molars 4 years later.

localized incipient periodontitis (stage I) and should have been referred to a periodontist. The progressive deterioration to a periodontitis stage III situation during orthodontic tooth movement remained unnoticed and indicates a lack of adequate monitoring. The loss of periodontal attachment necessitating comprehensive periodontal treatment after referral to our specialist clinic (Figure 6C–E) could have been avoided.

3.5 | Periodontally healthy but periodontitis-susceptible patient undergoing orthodontic treatment

In this scenario, the patient who is diagnosed as periodontally healthy before initiation of orthodontic tooth movement may develop periodontitis in the course of treatment. Periodontal inflammation can result from the increased bacterial load associated with the fixed orthodontic appliances in highly susceptible patients, who often present at a young age. This scenario presents a special challenge to the orthodontist as she/he may be the first to detect signs of periodontitis and has the responsibility to refer the patient to a periodontist. Thus, regular monitoring of the periodontal status is key in preventing rapid periodontal destruction in highly susceptible patients.

3.6 | Prevention of treatment errors related to OT

In order to prevent the treatment errors described above, the following recommendations have been made for periodontally susceptible patients undergoing OT.[66,88] As routine, prior to commencement of orthodontic treatment, patients have to be carefully examined for signs of periodontal disease, followed by individual oral hygiene instructions and dietary education. Orthodontic fixed appliances make maintenance of proper hygiene more difficult and have a major effect on the plaque and bleeding indices documented, resulting in subsequent mild inflammation of the oral tissues. Professional check-up visits with a dental hygienist need to be performed every 3 months and the time intervals between such visits should be decreased according to the individual needs of the patient. The orthodontist should be aware of early-onset periodontitis, which may not

be present at initiation of OT but may develop in patients during or after the course of therapy. For such patients, a periodontist should be involved.

Disease progression in patients with periodontitis depends on the extent and severity of the microbial biofilm challenge. Hence, to avoid increased tissue loss resulting from an unfavorable combination of periodontitis-associated inflammation with orthodontically induced inflammation, periodontal disease needs to be under control before starting orthodontic treatment. During and after orthodontic tooth movements, stringent monitoring of all periodontal parameters, including biofilm control, must be performed because an increased microbial load as a result of plaque accumulation around orthodontic appliances can induce periodontal inflammation, which possibly may be followed by destruction of surrounding tissues during tooth movement, depending on the individual susceptibility of the patient. To maintain the status of a successfully treated periodontitis patient,[2] guidelines for periodontal care and follow-up during OT are shown in (Table 2) and aim at achieving a plaque index of <20%, periodontal probing depths of <5 mm, and absence of bleeding on probing.[3,66,88]

To detect initial signs of deterioration, the periodontal status must be closely monitored by the hygienist/periodontist on a three monthly basis and, in most patients, adjustment of oral hygiene tools becomes necessary. As routine, a complete periodontal examination should be performed every 6 months, and in the event of recurrent disease, both treatments need to be adjusted accordingly. Nonadherence to oral hygiene protocols requires a temporary halt of active tooth movements. In some patients, removal of appliances should be considered until all signs of periodontal disease are resolved. In general, procedures initiating tooth movements should not be performed until periodontal inflammation is controlled and the patient is able to perform personal meticulous oral hygiene measures (Table 3).

Based on a randomized clinical trial[89] and a systematic review,[90] as well as on expert opinion, the following expert consensus-based recommendation (R7.7) was given in the new EFP S3 level clinical practice guideline[32]:

> "We recommend that during OT the patient's periodontal status is closely monitored and managed, ideally at each orthodontic appointment. If signs of periodontitis recurrence are detected, active OT should be interrupted, and the affected teeth should be maintained passively, while rendering proper periodontal treatment and oral hygiene reinforcement. Once periodontal health/stability has been re-established, OT can be re-instituted. We recommend that after completion of OT, life-long supportive periodontal care and life-long orthodontic retention are provided tailored to the individual needs/risk profile of the patient."

Furthermore, to facilitate treatment planning and execution, and interdisciplinary cooperation between periodontists and orthodontists, a treatment algorithm has been developed[32] (Figure 7).

In summary, OT can be safely performed in successfully treated periodontitis patients with no detrimental effects on periodontal tissues. However, conducting orthodontic tooth movements in patients with undetected, untreated periodontitis and continuing OT despite periodontitis developing during treatment should be viewed as treatment errors. Most importantly, to avoid these treatment errors, periodontal health and adequate oral hygiene must be assured prior to starting OT and constantly monitored throughout treatment. Failure to do so and failure to provide adequate supportive therapy should be viewed as treatment errors.

4 | OT IN RELATION TO MUCOGINGIVAL CONDITION

It has been known for a long time that mucogingival conditions can be affected by OT.[91] Mucogingival conditions comprising gingival recessions and periodontal phenotypes have been newly defined by the 2018 World Workshop Classification of Periodontal Diseases and Conditions.[1,12,92]

TABLE 2 Proposed guidelines for periodontal follow-up care during orthodontic therapy in patients susceptible to periodontitis, based on publications of Levin et al,[88] Pini Prato and Chambrone,[66] and Herrera et al.[32]

	Before orthodontic treatment	During orthodontic treatment	Following orthodontic treatment
Plaque control	+	+	+
Periodontal probing	+	+ Every 6 months	+ Once a year
Bitewings/parallel periapical radiographs	+	+ Once a year unless there is a pathological finding	+ Once a year unless there is a pathological finding
Referral to a periodontist	1. In the event of pathologic periodontal pockets or radiographic bone loss 2. In the event of doubt regarding the periodontal condition		

TABLE 3 Proposed actions required following different periodontal findings, based on Levin et al,[88] Pini Prato and Chambrone,[66] Sanz et al[3] and Herrera et al.[32]

Periodontal finding	Action required
Probing depths >5 mm with no bleeding on probing	Oral hygiene reinforcement; shorten interval between maintenance appointments to 4–6 weeks
Probing depths ≥5 mm with bleeding on probing	Stop active orthodontic treatment; refer to a periodontist; only after resolution of the periodontal inflammation continue orthodontic treatment with special care and follow-up
Root resorption ≥3 mm apparent on radiographs	Consider applying lighter forces; consider interruption of active tooth movement; radiographic follow-up every 6 months

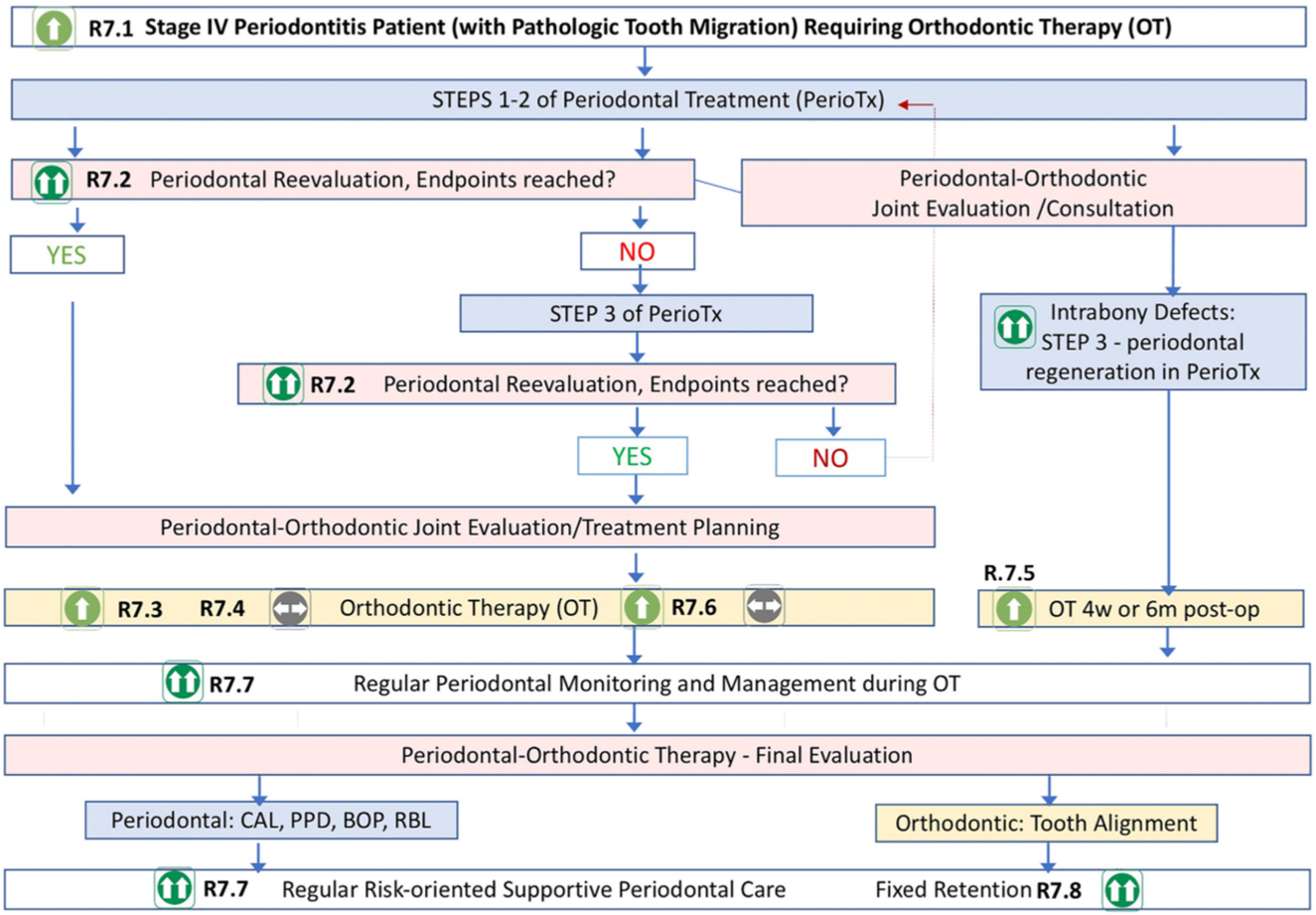

FIGURE 7 Flowchart illustrating how orthodontic therapy (OT) of patients with stage IV periodontitis can be integrated into the overall periodontal treatment plan with reference to recommendations R7.1–R7.8 of the S3-Level clinical practice guideline for the treatment of stage IV periodontitis. The steps of periodontal therapy were as described in the S3-Level clinical practice guideline for the treatment of periodontitis stages I–III.[3] CAL, clinical attachment loss; PPD, probing pocket depth; BOP, bleeding on probing; RBL, radiographic bone loss; 4 w, 4 weeks; 6 m, 6 months; post-op, post-operative.[32]

4.1 | Gingival recession

Gingival recession is defined as the apical shift of the gingival margin with respect to the CEJ; it leads to attachment loss and can be associated with various alterations of the root surface exposed to the oral environment. The new classification considers the interproximal attachment level[93] as well as the condition of the exposed root surface.[94]

Gingival recession affects a large proportion (22–88%) of people, with the proportion affected increasing with age.[95] Among anatomical variables, subjects with thin tissue and absence of attached gingiva (thin gingival phenotype) tend to have a higher incidence of gingival recession.[12,92,96,121] Further risk factors are the shape of a tooth, the presence of dehiscence/fenestration, an aberrant path of eruption, or thickness of the alveolar bone due to tooth position in the alveolar process.[97,98]

Gingival recession is associated with several clinical problems, such as dentin hypersensitivity, the development of caries and non-carious cervical lesions on the exposed root surface, and impaired esthetics, all of which are tooth-related conditions that concern the patient and the clinician.[12,99] A recent meta-analysis assessed the long-term outcomes of untreated facial gingival recession defects. The authors concluded that facial gingival recession in subjects with good oral hygiene is likely to result in an increase in the recession depth during long-term follow-up. In general, progression of gingival recession does not seem to impair the long-term survival of teeth.[100]

Life expectancy is increasing, and people are retaining more teeth. Consequently, both gingival recession and the related damage to the root surface are likely to become more frequent. Therefore, it is important to define predisposing conditions or treatments that are associated with the occurrence of gingival recession.

Depending on the direction of orthodontic tooth movement, gingival recession may develop or progress during or after OT. The prevalence of gingival recession varies from 5% to 12% at the end of treatment, with the number of recessions (prevalence up to 47%) increasing over time (Figure 8A). Mandibular incisors are the tooth type most commonly affected, followed by maxillary canines, premolars, and molars[101] (Figure 8B).

4.2 | Periodontal phenotype

The new term "periodontal phenotype" was adopted to describe the combination of gingival phenotype (gingival thickness, keratinized tissue width) and bone morphotype (thickness of the buccal bone plate).[12] Gingival phenotype can be assessed by using a periodontal probe to measure the gingival thickness observing visibility of the periodontal probe through gingival tissue after being inserted into the sulcus: (1) Probe visible: thin (≤1 mm); (2) Probe not visible: thick (>1 mm). Additional information on the three-dimensional gingival volume can be obtained by measuring the keratinized tissue width from the gingival margin to the mucogingival junction. Bone morphotypes have been measured radiographically using CBCT. There is evidence reporting a correlation between gingival thickness and

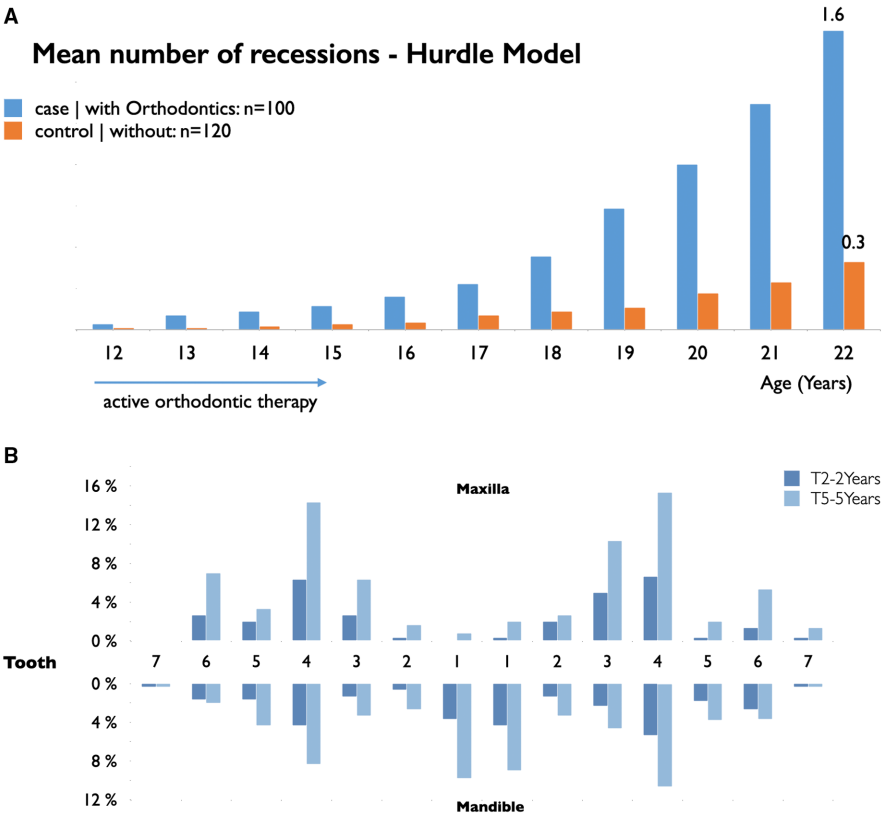

FIGURE 8 A, Mean number of labial gingival recessions in patients treated with orthodontic therapy (cases) and in untreated controls. Using the hurdle model, overall, the OR for cases compared with controls to have recessions is 4.48 ($P < .001$; 95% CI: 2.61–7.70). The OR for the increase of age by 1 year to have recessions is 1.53 ($P < .001$; 95% CI: 1.38–1.70). For those estimated to have recessions, the mean number of recessions for cases is estimated to be 142% higher than for controls ($P = .013$; 95% CI: 21–385). The estimated increase in the number of recessions by increasing age, for those with recessions, was not statistically significant. This increase was estimated to be 10% ($P = .231$; 95% CI: 6–28). Adapted from Renkema et al.[101] B, Frequencies (%) of gingival recessions per tooth at two time-points: T2 (at completion of orthodontic treatment) and T5 (5 years after completion of orthodontic treatment). Mandibular incisors seem to be the tooth type most vulnerable to the development of gingival recessions, followed by maxillary canines, premolars, and molars. Adapted from Renkema et al.[102]

buccal bone plate thickness.[103,104] A thin gingival phenotype increases the risk for gingival recession, and thin phenotypes are more prone to develop gingival recession of greater severity.[12,100,105]

4.2.1 | Gingival thickness

The assumption that the direction of tooth movements and the buccolingual thickness of the gingiva may play an important role in soft tissue alteration during orthodontic treatment was systematically reviewed.[106] The authors concluded that there is a higher probability of recession during tooth movement in areas with thin gingiva and that gingival augmentation can be indicated before the initiation of orthodontic treatment in such areas. Furthermore, studies have demonstrated a correlation between labial movement, post-treatment proclination, and a thin periodontal phenotype of mandibular incisors with tooth surface exposure during and after orthodontic treatment.[106–109]

Thus, the occurrence of gingival recession—in particular at mandibular incisors with post-treatment proclination—has to be viewed as a common side effect/complication of OT. If post-orthodontically a recession defect almost reaches the apex, the risk of tooth loss cannot be disregarded (Figure 9). Failure to monitor patients regularly and not intervening when there is an increase of recession and loss of the attached gingiva apical to the recession should be regarded as a treatment error.

4.2.2 | Keratinized tissue width

A retrospective study on the effects of OT examined the width of keratinized tissue in young patients.[110] A greater incidence of complete loss of keratinized tissue was found after OT on teeth with <2 mm of width of keratinized tissue (6.1% loss) than on teeth with >2 mm width of keratinized tissue (0.1% loss). Teeth with a lack of keratinized tissue before orthodontic treatment did not form new keratinized tissue after the orthodontic treatment. An example of a patient with lower incisors and canines affected is presented in Figure 10.

4.2.3 | Bony envelope

A histological study in experimental animals demonstrated that the movement of teeth to positions outside the labial or lingual alveolar plate could result in thinning of the alveolar plate or even dehiscence formation.[111] This observation was confirmed by a systematic review of clinical studies showing a higher occurrence or increased severity of gingival recession in more proclined teeth compared with less proclined or untreated teeth. The authors concluded that movement of incisors out of the osseous envelope of the alveolar process can be associated with a higher tendency for development of gingival recessions.[112] Recent cone beam CBCT studies confirm a higher incidence of bony dehiscence and gingival recession in teeth exposed to orthodontic forces that result in movement of teeth outside the bony housing, for example, after arch expansion.[113] Clearly, such outcomes should be considered at least as treatment complications if not as treatment errors. Examples of patients before and after orthodontic arch expansion are shown in Figures 11 and 12.

4.3 | Postorthodontic treatment changes in tooth position

Changes in tooth position that are related to orthodontic tooth movement but occur during the retention phase, after completion of active treatment, can also have unfavorable effects on mucogingival conditions. Many clinicians use fixed flexible spiral wire retainers in the anterior regions of the maxilla and mandible but there is limited evidence regarding stability of orthodontic

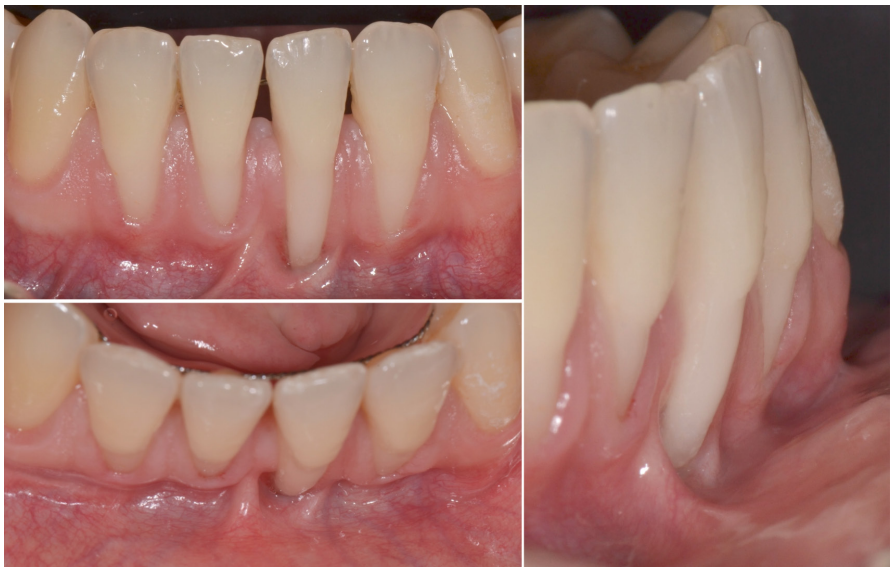

FIGURE 9 A 20-year-old postorthodontic patient presenting with a very advanced recession defect reaching the apex. The risk of tooth loss cannot be disregarded.

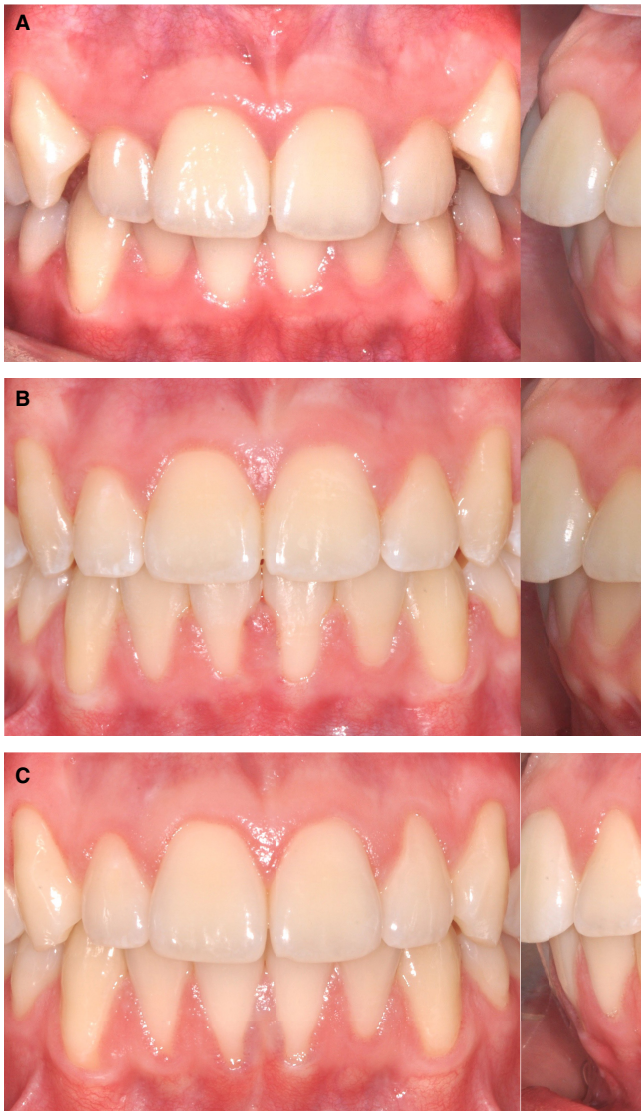

FIGURE 10 A, Clinical situation of a 12-year-old healthy male patient before orthodontic treatment, with anterior crowding in the mandibular front. Thin gingival tissues with an inadequate zone of marginal gingiva (width of keratinized tissue less than 2 mm) are present locally at the lower central incisors and canines. B, Situation at the age of 17. C, Distinct gingival recessions at the lower central incisors and canines at the age of 18 after debonding; the patient is now complaining about hypersensitivity. Orthodontic therapy can influence the development of gingival recessions during orthodontic therapy involving teeth that have an inadequate zone of the marginal gingiva (width of keratinized tissue less than 2 mm) present locally at the lower central incisors and canines.

alignment of the mandibular anterior teeth retained using these canine-to-canine lingual retainers. Therefore, Renkema[114] assessed the long-term effectiveness of mandibular canine-to-canine wire retainers bonded to all six teeth in a large consecutive group of patients; they found that at 5 years post-treatment, alignment of the mandibular anterior teeth was stable in 90.5% of the patients, whereas 9.5% experienced an increase in the main irregularity index. The increase of irregularity was strongly

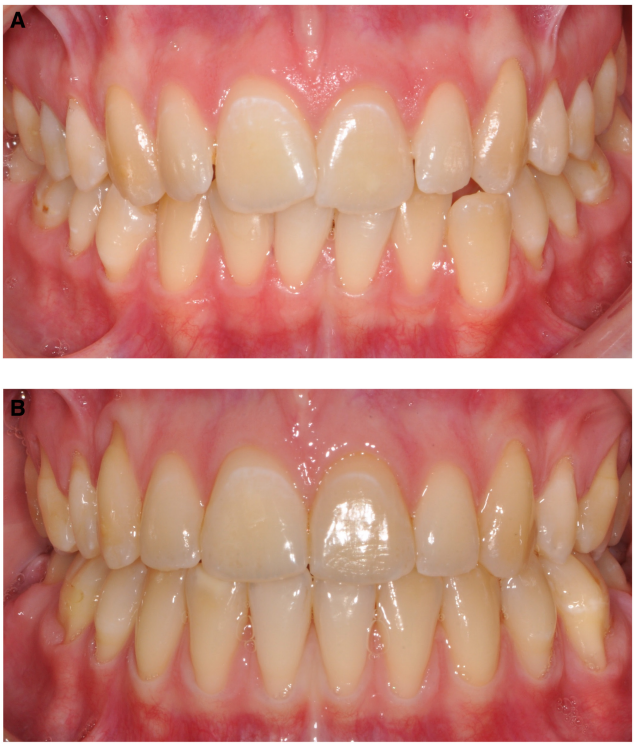

FIGURE 11 A, Clinical situation of a 16-year-old healthy female patient practicing good oral hygiene. Thin gingival tissues, narrow gingival width, and gingival recessions already present before orthodontic treatment. B, Five years after arch expansion, the patient presented with generalized gingival recessions, in spite of the extraction of four premolars in the course of the treatment.

related to the bonding failures of the retainer. In six (2.7%) patients, unexpected post-treatment complications (torque differences of the incisors, increased buccal canine inclination) were observed. These unexpected findings are confirmed by another publication[115] from the same group and are attributed to an active component of the wire due to either an elastic deflection caused by the clinician or a mechanical deformation from masticatory forces because the complications observed after orthodontic treatment were not present before this treatment was started. Forces generated by lingual fixed retainer wires were assessed in vitro and recorded during simulated intrusion–extrusion and buccal–lingual movements. High forces that exceed 1 N might be generated, and such forces are large enough to produce unwanted tooth movement during retention. Accordingly, gingival recession with root exposure might occur as a consequence of a root position outside the bony envelope.[116] This "wire syndrome" may present in different degrees of severity and depends on the amount of incisor inclination and torque differences.[117] Reported prevalence rates are up to 30%.[118]

These unwanted adverse effects of OT in the retention phase should be viewed at least as complications and if left unnoticed and/or unaddressed in their early stages should be considered as treatment errors. Various solutions, including removal of the bonded lingual retainer,[119] may be necessary (Figures 13 and 14).

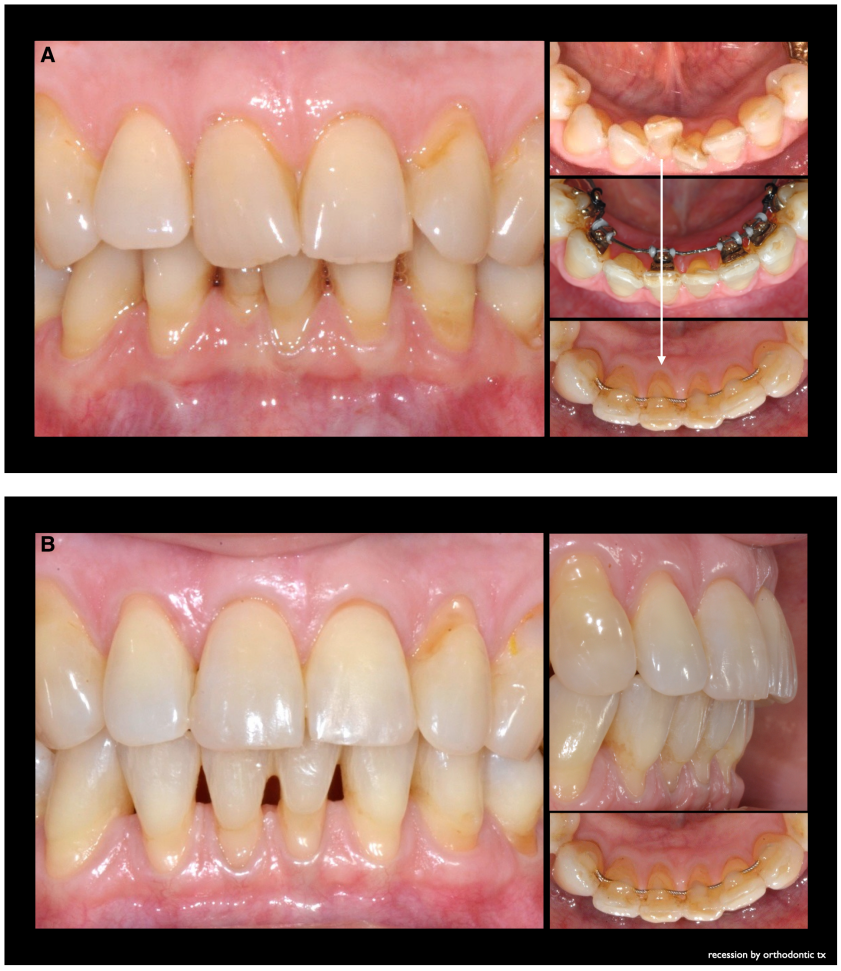

FIGURE 12 A, Clinical situation of a 45-year-old healthy female patient who reported difficulty in cleaning her crowded lower incisors and problems with food impaction; tooth #41 shows minimal recession. B, After orthodontic tooth alignment (arch expansion) for oral hygiene improvement. Proclination of tooth #41 and alignment was accompanied by a distinct increase of recession and loss of attached gingiva.

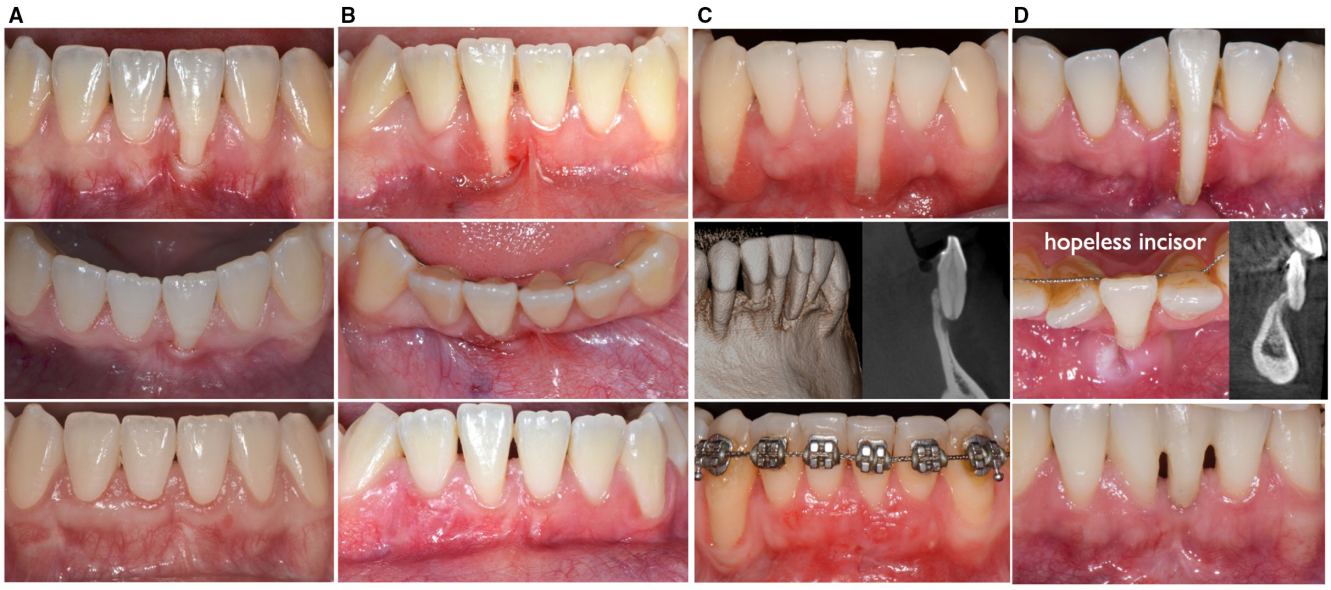

FIGURE 13 Four clinical situations (A-D) of "Wire syndrome" characterized by an increased buccal inclination of incisors and movement of mandibular canines. Irregularity can be graded according to differences in position of the root tip and/or coronal third of the root inside/outside the bony envelope. A, Incisor 31 root tip within the bony envelope, coronal third 1 mm outside the bony envelope, and 3 mm of soft tissue dehiscence: 100% root coverage possible without orthodontics. B, Incisor 41 root tip within the bony envelope, coronal third 2 mm outside the bony envelope, and 5 mm of soft tissue dehiscence: partial root coverage possible without orthodontics. C, Incisor 31 with root tip and coronal third >2 mm visible and outside the bony envelope: root coverage without orthodontics impossible. D, Hopeless lower incisor 31: root tip and coronal third visible on the mesial aspect, completely outside the bony and soft tissue envelope. A, B, C : Recession coverage by Karin Jepsen; D: Extraction of the root by Anton Sculean.

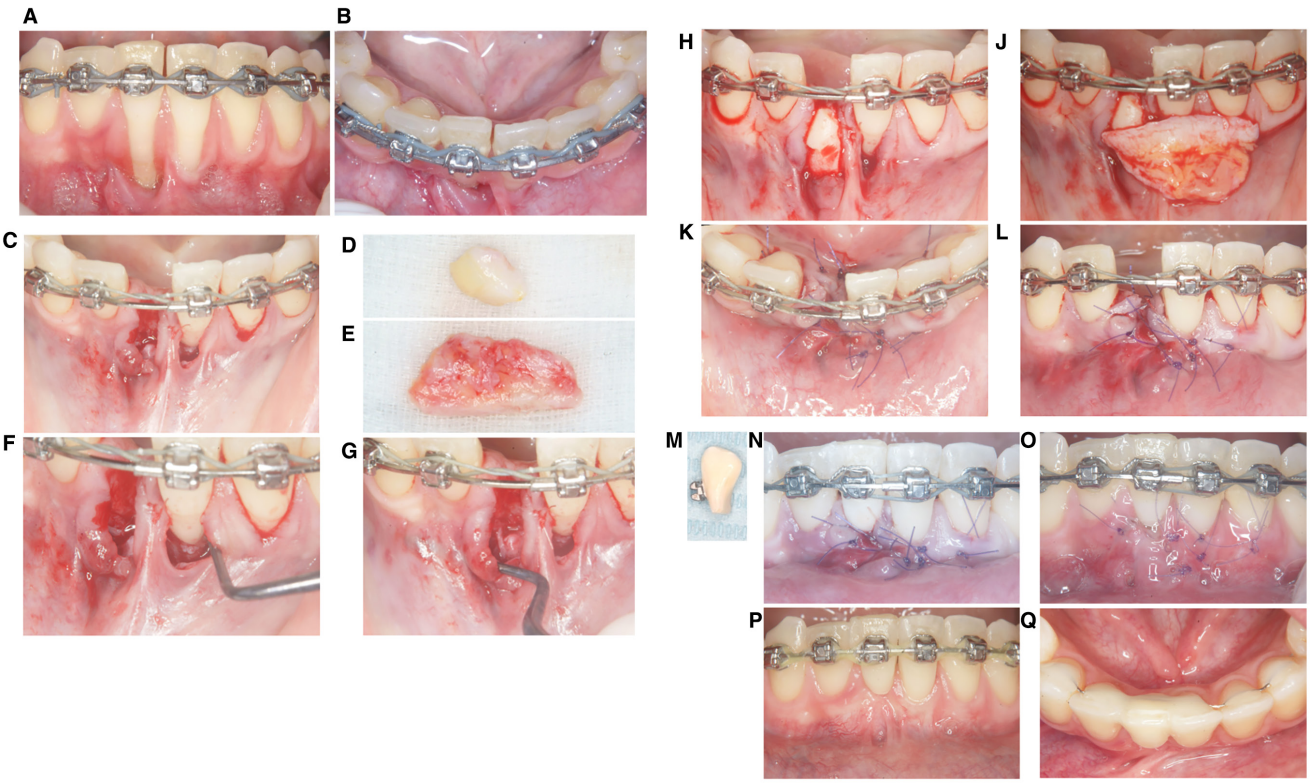

FIGURE 14 Clinical situation of the lower sextant in a 29-year-old healthy female patient undergoing orthodontics for the third time in her life. A, Frontal view. B, Occlusal view. The reason for referral was a progressive, severe recession in the lower anterior sextant. Repeated acute infections had occurred as a result of inadequate oral hygiene. After comprehensive examination it became evident that tooth #41 had become nonvital and was diagnosed as nonmaintainable. Orthodontic space closure after extraction of tooth #41 was not considered because of missing bone and an anticipated unfavorable anterior occlusal relationship (Case courtesy of Giles de Quincey—Department of Periodontology, University of Bern, Switzerland). C, After an initial phase involving scaling and root planing, and oral hygiene instructions, planned surgical treatment was scheduled. Tooth #41 was removed. D, The residual socket was de-epithelialized and sealed using a root-shaped, partially epithelialized connective tissue graft from the tuberosity. Within the same procedure, a palatal subepithelial connective tissue graft was harvested from region #32–42 (using the MCAT (modified coronally advance tunnel)-/LCT (LCT, laterally closed tunnel) technique) (E) for subsequent use in a modified coronally/laterally advanced tunneling technique, with the aim to provide root coverage (F) and gingival augmentation (G). H, Root debridement and EDTA-conditioning was followed by the application of amelogenins to the rinsed and dried root surfaces of teeth #31–32. The root-formed, partially epithelialized connective tissue graft was then sutured in place to the lingual gingiva using resorbable 6-0 sutures. The subepithelial connective tissue graft was inserted into the tunnel (J) and sutured using resorbable and non-resorbable 5-0/6-0 sutures sutures (K, L). M, N, Tooth #41 was temporarily replaced by attaching the autogenous crown to the orthodontic appliance. A nonsteroidal anti-inflammatory drug and chlorhexidine rinse were prescribed postoperatively. Healing was uneventful at 1 week (O) and 8 weeks (P), with limited morbidity at the palatal donor site. Q, Long-term replacement of tooth #41 was achieved by using the autogenous crown as an adhesive bridge combined with a lingual wire retainer. Orthodontic treatment was continued and, 13 months after surgery, tooth #41 was converted to a semi-permanent autogenous adhesive bridge, attached to the orthodontic wire retainer from teeth #33 to 43 and—to prevent rotation—a second wire retainer from teeth #31–42.

4.4 | Periodontal phenotype modification for patients receiving orthodontic treatment

In light of the reported risk for recessions in patients undergoing OT, an American Academy of Periodontology best-evidence consensus statement[120] focused on the question: "Is periodontal phenotype modification therapy beneficial for patients receiving orthodontic treatment?" Based on a best-evidence review of the literature,[96] the authors concluded that periodontal phenotype modification involving hard and/or soft tissue augmentation may provide clinical benefits to patients undergoing orthodontic treatment. They emphasized that the evidence is still limited and based on only a few studies. These studies were unable to provide a definitive answer as to when it is best to perform hard and soft tissue augmentation—before, during, or after orthodontic treatment. It would be reasonable to suggest augmentation before any labial tooth movement, especially in the presence of a thin phenotype or when there is less than 2 mm width of keratinized tissue.[96]

Examples of phenotype modification involving hard and soft tissue augmentation are presented in Figures 15 and 16. A procedure for phenotype modification using soft tissue augmentation before orthodontic treatment is described in Figure 17.

Alternatively, a novel interdisciplinary orthodontic–mucogingival approach for isolated gingival recession defects affecting

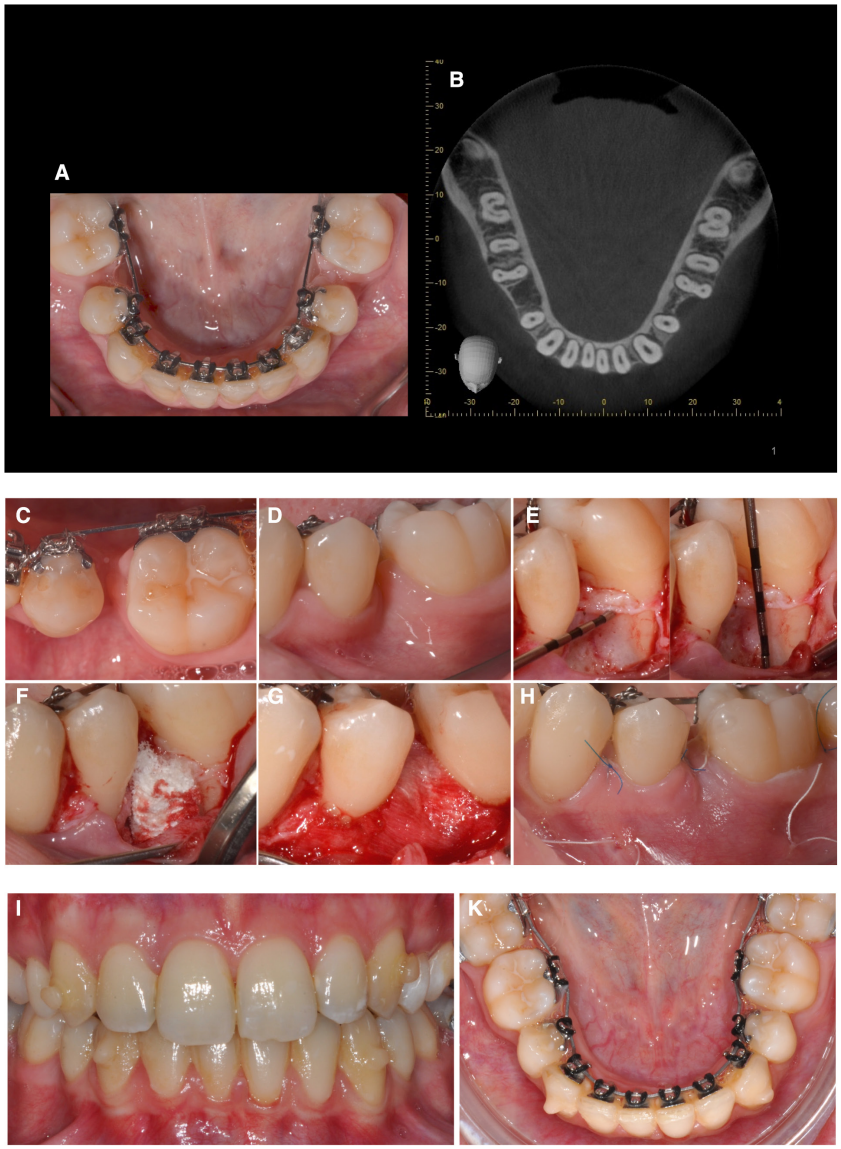

FIGURE 15 This patient is presenting with a thin periodontal phenotype accompanied by a space-closure problem after the loss of a deciduous molar (regio #35). Corticotomy-assisted orthodontic therapy combined with simultaneous bone and soft tissue augmentation (for modification of the periodontal phenotype) was performed to prevent attachment loss/recession. A, Occlusal view showing a splace closure problem in area #35, which is more distinct in the left than in the right quadrant. B, A narrow bony architecture of the collapsed alveolar process is visible in a three-dimensional image of the occlusal view. C, Situation at baseline, occlusal view. D, Situation at baseline, buccal view. E, Postextraction site, 9 mm wide; bone fenestration, 9 mm deep. F, After partial decortication on the mesial aspect of tooth #34 and to the distal aspect of tooth #36 using a deproteinized bovine bone mineral (BioOss®; Geistlich, Wollhusen, Switzerland) to enhance the bony architecture. G, A collagen matrix (Mucograft®; Geistlich) was placed to enhance the soft tissue volume. H, Flap closure. I, Within 3 months, tooth #36 could be moved to close the gap with no loss of periodontal attachment (frontal view). K, Occlusal view (Periodontal surgery: Karin Jepsen; orthodontic therapy: Nikolaos Daratsianos).

mandibular incisors with a labially displaced root has been proposed. This includes selective correction of tooth malposition and subsequent surgical recession coverage with coronally advanced flap and connective tissue graft. In a case series of 20 patients, favorable 1-year results were reported.[122]

4.5 | Gingival clefts

Gingival clefts develop frequently during orthodontic space closure and may compromise the treatment outcome. A randomized trial[123] indicated that the time-point when orthodontic space closure is initiated after permanent tooth extraction affects the incidence of gingival cleft development and that development of a gingival cleft seems to occur more frequently following early initiation of tooth movement and in "fast movers". The development of gingival clefts should be considered as a complication. A severe case of gingival cleft formation in a 23-year-old pre-orthodontic patient with an extraction space #13 to be closed is shown in Figure 18.

4.6 | Loss of interdental papilla

Gingival recession can also be a consequence of periodontal therapy as a result of the shrinkage of soft tissues during the resolution of inflammation. The pattern of interdental tissue support can have a major esthetic effect and may also influence the outcome of periodontal attachment regeneration. The appearance of so-called "black triangles" is generally considered to be esthetically unacceptable because these are undesirable in an esthetic smile.[124] The absence of an interdental papilla is one of the most challenging and troubling dilemmas in the treatment of periodontal disease. Besides esthetics, patients complain about functional and phonetic problems, leading to difficulties in personal relationships, self-esteem, and self-perception.[125]

FIGURE 16 A, Typical example of a progressive recession with gingival inflammation following retainer-enhanced plaque accumulation: recession is labial and mesial, frenum attachment, bony dehiscence is present between incisors, and the patient has a thin gingival phenotype. All images in (A) show the situation after repeated oral hygiene instructions and professional tooth cleaning. B, Surgical procedures, including lateral bone augmentation between the central incisors using a deproteinized bovine bone mineral (BioOss®; Geistlich), application of enamel matrix derivative (Emdogain®; Straumann, Basel, Switzerland), and positioning of a connective tissue graft (CTG) underneath a coronally positioned flap, were performed C, One year result: images show complete coverage after coronal positioning of the flap (Periodontal surgery: Karin Jepsen).

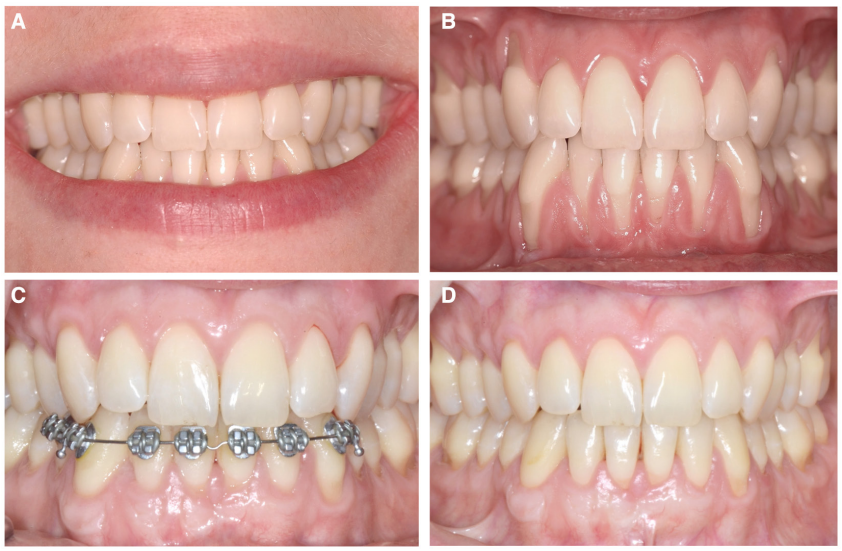

FIGURE 17 A, Smile line of a 30-year-old female patient. She was more worried about a prominent lower right canine from an esthetic point of view than about the severe recession. B, Severe recessions up to 8 mm deep were present on the lower right canine (tooth #43). C, Situation after phenotype conversion before the initiation of orthodontic therapy. Active tooth movements started 1 year after recession coverage procedures. D, Five years after debonding (Orthodontic therapy: Christoph Reichert. Periodontal surgery: Karin Jepsen).

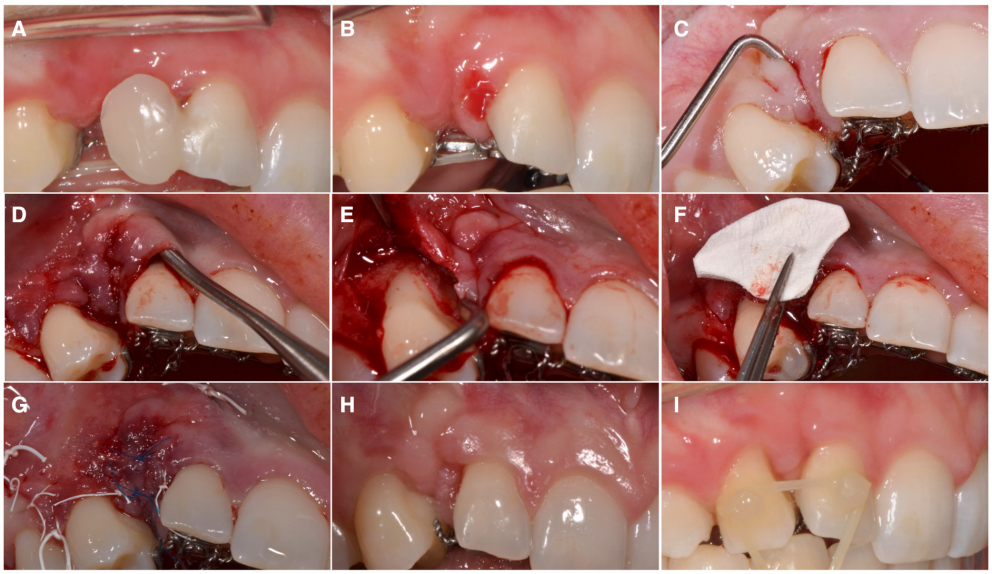

FIGURE 18 Clinical situation of a 23-year-old healthy female patient with a missing canine tooth (extraction space #13). Having been severely displaced, the tooth had to be removed surgically. A, Situation immediately after removal of the retained and displaced canine. B, During movement of tooth #14 toward the right segment, an invagination became evident in the middle of the gap. C, Situation before periodontal surgery; a buccopalatal through-and-through cleft-like defect was present. D, Full-thickness flap with maximal soft tissue preservation, a vertical incision was made on the distal aspect of tooth #14 for access. E, Bony defect visible on the mesial aspect. F, Placement of a collagen matrix and a bone substitute (BioGide®, BioOss®; Geistlich). G, Wound closure. H, Two weeks postoperatively. I, Six months postoperatively. The space had almost closed and there was no additional periodontal damage (Periodontal surgery: Karin Jepsen; orthodontic therapy: Nikolaos Daratsianos).

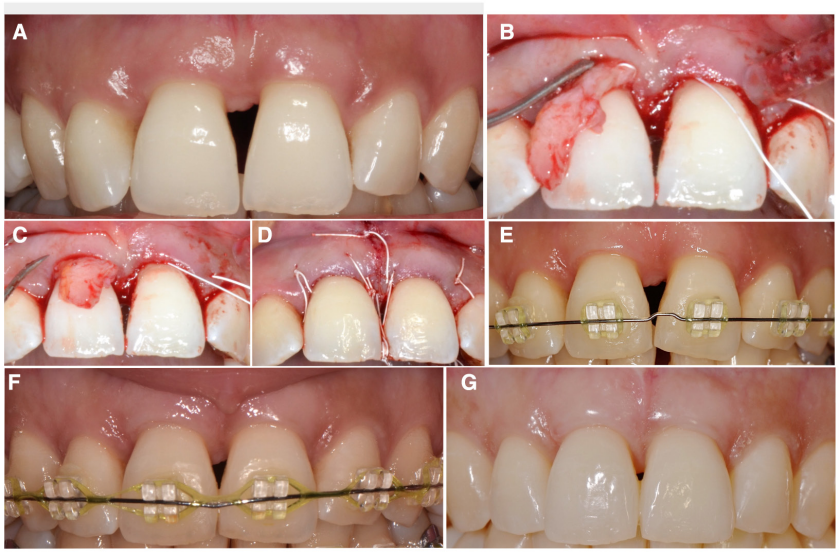

FIGURE 19 A, Clinical situation after a 23-year-old female patient with the diagnosis periodontitis stage IV, grade C, presenting with a complete loss of the papilla, class 3, between the maxillary central incisors, underwent nonsurgical therapy. Tunnel preparation (B) and placement of a subepithelial connective tissue graft (C). D, Microporous, monofilament suture (Goretex 5-0; W. L. Gore & Associates, Flagstaff, AZ, USA). E, Tooth intrusion and mesial torquing movements. F, In addition to the previous treatments, interdental stripping reduces the bone crest—contact point distance. G, Clinical situation 1 year after debonding, demonstrating that the soft tissues adapted to the new emergence profiles of the teeth as the interproximal spaces were reduced (Periodontal surgery: Karin Jepsen; orthodontic therapy: Andreas Jäger).

Nordland and Tarnow[126] described a classification for papilla loss that was based on three identifiable anatomical landmarks. Tarnow et al[127] found that when the distance between the bone crest and the contact point was 5 mm or less, a papilla was present in 98% of cases; this dropped to 56% and 27% when the distance from the bone crest to the contact point was 6 and 7 mm, respectively. Wu et al[128] reached a similar conclusion. In general, this means that the shorter the distance between the interproximal

contact and the bone crest, the less likely it is that a papilla will be missing.

Adjunctive orthodontic tooth movement can be beneficial in reducing interdental papilla loss. Closing the interdental contacts by conventional orthodontic movement, with or without interdental stripping, reduces the bone crest—contact point distance. However, the length of treatment, the need for appliances, and cost are limiting factors. Burke et al[129] recommended bringing the roots closer using a mesial torquing movement in an attempt to compensate for missing papillae. In conjunction with orthodontic treatment, proximal enamel can be recontoured to change the contact area to a broader surface, along with relocating the contact more apically. Cardaropoli et al[130] presented a study evaluating a combined approach of orthodontic and periodontal treatment to reconstruct the interdental papillae between upper central incisors, demonstrating that the soft tissues adapted to the new emergence profiles during intrusion of the teeth as the interproximal spaces were reduced. A clinical example of a combined periodontal-orthodontic approach, including proximal enamel reshaping together with soft tissue augmentation and orthodontic tooth approximation, is presented in Figure 19.

In summary, OT can have adverse effects on mucogingival conditions. Especially in situations with a thin gingival phenotype, labial tooth movement—in particular of mandibular incisors with proclination—can result in recession defects. This should be regarded as a common complication of which the patient needs to be informed prior to therapy and preventive phenotype modifications considered. Advanced recession defects resulting from tooth movements outside the bony envelope—often associated with rapid arch expansions—may be considered as treatment errors. Postorthodontic changes of tooth position can occur as a result of the use of nonpassive retention devices leading to increasing recession defects/root exposure ("wire syndrome"). These adverse events should be considered at least as complications, but, if left unnoticed and/or unaddressed, as treatment errors. The development of gingival clefts following orthodontic space closure occurs frequently and is viewed as a common complication. By contrast, orthodontic tooth movement can facilitate treatment of the "loss of interdental papilla" through a combined periodontal–orthodontic approach.

In conclusion, the present review has identified several areas of concern, in which OT can have unwanted adverse effects on periodontal/mucogingival conditions, but also areas with great potential for synergies between orthodontic and periodontal therapy.

Altogether, these call for close consultation and offer excellent opportunities for cooperation between the two specialties for the benefit of patients affected by tooth malpositioning and periodontal or mucogingival problems.

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