Phenotype Modification Therapy: New Challenges and Opportunities

Richard T. Kao, Donald A. Curtis, George A. Mandelaris, Colin Richman, Shan-Huey Yu, Katie Goss, and Guo-Hao Lin

Key Findings

Phenotype Modification Therapy (PhMT) is the utilization of a wide possibility of surgical procedures which can improve periodontal/peri-implant health. Additionally, it has the potential of enhancing, preventing, and withstanding remodeling effects associated with restorative and orthodontic treatments.

The concept of thick vs. thin gingiva was first introduced in a commentary regarding tissue responses to restorative treatments [1]. This resulted in an improved appreciation and understanding of tissue biotypes. With only free gingival grafts as the primary surgical therapeutic treatment, the options for modification of gingival tissues were initially limited. However, with recent surgical advances in gingival grafting and periodontal regeneration, many options are now available to modify periodontal tissue characteristics to better support health and stability. This chapter summarizes how surgical modification procedures called Phenotype Modification Therapy (PhMT) can improve periodontal/peri-implant health, particularly in association with restorative and orthodontic treatments [2].

R. T. Kao (✉)
Orofacial Sciences, University of California San Francisco, San Francisco, CA, USA

Private Practice, San Jose, CA, USA

D. A. Curtis
Preventive and Restorative Dental Science, University of California San Francisco, San Francisco, CA, USA

G. A. Mandelaris
Periodontics and Oral Medicine, University of Michigan, Ann Arbor, MI, USA

Private Practice, Chicago, IL, USA

C. Richman
Private Practice, Atlanta, GA, USA

S.-H. Yu
Periodontics and Oral Medicine, University of Michigan, Ann Arbor, MI, USA

K. Goss
American Academy of Periodontology, Chicago, IL, USA

G.-H. Lin
Orofacial Science, University of California San Francisco, San Francisco, CA, USA

Evolution of Appreciation of Gingival Tissues

Thick gingival tissue is probably the image most associated with periodontal health (Fig. 1a). This tissue is dense in appearance with a wide zone of attached gingiva. The gingival topography is relatively flat with the suggestion of a thick underlying bony architecture. Surgical evaluation of these areas often reveals relatively thick underlying osseous forms (Fig. 1b). Conversely, thin gingival tissue tends to be delicate and almost translucent in appearance (Fig. 1c). The tissue appears friable with a minimal zone of attached gingiva. The soft tissue is highly accentuated and often suggestive of thin or minimal bone over the labial roots. Surgical evaluation often reveals thin labial bone with the possible presence of fenestrations and dehiscences (Fig. 1d).

The concept of thick vs. thin gingival biotype was expanded to include the differential manner in which these tissue biotypes respond to inflammation, restorative trauma, and parafunctional habits (Tables 1 and 2) [3]. Defects resulting from these traumatic events dictate the necessary treatment/management modalities used to support health and manage/treat pathosis. Gingival biotypes also dictate different procedures for implant site preparation [4, 5].

As this concept of gingival biotype has evolved, recent literature has defined periodontal and peri-implant health based on anatomic characteristics of components of the masticatory complex, including (1) gingival thickness (GT) or peri-implant tissue thickness and keratinized tissue width (KTW); (2) bone morphotype; and (3) tooth dimensions. With the introduction of the 2018 Classification of Periodontal and Peri-Implant Diseases and Conditions, a new term—***periodontal phenotype***—was adopted to describe

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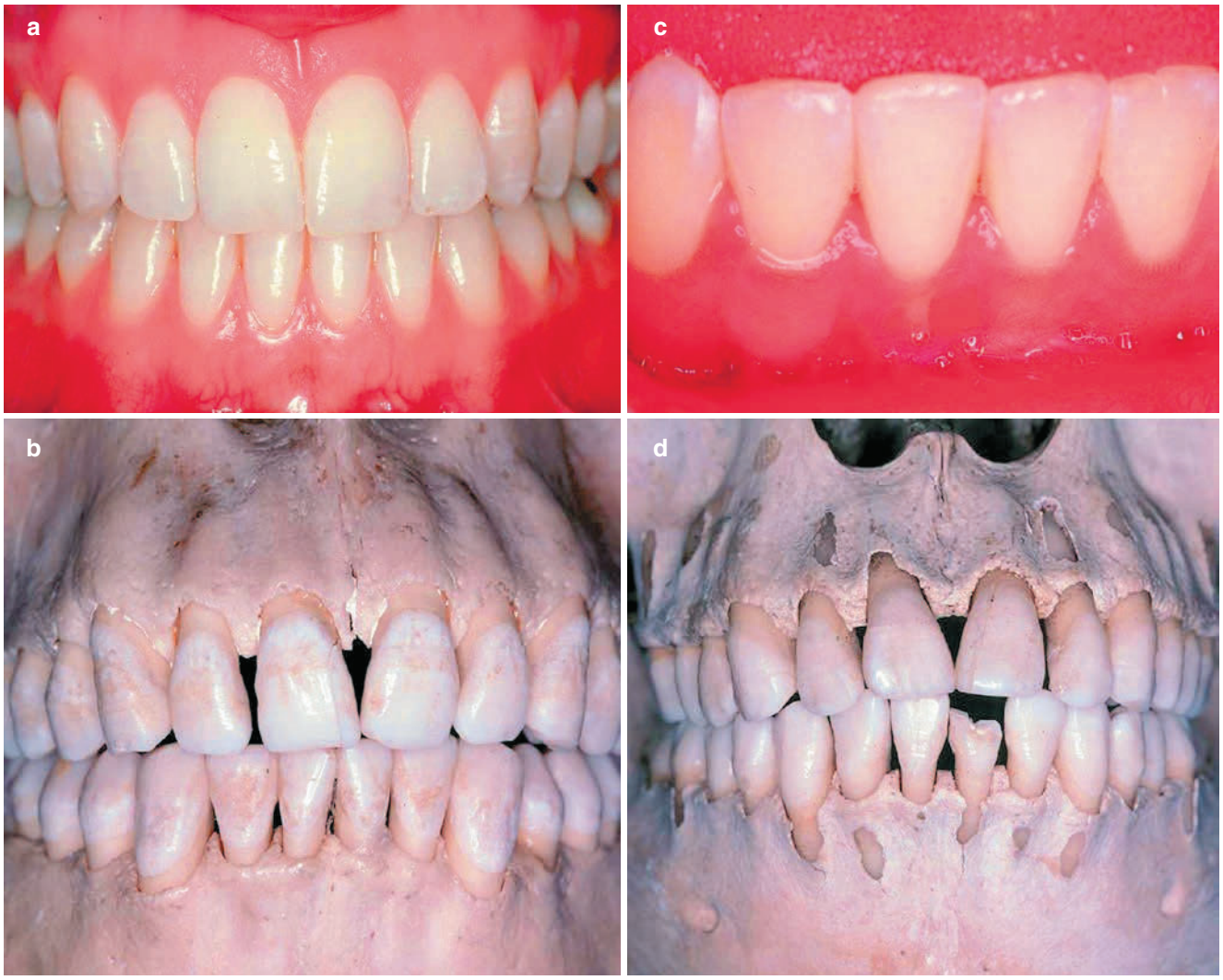

Fig. 1 Clinical and skeletal presentation of thick vs. thin gingival phenotypes. (**a**) Clinical presentation of thick gingival phenotype; (**b**) the typical underlying osseous architectures; (**c**) clinical presentation of thin phenotype; and (**d**) the typical underlying osseous architectures with presence of marginal bone loss, fenestrations, and dehiscences

Table 1 Comparison of thick vs. thin gingival biotype characteristics

Thick gingival biotype	Thin gingival biotype
• Relatively flat soft tissue and bony architecture • Dense fibrotic soft tissue • Relatively wide zone of attached gingiva • Thick underlying osseous form • Relatively resistant to acute trauma • Reacts to disease with pocket formation and intrabony defect formation	• Highly scalloped soft tissue and bony architecture • Delicate friable soft tissue • Minimal amount of attached gingiva • Thin underlying bone characterized by bony dehiscence and fenestration • Reacts to insults and disease with gingival recession

the combination of gingival biotype (three-dimensional gingival volume) and bone morphotype (thickness of the bone plate) [6, 7]. This term has been expanded to include peri-implant dimensions to describe the peri-implant phenotype (see Table 3) [2, 8].

This chapter focuses on how a thick or thin gingival/peri-implant phenotype influences a response from restorative procedures or trauma. The dimensions of periodontal and peri-implant phenotype differ in healthy patients and those at risk for development of recession and periodontal/peri-implant marginal bone loss (see Table 3). It should be noted

that a thin phenotype has an increased risk for pathosis (recession, inflammation, periodontitis/peri-implantitis). Over the past 25 years, advances in surgical interventions to improve soft and hard tissues have expanded the clinician's options for managing/treating these problems [9–14]. These surgical interventions, collectively called phenotype modification therapy (PhMT), can improve the dimensions of soft tissue, bone, or both (Table 3). In healthy patients and in those with pathosis, inflammation and acute trauma (i.e., viral lesion along the gingival margin or acute gash with chicken bone) may result in attachment loss and gingival recession. With a thick phenotype, the tissue usually will heal and with chronic inflammation, pockets may form and intrabony defects may result. With a thin phenotype, the reparative response is usually the appearance of attachment loss and gingival recession. There are many gingival grafting procedures that can be performed to prevent or correct gingival recession [9, 10]. While these procedures may prevent or minimize future attachment loss, orthodontic movement generates chronic forces which increase the potential for loss of the bony housing and associated soft tissue. Therefore, PhMT may require augmentation of the osseous housing and, at times, a concurrent increase in gingival tissue thickness. This is necessary so that orthodontic tooth movement does not result in increased development of bony dehiscence/fenestration, gingival recession, mobility, and risk for future recession-based attachment loss.

The purpose of this chapter is to define parameters for periodontal and peri-implant health and describe how PhMT can help maintain or improve dental health, particularly prior to extensive restorative and orthodontic treatment.

Table 2 Differential response to trauma, inflammation, and chronic diseases by thick vs. thin biotypes

	Thick gingival biotype	Thin gingival biotype
Inflammation	• Soft tissue: Marginal inflammation; cyanosis; bleeding on probing; edema /fibrotic changes • Hard tissue: Bone loss with pocket formation/ intrabony defects	• Soft tissue: Thin marginal redness and gingival recession • Hard tissue: Rapid bone loss associated with soft tissue recession
Surgical response	• Predicable soft tissue and hard tissue contour after healing	• Difficult to predict where tissue will heal and stabilize
Tooth extraction	• Lower risk for ridge atrophy	• Increased risk for ridge resorption in the apical and lingual direction due to the loss of bundle bone
Implant placement	• Easier to achieve implant stability due to less tendency for ridge atrophy	• Increased risk for complication due to resorption and post-extraction remodeling
Implant maintenance	• Easier to maintain peri-implant tissue stability	• Increased risk for recession and marginal bone loss

The *Cognitive* Clinical Approach to PhMT

So how does one assimilate the knowledge accumulated to date regarding the dimensions of periodontal and peri-implant phenotype and apply it in clinical practice? First, the clinician must appreciate that this knowledge represents the culmination of histological and clinical studies which best support tissue health [2]. There are average tissue dimensional relationships that are associated with health. As such, just like our understanding that supracrestal tissue attachment (previously biologic width), in his studies of human cadavers, Gargiulo found that a connective tissue attachment of 1.07 mm and a junctional epithelium of 0.97 mm best supported periodontal health when subgingival restorations were in proximity of the supracrestal tissue attachment [15]. As such these are dimensional means of the gingival width, thickness, and bone morphotype in health. When tissues are subject to inflammation, trauma, close proximity to restorative margins, or orthodontic treatment, these periodontal phenotype structures are *challenged.* Though we know most

Table 3 Phenotype modification therapy for improving periodontal and peri-implant tissue quality

	Dental thick phenotype	Dental thin phenotype	Dental PhMT	Peri-implant thick phenotype	Peri-implant thin phenotype	Peri-implant PhMT
Keratinized tissue width	5.09–6.65 mm (mean 5.72 mm) >2 mm[a]	2.75–5.44 mm (mean 4.15 mm)	FGG, SCTG	>2 mm[a] SxD	<2 mm SxD	SCTG, FGG
Gingival thickness	1.24–1.79 mm >1 mm[a]	0.63–1.24 mm (mean 0.80 mm)	FGG, SCTG, filler substitutes	>2 mm[a] SxD	<2 mm	SCTG, FGG, filler substitutes
Bone thickness	0.75 mm mean	0.34 mm mean	PAOO, SFOT, or Wilcodontic's	>2 mm[a] SxD	<2 mm SxD	GBR, filler substitutes, bone graft, combination of above

FGG free gingival graft, *SCTG* subepithelial connective tissue graft, *PAOO* periodontally accelerated osteogenic orthodontics, *SFOT* surgically facilitated orthodontic therapy, *SxD* surgically determined/modified at the time of placement, *GBR* guided bone regeneration
[a]Therapeutic goals

thick phenotypes will respond predictably to the rules of supracrestal tissue attachment, thin or "thick appearing" phenotypes generally will not respond in a predictable fashion. The final healing position of thick gingival tissue after a crown lengthening procedure is more predictable than with a thin phenotype. Additionally, thin phenotype tissue will require more time to achieve maturation and stability. When the clinician determines that the tissue dimension is not ideal, PhMT strategies should be applied. Even when the dimensions are optimal as outlined in Table 3, it is better to overcompensate since tissues do not always follow the norm.

Following is a case example that illustrates this point. In Fig. 2, an Asian patient came to the practice complaining about the appearance of the front bridge he received 6 years earlier. On initial analysis, one might conclude that the previous clinician did a poor job and the bridge needs to be redone. The restorative margin of #9 is defective and the bridge was cemented short of the preparation margin. Further examination of the thickness of the gingival margin of #8 suggests it is reasonably thick. The #8 pontic is long and there appears to be a soft tissue deficiency on the apical aspect. The crown margin is probably acceptable; however, there is recession associated with this restorative margin. No other recession is readily visible in this case. Re-doing this case may yield a similar result for the patient.

The *cognitive* clinician should learn not just the average phenotype dimension but appreciate that in some healthy patients—and even more so in those with pathosis (increased inflammation and plaque accumulation with crown margins)—tissue responses may be amplified. This is actually a thin periodontal phenotype. If the clinician studied the response of the tissue beneath the pontic, he/she would realize that the longer length suggests that there was quite a bit of remodeling of the underlying bone. Further remodeling after the placement of the bridge resulted in the soft tissue deficiency. In other words, this "thick" appearing case is actually a thin case. The previous bridge was placed too quickly post-extraction. As a result, the tissue continued to remodel, resulting in further bone loss under the pontic. The response of the tissue may have been more apparent if ideal provisionalization was fabricated. What was needed in this case was longer provisionalization and more than the conventional 2–3 months of soft tissue healing. This would also explain the recession seen on #7 and #9. When a restoration is placed at the extraction site in a thin periodontal phenotype, the healing time needs to be extended beyond the routine 2–3 months associated with a thick phenotype.

Another consideration beyond the thin periodontal phenotype is the fact that the patient is Asian. Though there is no evidence that correlates sex and age with gingival phenotype, there is some evidence that there are differences in gingival thickness between ethnic groups [16]. While current knowledge regarding dental morphology is based on characteristics found in white subjects, there are studies that suggest that gingival biotypes are more prevalent in Asians [17, 18]. Asians may be more prone to gingival recession and encounter more difficulties with esthetic reconstruction. Conversely, a recent report on biotypes in African-Americans suggests this population not only has a thick biotype but also may have a wider attached gingival zone with lower incidence of gingival recession [19]. Therefore, it is prudent to consider these ethnic differences in gingival phenotypes and how these variations may influence the clinician's ability to triage and treatment plan.

So how does the cognitive clinician avoid these problems? First, he/she needs to define the tissue phenotype. There are several rules about making a *tentative diagnosis* of thick vs. thin phenotype [2, 16, 20, 21]; however, the tissue response to pathologic insults (recession, inflammation, periodontitis/peri-implantitis) may vary. Additionally, with the advent of PhMT, we now have surgical intervention strategies to make the tissue phenotype more resistant to remodeling effects. One can respond *reactively*, in which case the results discussed in Fig. 2 will continue to recur until the clinician takes the time to appreciate the tissue changes. Alternatively, the cognitive clinician can respond *proactively,* using a surgical strategy such as gingival grafting, bone grafting/augmentation, or corticotomy-bone grafting (surgically facilitated orthodontic therapy/periodontally accelerated osteogenic orthodontics, SFOT/PAOO) to alter the tissue phenotype in preparation for *possible* pathologic/iatrogenic insults. Some consequences of the reactive approach are easy to correct such as gingival recession. However, the damaging results associated with peri-implantitis or orthodontic movement of teeth out of the bony housing may have significantly negative impacts. As you explore this topic further in this chapter, consider that many of the following procedures should be undertaken to avoid clinical complications.

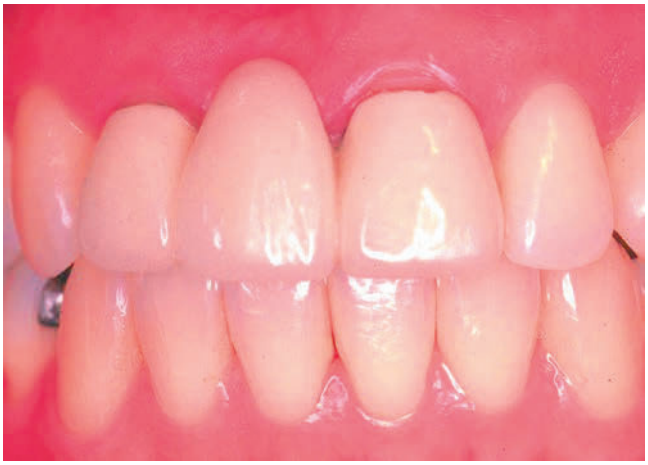

Fig. 2 Tissue response in thin phenotype in response to previous restorative treatment. How did this happen?

Improving Gingival Phenotype for Restorative Treatment

Restoratively Driven Needs for Phenotype Conversion

Surgical intervention to modify the gingival tissue from a thin to a thicker gingival phenotype is often considered for esthetic and functional reasons. The restorative dentist should carefully evaluate the potential benefits of a phenotype conversion, taking into consideration the planned restorations, materials to be used, esthetic requirements, rate of soft tissue changes, anticipated access for cleaning, and patient comfort. Adding soft tissue through free gingival grafts (FGG), subepithelial connective tissue grafts (SCTG), or adding keratinized tissue (KT) can improve the symmetry of the soft tissue as well as limit the impact of restorative interventions or toothbrush abrasion by the patient. There should be compatibility between the planned restorative procedure and the supporting soft tissues. In patients with a thin tissue phenotype, the impact of restorative procedures needs to be carefully evaluated.

In patients with a thin tissue phenotype, restorative procedures will have a different impact in a natural dentition than in implant-supported restorations. For example, when planning an all-ceramic crown in a dentition with a thin tissue phenotype and minimal attached tissue, the clinical situation can often remain stable if the patient has good oral hygiene. In contrast, when planning an implant-supported crown on a patient with a thin tissue phenotype and limited keratinized tissue, it is more often necessary to add keratinized tissue. This is because the tissues around an implant-supported restoration are often sensitive and grafting a 2-mm band of keratinized tissue allows for improved comfort when the patient is completing oral hygiene maintenance [20, 21].

It is important to remember that soft tissues are constantly adapting and responding to stimuli. Restorative procedures will elicit different reactions depending on the type of restoration and the soft tissue characteristics. Therefore, the goal is to modify the soft tissue characteristics to be compatible with the planned restorative procedures. The soft tissue modification should ideally be completed prior to the restorative treatment. Additionally, extra care should be afforded patients with a thin tissue phenotype. Mistakes often have a more pronounced impact in patients with a thin tissue phenotype.

For example, Fig. 3 illustrates a patient with a thin tissue phenotype and a distal extension partial denture fabricated that includes an inappropriate fulcrum, resulting in severe trauma to the patient's tissues. The impact is magnified in the thin tissue phenotype patient.

In this thin tissue patient, several unfavorable soft tissue adaptations can be seen (Fig. 4). Toothbrush abrasion primarily on the patient's left side has resulted in significant recession, especially around the endodontically treated tooth #10. Recession has also exposed the margin of the fixed prosthesis on tooth #8. The rapid rate and degree of recession are more likely to occur in a patient with a thin tissue phenotype.

Patients will often present with a non-carious cervical lesion (NCCL) and have esthetic concerns or symptoms consistent with dentin sensitivity (Fig. 5) [22]. NCCLs are more frequently seen with increasing age, in the maxillary arch, in the premolar area and most frequently related to toothbrush abrasion [9]. The patient will often ask if a restoration can be placed to cover the yellow appearance of the exposed dentin. However, a far better option is to consider having the periodontist blend the apical aspect of the NCCL with odontoplasty and place a connective tissue graft approximately 1–2 mm coronal to that margin. If after 2–3 months of healing the tissue is stable, the clinician can consider restoring

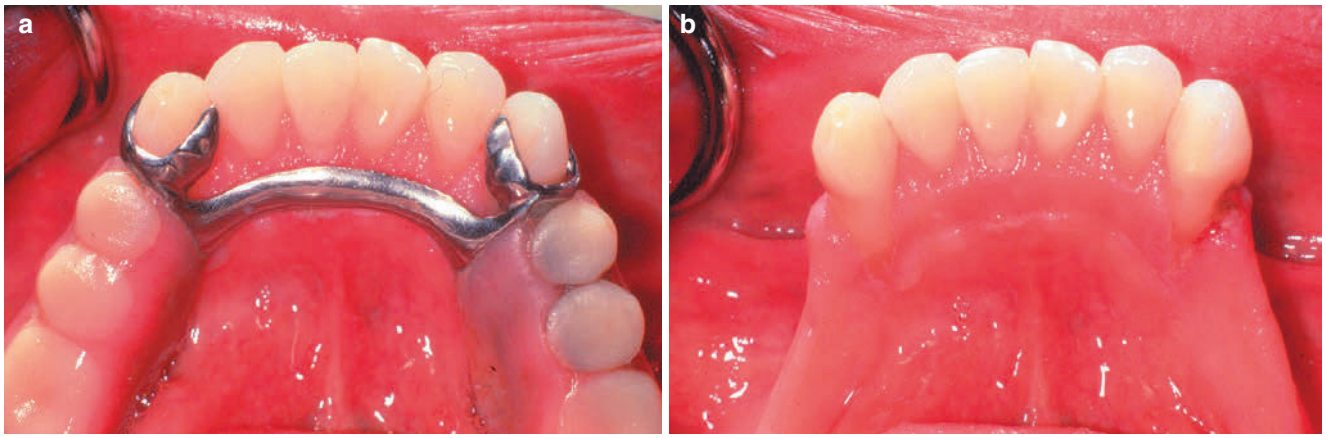

Fig. 3 This is a case of long-termed use of a partial denture where monitoring was neglected. Settling of the denture resulted in gingival recession. Resolution is possible with subepithelial connective tissue grafting but this often result in a period where the prosthesis cannot be used during the healing period

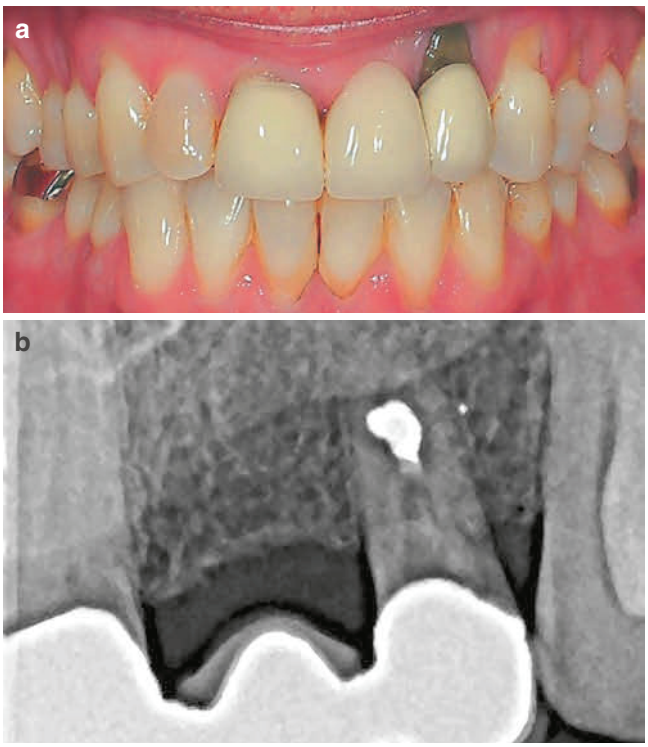

Fig. 4 What happened? The area of #10--12 has thin gingival phenotype which is reflected by generalized recession in this area due to toothbrush abrasion (a). After the prosthesis was placed, #10 displayed continual increase in gingival recession. Radiographs reviewed that #10 was treated with retrograde procedure but the canal has not been obturated (b). The endodontic lesion resulted in venting of the infection resulting in additional attachment loss and gingival recession

the coronal aspect of the NCCL with fluoride containing a glass-ionomer composite. Adding soft tissue can help stabilize and limit future restorative needs in patients with NCCL. For example, in patients with NCCLs followed for 25 years, those who had root coverage procedures were found to need fewer restorations [22].

In patients with thin tissue, greater care needs to be exercised by the restorative dentist with regard to tissue trauma during tissue retraction, the use of provisional restorations that are carefully adapted, and when selecting the restoration. This is because trauma will result in recession in a thin tissue phenotype patient.

Restoratively Driven Needs for Phenotype Conversion in Patients with Dental Implants

PhMT for soft tissue modification (PhMT-s) around implants usually includes FGG, SCTG procedures, or the use of substitutional material (i.e., collagen matrix or acellular dermal matrix) while PhMT for bone morphotype modification (PhMT-b) around implants is usually achieved via guided bone regeneration (GBR) procedures.

In a patient with a thin tissue phenotype adjacent to dental implants, midfacial recession has been reported to occur in an average of 26% of patients [23]. Midfacial recession can be minimized by using a connective tissue graft for PhMTs [24]. A good general guideline is that when the facial soft tissue thickness is less than 2 mm and esthetics is important, then a connective tissue graft and zirconia abutment should be considered [25]. The problem with titanium abutments is that they can give a grayish color to the restoration. The restorative material needs to be matched to the gingival thickness and the esthetic requirements. There is also some evidence platform switching in thin tissue phenotype patients will help preserve bone levels [26]. A comprehensive assessment of biologic complications can help in educating the patient and in ensuring the various risks has been considered [27, 28].

A peri-implant phenotype is composed of soft tissue components and the bone morphotype. Restoratively, ideal soft tissue components include adequate connective tissue thickness, keratinized tissue width, and supracrestal tissue attachment. Clinical experiences and research studies have suggested surgical modification of peri-implant phenotype from a thin to a thick peri-implant phenotype could lower the risk of future recession around dental implants and improve esthetics [20, 29].

PhMT-s with FGG, SCTG, or substitutes are mostly utilized to increase tissue thickness or width of keratinized gingiva. Reviews indicate an average gain of 1 mm tissue thickness can be expected from various soft tissue grafting procedures with autogenous grafts [15, 20]. Following is evidence supporting the benefits of PhMT-s:

- Since peri-implant tissue is lacking in periodontal ligament which provides a protective fibrous seal, increased peri-implant tissue thickness is required to minimize the risk of future recession and maintain esthetics [30].
- A prospective study followed implants lacking keratinized gingiva that were either treated with or without free gingiva grafts. After 18 months, there was a significant gain in width of keratinized tissue in the implants that received FGG versus the control group. Additionally, the crestal bone level in the FGG group was more stable compared with the control group [31].
- A recent systematic review confirmed that PhMT with SCTG is expected to yield an increase of approximately 1 mm of tissue thickness based on the meta-analysis [19]. Zucchelli et al. reported 5-year results from a prospective cohort study correcting single dental implant soft tissue dehiscence with coronally advanced flap in combination with SCTG and crown replacement [32]. At the end of the study period, soft tissue dehiscence was completely covered in 15 out of 19 cases (79%). In addition, there was a statistically significant increase in buccal soft tissue thick-

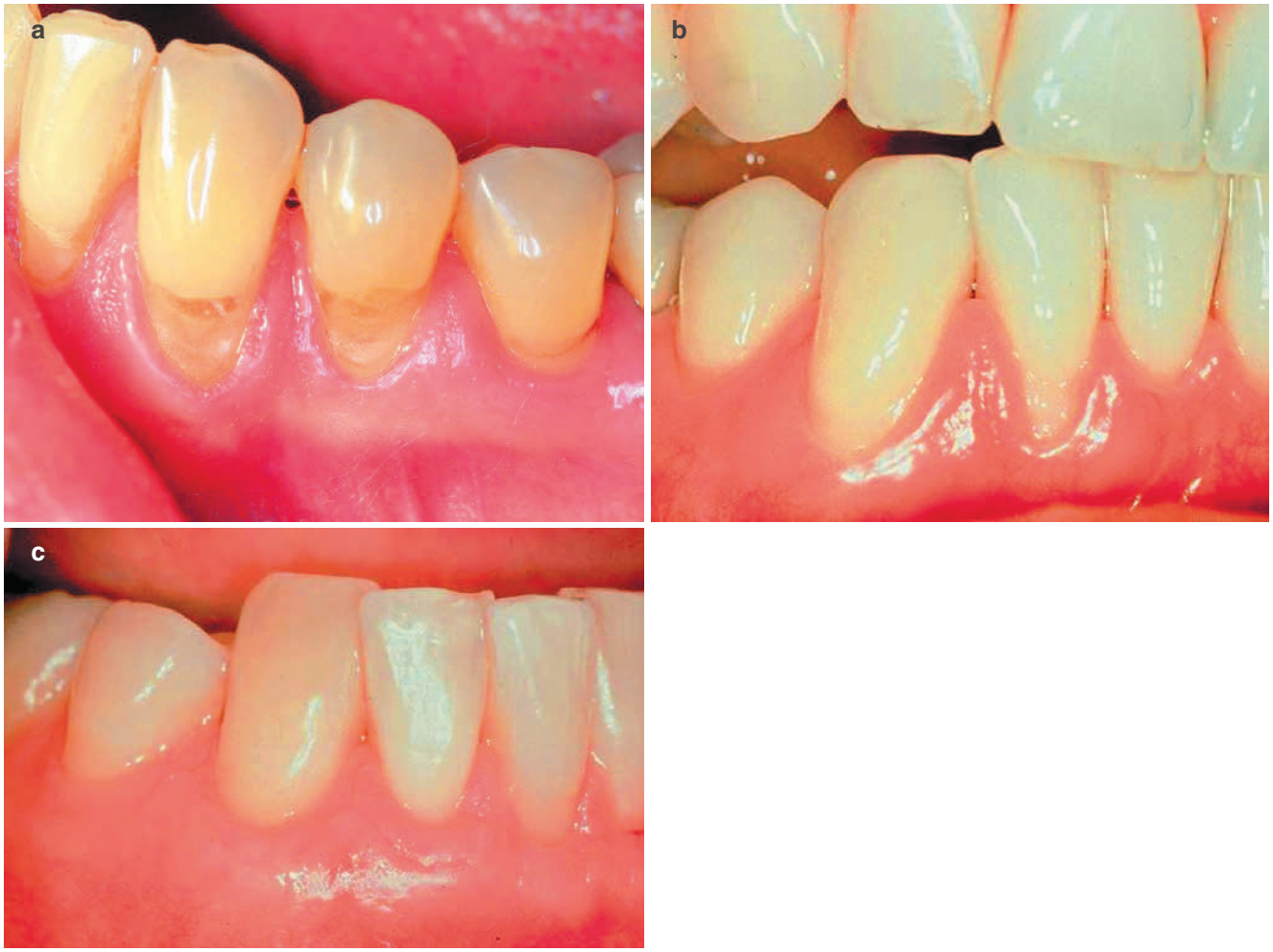

Fig. 5 (**a**) NCCL provides a clinical challenge in that in advance cases, there is loss of both coronal and root structures. Restorations require a combination of SCTG followed by restorative treatment for the coronal tooth structure loss. (**b**, **c**) Minor NCCL can be managed with odontoplasty and SCTG

ness as well as keratinized tissue height at 1-year post-treatment and the tissue phenotype was maintained throughout the 5-year follow-up [32]. To achieve a favorable outcome, a pre-surgical prosthetic phase consisting of removal of the crown and reduction of the implant abutment was suggested by the authors to allow an increase in soft tissue volume between the abutment and the adjacent teeth [32]. The final restorations were fabricated and delivered 8 months after the surgical procedures.

- In a recent study, it was reported that sites with PhMT-s gained 34% tissue thickness after 2 years of follow-up, whereas sites without a connective tissue graft lost 10% of tissue thickness [33]. Therefore, performing PhMT-s to change soft tissue thickness seems to be an enduring and predictable approach. A thickened soft tissue phenotype can help decrease the soft tissue discoloration and show-through of the restorative material versus a thin tissue phenotype. The thickened tissue also provides more volume, enabling the clinician to develop more idealized crown contours, which has both esthetic and biologic advantages [34, 35].
- Rosen presented two cases of PhMT-s with SCTG around dental implants and demonstrated that phenotype modification could prevent further gingival recession and provide a more stable environment conducive to maintaining peri-implant health up to 5 years [36]. Additionally, it is strategically easier to manage cases by ***proactively*** performing PhMT-s by grafting prior to the appearance of recession and marginal bone loss as compared to ***reactively*** responding to the problem. Substitute materials are mainly used to increase tissue thickness but they rarely increase keratinized tissue width.

To conclude, PhMT-s to increase the keratinized width around implants are associated with greater reduction in gin-

gival and plaque indices when compared to non-grafted sites [37] and potentially prevent interproximal marginal bone loss around dental implants [20, 38]. PhMT-s to increase the tissue thickness around implants are also shown to be associated with less marginal bone loss and less amount of mucosal recession [20, 37].

In addition to the fact, PhMT-s provide a more stable peri-implant tissue environment, the resulting tissue is also restoratively more ideal. It has been reported that when the soft tissue phenotype is thin, ridge lapping restoration design is often necessary which limits access for cleaning [38]. By thickening the patient's soft tissue phenotype with PhMT, it is easier to avoid the ridge-lap of crown restorations and develop a crown emergence profile that is easier to facilitate patient's oral hygiene and maintain long-term tissue health and esthetics.

For implant stability, the bone morphotype needs to have adequate thickness so it is not prone to remodeling. Based upon the available evidence, most studies utilized 2 mm as the threshold thickness to negate peri-implant bone remodeling [25, 39, 40]. PhMT-b is probably to most critical for peri-implant health. While we can often "sneak" an implant into the bony housing, one needs to be cognizant that should there be any suspicion that there is inadequate bone volume as described above, it is critical to be proactive to include bone augmentation as part of treatment planning. For the reactive clinician, the problem is often not recognized until marginal bone loss and soft tissue recession have appeared. Unfortunately, it may take years for the damage to be apparent, so it is important to be proactive in assuring that there is adequate peri-implant bone thickness.

Even after implant placement, the bony housing may continue to remodel. This remodeling process may result in horizontal and vertical bone resorption which predisposes the implant to esthetic and biologic complications [41]. It is generally accepted that a minimal buccal bone width of 2 mm is needed to compensate for physiologic bone remodeling and prevent future complications such as tissue recession [42, 43]. PhMT-b around dental implants can be performed simultaneously to the implant surgery (proactively) or after the implant placement (reactively). Most research studies reported PhMT-b completed simultaneously to the implant placement. Jung et al. evaluated bone level changes and gingival recession around dental implants with small (≤5 mm in height) dehiscence after placement and were treated with either GBR or left for spontaneous healing [44]. The spontaneous healing group had more crestal bone loss than the PhMT-b group, which demonstrated stable bone levels. In the 18 months of follow-up, the authors did not report any significant difference regarding soft tissue recession depth between the two groups; however, given that the spontaneous healing group already presented less favorable bone levels at 1.5 years post-implant placement, it is highly likely that the implant with crestal bone loss could start to develop soft tissue recession. There are minimal well-controlled clinical studies that investigate PhMT-b correcting bony fenestration or dehiscence around dental implants after loading.

Orthodontic Driven Needs for Phenotype Conversion

The Biology of SFOT/PAOO

Orthodontic treatment is limited by the inherent regional hard and soft tissue anatomy within any individual. Should orthodontic treatment require the movement of teeth outside of the regional bony housing, treatment can still be possible with the use of PhMT in concert with orthodontic treatment.

Surgically facilitated orthodontic therapy (SFOT) involves procedures that can aid in the completion of more complicated orthodontic cases with minimal or inadequate soft and hard tissue volume [45–47]. This includes the use of corticotomy surgery plus dentoalveolar bone decortication with bone augmentation ± gingival grafting which has been popularized as periodontally accelerated osteogenic orthodontics (PAOO). As a broader category, SFOT may also include the use of temporary anchorage devices (TAD) and/or anchor plates. The term PAOO has been described by others under the patented name of accelerated osteogenic orthodontics (AOO) or Wilcodontics.™ In this chapter, SFOT will focus on the use of dentoalveolar corticotomy surgery with dentoalveolar bone decortication and bone augmentation to facilitate orthodontic treatment ± soft tissue grafting [45]. This procedure is efficacious in the treatment of malocclusion by increasing alveolar volume and enhancing periodontal phenotypes [45]. It can also be effective in the management of dentoalveolar deficiencies when tooth movement exceeds the orthodontic boundary conditions [48–51]. The benefits of SFOT include accelerated treatment time [48], greater stability of clinical outcome with less relapse [52], and enhanced scope of treatment for malocclusion [45]. Clinically, SFOT has been shown to resolve crowding of the natural dentition [46], facilitate eruption of impacted teeth [53, 54], accelerate canine retraction [46], correct intrusion and open bite [45, 48], and facilitate borderline orthognathic/orthodontic surgical cases [55]. When bone augmentation is performed in conjunction with the corticotomy surgery, the increased thickness of the alveolar bone is believed to enhance post-orthodontic stability of the dentition [56–58]. In a 2016 systematic review [51], corticotomy did not negatively influence periodontal health nor negatively impact periodontal probing

depth, gingival recession, clinical attachment levels, and alveolar crestal bone heights.

While treatment planning for these cases is complicated [51], the surgical aspects of the procedure are familiar and highly suited to periodontists with advanced training in this therapeutic modality. We recently published the collective experiences of at least 1500 cases and have developed a decision tree for SFOT [59].

SFOT Decision Process and Clinical Management

In the orthodontic management of thin phenotype cases, the critical decision is to determine if the periodontal phenotype has adequate soft tissue (keratinized tissue width and gingival thickness) with a bone morphotype that will be conducive to the planned orthodontic tooth movement. Critical stages for the management of PhMT in thin phenotype cases involve an interdisciplinary assessment of the osseous and mucogingival tissue, the scope of proposed orthodontic tooth movement, the actual corticotomy-bone augmentation procedure, and post-operative care as detailed in our decision tree (Fig. 5).

Orthodontic treatment limitations are usually based on the extent of the proposed orthodontic tooth movement and osseous boundaries as defined by CBCT analysis. The extent of tooth movement is somewhat subjective and variable. Proffit's envelope of discrepancy can provide some direction on malocclusion case management, but these numbers do not consider opportunities when dentoalveolar bone augmentation has been performed [60]. Ferguson et al. published on the scope of treatment with periodontally accelerated osteogenic orthodontic therapy and reported that certain tooth movements can be two to three times greater than that with conventional orthodontic parameters, from a horizontal (bucco-lingual) and/or vertical direction of orthodontic movement [45]. The extent of tooth movement and need for dentoalveolar bone augmentation are best evaluated with 3D simulation technology that allows the dentoalveolar bone complex to be scrutinized [55]. Seeing only the crowns of teeth in software simulations (whether for traditional orthodontic appliances or for aligner therapy) does not reveal the periodontium attached to the crowns nor provide transparency to unavoidable changes that occur within the dentoalveolar bone complex when tooth movement occurs. It is the periodontist's responsibility to define areas where hard and/or soft tissue augmentation will be needed and how much augmentation is possible to enhance dentoalveolar bone volume to which arch forms can be decompensated or corrected. This starts, in part, with an analysis of the mucogingival status with a focus on the keratinized tissue width, gingival thickness, and presence/risk for recession [16, 60]. Marginal tissue may be defined as thick vs. thin. Clinically, we found that the most convenient approach is to use the phenotype probe visibility test. (Hu Friedy). It is important to determine whether the areas of soft tissue deficiencies are localized or generalized. Limited localized areas can be managed during the corticotomy-bone augmentation procedure. If there is generalized thin soft tissue, there should be concern about whether the thin tissue can sustain the bone augmentation procedure. Should this be the case, a two-step approach is recommended. The thin phenotype can be thickened with the use of a subepithelial connective tissue graft. A small regional area of gingival thinness can be managed with either connective tissue grafts or allografts. The grafted site is usually allowed to heal and revascularize for 3–4 months prior to proceeding to the SFOT procedure.

The corticotomy-bone augmentation procedure is the critical part of this PhMT surgery. A clinical case is provided to detail these steps (Fig. 6). Interdental corticotomy and decortication of the alveolar housing create a transient demineralization phase within the dentoalveolar complex, and the resulting soft tissue-bone matrix permits an accelerated rate of tooth movement due to an altered physiologic response known as the regional acceleratory phenomenon (RAP) effect [52, 61, 62]. This injury results in acceleration of all processes involved in healing, including remodeling, cell turnover, metabolism, and repair. In individuals with thin bone morphotype, the movement of the teeth frequently results in bone loss (fenestrations, dehiscences) and future gingival recession. By combining soft tissue augmentation and bone grafting techniques, it is possible to convert a thin to a thick gingival phenotype and bone morphotype. The interdental corticotomy is performed primarily at interdental sites, with burs or piezo surgical incision to a depth of at least 2 mm. It is critical to extend the corticotomies beyond the cortical bone and into the medullary area. The corticotomies and bone augmentation should be provided on the facial bone in the same direction of the tooth movement, that is on the pressure side. For example, if arch expansion is undertaken, the treatment provided is for the facial alveolar bone only. Contrary to previous reports, the corticotomy-dentoalveolar bone decortication need not be done on both buccal and lingual sides as it has been shown that the tension side of orthodontic tooth movement will respond with bone deposition through the process of distraction. However, it should also be understood that injury to both sides may be required to achieve the desired tooth movement [63]. The greater the surgical insult around the tooth, the greater the magnitude of the RAP produced [62–64].

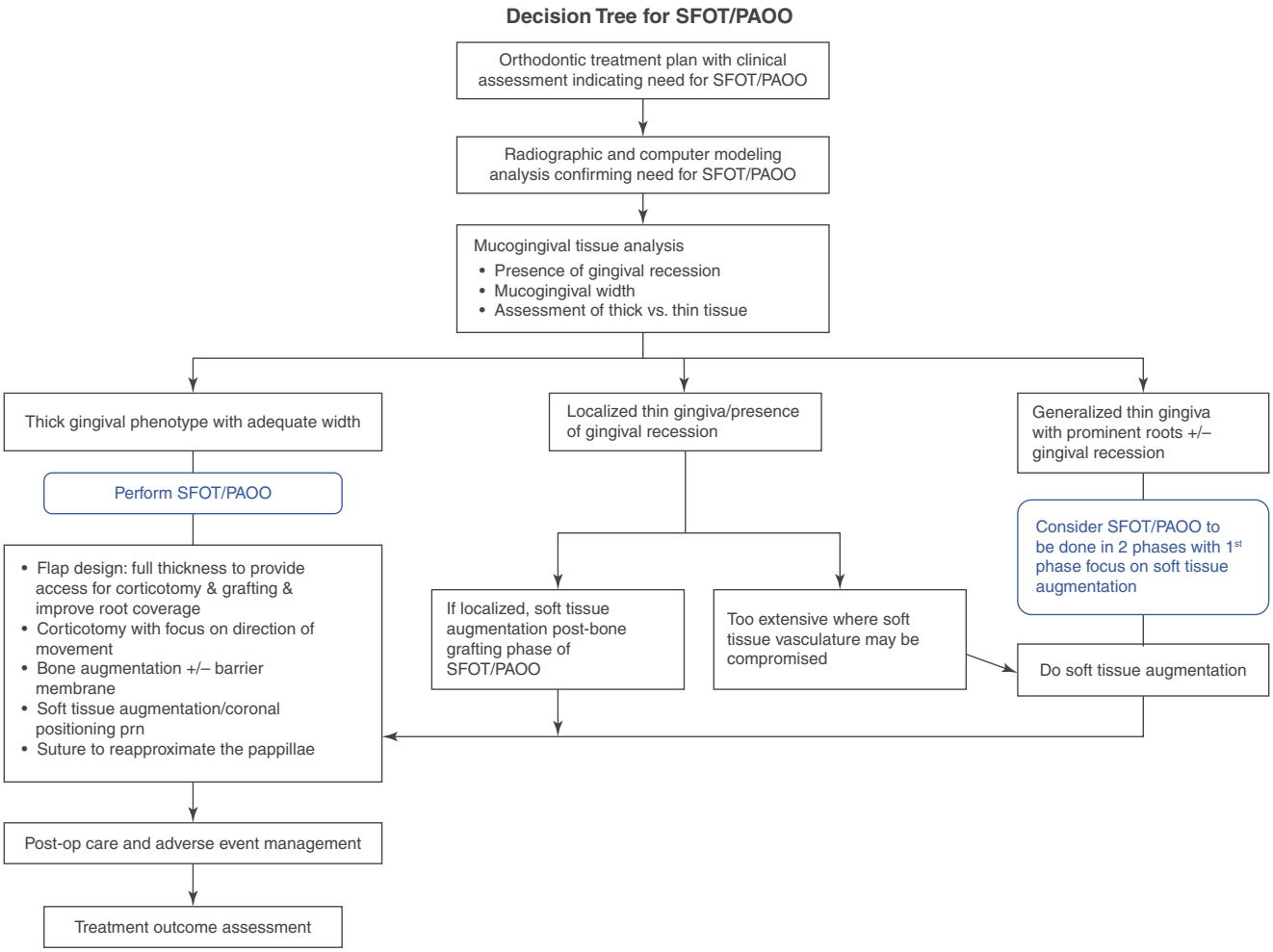

Fig. 6 Decision tree for implementation of SFOT for orthodontic treatment enhancement

Healing is consistent with most regenerative and mucogingival grafting surgeries. Infection is generally rare with the main adverse event consisting of incomplete root coverage. In extensive grafting with thin tissue thickness, superficial sloughing of the epithelium may be observed for the first few months, which requires monitoring but no immediate active management. Relapse of gingival recession, infrequently noted, is remedied with subsequent corrective mucogingival surgery such as additional subepithelial connective tissue grafts or augmentation with allograft substitute. The primary concern post-operatively is to activate orthodontic forces one to 10 days post-SFOT surgery. The arch wire should be activated within the first 2 weeks and usually changed every other week.

Post-SFOT monitoring—short-term and long-term—includes monitoring of pocket depth, relapse of gingival recession, soft tissue thickness, and increased appearance of bone thickness from CBCT views obtained one or more years posttreatment. As with any therapeutic approach, stability of long-term outcomes is important (Fig. 7).

SFOT is a PhMT that can open orthodontic treatment opportunities for a certain population of patients with malocclusion. The corticotomy-bone augmentation procedures are inherent to our periodontal training, but to master the use of SFOT in orthodontic treatment, an interdisciplinary team is essential to properly coordinate treatment sequencing, timing, and surgical protocols, and address the core problems of the patient (Fig. 8).

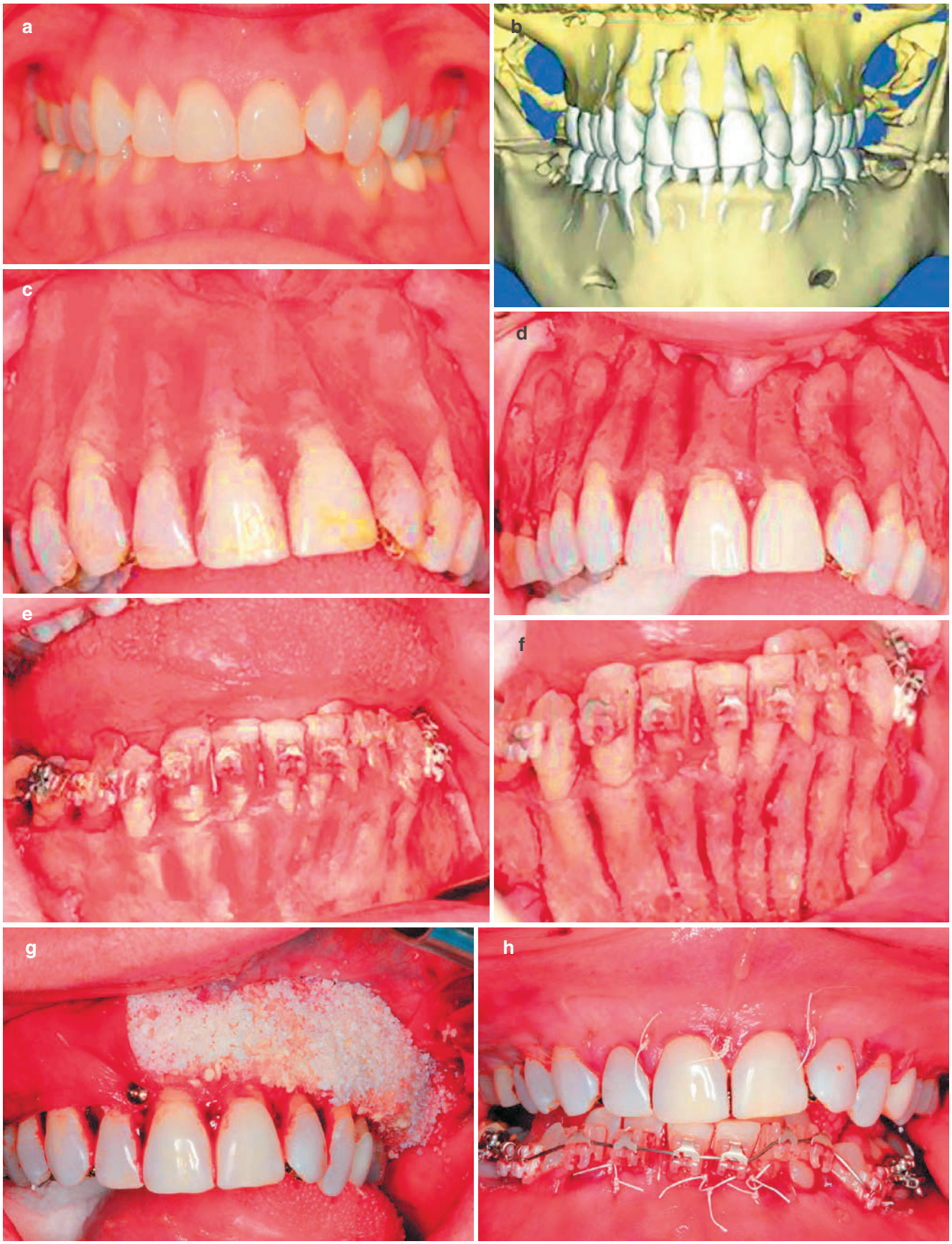

Fig. 7 Detailed steps of SFOT/PAOO. (**a**) pre-op clinical view; (**b**) pre-op computer analysis of CBCT (note the prevalence of fenestration and dehiscence- dentoalveolar deficiencies); (**c**) surgical view- maxillary view; (**d**) maxilla with corticotomy; (**e**) surgical view- mandibular view; (**f**) mandible with corticotomy; (**g**) bone augmented maxilla with portions covered with collagen barrier membrane; (**h**) closure with ePTFE sutures; (**i**) case completion; and (**j**) post-op computer analysis of CBCT

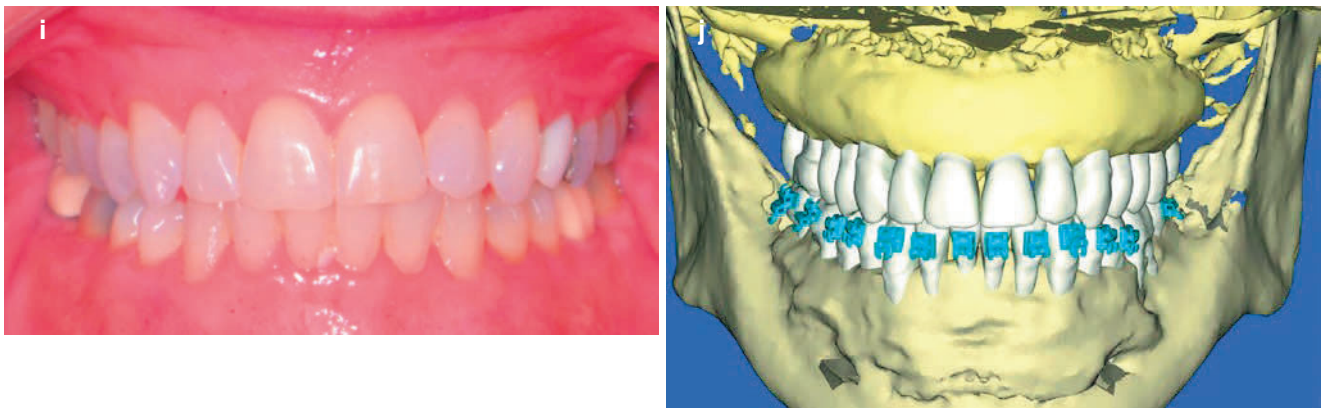

Fig. 7 (continued)

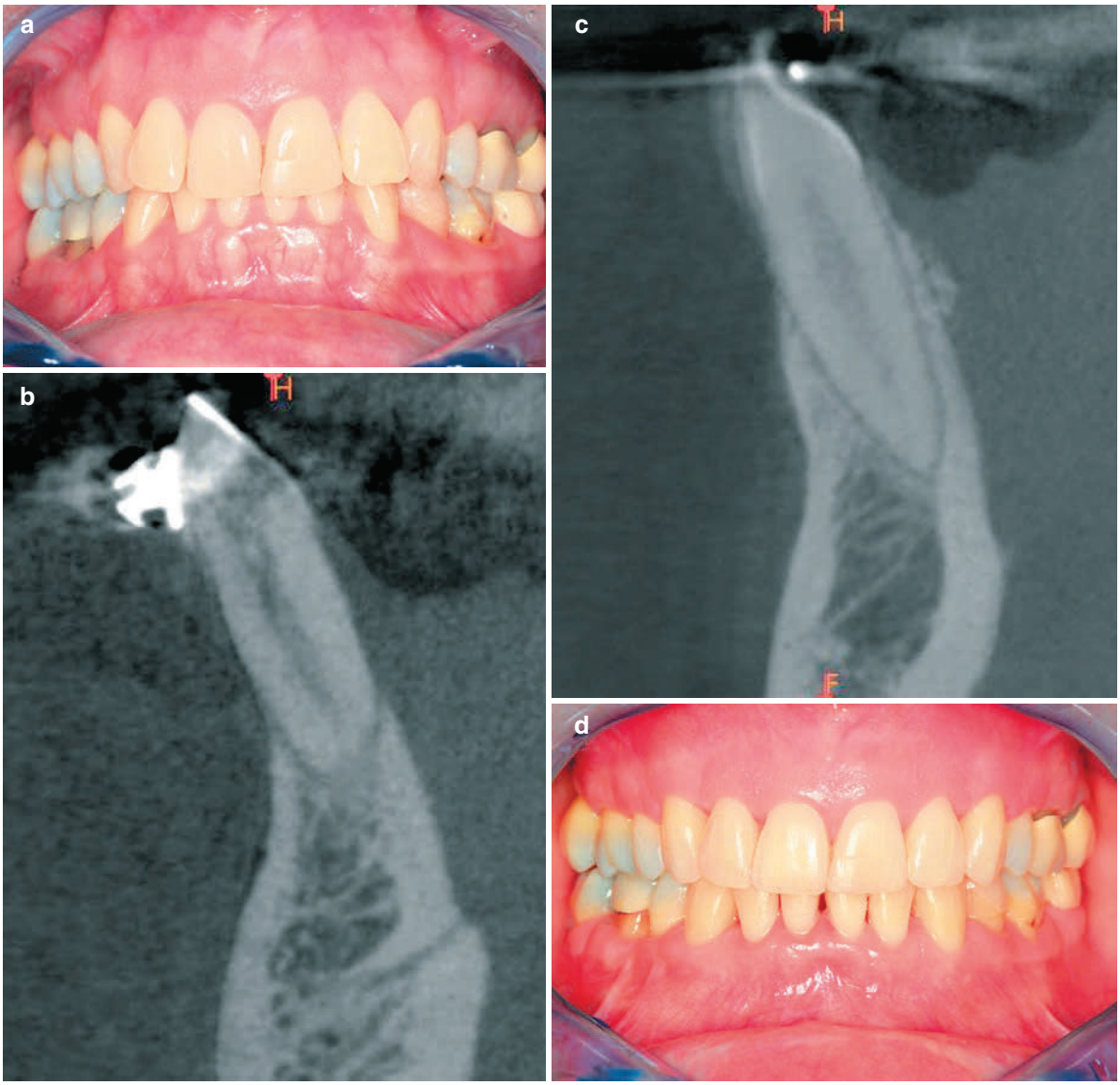

Fig. 8 This is a SFOT case with a 10 + yrs. follow-up. (**a**) pre-op view. (note the "corrugated appearance" due to root prominences for the anterior teeth); (**b**) CBCT of the lower anterior segment. (note the lack of supporting alveolar bone); (**c**) post-op CBCT of the same area 14 mos. After SFOT; and (**d**) clinical presentation after 10+ year. The "corrugated appearance" of the facial alveolar bone, seen in the pre-treatment image no longer evident in the posttreatment clinical vies, due to bone augmentation. The orthodontic treatment did not result in the appearance of gingival recession

Conclusion: Incorporation of PhMT to Improve Clinical Outcomes

This chapter reviews the importance of periodontal phenotype in the maintenance of periodontal/peri-implant tissue health. This health status is challenged with inflammation, trauma, and restorative procedures. A thin periodontal/peri-implant phenotype is particularly susceptible to increased risk for pathosis. With PhMT intervention, modification of soft tissue, bone, or a combination of both can help or improve dental health [2]. Current evidence supports the following:

- Periodontal phenotypes can influence susceptibility to pathosis as well as the clinician's ability to maintain the periodontium or improve periodontal health, especially when extensive restorative and orthodontic treatment is planned.
- Recent advances in oral care and surgical interventions such as PhMT can improve therapeutic outcomes in patients undergoing maintenance and in those requiring restorative, implant, and orthodontic treatment. PhMT intervention can involve surgical modification of soft tissue, bone, or a combination.
- In patients with thin gingival phenotypes, health may be maintained with good oral hygiene; however, iatrogenic, inflammation, or traumatic forces may result in recession and attachment loss. Patients with thin gingiva (<1 mm, measured from within the coronal one-third of the periodontal soft tissue) are more prone to future gingival recession. In these patients, PhMT may help maintain periodontal health and stability. Additionally, in anticipation of restorative or orthodontic treatment, PhMT can be pursued to increase the KTW and GT dimensions through gingival grafting and provide adequate/increased stability. However, with orthodontic treatment and tooth movement that exceeds the bony envelope, PhMT may require not only modification of the gingival tissue, but an increase in the volume of morphotype and to initiate the RAP effect. Recent advances in SFOT permit this to be performed effectively and safely.
- When clinicians are faced with situations where adverse outcomes may occur due to tissue phenotypes, it is important to employ PhMT *proactively* to support health and tissue stability. Responding *reactively* may result in pathosis that may impact an ideal treatment outcome. This is particularly true in regenerating tissue loss associated with peri-implantitis.
- Since the advent of the thick vs. thin gingival biotype concept, our understanding of the periodontal and peri-implant dimensions required for health has improved. More importantly, a variety of surgical interventions, which can be referred to as PhMT, can improve the periodontal/peri-implant environment to better maintain health. The profession still faces challenges, such as correcting defects associated with peri-implantitis and creating the ideal disease-resistant peri-implant environment. Great strides have been made, but clinicians should look forward to a better understanding of these challenges and mastery of therapies to address them.

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