

# Chapter 1

## Tooth Retention and Implant Placement: Developing Treatment Algorithms

*Paul A. Fugazzotto, DDS and Sergio De Paoli, MD, DDS*

### Outline

**Resective Therapy: Applicable Today?**  
**The Rationale for Pocket Elimination Procedures through  
the Use of Osseous Resective Techniques**  
**Results of Longitudinal Human Studies**  
**Clinical Example One**  
**Clinical Example Two**  
**Financial Algorithms**  
**Specific Clinical Scenarios**  
**Scenario One: The Single-Rooted Decayed Tooth**  
**Clinical Example Three**  
**Clinical Example Four**  
**Scenario Two: A Single Missing Tooth**  
**Clinical Example Five**  
**Clinical Example Six**  
**Scenario Three: Multiple Missing Adjacent Posterior  
Teeth**  
**Scenario Four: A Missing Maxillary First Molar, When  
the Second Molar Is Present**  
**Eliminating less predictable therapies through  
implant use**  
**Clinical Example Seven**  
**The influence of patient health on treatment plan  
selection:**  
**Conclusions**

There is no doubt that the introduction and evolution of regenerative and implant therapies affords clinicians the opportunity to provide patients with previously undreamt-of treatment outcomes. However, such therapeutic approaches must not be visualized as an end to themselves.

The goals of conscientious and comprehensive therapy remain the maximization of patient comfort, function, and esthetics in both the short and long terms. While it has become popular to speak of paradigm shifts in clinical dentistry, these shifts represent nothing more than alterations in

the treatment approaches utilized to attain the aforementioned therapeutic goals. In addition, efforts must be made to utilize the least involved and least expensive therapies possible for ensuring these treatment outcomes.

Maximization of oral health and amelioration of patient concerns remain the sine qua non of ethical practice. When considering the utilization of various regenerative or implant reconstructive approaches, it is important to listen to patient desires, determine patient needs, and ensure that the therapy to be employed is truly in the best interests of the patient. These interests may not always be optimally served through use of tooth extraction, complex regenerative therapies, and placement of multiple implants. Such treatment options should never be viewed as a means by which to supplant all other therapeutic approaches. Rather, a thorough understanding of the predictability of appropriately performed therapies around natural teeth is crucial to the formulation of an ideal treatment plan for a given patient. This treatment plan is based on a precise diagnosis of the patient's condition, and recognition of all contributing etiologies. Such a diagnosis takes into consideration the entire dentition, treating each site as both an individual entity, and a component in the masticatory unit.

Nowhere is this fact more evident than when considering management of the periodontally diseased dentition.

When faced with active periodontal disease, one of seven therapies may be employed.

- **No treatment:** Such a decision may be due to the patient's refusal of active therapy; or the patient's physical, financial, or psychological inability to undergo the necessary treatments. In such a scenario, it is imperative that the

## 2 Tooth Retention and Implant Placement

patient be made aware of the short- and long-term risks to both his or her oral and overall health represented by such a decision. It is important to realize that periodontal disease is a self-propagating disease. If no active therapy is carried out to halt disease progress, extension of the disease will result in tooth loss. When a patient chooses to pursue no active therapy, it is imperative that this concern be explained to the patient, and that every effort be made to both motivate the patient to seek treatment, and to adapt the treatment to the individual patient and the specific characteristics of his or her problems.

Regardless of which active therapeutic course is chosen, patients are always instructed in appropriate plaque control measures, so as to obtain an acceptable level of home debridement and bacterial control. A reevaluation is then carried out to determine which sites have healed through only the patient's plaque control efforts, and which areas still demonstrate signs of inflammation. Such a reevaluation is carried out in concert with a patient's specific risk assessment.

- **Subgingival debridement and institution of a regular professional prophylaxis schedule:** While this option seems attractive to many clinicians and patients, it is important to realize that, in many cases, such an approach does not halt the ongoing periodontal disease processes when significant pocketing is present. At best, the rate of attachment loss is slowed. This treatment option is indicated for patients who are physically, financially, or psychologically unable to undergo more comprehensive therapy, but who would at least agree to periodic debridement and prophylaxis in an attempt to delay tooth loss. This option is most appropriate for patients of an advanced age, who have demonstrated moderate attachment loss. Younger patients, or older patients with more aggressive periodontal disease problems, are less suited to actuarial therapeutic regimens. In addition, the potential dangers to adjacent teeth must be recognized and planned for.
- **Surgical therapies aimed at defect debridement and/or pocket reduction:** As explained above, these treatment approaches represent a significant compromise in therapy. A patient who has undergone surgical intervention is

left with a milieu which is highly susceptible to further periodontal breakdown. It is important to consider the need for retreatment and the potential damage to the attachment apparatus of adjacent teeth. This treatment option offers minimal advantages over the aforementioned treatment approach, and no advantages compared to the subsequent treatment approach.

- **Resective periodontal surgical therapy, including elimination of furcation involvements, in an effort to ensure a posttherapeutic attachment apparatus characterized by a short connective tissue attachment to the root surface, a short junctional epithelial adhesion, and elimination of probing depths greater than 3 mm:** This treatment approach offers the greatest chance of preventing reinitiation of periodontal disease processes. However, such a treatment regimen must be utilized appropriately. Osseous resective therapy that results in irreversible compromise of a given tooth, the initiation of secondary occlusal trauma due to reduced periodontal support and a poor crown to root ratio, or an esthetically unacceptable treatment result should not be considered ideal therapy. The advent of regenerative and implant therapies affords additional treatment options in previously untenable scenarios.
- **Periodontal regenerative therapy aimed at rebuilding lost attachment apparatus and surrounding alveolar bone:** Long viewed as an ideal to be strived for, periodontal regenerative therapy has a history of misunderstanding, misuse, and abuse. There is no doubt that predictable regenerative techniques are available for utilization in appropriate defects. There is also no doubt that the indications for the employment of these therapies are poorly understood. The net result is inconsistent treatment outcomes and condemnation of otherwise useful therapies by a large number of clinicians. When utilized in the appropriate manner in stringently selected defects, guided tissue regeneration yields highly predictable treatment outcomes. The advent of new materials offers the potential for even more impressive regenerative results. Unfortunately, the field of periodontal therapy continues to be handicapped by an incomplete understanding of diagnostic and technical criteria for success

with regenerative therapy. Many of these criteria have been elucidated in a previous publication (1). Advances in tissue engineering also offer preliminary regenerative results which are highly impressive. However, while the use of available growth factors is promising, the precise parameters of utilization, questions of cost, and reasonable treatment results are yet to be defined.

- **Tooth removal with either simultaneous regenerative therapy and implant insertion or guided bone regeneration with subsequent implant placement and restoration:** While highly predictable in almost every situation, regenerative and implant therapies must not be viewed as a panacea. To remove teeth, which may be predictably maintained through more conservative therapies and which will yield acceptable treatment outcomes, is unconscionable. However, to maintain compromised teeth which will eventually be lost, or to subject a patient to an inordinate amount of therapy or expense to keep teeth which may be more simply and predictably replaced by implants, is unacceptable.
- **A combination of the above therapies:** An uncomfortable and irresponsible dichotomy is developing in which the patient is viewed as either a "periodontal patient" or an "implant patient." A patient is neither.

Prior to the initiation of active therapy, a thorough examination and diagnosis must be carried out, and a comprehensive interdisciplinary treatment plan must be formulated. A high-quality full series of radiographs must be taken. When necessary, three-dimensional images are utilized as well. Panorex films are not utilized, as their accuracy is insufficient for providing useful information for comprehensive therapy. The components of a thorough clinical examination, including periodontal probing depths, hard and soft tissue examination, models and facebow records, are well established and will be discussed in subsequent chapters. However, it is important to realize that a thorough examination begins with an open discussion with the individual patient. It is crucial that the clinician determines the patient's needs and desires. In this way, treatment plans may be formulated which are in the best interest of the patient and which represent a greater value for the patient.

Prior to formulating a comprehensive treatment plan, all potential etiologies must be identified and assessed. In addition to systemic factors, these etiologies include periodontal disease, parafunction, caries, endodontic lesions, and trauma.

The treating clinician should always formulate an "ideal" treatment plan and present it to every patient. Appropriate and predictable treatment alternatives must be offered to the patient, thus allowing the patient to choose the treatment option to which he or she is best suited physically, financially, and psychologically.

Clinicians who fail to incorporate regenerative and implant therapies into their treatment armamentaria are depriving their patients of predictable therapeutic possibilities which afford unique treatment outcomes in a variety of situations.

Regenerative and implant therapies impact the partially edentulous patient in a number of ways, including:

- replacement of less predictable therapies
- replacement of more costly therapies
- augmentation of existing therapies
- introduction of newer therapies

Conversely, teeth which can be predictably restored to health through reasonable means should be maintained if their retention is advantageous to the final treatment plan. Clinicians who claim to be implantologists, performing only implant therapy while ignoring periodontal and other pathologies, do patients a disservice. Such clinicians include practitioners who either perform inadequate periodontal therapy to predictably halt the disease process, or remove teeth which could be treated through straightforward periodontal techniques.

It is inconceivable that any clinician would see only patients who require implant therapy, and demonstrate periodontal, endodontic, restorative, and occlusal health around all remaining teeth which are not to be extracted. This trend toward metallurgy at the expense of ethical, comprehensive care must be avoided at all times.

### **Resective Therapy: Applicable Today?**

Pocket elimination has long been advanced as one of the primary end points of periodontal therapy. An excellent review of the evolution of the

#### 4 Tooth Retention and Implant Placement

treatment modalities employed in pursuit of this goal has been published in the *Proceedings of the World Workshop in Clinical Periodontics* (2). A frequently utilized procedure when seeking pocket elimination is osseous resective surgery. Unfortunately, the ultimate objectives of this approach are rarely elucidated correctly and comprehensively.

The World Workshop states the objectives of osseous resective surgery as follows:

1. pocket elimination or reduction
2. a physiologic gingival contour that tightly adapted to the alveolar bone and apical to the presurgical position
3. a clinically maintainable condition

This formulation is incomplete. The primary goal of pocket elimination therapy is to deliver to the patient an environment which is conducive to predictable, long-term periodontal health, both clinically and histologically. With this fact in mind, the aforementioned objectives should be expanded to read:

1. Pocket elimination or reduction to such a level where thorough subgingival plaque control is predictable for both the patient and the practitioner.
2. A physiologic gingival contour is conducive to plaque control measures. This would include the elimination of soft tissue concavities, in the area of the interproximal col and elsewhere, soft tissue clefts, and marked gingival margin discrepancies.
3. The establishment of the most plaque-resistant attachment apparatus possible. This includes the elimination of long epithelial relationships to the tooth surface, where possible, and the minimization of areas of nonkeratinized marginal epithelium.
4. The elimination of all other physical relationships which compromise patient and professional plaque control measures. These include furcation involvements and subgingival restorative margins.
5. A clinically maintainable condition will evolve as a result of the previous four criteria having been met.

In short, pocket elimination is seen as a means of maintaining the plaque-host equilibrium in the host's favor by closing the window of host vulnerability as much as possible. While not always a realistic end point, this goal is most pre-

dictably maximized through pocket elimination procedures.

Two important questions present themselves:

- Are the principles behind pocket elimination conceptually sound?
- Does the clinical literature support the continued use of pocket elimination therapy?

#### **The Rationale for Pocket Elimination Procedures through the Use of Osseous Resective Techniques**

Periodontal pockets have long been recognized as complicating factors in thorough patient and professional plaque control. Waerhaug has shown that flossing and brushing are only effective to a depth of about 2.5 mm subgingivally (3). Beyond this depth, significant amounts of plaque remain attached to the root surface following a patient's oral hygiene procedures. Professional prophylaxis results are also compromised in the presence of deeper pockets. The failure of root planing to completely remove subgingival plaque and calculus in deeper pockets is well documented in the literature (4-8). Through the examination of extracted teeth which had been root planed until they were judged plaque-free by all available clinical parameters, Waerhaug demonstrated the correlation between pocket depth and failure to completely remove subgingival plaque (3). Instrumentation of pockets measuring 3 mm or less was successful with regard to total plaque removal in 83% of the cases. In pockets of 3-5 mm in depth, 61% of the teeth exhibited retained plaque after thorough root planing. When pocket depths were 5 mm or more, failure to completely remove adherent plaque was the finding 89% of the time. Tabita (9) noted that no tooth demonstrated a plaque-free surface 14 days after thorough root planing, if the pretreatment pocket depths were 4-6 mm. This was true even though patients exhibited excellent supragingival plaque control.

Reinfection of the treated site is a result of three different pathways (3, 9):

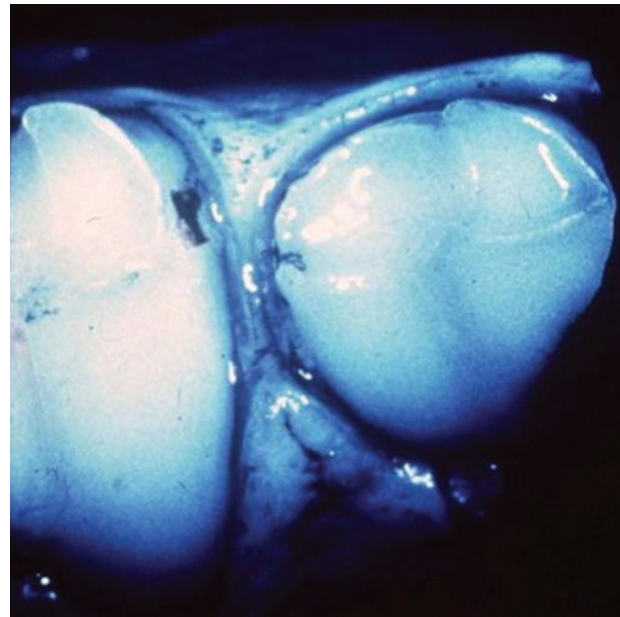
- (a) Plaque that remains in root lacunae, grooves, etc. will begin to multiply and repopulate the root surface following therapy.

- (b) Plaque which is adherent to the epithelial lining of the pocket will repopulate the root surface after healing. It has been demonstrated that, even if curettage is intentionally performed in conjunction with root planing, complete removal of the epithelial lining of the pocket is not a common finding (10–12).
- (c) Supragingival plaque will extend subgingivally, beyond the reach of the patient, and adhere to the root surface.

The magnitude of the limitations imposed upon proper plaque removal and control by pocket depths led Waerhaug to state: “If the pocket depth is more than 5 mm, the chances of failure are so great that there is an obvious indication for surgical pocket elimination” (3).

In the absence of deep probings, poor soft tissue morphology may contribute to increased plaque accumulation. Deep, sharp clefts, and marked soft tissue marginal discrepancies in adjacent areas have been implicated as factors contributing to inadequate patient plaque control (13). Interproximally, the morphology of the soft tissue col must be considered. If the buccal and/or lingual peaks of tissue are coronal to the contact point, the gingiva must “dip” under the contact point to reach the other side, resulting in a concave col form (14–16). When the col tissue touches the contact point, whether it is composed of natural tooth or restorative material, the epithelium does not keratinize (17 [Ruben MP, Personal communication, Boston, 1980], 18) (Figures 1.1 and 1.2). Such lack of keratinization is not an inherent property of either col or sulcular epithelium, as the ability of this tissue to keratinize when it is no longer in contact with the tooth, either as a result of periodontal therapy or eversion, is well documented (18–20). Nonkeratinized epithelium is less resistant to disruption and penetration by bacterial plaque than its keratinized counterpart (21, 22). When a concave, nonkeratinized col form is present, the patient must try to control an area which is conducive to plaque accumulation, and more easily breached by the aforementioned plaque and its byproducts (Figures 1.3 and 1.4).

Management of the soft tissue col form is predictably achieved through the proper use of osseous resective techniques. In addition to eliminating interproximal osseous craters, the buccolingual dimension of the alveolar process must be taken into consideration. If buccal osseous ledging is not



**Figure 1.1** A decalcified section demonstrating the concave nature of the interproximal soft tissue col.

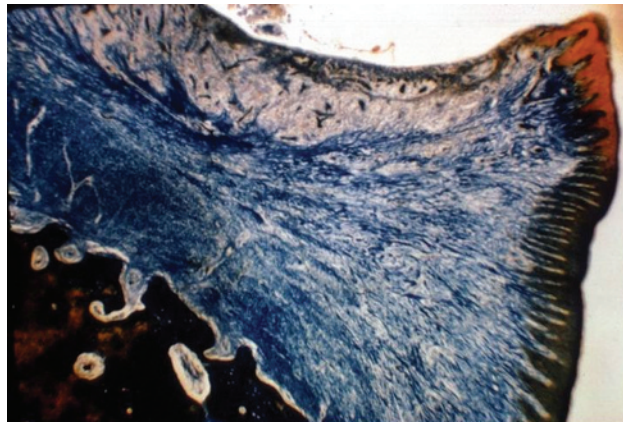
reduced adequately to allow for the smooth flow of soft tissues interproximally, without their first having to pass coronal to the contact point and “dip” underneath it, a concave col form will result (15, 23) (Figures 1.5 and 1.6). In addition, should the radicular bone be coronal to or at a height equal to the interproximal osseous septum, the soft tissues will not heal in tight adaptation to the underlying bone (16). Soft tissues will not heal in sharp angles, and will strive to regain a papillary form interproximally. All dimensions



**Figure 1.2** A histologic slide underscores the nonkeratinized nature of the col epithelium where it touches the contact point between the teeth.

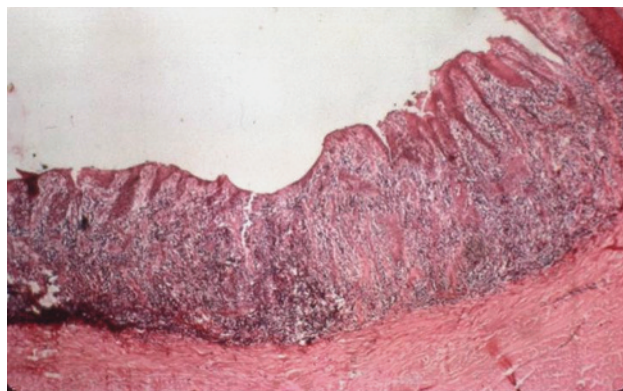


## 6 Tooth Retention and Implant Placement

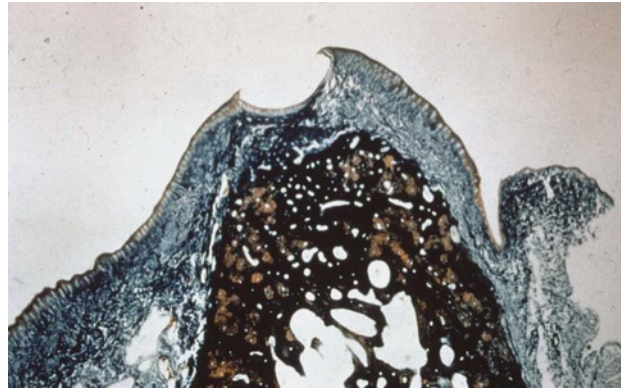


**Figure 1.3** The nonkeratinized concave col epithelium is especially susceptible to bacterial penetration and inflammatory breakdown.

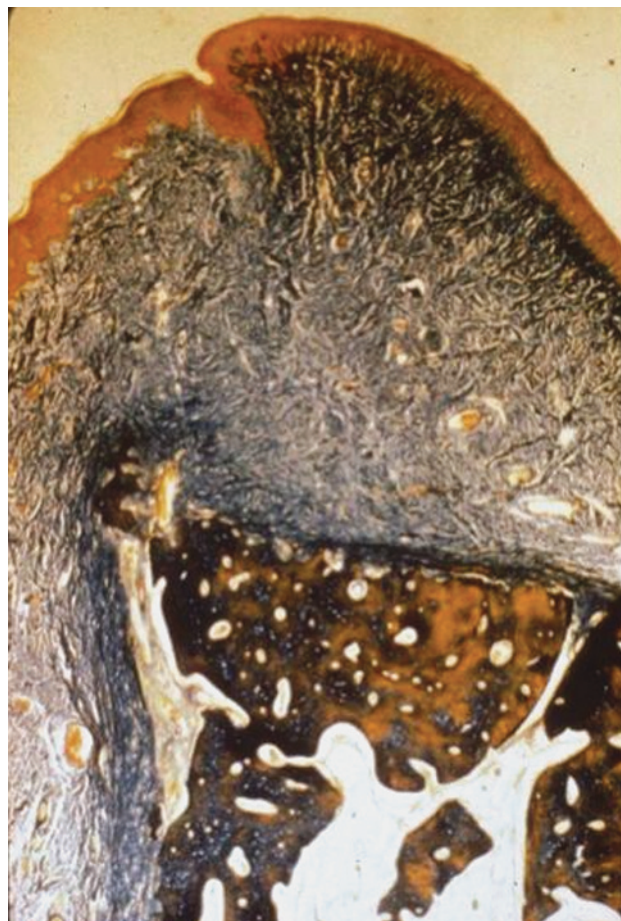
of the interproximal space (i.e., apico-occlusal, buccolingual, and mesiodistal) must be considered when evaluating the effects of existent osseous contours on the morphology of the overlying soft tissues. Matherson's work in monkeys demonstrated this fact clearly (24). The naturally occurring condition was one of a markedly concave soft tissue col. Replaced flap surgery without osseous therapy did not significantly alter the soft tissue col form. Interdental osteoplasty, resulting in the formation of an interproximal osseous peak, reduced the depth of the concavity in the col morphology. Osteoplasty which encompassed both the interproximal and radicular areas, thus reducing the buccolingual osseous ledging and eliminating reverse architecture, as well as forming an interproximal osseous peak, had the greatest effect on col



**Figure 1.4** As the inflammatory lesion progresses through the nonkeratinized col epithelium and into the connective tissue, marked tissue destruction is noted.



**Figure 1.5** Despite the convex nature of the interproximal alveolar bone, the soft tissue col is concave due to its contacting the contact point between the teeth.



**Figure 1.6** If the interproximal soft tissues are apical to the contact point, the convex interproximal bone contours are mimicked by covering keratinized soft tissues.



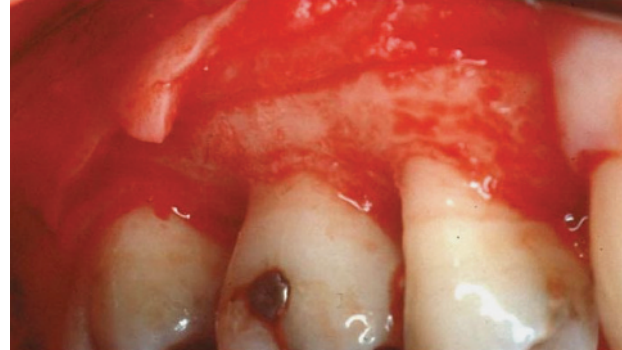
**Figure 1.7** A patient presents with 6 mm pockets interproximally, which bleed upon gentle probing.

morphology. Formation of a convex col form postoperatively was limited by the contours of the monkeys' teeth. Their contact points are broader buccolingually and more apically placed than those found in man. Odontoplasty would have been necessary to allow for sufficient space for the regeneration of the interproximal soft tissues apical to the contact points of the natural teeth. There is no doubt, contrary to published interpretations (2), that osteoplasty affected the postsurgical col morphologies in the precise manner which would be expected by proponents of osseous resective surgery (Figures 1.7-1.9).

While keratinization of the col tissues and alteration of their morphology to one more conducive



**Figure 1.8** Flap reflection reveals extensive osseous ledging. Failure to eliminate this ledging will result in these soft tissues having to "dip under" the contact point, and the reestablishment of a nonkeratinized concave soft tissue col form.



**Figure 1.9** The appropriate osteoplasty has been performed. The soft tissues may now be replaced at osseous crest, and will heal in a concave, keratinized manner apical to the contact points between the teeth.

to plaque control is achievable, this is not the case with the sulcular epithelium. Even if the sulcular epithelium could be predictably keratinized, it would serve no purpose, as the junctional epithelium is incapable of keratinization (25). The junctional epithelium is markedly different than other epithelia found in the oral cavity. In both keratinized and nonkeratinized oral epithelia, differentiation between the basal and superficial layers is a consistent finding (i.e., a decrease in Golgi vesicle and rough endoplasmic reticulum volumes, and an increase in tonofilament volume), as is a modification of the intercellular substance in the superficial layers, thus forming a permeability barrier (25). No evidence of differentiation is noted in the junctional epithelium. It has been suggested that this is due to the unique function of the junctional epithelium, which is to adhere to dissimilar tissues (26). If junctional epithelium was differentiated highly enough to keratinize, it would lose the ability to perform its primary function. Barnett (27) notes that, even in the presence of a keratinized sulcular epithelium, the junctional epithelium would still present a relatively easy mode of entry to the underlying structures for bacterial byproducts. Squiers (25) stated that "...it is reasonable to accept the junctional epithelium as a tissue which, by virtue of its adherent properties, is probably intrinsically permeable."

Saito et al. (28) examined clinically normal junctional epithelium in dogs via freeze-fracture and thin sectioning. Junctional epithelium was found to contain fewer desmosomes than other oral epithelium (5% in its most coronal aspect



## 8 Tooth Retention and Implant Placement

and only 3% apically). Very few cytoplasmic filaments were noted. Numerous gap junctions were noted, many of which were large in size. Tight junctions were only noted in freeze-fracture replicas, and these were underdeveloped or discontinuous in nature. These findings were in agreement with those of other researchers (29), and suggest that these areas leak, thus forming inadequate permeability barriers (30, 31). Saito et al. state that "...it is doubtful that the epithelium provides a complete barrier function because of the vast extent of the intercellular spaces and the sparseness of desmosomes" (28). Numerous studies have demonstrated the permeability of the junctional epithelium to a variety of substances (31–35). The relative impermeability of the sulcular epithelium, when compared to the junctional epithelium, has also been well documented. Substances were shown to penetrate the junctional epithelium, but not the sulcular epithelium (32, 33, 36).

The tenuous nature of the epithelial adherence to the tooth, and the ease with which it is separated, are well known (37). Listgarten (38) and others (39–43) have consistently shown that, in the presence of inflammation, the periodontal probe passes beyond the ulcerated junctional epithelium, stopping at the most coronal position of intact connective tissue fiber insertion into the root surface. This is not the case in noninflamed situations (44–46). The junctional epithelium therefore presents a dual-fold compromise. Not only is it more easily penetrated by bacterial enzymes, but it is also more easily detached in the presence of inflammation than inserted connective tissue fibers. In the stages of periodontal disease development, the "initial" lesion is seen as developing as follows:

1. bacterial accumulation in the gingival sulcus
2. an increase in the concentration of specific bacterial products
3. diffusion of these products through the more permeable junctional epithelium into the underlying connective tissue
4. dilation of the intercellular spaces of the junctional epithelium, and the presence of polymorphonuclear and mononuclear cells
5. perivascular collagen destruction
6. progression to the "early" lesion

Ideally, the expanse of the junctional epithelial adhesion to the tooth should be minimized in light of its relative biologic and mechanical inferiority

when compared to connective tissue attachment to the root surface.

Following appropriate osseous resective surgery with apically positioned flaps, an attachment apparatus is formed which consists of approximately 1 mm of connective tissue fiber insertion into the root surface, followed by 1 mm of junctional epithelial adhesion coronally (47, 48). The connective tissue attachment is derived from a combination of outgrowth of the periodontal ligament and resorption of osseous crest (49). This is markedly different than the postsurgical attachment apparatus obtained with either curettage or replaced flap (modified Widman or open flap curettage) surgery. These procedures have all demonstrated healing to previously periodontally affected root surfaces by the formation of a long junctional epithelium (50–68). New connective tissue attachment supracrestally has not been a consistent finding, nor has cementogenesis (69). The components of the postoperative attachment apparatus of open flap curettage procedures without osseous resection are the same; connective tissue insertion for the first millimeter supracrestally, followed by a long junctional epithelium. The length of the junctional epithelium is dependent upon the distance between the osseous crest and the margin of the soft tissue. Only pocket elimination surgery will consistently result in a short junctional epithelium, and thus avoid the compromises inherent in a longer epithelial relationship.

Proper pocket elimination therapy is not only concerned with pocket depths, but also with plaque accumulation in a vertical direction. Horizontal destruction of periodontal support, resulting in furcation involvements, will lead to a major compromise in therapy if left untreated. The inaccessibility of even early furcation involvements to proper plaque control measures is well documented (3, 70–73). A review of the literature also underscores the inadequacy of many therapies in the treatment of the furcated tooth. "Maintenance" care, open and closed debridement, chemical treatment of the root surface, and placement of particulate materials without membrane use have failed to demonstrate predictable success in the treatment of the periodontally involved furcation. Removal of the vertical periodontal pocket, without eliminating the horizontal component of a furcation involvement, results in a compromised environment for the removal of plaque by the patient, leading to



continued periodontal breakdown. This topic will be discussed in greater detail in Chapter 9.

Restorative margin position may also influence long-term periodontal health. Plaque accumulation at the restorative margin-tooth interface is a consistent finding in both research and clinical practice (74–81). If this margin is subgingival, the resultant increased plaque accumulation may lead to acceleration of periodontal breakdown and recurrent caries (81, 82) (Figure 1.10). This fact becomes more critical if the attachment apparatus attempting to maintain a healthy state includes a long junctional epithelium. The increased permeability and detachability of a long junctional epithelial adhesion in the face of inflammation lend the long junctional epithelium a greater vulnerability to the increased inflammatory insult inherent in subgingival margin placement.



**Figure 1.10** Recurrent caries is noted at the most apical extent of a deep subgingival interproximal restoration.

## Results of Longitudinal Human Studies

Numerous clinical studies have attempted to compare short- and long-term results of various treatment modalities. The most widely read are probably those of Ramfjord and coworkers (83–91). As time progressed, these studies became more sophisticated in response to design shortcomings which were recognized by the authors. The first study, published in 1968 (83), compared the results of curettage versus pocket elimination in the treatment of periodontal pockets. The authors concluded that “subgingival curettage was followed by more favorable results than surgical elimination of periodontal pockets.”

Being the first longitudinal study of this type, there were significant design flaws which the authors attempted to correct in subsequent studies. A split mouth design was not adopted until the third year of the study. For the first two years of data compilation, individual host response to therapy was an unaccounted for variable. Pockets were treated via gingivectomy procedures, if this could be accomplished within the bounds of the existing attached gingiva, if pocket depths were 5 mm or less and if extensive bone recontouring was not required to obtain acceptable gingival contours. This approach did not demonstrate a proper understanding of the rationale for pocket elimination therapy with osseous resection. Soft tissues will tend to reform interproximal papillae after periodontal surgery (92, 93). By eliminating interproximal osseous craters and reverse architecture, the clinician strives to achieve a closer adaptation of the reforming soft tissues to the underlying bone, helping to ensure the development of a postoperative attachment apparatus consisting of a connective tissue fiber insertion, followed by a short junctional epithelial adhesion. If interproximal osseous craters remained, which would have been the case where gingivectomy procedures were performed in the face of osseous defects, the long-term benefits of resective osseous therapy could not be properly assessed. In the 1968 study, no mention was made of the extent to which osteoplasty was carried out to eliminate buccal osseous ledging. If buccal ledging was allowed to remain, the resultant interproximal soft tissue morphology would be that of a concave col, due to the influence of the contact point. As

## 10 Tooth Retention and Implant Placement

previously discussed, this col would be more susceptible to inflammatory breakdown than the convex, keratinized interproximal soft tissues which would result from properly performed osseous resective therapy with apically positioned flaps.

Pocket measurements were taken at the “mesial side of the tooth,” with no mention being made of probe angulation. Watts (94) has demonstrated that even small variations in probe angulation will result in significant probing errors. While 60% of the probing measurements were reproduced, the number dropped to 23% for reproducible site configurations. The most important source of probing error was variation of the probe position in a transverse plane, despite the use of a stent. If stents were not used, as is the case in the 1968 Ramfjord study, errors would be magnified. Measurements taken in the manner described do not accurately measure the differences between the attachment apparati obtained via pocket elimination surgery and curettage. One difference in these two attachment apparati is that of a short junctional epithelium following pocket elimination surgery, and a longer junctional epithelium following curettage. This difference is not as significant at the line angles of the teeth as it is interproximally between the base of an osseous crater and the most coronal extent of the junctional epithelial adhesion. If measurements are taken at the line angles of the teeth, the relative stabilities of the different attachment apparati over time are not taken into account.

Another significant weakness in the 1968 study is one of execution. The first postoperative measurements were recorded at one year. The mean pocket reduction following pocket elimination surgery was 1.6 mm, resulting in residual mean pocket depths greater than or equal to 2.4 mm. When the data were broken down, the range of residual pocket depths became evident. In initial pockets of greater than 6 mm, approximately a 0.4-mm change occurred, leaving residual pocket depths greater than or equal to 5.6 mm. One of the basic postulates of pocket elimination surgery is the inability of the patient to exhibit adequate subgingival plaque control in areas probing greater than 3 mm. By leaving pockets of greater than 5.6 mm after therapy, the efficacy of pocket elimination therapy was not tested. The 1973 study by Ramfjord and coworkers had an identical design to that of 1968, and thus suffered from the same problems (84).

In 1975, the study was expanded to include the modified Widman procedure (85) and patients were followed over time (86, 89). The modified Widman procedure employed, as described in 1974 (94), was essentially replaced flap curettage, with osseous therapy as needed to facilitate interproximal flap coaptation.

The authors concluded that pocket elimination surgery did not offer any long-term benefits with regard to pocket depth or progression of disease, and that “although all three methods result in gain of attachment in moderately deep pockets, the long-term gain is significant only after curettage and modified Widman flap” (89).

As already discussed, design and execution flaws masked the differences between pocket elimination therapy and curettage or modified Widman surgery.

Interproximal pocket depth measurements were recorded “at the mesio- and distobuccal surfaces close to the contacts and without tilting the probe” (89). Thus, the measurements were taken at the wrong positions to measure the differences between the attachment apparati of the various treatment modalities. Due to the limited buccal and/or lingual osseous resection performed with the modified Widman procedure, the attachment apparati at the line angles of the teeth were similar for both procedures. The only difference in underlying osseous morphologies existed in the interproximal craters. Measurements purporting to compare the two therapies must record these differences.

Appropriate osseous resection to eliminate defects and reverse architectures, followed by apically positioned flaps, routinely results in pocket depths of less than 3 mm. Such was not the case in these studies. In pockets which probed 4–6 mm initially, probing depths of 1.7–3.7 mm are noted one year postoperatively. Where pockets probed 7–12 mm before therapy, residual pocket depths were 2.6–7.6 mm. These readings are not indicative of pocket elimination having been achieved. What was tested was not pocket reduction (modified Widman) versus pocket elimination; but rather pocket reduction versus pocket reduction. It would be unusual if both situations did not behave identically over time.

Ramfjord and coworkers felt that “the fact that pockets and attachment levels on the four tooth surfaces behaved similarly when the initial severity was constant made it possible to collapse

the data from the four surfaces and report the means" (89). This conclusion was based on the fact that all four tooth surfaces behaved the same with regard to pocket reduction and attachment gain one year postoperatively (95). However, one year is too short a time for proper evaluation of therapeutic results. Waerhaug has demonstrated the seemingly slow progression of untreated periodontal disease in data consisting of a large number of sites, and stated that a minimum of 3–5 years is necessary to evaluate treatment efficacy (3).

What was gained histologically following the various treatments was a short connective tissue insertion and a junctional epithelium of varying lengths. Where interproximal osseous craters are present, the junctional epithelium will be relatively longer; where there is a shorter distance from the osseous crest to the tissue margin (the buccal and lingual midradicular areas in most instances), the junctional epithelium will be relatively shorter. While areas of the same preoperative probing depth may appear to behave the same initially with regard to clinical response to therapy, they bear no resemblance to each other histologically. Collapsing the data in this manner masks the differences between the two clinical approaches.

One of the basic principles of pocket elimination therapy was ignored; that of the greater resistance of connective tissue fiber insertion than junctional epithelial adhesion to inflammatory breakdown. Buccal and lingual areas of long junctional epithelium are not subject to the same challenges as interproximal areas. Patient plaque control is easier and there are no concave col forms with retractable soft tissue peaks to trap plaque. Furthermore, restorative margins are more easily cleaned buccally than interproximally.

Ramfjord and coworkers also stated that "since the pockets and attachment levels from one year after treatment behaved essentially in a linear fashion, a grouping according to severity was adopted" (89). The progression of periodontal disease does not behave in a linear fashion, but rather is characterized by bursts of activity in specific sites, followed by periods of quiescence (96). The reporting of running medians is less effective in detecting site-specific changes in longitudinal periodontal studies than other statistical methods (97–99). By reworking statistics that reported no periodontal changes over time posttherapy, Lindhe was able to demonstrate the masking effect of reporting mean values (100).

The influence of furcations on the progression of periodontal breakdown was also ignored in the aforementioned studies. One facet of pocket elimination therapy is the elimination of furcation involvements through odontoplasty or root resection (101–104). Failure to eliminate the involved furcal areas renders complete plaque removal impossible due to local anatomy (105–108). Even with flap reflection, thorough debridement of an involved furcation is not a consistent finding (109, 110). An affected furcation will contribute to further periodontal breakdown both within the furcation itself and in adjacent structures. As the inflammatory lesion in the furcation spreads, it may also act in a "back door" manner, emerging from the internal aspect of the furcation to cause destruction of the attachment apparatus.

The effects of furcation involvements on the pathogenesis of periodontal disease were evident. Maxillary molars exhibited the greatest degree of periodontal breakdown following therapy, followed by mandibular molars and maxillary bicuspids.

The same limitations were evident in two studies carried out by Hill et al. and Ramfjord et al. (90, 91). Waerhaug's admonition with regard to leaving furcation involvements after therapy was borne out, as 16 of the 17 teeth lost in these studies were molars.

Pihlstrom et al. (111, 112), when comparing root planing alone and flap surgery with root planing, demonstrated greater pocket reduction initially with the flap procedure as a result of clinical attachment "gain." Repocketing of the areas treated with flap surgery, to the level of the root-planed sites, occurred within three years postoperatively. This is to be expected, as root planing and open flap curettage demonstrate the same compromised attachment apparatus posttherapy.

Disturbing findings with all longitudinal studies evaluating treatment modalities which yield a long junctional epithelium as a posttherapeutic attachment apparatus (root planing, curettage, modified Widman, flap curettage without osseous therapy, etc.) were repocketing and continued attachment loss (90, 91, 113, 114).

Proponents of pocket elimination therapy contend that, when carried out and evaluated properly, pocket elimination is superior to pocket reduction with respect to patient maintainability and long-term periodontal health. Do longitudinal studies exist which support these contentions?

## 12 Tooth Retention and Implant Placement

Ammon's group published two papers, one being a five-year follow-up of the initial patient data (115, 116), evaluating the relative efficacies of appropriately executed osseous resection with apically positioned flaps, and the other being apically positioned flaps with only root planing. Design modifications were made from the Ramfjord studies to help eliminate the problems already discussed. Data were first pooled by pocket depth, and then subdivided into tooth surfaces within a given pocket depth, to help elucidate the strengths and differences of the postsurgical attachment apparatus. Mesial and distal probing depths were recorded with the probe placed as far interproximally as possible, angulated to follow the long axis of the tooth. Only lesions which were amenable to resective therapy, and could therefore properly evaluate its applicability, were treated in such a manner. Finally, surgical photographs were published which demonstrated the techniques employed.

Greater interproximal soft tissue cratering was noted upon initial healing following open flap curettage, as compared with osseous surgery. Six weeks postoperatively, the cratering had disappeared. This finding is in agreement with Lindhe and Nyman (117). Pocket reduction at six months was the same for sites treated by either modality; flap curettage reduction being a result of attachment "gain" while osseous surgery reduction was due to pocket elimination procedures. The attachment "gain" was a function of papillary regrowth and a subsequent long epithelial relationship to the root, as a connective tissue fiber attachment cannot be expected following flap curettage (51, 56, 69). Five years postoperatively, statistically significant interproximal pocket depth differences were noted between the sites treated with and without osseous therapy. Pocket depths in the flap curettage areas were approaching preoperative values while the pocket elimination attained with osseous therapy was maintained. On the buccal and lingual surfaces, pocket elimination was maintained with both treatment approaches. These results underscore both the fragility of the junctional epithelial adhesion and the danger of collapsing data. Radicularly, where patient plaque removal was easier and the junctional epithelium was shorter, pocket elimination was maintained following both therapies. In interproximal areas of more difficult plaque removal, coupled with a longer junctional epithelial relationship due to the presence of osseous craters, repocketing occurred in sites treated with

open flap curettage. Flap curettage sites which initially probed 4 mm underwent repocketing at five years three times more often than sites treated via osseous resection. If initial probing depths were 5 mm, flap curettage sites repocketed 3.6 times as often as those treated with osseous resection. With initial probings of 6–8 mm, repocketing was 6 times as likely to occur with open flap curettage. When all sites with a preoperative probing depth greater than or equal to 4 mm were considered, bleeding upon probing was encountered 2.3 times more often in sites treated with open flap curettage than with osseous resection, five years postoperatively. There was a 91% correlation between the presence of subgingival plaque and bleeding upon probing.

Other authors have demonstrated the long-term efficacy of pocket elimination therapy. Lindhe and Nyman (100) reported the 14-year results of pocket elimination therapy in 61 patients with advanced periodontal disease preoperatively. All patients had remained on regular maintenance schedules. Only 0.49 teeth were lost per patient over 14 years. Disease progression was shown to be 20–30 times slower than in Swedes with untreated periodontal disease (118). Nabers et al. (119) reported the results of 1,435 patients treated via pocket elimination therapy. The patients lost an average of 0.29 teeth over a mean postoperative time of 12.9 years.

In contrast, McFall (120) demonstrated an average tooth loss of 2.6 teeth per patient 19 years posttherapy. Goldman et al. (121), 22.2 years postoperatively, documented a tooth mortality of 3.6 teeth per patient. Both of these studies employed treatment modalities which did not include pocket elimination therapy.

Kaldahl et al. (122, 123) compared treatment results in 82 periodontal patients treated in a split mouth design with either coronal scaling, root planing, modified Widman surgery, or flap surgery with osseous resection. All therapies produced mean pocket depth reductions, and there were no differences between the therapies with regard to residual pocket depths at the end of two years in sites which initially probed 4 mm or less. Subsequent breakdown of sites during supportive maintenance care of up to seven years was greater in areas treated with modified Widman surgery and scaling and root planing than in areas treated with osseous resective therapy. These differences in the number of sites breaking down increased as initial pocket depth increased, underscoring the



superiority of osseous resective therapy as a clinical modality for eliminating pockets and rendering areas maintainable over time by patients. Shallower pocket depths, coupled with a biologically stronger attachment apparatus of a short connective tissue attachment and a short junctional epithelium attained after osseous resection, proved more resistant to subsequent breakdown during maintenance than an attachment apparatus of a short connective tissue attachment and a long junctional epithelial adhesion obtained following root planing or modified Widman surgery. As expected, these differences were greater in areas with deeper initial pocket depths, as the difference in posttherapeutic attachment apparatus would have been more marked in these areas than in their shallower counterparts.

The differences in tooth retention can be traced to the ability of the patient and the clinician to successfully and predictably effect thorough plaque removal. Properly performed pocket elimination therapy provides an environment of minimal probing depth which is conducive to plaque removal. Even in the face of excellent supragingival plaque removal, we know that the patient is only effective at removing plaque to a subgingival depth of 2.5 mm (3). Lindhe et al. have demonstrated that there is no relationship between supragingival plaque control and changes in probing depths or attachment levels (124), or between supragingival plaque control and bleeding upon probing. The clinician must not be misled by the supragingival scenario. Waerhaug spoke of the existence of subclinical inflammation (3), where the tissue appears healthy, but periodontal destruction is occurring subgingivally. Ammons and coworkers (116) found a direct correlation between pocket depth and bleeding upon probing. Greater postsurgical pocket depths resulted in a higher incidence of bleeding upon probing. Coupled with the previously discussed 91% correlation between bleeding upon probing and the presence of subgingival plaque, the problems inherent in deeper postoperative probing depths are obvious. Badersten et al. (125, 126) noted that bleeding upon probing was directly related to pocket depth, with deeper areas bleeding more often. Waite (127) found that areas with deeper probing depths exhibited a higher frequency of bleeding upon probing and a greater degree of inflammation. Additionally, the same limitations which apply to subgingival root planing in the face

of pocket depths must be considered in the maintenance phase of therapy.

The deeper the residual probing depths, the more difficult debridement and maintenance become for both the patient and the dental professional (3, 128–137). Numerous longitudinal studies have demonstrated that sites with probing depths of greater than or equal to 6 mm are at significantly higher risk for future deterioration and development of additional attachment loss as a result of disease activity, if left untreated (138–143).

The scenario for continued loss of attachment in the face of posttherapeutic pocketing is as follows:

1. The patient presents with pocket depths in excess of 3 mm.
2. Patient plaque control removes plaque up to 2.5 mm subgingivally.
3. Subgingival scaling is increasingly less effective in areas probing greater than 3 mm.
4. Plaque left behind subgingivally following root planing begins to grow and repopulate the root surface within 14 days.
5. As the plaque front proceeds further subgingivally, its removal is less effective.
6. The attachment apparatus which results from curettage, modified Widman surgery, flap curettage, etc. has a long junctional epithelial component.
7. This epithelial adhesion exhibits greater permeability to plaque than a connective tissue fiber insertion.
8. Junctional epithelium is easily detached from the root in the presence of inflammation.
9. As the pocket deepens, the problems with plaque removal are exacerbated.
10. The presence of furcation involvements and/or subgingival restorations makes plaque removal even more difficult.
11. The result is continued periodontal breakdown.

Such continued periodontal breakdown following active therapy is avoidable. The technical aspects of osseous resective surgery have been clearly elucidated (16, 23). Employed in conjunction with selective extractions, root resective therapy, and prosthetic reconstruction, these techniques afford a high degree of predictability (23), albeit with significant temporal and financial costs.

## 14 Tooth Retention and Implant Placement

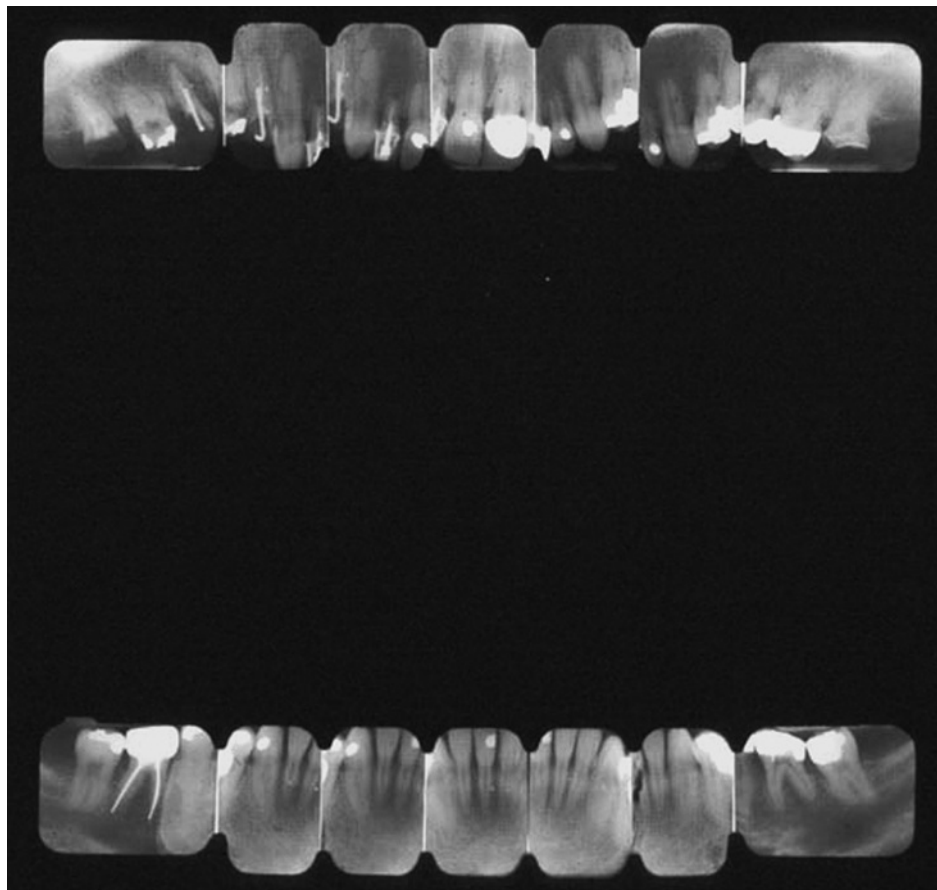
### Clinical Example One

In 1981, a 26-year-old female presented with a number of periodontal and restorative concerns. Postorthodontic blunting of the roots was noted (Figure 1.11). Class I furcation involvements were present on all maxillary and mandibular molars. Subgingival caries was present in many areas. Osseointegrated implants were not a viable treatment option at the time of patient examination.

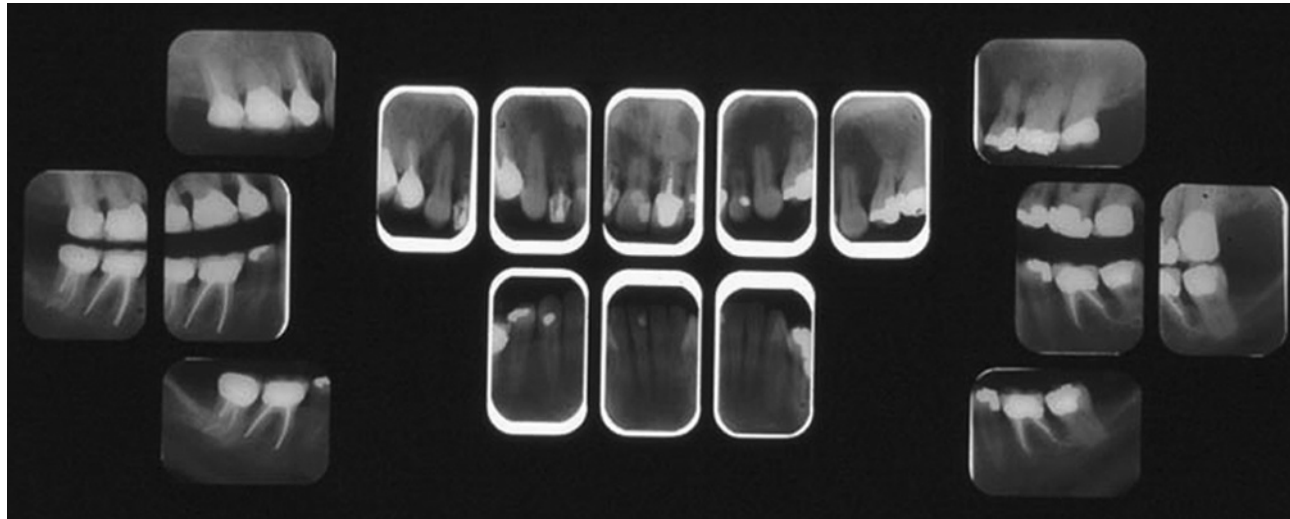
The combination of the patient's young age, short root structures, and active periodontal and restorative pathologies mandated a comprehensive, coordinated effort in order to afford her with a predictable treatment outcome. The performance of periodontal surgical therapies which would not eliminate deeper pockets and furcation involvements, and render all caries and defective restora-

tive margins supragingival for the restorative dentist's intervention, would be ill advised. When treating such a patient, the clinician has "one shot" at restoring the patient to health. The patient's limited attachment apparatus could not afford to withstand multiple surgical insults, nor be subject to continued periodontal breakdown following active care.

The patient was treated with an osseous resective approach. All furcation involvements were eliminated through odontoplasty. Tissues were positioned in such a manner as to allow placement of restorative margins supragingivally or intracrevicularly. A full series of radiographs taken 25 years after active therapy had been completed demonstrate the maintenance of periodontal support around the teeth, and the high degree of predictability afforded this patient through appropriate, coordinated care (Figure 1.12).



**Figure 1.11** A patient presents with numerous oral health concerns including significant caries, blunted roots, and early-to-moderate periodontal destruction. Class I furcation involvements are noted on all molars.



**Figure 1.12** Twenty-five years after completion of active periodontal and restorative therapies, the patient demonstrates excellent periodontal and restorative stability.

While the therapy employed proved highly predictable, the question facing today's clinician is whether or not to perform such therapy on severely compromised teeth, or to remove selective teeth and utilize an implant reconstructive approach. This question is paramount when considering root resection therapy.

Root resective therapy is a highly predictable therapeutic modality in specific situations.

While various authors have reported a wide range of success and failure, this was often due to utilization of root resective therapies in less than ideal scenarios. It is imperative that the forces being placed upon a root-resected tooth be managed appropriately if a reasonable degree of predictability is to be attained. When this is accomplished, long-term treatment results rival those of osseointegrating implants. Seven hundred one root-resected molars were followed for a period of up to 15-plus years in function. The cumulative success rates of the root-resected teeth in function were 96.8% (144).

However, while such a treatment approach may yield a high degree of predictability, the technical acumen and financial commitment required for such care often prove daunting and unrealistic.

### Clinical Example Two

A 41-year-old female presented with severe periodontal disease, characterized by moderate bone

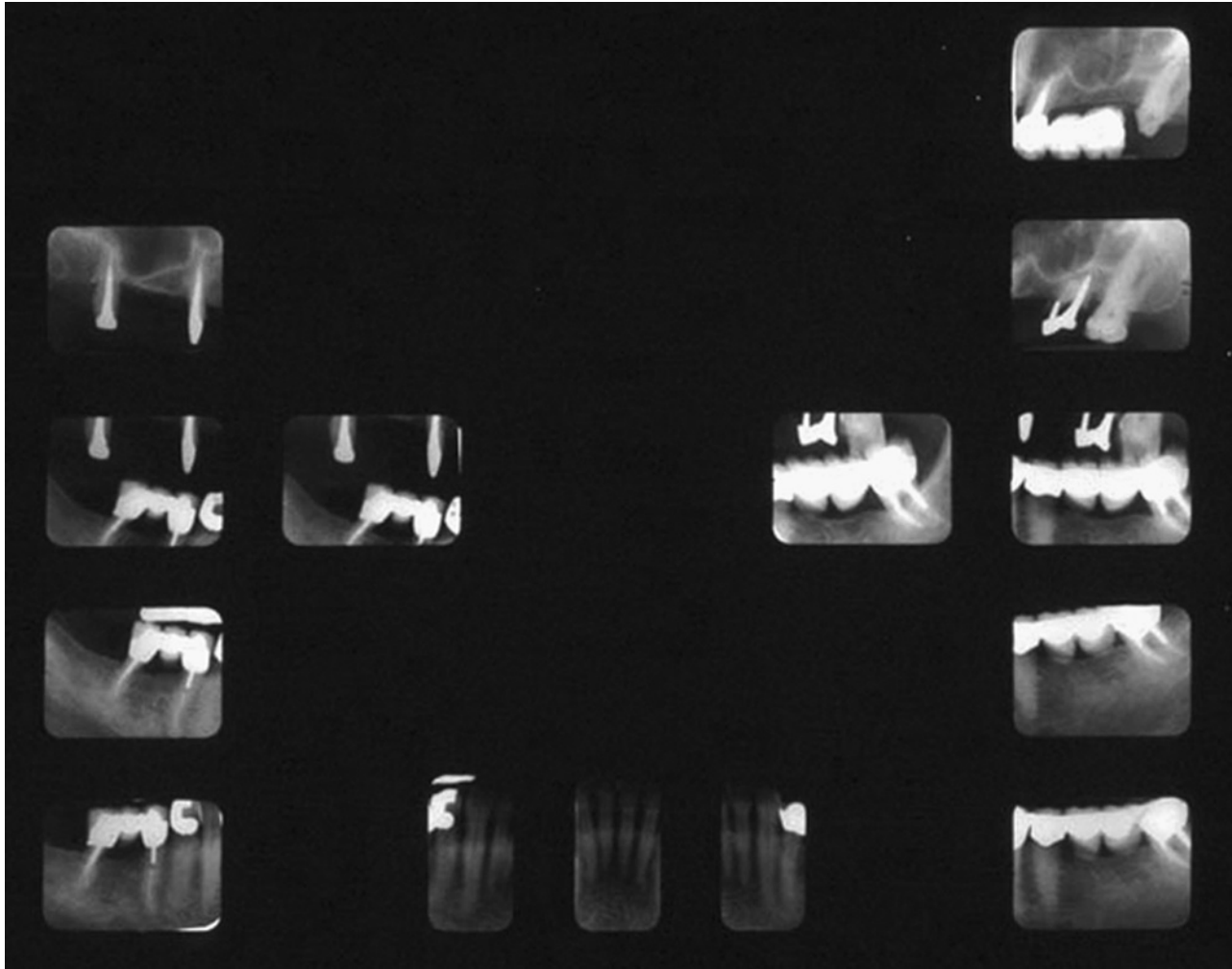
and attachment loss, Class II and III furcation involvements on all molars, and significant mobility patterns. The patient was temporized, underwent comprehensive periodontal therapy, including root resections and retention of a palatal root in the maxillary right second molar position; the mesiobuccal and distal buccal roots of the maxillary left first molar; and the distal root of the mandibular right first molar (Figure 1.13). The maxillary right cuspid was missing.

A maxillary full fixed reconstruction and a mandibular posterior reconstruction were carried out (Figures 1.14 a-f). The patient remained on a regular maintenance schedule. Radiographs taken 15 years after therapy had been performed, demonstrated stability of both the prosthesis and the supporting periodontium around the remaining teeth and or portions of teeth, despite the lack of a maxillary right cuspid (Figure 1.15).

After 15 years in function, the patient underwent significant life changes. The patient was not seen for one year, and had begun to clench and grind heavily. The net result was that the abutments in the maxillary right quadrant fractured. These abutments were most prone to parafunctional overload, as no cuspid was present. The loss of the established force equilibrium resulted in root fracture, tooth loss, and loss of the maxillary prosthesis.

While it is impossible to predict the future with regard to trauma and/or increased parafunction, the utilization of implants affords the

**16** Tooth Retention and Implant Placement



**Figure 1.13** A patient who presented with severe periodontal disease has been temporized and treated with resective periodontal therapy, including root resections. The palatal root of the maxillary right second molar; the mesiobuccal and distal buccal roots of the maxillary left first molar; and a distal root of the mandibular right first molar have been maintained.

opportunity to build a greater margin of safety into reconstructive therapy.

**FINANCIAL ALGORITHMS**

Assessment of various treatment options in a given clinical scenario must also take into account the financial commitment entailed with each therapeutic approach. A recent survey polled over 100 periodontists and their referring dentists in 20 metropolitan areas regarding the costs for various therapies (145). The costs for periodontal surgical therapies, endodontic therapy on single- and multirooted teeth, posts and crowns on natural teeth, tooth extraction, implant placement, and implant

abutments and crowns were assessed relative to a given value X (Table 1.1). Such information must be available to the clinician when formulating and presenting various treatment options to the patient.

**SPECIFIC CLINICAL SCENARIOS**

**Scenario One: The Single-Rooted Decayed Tooth**

When faced with a tooth which is decayed subgingivally at or near the osseous crest, the following treatment options present themselves:

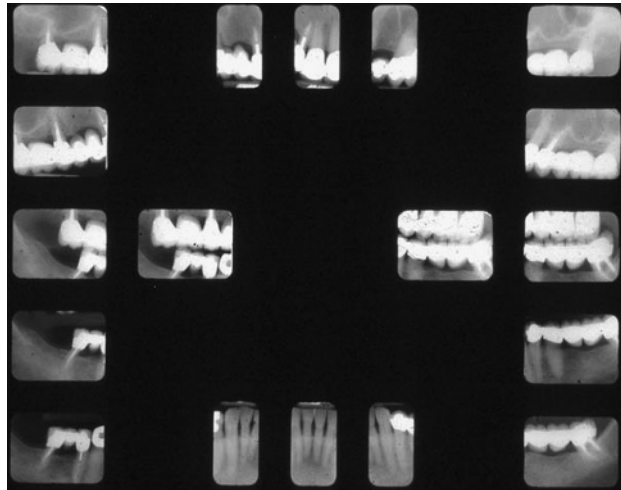
- (a) Crown-lengthening osseous surgery followed by endodontic therapy and post and





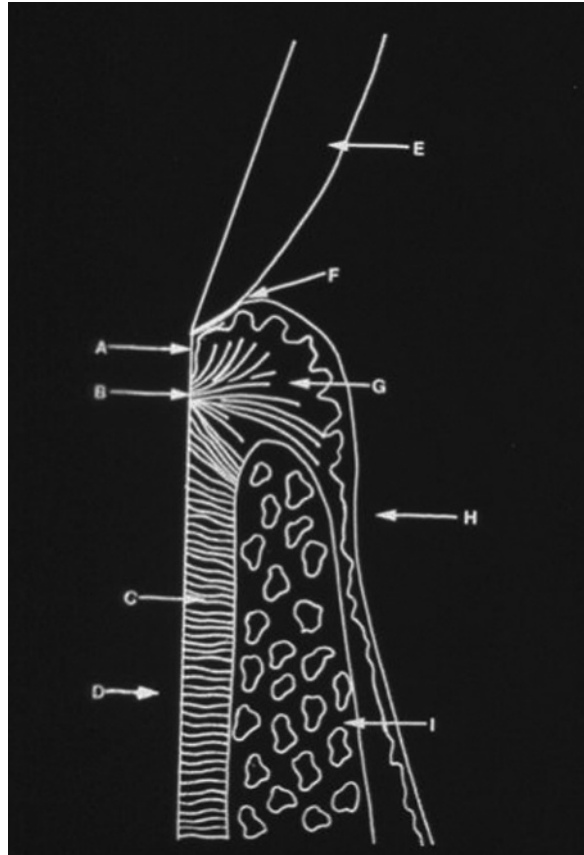
**Figure 1.14** (a–f) Buccal and clinical views of the completed reconstruction after 10 years in function. Note the lack of a cuspid in the maxillary right quadrant. The patient's home care and soft tissue health are excellent.

**18** Tooth Retention and Implant Placement



**Figure 1.15** A full series of radiographs taken 10 years after completion of therapy demonstrate the stability of the periodontium and the prostheses which are in place.

core buildup if necessary, and the appropriate restoration: The predictability of crown-lengthening osseous surgery is well established. When performed appropriately, crown-lengthening surgery results in both adequate clinical crown for restoration of the tooth in a maintainable manner, and the development of a predictable attachment apparatus consisting of approximately 1 mm of connective tissue attachment, 1 mm of



**Figure 1.16** (A) Junctional epithelial adhesion; (B) connective tissue attachment; (C) periodontal ligament; (D) tooth root; (E) enamel; (F) gingival sulcus; (G) gingival connective tissue; (H) outer epithelium; (I) alveolar bone.

**Table 1.1** Relative fees for various therapies.

Therapy	Fee
Endodontic—single root	0.9X
Endodontic—multiple root	1.3X
Core buildup—natural tooth	0.6X
Crown—natural tooth	1.4X
Pontic	1.4X
Crown-lengthening periodontal surgery	1.1X
Regenerative periodontal surgery	1.9X
Orthodontic supereruption	2.8X
Extraction	0.3X
Implant	2.1X
Implant abutment (stock) and crown	2.2X
Implant abutment (custom) and crown	2.7X
Regenerative therapy at tooth extraction	0.7–1.4X
Sinus augmentation	2.5X

junctional adhesion, and a 1- to 1.5-mm-deep sulcus (Figure 1.16). It is imperative that such therapy be performed in a manner which ensures both the maintenance of the attained hard and soft tissue morphologies, and the ability of the patient to perform appropriate plaque control measures around the final restoration. Advocates of “minimal approach surgery,” consisting of use of a laser or rotary instrumentation to “attain biologic width” only at the site of subgingival caries without ensuring a confluence with the adjacent hard and soft tissues, fail to understand the three-dimensional nature of tissue biodynamics and healing. Utilization of these limited access therapies results in eventual reformation of the presurgical soft tissue form and the presence of deep subgingival restorative margins. These problems

are avoided through the employment of techniques which are well documented in the literature (146–149).

The precise position and extent of the carious lesion and/or tooth fracture to be uncovered through crown-lengthening osseous surgery must be assessed prior to initiation of surgery. The advisability of performing such treatment is directly dependent upon whether the lesion to be uncovered is buccally, lingually, or interproximally placed, and its proximity to adjacent roots and/or furcation entrances.

Prior to performing crown-lengthening osseous surgery, a number of factors must be considered including:

1. The effect of therapy on teeth adjacent to the tooth to be crown lengthened: Depending upon the tooth preparation technique to be employed, 3–4 mm of tooth must be exposed between the alveolar crest and the planned position of the final restorative margin. In situations where a patient presents with a short root form, or caries on the root surface which would require removal of extensive amounts of osseous support, the tooth may be unduly compromised following crown-lengthening osseous surgery. If such a procedure will result in periodontal instability, or the development of secondary occlusal trauma, crown-lengthening surgery should not be employed.
2. The effect of crown-lengthening osseous surgery on the entrance to a furcation of a multirrooted tooth to be crown lengthened: If attainment of an adequate amount of exposed tooth structure for restorative intervention and development of a healthy attachment apparatus results in the development of an untreatable furcation involvement, such a therapeutic approach is ill advised. Should a Class I furcation involvement result following crown-lengthening osseous surgery, it is easily eliminated through odontoplasty, as will be discussed in Chapter 9. However, development of a furcation of any degree greater than Class I should be avoided at all costs.
3. The effect of crown-lengthening osseous surgery on the furcation entrances of

adjacent teeth: As previously mentioned, if the necessary osseous resection will result in a significant furcation involvement on an adjacent tooth, it should be avoided. In addition, care must be taken to assess the extent of osseous support which will be removed from adjacent single- and multi-rooted teeth during the performance of crown-lengthening osseous surgery. It is illogical to significantly compromise the periodontal health of adjacent teeth so as to afford adequate clinical crown length for appropriate restoration of a severely decayed tooth.

4. The effect of crown-lengthening surgery on the patient's esthetics: While palatal caries on a maxillary anterior tooth may be safely exposed for restoration, the same procedure performed interproximally or buccally often results in an unacceptable esthetic treatment outcome. In such situations, other treatment options should be explored.

If a decayed single root tooth is to be crown lengthened and restored, the need for endodontic therapy, as well as the ease and predictability of such therapy, must be carefully considered prior to initiation of care. Should the clinician have any questions regarding these points, appropriate consultations should be sought.

It is also imperative that the ability to predictably restore a specific decayed tooth is assessed prior to the initiation of care. Both the extent and position of the carious lesion will be paramount in determining the feasibility of maintaining the tooth in question.

### Clinical Example Three

A 51-year-old male presented with a buccal fracture on a mandibular left first molar (Figure 1.17). Radiographic examination demonstrated the short root trunk of the fractured tooth (Figure 1.18). Crown-lengthening osseous surgery would have led to significant invasion of the buccal furcation of the first molar, due to both its short root trunk and the position of the buccal fracture in relation to the furcation entrance. As a result, this tooth

## 20 Tooth Retention and Implant Placement

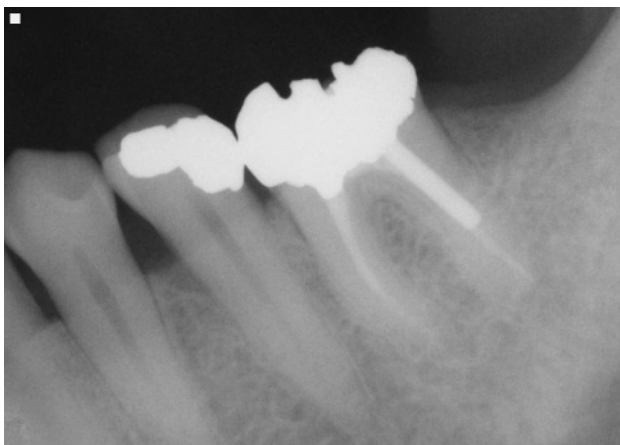


**Figure 1.17** A patient presents with a subgingival buccal fracture of a mandibular first molar.

must be removed and replaced with an implant at the time of tooth extraction, with concomitant regenerative therapy; this technique will be discussed in Chapter 9. Carious lesions which appear similar clinically often present with widely disparate prognoses when a radiographic examination is carried out.

### Clinical Example Four

A 31-year-old female presents with subgingival caries on the distal and palatal aspects of her maxillary



**Figure 1.18** A radiograph demonstrates the short root trunk of the fractured mandibular first molar. Crown-lengthening osseous surgery would lead to invasion of the entrance to the buccal furcation and a compromised long-term prognosis for the tooth.



**Figure 1.19** A patient presents with subgingival caries on the distal and palatal aspects of a maxillary right second bicuspid. Crown-lengthening osseous surgery would require removal of approximately 4 mm of bone at the area of the entrance to the mesial furcation of the first molar, and would unduly compromise the first molar.

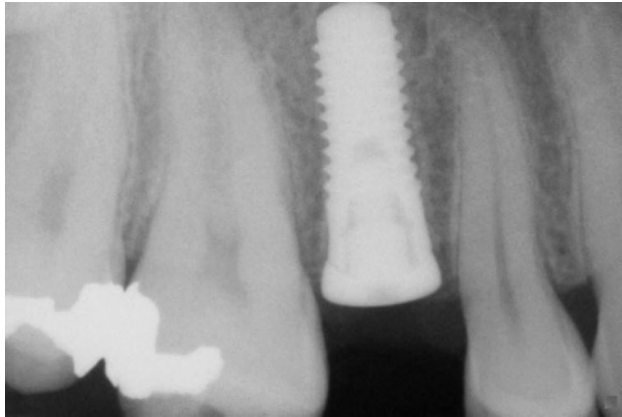
lary right secondary bicuspid (Figure 1.19). Appropriate crown-lengthening surgery would require removal of approximately 4 mm of bone at the area of the entrance to the mesial furcation of the first molar. Such therapy would compromise the prognosis of the first molar. Removal of 4 mm of bone from the distal aspect of the second bicuspid would also significantly alter its crown to root ratio and adversely affect the long-term prognosis of the tooth.

Due to these considerations, the maxillary second bicuspid was extracted and an implant was placed at the time of tooth removal. Following osseointegration, the implant is ready for restoration with a stock abutment and crown (Figure 1.20).

Figure 1.21 demonstrates a mandibular left first molar with caries on its distal aspect. The position of the caries with relation to both the interproximal osseous crest and the entrances to the furcations of the first molar renders it an excellent candidate for crown-lengthening osseous surgery and subsequent restoration.

In contrast, Figure 1.22 is a radiograph of another mandibular first molar which presents with distal subgingival caries. Both the more apical extent of the carious lesion interproximally and the fact that the mesial apical aspect of the lesion is approaching the entrance of the buccal furcation of the mandibular first molar render the tooth's





**Figure 1.20** The decayed second bicuspid has been extracted and replaced with an implant at the time of tooth removal. Following completion of osseointegration, this implant is ready for restoration with a stock abutment and crown.



**Figure 1.21** A patient presents with subgingival caries on the distal aspect of a mandibular first molar. The position and extent of this caries renders the tooth an excellent candidate for crown-lengthening osseous surgery and subsequent restoration.



**Figure 1.22** A patient presents with subgingival caries on the distal aspect of a lower first molar. The apical and buccal extents of the caries render this tooth a poor candidate for crown-lengthening osseous surgery. Such a procedure would unduly compromise the second molar and would invade the buccal furcation of the first molar.

prognosis poor. Attempts at crown-lengthening osseous surgery will unduly compromise the second molar and involve the entrance to the buccal furcation of the first molar. This tooth must be removed and replaced with an implant, abutment and crown.

The esthetic ramifications of crown-lengthening osseous surgery must be considered as well. Figure 1.23 demonstrates a fractured



**Figure 1.23** Attempts to crown lengthen the fractured lateral incisor would result in an esthetically unacceptable treatment result. If this tooth is to be maintained, orthodontic supereruption must first be carried out.

## 22 Tooth Retention and Implant Placement

maxillary left lateral incisor. Appropriate crown-lengthening osseous surgery around this tooth would result in a highly unesthetic situation for the patient. If this tooth is to be maintained, orthodontic supereruption must be considered prior to crown-lengthening osseous surgery.

- (b) Orthodontic supereruption with or without crown-lengthening osseous surgery: Supereruption of a decayed tooth affords the opportunity to minimize the removal of osseous support from adjacent teeth during crown-lengthening osseous surgery. In addition, the esthetic compromise of such surgery is significantly diminished. Finally, the need for crown-lengthening surgery may be obviated through severance of the periodontal ligament fibers at three-week intervals during the supereruption process. Such fiber separation often prevents the attachment apparatus from supererupting along with the orthodontically treated root, resulting in “nonsurgical crown lengthening.”

When orthodontic supereruption is contemplated, it is imperative that a number of factors be considered including:

1. The effects of orthodontic supereruption and subsequent crown lengthening on the treated tooth: Appropriate assessment of the expected root length following active therapy is crucial prior to the initiation of orthodontic supereruption. The patient is ill served by a supererupted, crown lengthened, and restored tooth which is unstable due to a poor crown to root ratio.
  2. The time involved in orthodontic supereruption: When assessing the advantages and disadvantages of various treatment approaches, the number of patient visits and the overall length of therapy must be openly discussed.
  3. The cost of orthodontic supereruption: As noted in Table 1.1, the use of orthodontic supereruption prior to crown-lengthening surgery and tooth restoration, with or without endodontic intervention, significantly impacts the cost/benefit ratio to the patient.
- (c) Tooth extraction, implant placement, and restoration: While this treatment approach eliminates the need for endodontic therapy and crown-lengthening osseous surgery, and

theoretically addresses concerns regarding the effects of osseous resection on adjacent teeth, its utilization assumes a number of conditions. The tooth must be extracted in a minimally traumatic manner with as little bone removal as possible. In addition, it is highly advantageous to utilize extraction techniques which will result in the least post-operative bone resorption and remodeling. If high-speed rotary instrumentation is necessary to effect tooth extraction, the resorptive phase of bone remodeling will be significantly increased. In such a scenario, the clinician may contemplate a two-stage procedure, performing regenerative therapy at the time of tooth removal, and placing the implant at an additional visit. Such rotary instrumentation is ideally avoided at all times. If necessary, piezosurgery is employed to help effect minimally traumatic root removal. Single-rooted teeth are always removed with a flapless technique, as will be discussed in Chapters 10 and 11. A decision is made after tooth removal as to whether or not buccal and/or palatal/lingual flap reflection are necessary.

Prior to contemplating implant placement at the time of tooth removal, the patient's biotype and the esthetic risks involved must be diagnosed and considered, as will be discussed in detail in Chapters 10 and 11. The clinician must be familiar with various osteotomy preparation and implant insertion techniques that ensure ideal implant positioning at the time of removal of single-rooted teeth. Finally, the need or lack of need for concomitant regenerative therapy, must be considered, with regard to complexity, duration, and cost of care.

In the case of multirrooted teeth, it is imperative that the clinician assesses the feasibility of placing an implant in an ideal restorative position at the time of tooth removal, the need for concomitant regenerative therapy, or the necessity of performing regenerative therapy and placing the implant at a second surgical visit. These considerations significantly impact the time and cost of therapy and the decision-making process regarding selection of the appropriate treatment modality. Chapters 8 and 9 will discuss these topics in depth.

**Table 1.2** Treatment options for a decayed single-rooted tooth.

Treatment option	Advantages	Disadvantages
Crown-lengthening osseous surgery with endodontic therapy, if necessary, followed by restoration	<ol style="list-style-type: none"> <li>1. Tooth retention</li> <li>2. Lesser cost of therapy</li> </ol>	<ol style="list-style-type: none"> <li>1. Decreased periodontal support for the treated tooth</li> <li>2. Possible decreased periodontal support for adjacent teeth</li> <li>3. Possible esthetic compromise</li> </ol>
Orthodontic supereruption with crown-lengthening osseous surgery followed by restoration	<ol style="list-style-type: none"> <li>1. Tooth retention</li> <li>2. Lessen effects on adjacent teeth</li> <li>3. Ameliorate esthetic concerns</li> </ol>	<ol style="list-style-type: none"> <li>1. Reduced periodontal support around treated tooth</li> <li>2. Protracted course of care</li> <li>3. Greatest cost of therapy</li> </ol>
Tooth removal, implant placement, and restoration	<ol style="list-style-type: none"> <li>1. A high degree of predictability</li> <li>2. No adverse effects on adjacent teeth</li> </ol>	<ol style="list-style-type: none"> <li>1. Tooth loss</li> <li>2. Slightly greater potential cost of therapy than option 1</li> </ol>
Tooth extraction, implant placement, concomitant regenerative therapy, and subsequent restoration	<ol style="list-style-type: none"> <li>1. A high degree of predictability</li> <li>2. No adverse effect on adjacent teeth</li> </ol>	<ol style="list-style-type: none"> <li>1. Tooth loss</li> <li>2. Greater cost of therapy than option 1</li> <li>3. Slightly protracted course of therapy</li> </ol>

The advantages and disadvantages of each treatment approach are detailed in Table 1.2.

In addition to the clinical advantages and disadvantages of the above treatment approaches, a cost-benefit analysis must be carried out to help ensure appropriate patient care (Table 1.3). Interestingly, with the exception of the introduction of supereruption or significant regenerative therapy at the time of tooth removal, the differences in

therapeutic costs are not enough to warrant selection of one treatment modality over the other. Rather, the site-specific considerations previously discussed are the overriding factors in the decision-making process in these situations.

Assessment of the aforementioned clinical, temporal, and financial variables affords the ability to construct a logical decision tree for therapy when faced with a single decayed tooth (Flow chart 1.1).

**Table 1.3** Cost analysis of treatment options for a decayed single-rooted tooth.

Treatment option	Cost as a factor of "X"
Crown-lengthening osseous surgery followed by restoration	2.5X
Crown-lengthening osseous surgery followed by endodontic therapy and restoration, single-rooted tooth	4.0X
Crown-lengthening osseous surgery followed by endodontic therapy and restoration, multirooted tooth	4.4X
Orthodontic supereruption followed by crown-lengthening osseous surgery and restoration	5.3X
Orthodontic supereruption followed by crown-lengthening osseous surgery, endodontic therapy, and post and core buildup, single-rooted tooth	6.8X
Tooth extraction, implant placement, and restoration with a stock abutment	4.6X
Tooth extraction, implant placement, and restoration with a custom abutment	5.1X
Tooth extraction, implant placement, regenerative therapy, and restoration with a stock abutment	5.7X–6.0X
Tooth extraction, implant placement, regenerative therapy, and restoration with a custom abutment	6.2X–6.9X

## 24 Tooth Retention and Implant Placement

If a tooth may be easily crown lengthened without unduly compromising either adjacent teeth, its own periodontal support, or the patient's esthetic profile, and no endodontic therapy is required; it is logical to perform crown-lengthening osseous surgery and restore the tooth appropriately.

However, if either the support of the tooth to be crown lengthened or the adjacent teeth will be unduly compromised, or the esthetic treatment outcome will be unsatisfactory, the tooth should be removed and replaced with an implant. Concomitant regenerative therapy is performed if necessary.

If a tooth may be safely crown lengthened without affecting its support or that of the adjacent teeth, and patient esthetics will not be unduly compromised, but endodontic therapy will be required, it is still more logical to remove the tooth and place a single implant, assuming significant regenerative therapy will not be necessary. In such a scenario, the patient is provided with a higher degree of long-term predictability without a significant increase in the overall cost of care.

Finally, if a tooth may be safely crown lengthened without affecting its support or that of adjacent teeth, the esthetic treatment outcome will be satisfactory, and tooth extraction and implant placement will require significant regenerative therapy, the patient may be logically treated by either of the aforementioned means. In such a situation, a clinician's understanding of therapeutic potentials and treatment philosophy will often be the determining factor in treatment selection. Nevertheless, it is logical, if all three therapies will be required around a natural tooth (i.e., crown-lengthening surgery, endodontic therapy, and subsequent restoration), to remove the tooth and replace it with an implant, due to both long-term predictability and cost considerations.

The use of orthodontic supereruption followed by crown-lengthening osseous surgery and restoration, with or without endodontic therapy, is rarely indicated. The significantly protracted course and increased cost of therapy make it hard to justify such a treatment approach. However, orthodontic supereruption is often indicated in cases where it is impossible to attain an acceptable esthetic treatment outcome through crown-lengthening osseous surgery and restoration, or tooth extraction, implant placement, and restoration without orthodontic intervention to "supererupt" the interproximal and/or buccal hard and soft tissues.

### Scenario Two: A Single Missing Tooth

Nowhere has the paradigm shift brought about by the advent of predictable regenerative and implant therapies been felt as strongly as in the replacement of a single missing tooth with natural teeth on either side. Available treatment options are as follows:

- (a) A three-unit fixed prosthesis: The advantages cited for such a treatment approach have traditionally included the alacrity of care and the ability to avoid surgical therapy. However, the introduction of newer implant surfaces has rendered the temporal differences meaningless. Implants placed in sites where regenerative therapy is not required can predictably be restored 2–4 weeks after insertion. In situations where a single tooth is replaced, the implant is often temporized at the time of placement. The time between implant placement, impressioning, and abutment and crown insertion is the same as the time between natural tooth preparation, impression taking, and fixed prosthesis insertion. The number of visits and overall time required for restoration of a single implant are less than those required for placement of a conventional three-unit fixed splint on natural teeth, as no framework try-in is required for single implant restoration.

Proponents of three-unit fixed bridges to replace a single tooth will often cite the conditions of the adjacent teeth as a determining factor in treatment selection. While at first glance it may appear that, if the single tooth edentulous site is bordered by restored teeth on one or both sides, it would be logical to place a three-unit fixed bridge, as "virgin" teeth are not being compromised. This philosophy would appear especially cogent if one or both of the adjacent teeth required restorations.

However, a close examination of the situation demonstrates that such thinking is inherently flawed. Teeth which have been restored, or which require restoration, exhibit a higher degree of probability to need endodontic intervention. Removal of older, large restorations and underlying tooth structure often mandates endodontic intervention and core buildup prior to restoration. In addition, teeth with significant carious lesions

often require endodontic therapy. The argument could be made that such teeth should be treated prophylactically with endodontics if they are to serve as abutments for fixed prostheses, so as to avoid future problems.

Numerous studies have demonstrated the inadvisability of assuming that a three-unit fixed prosthesis will predictably remain intact for 20 years or more. Should one of the abutments of a fixed prosthesis become problematic, the entire prosthesis must be replaced. However, should a single implant or the crown it supports develop problems, this site may be addressed individually. From the point of view of predictability, it is more logical to place a three-unit fixed splint utilizing "virgin" teeth as abutments, than to depend upon the teeth which have been previously restored or exhibit active carious lesions.

### Clinical Example Five

A 37-year-old male presented with a severely decayed mandibular left second bicuspid (Figure 1.24). The prognosis for this tooth was very poor.

Reasonable treatment options included tooth extraction with simultaneous implant placement and eventual restoration, or tooth extraction with fabrication of a three-unit fixed splint including the first molar and first bicuspid.



**Figure 1.24** The mandibular second bicuspid is hopeless. It is best replaced with an implant abutment and crown. Placement of a three-unit fixed prosthesis would mandate endodontic therapy on the first molar.

The conventional argument would be that placement of a three-unit fixed bridge is indicated in this area, as the first molar presented with a significant amalgam restoration. However, because of this fact it is actually more logical to utilize a single implant, abutment and crown to replace the hopeless second bicuspid. Incorporation of the first molar into a three-unit fixed splint would undoubtedly result in the need for endodontic therapy on this tooth, thus increasing both the complexity and cost of care. In addition, the patient would be left with an area which would be more problematic with regard to appropriate plaque control measures.

### Clinical Example Six

A 61-year-old male presented with recurrent decay around a crown on a maxillary right second bicuspid, the terminal abutment for a two-unit cantilevered fixed prosthesis (Figure 1.25). Significant osseous loss was noted around this bicuspid abutment which presented with a Class II mobility. In addition, the maxillary right first molar required crown-lengthening surgery and a new restoration.

Adequate bone remained around the maxillary right second bicuspid to maintain it following



**Figure 1.25** A patient presents with recurrent caries on the maxillary first molar and second bicuspid, and moderate periodontal destruction around the second bicuspid. The first bicuspid could be replaced with a fixed prosthesis. However, such therapy would almost certainly involve endodontic treatment of one or both abutments. Following periodontal therapy to rebuild damaged alveolar bone around the second bicuspid, an implant was placed in the first bicuspid position, and the implant, second bicuspid and first molar were restored with single crowns.



## 26 Tooth Retention and Implant Placement

amelioration of excessive traumatic forces and performance of a conservative periodontal regenerative procedure. The question now became whether to replace the missing maxillary first bicuspid with an implant, abutment and crown, or through the use of a three- or four-unit fixed prosthesis including the second bicuspid and cuspid, and possibly the first molar.

If a three- or four-unit fixed prosthesis was utilized, the intact cuspid would be significantly involved. As a result, it was more logical to perform the aforementioned periodontal surgical therapy around the first molar and second bicuspid, place an implant in the position of the first bicuspid, and restore the implant, the second bicuspid and first molar with individual crowns. The end result will be greater ease of plaque control efforts and a highly predictable long-term prognosis.

From an ethical point of view, it is difficult to justify preparation of two adjacent "virgin" teeth to place a three-unit fixed splint when utilization of a single, implant abutment and crown will leave these teeth intact and uncompromised.

Patient hygiene capabilities are also enhanced when a single, implant abutment and crown are placed, as compared to a three-unit fixed splint. This fact once again offers a higher degree of long-term predictability to a single implant and crown as compared to a three-unit fixed bridge.

These rationales do not mean that implant placement is the ideal treatment of choice in all areas where a single tooth is missing and natural teeth are present on either side of the edentulous

space. Specific site considerations must be assessed prior to committing to an implant therapeutic approach. The questions which must be asked include the following:

- Are the root angulations of the adjacent teeth appropriate for implant placement between them?
- Is adequate space available mesiodistally for retention of the bone and covering soft tissues between the implant and the adjacent teeth?
- Does the position of the inferior alveolar canal or the mental foramen preclude implant placement in the desired position?
- Will concomitant horizontal augmentation therapy be required to place the implant in the appropriate buccolingual position, and to ensure it is housed in bone of sufficient dimension to withstand functional forces over time?
- Can augmentation therapy be performed at the time of implant placement, or must the patient undergo two surgical sessions?
- Is sinus augmentation therapy necessary to effect appropriate implant placement?
- Can sinus augmentation therapy be performed at the time of implant placement, or must the patient undergo two surgical sessions?

The advantages and disadvantages of each treatment approach are outlined in Table 1.4.

Finally, financial assessment of each treatment option must be carried out to ensure the

**Table 1.4** Treatment options for a single missing tooth in a tooth-bounded space.

Treatment option	Advantages	Disadvantages
Three-unit fixed bridge	<ol style="list-style-type: none"> <li>1. Avoid surgical therapy</li> <li>2. Avoid vital structures</li> <li>3. Eliminate the need for regenerative therapy</li> <li>4. Slightly lesser cost of therapy than implant placement and restoration, if no endodontic therapy is required on abutment teeth</li> </ol>	<ol style="list-style-type: none"> <li>1. Involvement of adjacent teeth</li> <li>2. Potential for endodontic therapy</li> <li>3. Greater cost of treatment if endodontic therapy is required</li> <li>4. More difficult to perform adequate home care</li> </ol>
Implant placement and restoration with a stock abutment and crown	<ol style="list-style-type: none"> <li>1. No involvement of adjacent teeth</li> <li>2. Greater ease of home care</li> <li>3. Greater long-term predictability</li> </ol>	<ol style="list-style-type: none"> <li>1. Need to avoid vital structures</li> <li>2. Potential need for regenerative therapy</li> <li>3. Possibility of second surgical visit</li> </ol>

**Table 1.5** Cost analysis of treatment options for a single missing tooth in a tooth-bounded space.

Treatment option	Cost as a factor of "X"
Three-unit fixed bridge	4.1X
Three-unit fixed bridge with endodontic therapy and buildup on one abutment	5.6X–6.0X
Three-unit fixed bridge with endodontic therapy and buildups on two abutments	7.1X–7.5X
Implant placement with a stock abutment and crown	4.3X
Implant placement, regeneration, stock abutment and crown	5.0X–6.4X

patient is attaining the greatest monetary benefit from the care to be delivered (Table 1.5).

Once these factors have been taken into consideration, a simple, logical decision tree may be formulated (Flow chart 1.2).

### Scenario Three: Multiple Missing Adjacent Posterior Teeth

A long span fixed prosthesis, defined as a prosthesis with more than one adjacent pontic, is rarely indicated due to the advent of predictable regenerative and implant therapies. Utilization of such a long span prosthesis represents a significant compromise in patient hygiene capabilities and long-term predictability of therapy. The increased stresses placed upon the abutment teeth in these scenarios result in an unacceptably high incidence of abutment and hence prosthesis failure. In addition, flexure of the prosthesis over time often leads to cement washout and recurrent caries beneath the crowns on the abutment teeth. As a result, the biomechanical prognosis is very poor.

The only indications for such a prosthesis are in situations where the positions of vital structures, combined with severe ridge atrophy, render appropriate implant placement impossible, even following extensive regenerative therapy. It must be cautioned that the clinician should not accept such a diagnosis too quickly. Simple, predictable regenerative techniques are available to sufficiently augment all but the most atrophic ridge. This fact, combined with the utilization of shorter implants with specific designs, makes it rare to encounter a site which may not be rendered suitable for implant reconstructive therapy, as will be seen in Chapters 2 and 7.

The only other rationale for placing a long-span fixed prosthesis instead of an implant-supported prosthesis is a patient who is medically

unable to undergo any type of oral surgical procedure. Once again, such situations are rare.

### Scenario Four: A Missing Maxillary First Molar, When the Second Molar Is Present

The reduced success rates of smooth surface threaded implants in the maxillary posterior region initially led clinicians to avoid such therapy, and place conventional fixed prostheses to replace missing maxillary first molars. However, rough surface implants of various topographies and formulations have demonstrated short- and long-term success rates equal to those of osseointegrated implants in other areas of the mouth. As a result, the maxillary posterior region must no longer be viewed as an undesirable site for implant reconstructive therapy. The decision as to whether to place a single implant abutment and crown or a three-unit fixed splint should be grounded in previously discussed considerations, including length and cost of therapy and long-term predictability of care.

As previously detailed, the belief that a three-unit fixed bridge is indicated over an implant abutment and crown when one or both of the adjacent teeth are either restored or require restoration, is a fallacy. The opposite is true. When the planned abutment teeth require removal of large older restorations, or treatment of significant caries lesions, the incidence of endodontic therapy increases dramatically, as do the complexity and cost of care. Significant involvement of the planned abutment teeth is actually an indication for placement of a single implant, abutment and crown rather than a three-unit fixed prosthesis.

In addition to the already discussed compromise of greater difficulty in performing adequate home care measures around a three-unit fixed

## 28 Tooth Retention and Implant Placement

bridge as compared to a restored implant, the suitability of a maxillary second molar to serve as a terminal abutment for a fixed prosthesis must be considered. The root morphology of the maxillary second molar is often conical and/or fused. In addition, care must be taken to ensure that a distal wedge periodontal surgical procedure is performed, if necessary, to eliminate redundant soft tissues on the distal aspect of the maxillary second molar. Failure to do so will result in a short preparation wall and a compromise in crown retention, and a milieu which will pose a further difficulty in plaque control efforts. When faced with such a short preparation wall, the clinician must choose between two unacceptable treatment options. Either the preparation extends further subgingivally, often encountering undercuts in the anatomy of the tooth, or the restoration ignores these undercuts as it extends subgingivally, resulting in a restorative overhang in the furcation area. If the preparation does not extend in this manner, the final restoration will have a short axial wall, resulting in cement washout and prosthetic failure.

The question of which therapeutic approach to adopt usually hinges upon the need or lack of need for concomitant or prior regenerative therapy, and the extent of the regenerative therapy which will be required. In order to fully address this topic, an in-depth discussion must be carried out regarding various treatment approaches for augmentation of the posterior maxilla, the indications for each treatment approach, and the minimum implant lengths suitable in maxillary posterior reconstructive scenarios. This discussion is the focus of Chapter 6.

In summary, the treatment options available for replacement of a missing maxillary first molar when the second molar is present are as follows:

- (a) A three-unit fixed splint with endodontic therapy if required.
- (b) Placement of a single implant without concomitant regenerative therapy. The implant is subsequently restored with a stock abutment and crown.
- (c) Placement of a single implant with osteotome therapy. The implant is subsequently restored with a stock abutment and crown.
- (d) Placement of a single implant with concomitant sinus augmentation therapy. The im-

plant is subsequently restored with a stock abutment and crown.

- (e) Placement of a single implant with concomitant sinus augmentation and buccal ridge augmentation. The implant is subsequently restored with a stock abutment and crown.
- (f) Osteotome therapy followed by implant placement in a second stage surgery, and subsequent restoration with a stock abutment and crown.
- (g) Sinus augmentation therapy, with concomitant buccal augmentation therapy if necessary, followed by implant placement at a second stage surgery, and subsequent restoration with a stock abutment and crown.

While the focus of the present discussion is not when to select a given regenerative therapy, the above outline allows comparisons to be made between three-unit fixed prostheses, implant placement and restoration, and regenerative and implant therapies followed by implant restoration (Tables 1.6 and 1.7).

A cost-benefit analysis of each treatment option is offered in Flow chart 1.3.

If no augmentation therapy is necessary, both clinical and financial determinants point to the most logical option as being that of implant placement and restoration with a stock abutment and crown. Even when an osteotome lift must be performed at the time of implant placement, it is inappropriate to look toward a three-unit fixed bridge. The performance of a concomitant osteotome procedure is atraumatic and adds at most 3–5 minutes to the overall time of the surgical visit.

Should simultaneous sinus augmentation therapy (with or without concomitant buccal ridge augmentation therapy) be required at the time of implant placement, the clinician's clinical philosophy and facility with various procedures will most likely dictate the chosen course of therapy. Performed appropriately, a sinus augmentation procedure takes 15–20 minutes and is not problematic for the patient either during the course of treatment or postoperatively. If such augmentation can be accomplished at the time of implant placement, the most ideal therapeutic approach is still implant utilization as opposed to a three-unit fixed bridge. However, if the treating clinician is not fluent in sinus augmentation procedures, and

**Table 1.6** Treatment options for a missing maxillary first molar.

Treatment option	Advantages	Disadvantages
Three-unit fixed bridge	<ol style="list-style-type: none"> <li>1. Avoid potential regenerative therapy</li> <li>2. Slightly lesser cost of therapy</li> <li>3. Significantly lesser cost of therapy if regenerative therapy is required for implant placement</li> </ol>	<ol style="list-style-type: none"> <li>1. Possible need for endodontic intervention</li> <li>2. Greater difficulty in plaque control efforts</li> <li>3. Potential need for periodontal surgical therapy on the second molar</li> <li>4. Second molar is often ill suited to serve as a terminal abutment</li> </ol>
Implant placement without regenerative therapy followed by restoration with a stock abutment and crown	<ol style="list-style-type: none"> <li>1. No involvement of adjacent teeth</li> <li>2. No need for endodontic therapy</li> <li>3. Greater ease of plaque control efforts</li> <li>4. Greater long-term predictability</li> </ol>	<ol style="list-style-type: none"> <li>1. Slightly higher cost of therapy than a three-unit fixed bridge without endodontic therapy</li> </ol>
Implant placement with concomitant osteotome use followed by restoration with a stock abutment and crown	<ol style="list-style-type: none"> <li>1. No involvement of adjacent teeth</li> <li>2. No need for endodontic therapy</li> <li>3. Greater ease of plaque control efforts</li> <li>4. Greater long-term predictability</li> </ol>	<ol style="list-style-type: none"> <li>1. Slightly higher cost of therapy than a three-unit fixed bridge without endodontic therapy</li> </ol>
Implant placement with concomitant sinus augmentation therapy followed by restoration with a stock abutment and crown	<ol style="list-style-type: none"> <li>1. No involvement of adjacent teeth</li> <li>2. No need for endodontic therapy</li> <li>3. Greater ease of plaque control efforts</li> <li>4. Greater long-term predictability</li> </ol>	<ol style="list-style-type: none"> <li>1. Greater cost of therapy than a three-unit fixed bridge without endodontic therapy</li> </ol>
Sinus augmentation therapy followed by implant placement at a second surgical visit followed by restoration with a stock abutment and crown	<ol style="list-style-type: none"> <li>1. No involvement of adjacent teeth</li> <li>2. No need for endodontic therapy</li> <li>3. Greater ease of plaque control efforts</li> <li>4. Greater long-term predictability</li> </ol>	<ol style="list-style-type: none"> <li>1. Greater cost of therapy than a three-unit fixed bridge without endodontic therapy</li> <li>2. Need for a second surgical visit</li> </ol>

**Table 1.7** Cost analysis of treatment options for a missing maxillary first molar.

Treatment option	Cost as a factor of "X"
Three-unit fixed bridge	4.1X
Three-unit fixed bridge with crown-lengthening surgery	5.2X
Three-unit fixed bridge with one endodontic therapy	5.0X–5.4X
Three-unit fixed bridge with two endodontic therapies	6.3X
Implant placement and restoration with a stock abutment and crown	4.3X
Implant placement with concomitant osteotome therapy and restoration with a stock abutment and crown	4.3X
Implant placement with concomitant sinus augmentation therapy and restoration with a stock abutment and crown	6.8X
Sinus augmentation therapy followed by implant placement at a second surgical visit and restoration with a stock abutment and crown	6.8X

### 30 Tooth Retention and Implant Placement

views them as a major surgical event, a three-unit fixed bridge will be chosen as the appropriate therapy. Unfortunately, such an approach would leave the patient with the aforementioned compromises, and a lesser degree of long-term predictability than sinus augmentation, implant placement, and restoration.

Should endodontic therapy be required on one or more of the abutment teeth, sinus augmentation with simultaneous implant placement would be the appropriate course of therapy, from both clinical and financial points of view.

However, should sinus augmentation therapy have to be performed in a surgical visit prior to the time of implant placement, and should no endodontic therapy be required on the abutment teeth, a three-unit fixed splint is the therapeutic modality of choice, assuming the second molar is well suited to serve as a terminal abutment for a three-unit fixed bridge. Such an approach will eliminate the need for the patient to undergo two surgical sessions, and a protracted course of therapy. Before deciding upon this treatment approach, it is important to truly assess the need or lack of need for a separate sinus augmentation procedure, and to have a thorough understanding of the capabilities of implants of various lengths in replacing single missing maxillary posterior teeth.

#### *Eliminating less predictable therapies through implant use*

The predictability of regenerative and implant therapies affords the opportunity to avoid higher stress, less predictable treatment alternatives.

Long span fixed prostheses are rarely considered, and posterior cantilevers are never employed in fixed prosthetic situations. Distal cantilevers in posterior regions are only utilized when fabricating hybrid prostheses in edentulous arches.

#### **Clinical Example Seven**

A 36-year-old male presented with an inability to wear a maxillary removable partial prosthesis, and esthetic concerns regarding missing teeth in the maxillary bicuspid regions.

A full arch fixed splint was fabricated, employing two distal cantilevers in the maxillary right quadrant and one distal cantilever in the maxillary



**Figure 1.28** A temporary fixed prosthesis has been placed on three of the remaining maxillary teeth. Note the metal occlusal stops in the prosthesis, at the sites of the abutment teeth.

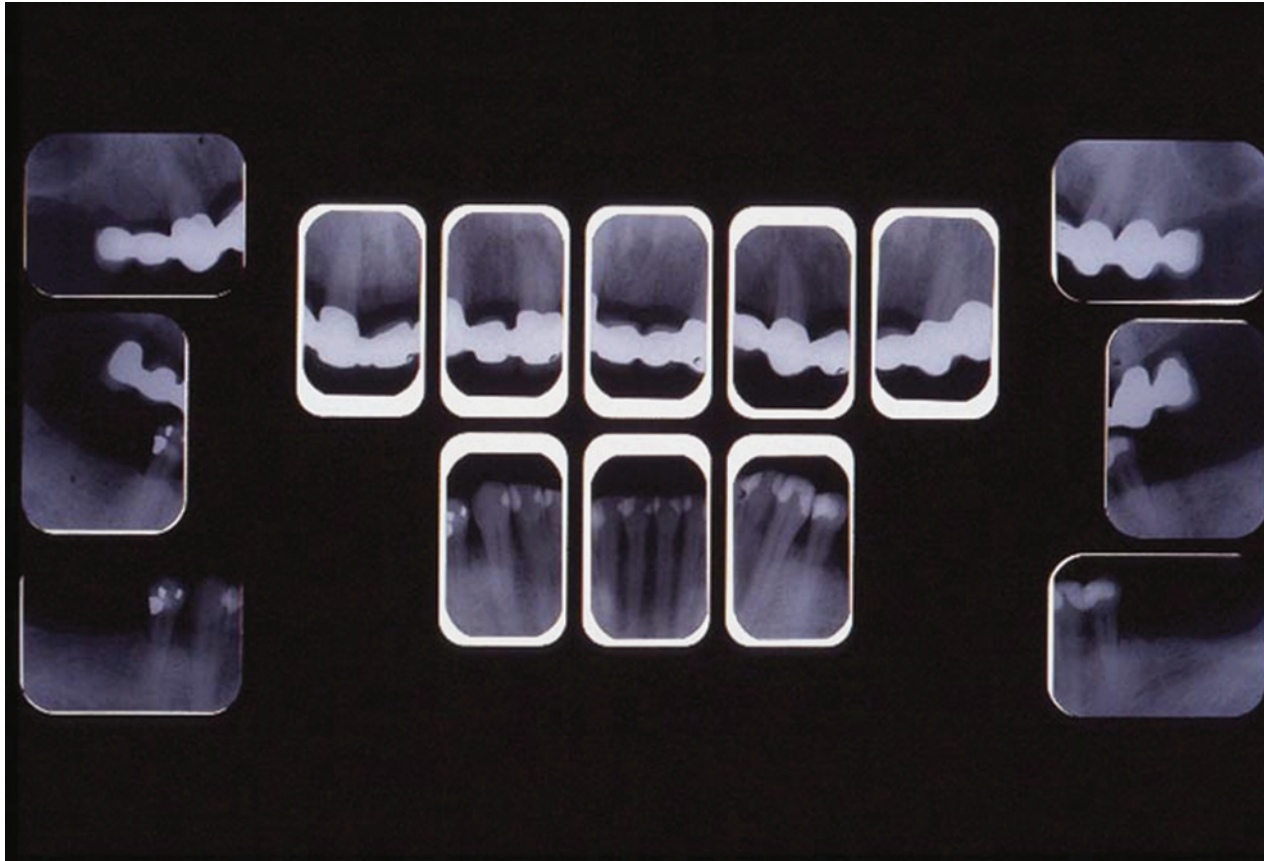
left quadrant. These cantilevers were not in contact with the opposing dentition and only served an esthetic purpose.

The patient was stable for over 10 years (Figure 1.26). Subsequently, the patient moved out of the area and another practitioner reconstructed the mandibular arch, with a cantilevered posterior fixed prostheses, which occluded with the maxillary cantilevers already in place (Figure 1.27). Within one year of this therapy being completed, accelerated bone loss and root fractures were noted around the maxillary abutments, undoubtedly due to the greater forces being placed upon them.

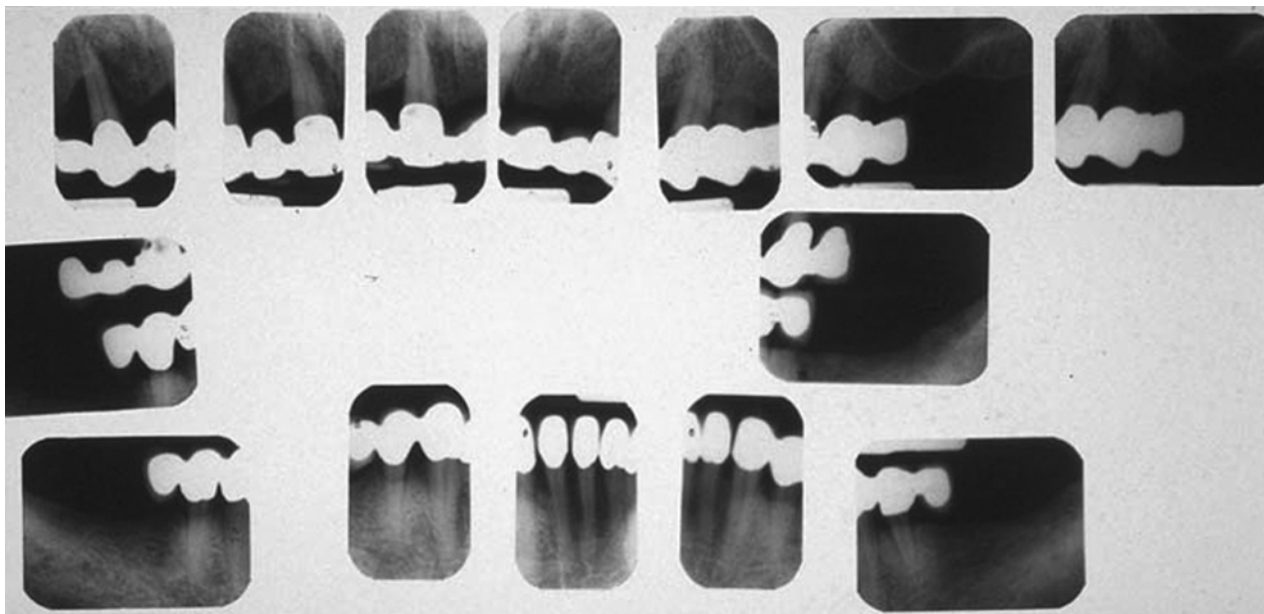
As the patient refused to wear a removable prosthesis at any time during therapy, treatment proceeded as follows: A maxillary temporary fixed splint was fabricated which was supported by three of the remaining maxillary teeth (Figure 1.28). Metal stops were evident on the temporary fixed prosthesis at the sites of the abutment teeth.

All other maxillary teeth were extracted and implants were placed. Subsequent to osseointegration of these implants, an impression was taken and an implant-supported temporary fixed prosthesis was fabricated. The remaining natural teeth were extracted, additional implants were placed, and the temporary prosthesis was inserted. Following completion of osseointegration, impressions were taken and a full arch, implant-supported fixed prosthesis was fabricated (Figure 1.29). No angled abutments were necessary, and all screw holes exited the prosthesis in ideal positions. A buccal clinical view of the prosthesis in place demonstrates the





**Figure 1.26** A patient has been reconstructed with a maxillary fixed prosthesis which includes two cantilevers in the maxillary right quadrant and one cantilever in the maxillary left quadrant. These cantilevers are not in function.



**Figure 1.27** After more than 10 years of stability, a subsequent practitioner placed a mandibular fixed prosthesis with cantilevers which occluded with the maxillary cantilevers. Within one year of its placement, the maxillary abutments demonstrated accelerated periodontal destruction and root fractures.

## 32 Tooth Retention and Implant Placement



**Figure 1.29** Following sequential implant placement and temporization, the final maxillary fixed prosthesis has been inserted. Note the ideal positions of the screw holes in the prosthesis.

patient's satisfaction with the esthetic outcomes of therapy (Figure 1.30).

### *The influence of patient health on treatment plan selection:*

It is critical that the roles played by various systemic diseases and/or patient factors in the healing and long-term predictability of different treatment approaches be well understood. Should any questions arise, the patient's physicians must always be consulted.

Numerous comprehensive texts are available which discuss this topic in depth. There is no need to regurgitate the information here.



**Figure 1.30** A buccal clinical view demonstrates the patient's esthetic satisfaction.

However, there are three common health concerns clinicians face every day, which are often misunderstood.

1. **Diabetes:** The presence of diabetes is not an absolute contraindication to therapy. The literature has demonstrated that success rates of regenerative and implant therapies in well controlled diabetics are essentially identical to those reported upon in nondiabetic patients (148). The problem arises in defining a controlled diabetic. Ideally, a consultation with the patient's physician should yield the information that the patient in question has had his or her diabetes under control for a minimum of one year. If this is not the case, it is prudent to have the patient demonstrate this level of control prior to the initiation of regenerative and/or implant therapies.
2. **Intravenous bisphosphonates:** Intravenous bisphosphonates (BIS), which are used to reduce bone pain and hypercalcemia of malignancy, have been linked with spontaneous bisphosphonate-associated osteonecrosis (BON). The ramifications of such problems are often severe and must be viewed as an absolute contraindication to periodontal or implant surgical therapy, unless the patient presents with an acute situation requiring intervention. Patients with a history of intravenous BIS therapy must be treated with care, as the potential for development of severe BON is significant. Current dental protocols suggested by Marx for patients who will receive or are receiving intravenous BIS therapy include:

#### **Before initiating intravenous BIS therapy:**

Due to the recognized high level of comorbidity of dental diseases with BIS therapy (84% of the patients followed by Marx and coworkers demonstrated periodontal disease, and 28.6% of these patients demonstrated dental caries), it is imperative that appropriate dental examination and diagnosis be carried out before the initiation of BIS therapy. Once a thorough examination with radiographs has been accomplished, necessary treatment is aimed at eliminating periodontal disease, active caries, and endodontic lesions, thus helping ensure that invasive dental procedures will not be

necessary in the near future. Dental implants should not be placed in these patients. The fit of all existing prostheses must be checked, and the prostheses adjusted or replaced as necessary to minimize trauma to underlying hard and soft tissues. Where possible, removable prostheses should be replaced with fixed appliances. Finally, a thorough prophylaxis should be performed before the initiation of BIS therapy, and the patient should be placed on a comprehensive four-month maintenance schedule to ensure their continued periodontal and restorative health.

**During intravenous BIS therapy:** Patients should be seen by their periodontist and restorative dentist so that the dental team can evaluate the oral cavity for the presence or absence of the aforementioned diseases and/or ill-fitting prostheses, and ensure that no exposed bone is present. A dental cleaning and fluoride treatment should be carried out, and the patient should be placed on a four-month maintenance schedule to ensure continued periodontal and restorative health. Teeth should only be extracted as a last resort. Nonrestorable teeth should be prepared to the gingiva and have their pulps extirpated, as such therapy is less risky than tooth extraction. Teeth with mild to moderate mobilities should be splinted together rather than removed. Teeth with extreme mobility should be extracted, as osteonecrosis is probably already present and merely hidden by the granulation tissue at the apex of the highly mobile tooth. Once extraction is carried out, appropriate measures must be taken with regard to debridement, tissue management, and antibiotic coverage to help minimize the risk of developing further osteonecrosis. Implants should not be placed in these patients. If BIS-induced osteonecrosis does occur, it is important to realize that such osteonecrosis may not be successfully treated by the modalities utilized for treatment of osteoradionecrosis, such as hyperbaric oxygen. Rather, efforts must be made to control infection and render palliative treatment

to patients in the areas of the exposed bone.

3. **Oral BIS:** Oral BIS, which are utilized in the treatment of osteoporosis and osteopenia, are of relatively widespread use in postmenopausal females. Twenty-two million prescriptions for one of the oral BIS (Alendronate) were written between May 2003 and April 2004 alone.

The question is whether or not oral BIS use predisposes a patient to the development of BON. This issue came to light following publications by Marx and coworkers (149) and Migliorati and coworkers (150). Each of these reports documented patients who had been taking oral BIS and demonstrated BON. It is important to realize that the patients in both of these studies had been referred to the institutions in question, thus making it impossible to assess the size of the patient pool taking oral BIS from which these patients were drawn. As a result, no statements could be made regarding the incidence of problems following tooth extraction in patients taking oral BIS, based wholly upon these studies.

Jeffcoat (151), in a single masked controlled study, assessed the response of patients taking oral BIS for 1–4 years with a mean time of 3 years who received implant therapy, compared to age-matched controls taking no oral BIS. Three years postimplant placement, no implants had been lost and no BON had been reported in the 25 patients taking oral BIS. A recent study conducted in two private practices (152) evaluated 61 patients taking oral BIS for 1–5 years with a mean time of 3.3 years, who had implants placed in intact ridges or at the time of tooth extraction. None of these patients demonstrated complications post therapy. All implants were functioning successfully by the Albrektsson criteria 12–24 months postinsertion.

Both of these studies seem to indicate that, in appropriately treated patients, a history of oral BIS does not increase the incidence of postoperative osteonecrosis or other complications. However, no definitive control studies have been published on this point. Naturally, prior to initiating therapy, patients must be informed of the likely risks and benefits of care. It is important to be cognizant



### 34 Tooth Retention and Implant Placement

of this history and to treat such patients in an appropriate manner.

Certain comorbidities may increase the chances of BON. Poor plaque control, a smoking habit, endodontic or carious pathologies, and overlying removable prostheses have all been implicated in the development of BON in patients with a history of oral BIS use. A recent article by Levin et al. (153) presented a patient with a history of oral BIS use who was wearing a maxillary removable partial prosthesis. This patient developed severe BON in the area of impingement of the prosthesis on the underlying hard and soft tissues.

In addition to eliminating the aforementioned pathological or habitual comorbidities, patients benefit greatly from removing the torquing forces of distal extension removable prosthesis from underlying hard and soft tissues. This may be especially true in patients with a history of oral BIS use.

All too often implant therapies are viewed as an all or none scenario. A patient is either a “full implant patient” or is “not an implant patient.” Such an artificial dichotomy does a disservice to our patients. Implants may be very predictably utilized to improve patient treatment plans without the substantial temporal and financial commitments commensurate with full mouth reconstructions.

Placement of individual implants in areas of a removable prosthesis’ distal extensions affords a number of advantages:

- Prosthetic retention is improved.
- The need to clasp anterior teeth to provide retention is significantly decreased or eliminated, thus improving the prognoses of these teeth.
- The prosthesis rests upon the implants rather than the hard and soft tissues, thus lessening bone atrophy beneath the prosthesis.
- The lever arm of the prosthesis is significantly reduced both immediately and over time. The immediate reduction in lever arm forces is obvious. However, continued prosthesis use in a distal extension situation results in bone atrophy and further rotation and levering of the removable prosthesis in the absence of implants. Utilization of a single implant in each distal extension area significantly lessens this problem.



**Figure 1.31** Implants have been placed in each distal extension area and restored with locator attachments.

A 51-year-old patient presented with a distal extension removable partial prosthesis. One implant was placed in each distal extension area. Locator attachments were utilized to help support the removable partial prosthesis, thus providing increased retention, and ameliorating the destructive lever arms of the distal extension prosthesis (Figures 1.31 and 1.32).

Early work suggests that the risk of development of BON may be assessed through a CTX blood test. Marx and coworkers (154) have noted a correlation between CTX blood test values and the development of postoperative complications in patients taking oral BIS.



**Figure 1.32** A view of the “female” components in the removable partial prosthesis.

They have proposed that a patient with a CTX value higher than 150 is at a minimal risk; a patient with a value between 100 and 150 is at moderate risk; and a patient with a CTX blood test value less than 100 is at a high risk for developing postoperative complications. However, the validity of this proposal has not yet been established through large-scale studies. While the need for further research and data regarding incidence of complications and suggested treatment protocols for intravenous and oral BIS patient is obvious, the available literature would point to the need for absolute care when treating patients with a history of intravenous BIS use, and comprehensive but undeterred care when treating patients with a history of oral BIS use.

4. **Smoking:** Smoking is not an absolute contraindication to regenerative or implant therapies. Nevertheless, the literature has demonstrated that various thresholds of smoking are more deleterious to short- and long-term treatment outcomes. A reasonable suggestion is that patients reduce their smoking habit to less than 10 cigarettes per day prior to any type of implant or regenerative therapy. No sinus augmentation, other than osteotome use, is carried out in patients who smoke. The desired level of smoking reduction or cessation must be attained and maintained for a minimum of three months prior to the initiation of therapy and a minimum of three months post-therapy.
5. **Parafunctional habits:** The forces generated from such habits significantly increase the chances of implant and/or prosthetic failure. The biological ramifications of such force application have been well established with regard to bone loss and eventual implant disintegration. Prosthetic failures as a result of biomechanical inability to withstand such excessive force application have been documented throughout the literature.

Significant time should be spent with the patient discussing concerns regarding uncontrolled diabetes, smoking, and other systemic conditions. Efforts should be made at behavioral modification rather than chastisement. It is illogical to tell a patient who has been smoking 20–30 cigarettes a day

for decades that he or she “must stop completely.” It is much more effective to work with this patient in an effort to decrease smoking to a level below 10 cigarettes per day. More often than not the clinician will find that the patient continues to decrease his or her smoking level until the habit ceases all together.

## Conclusions

Claims of therapeutic success, regardless of the treatment modality employed, demand the ability to answer the following questions in the affirmative:

- Is the patient better off than before undergoing therapy?
- Has the longevity of the teeth been extended where practical and in the best interests of the patient?
- When natural teeth are to be maintained, has the longevity of the teeth been extended for as long as therapeutically possible?
- If regenerative and/or implant reconstructive therapies have been carried out, have they been utilized in the best interests of the patient, and in a manner by which to ensure maximization of long-term treatment outcomes?

Patients are human beings who have come to us and entrust us to provide appropriate care for them. The challenge facing the conscientious clinician today is not that of mastering available techniques. Such mastery is easily attained through education and practice. The challenge we all must meet is the determination of when to perform which therapy for an individual patient, in a given situation.

## References

1. Fugazzotto PA. 1999. Guided tissue regeneration: Maximizing clinical results. *Compend Cont Dent Educ* 6(Suppl):1–42.
2. Caffesse RG. 1989. *Resective Procedures*. Chicago, IL: American Academy of Periodontology. Proceedings of the World Workshop in Clinical Periodontics.
3. Waerhaug J. 1978. Healing of the dento-epithelial junction following subgingival plaque control. II: As observed on extracted teeth. *J Periodontol* 49:119–134.



### 36 Tooth Retention and Implant Placement

4. Stambaugh RV, Drago M, Smith DM, and Carosali L. 1981. The limits of subgingival scaling. *Int J Periodontics Restorative Dent* 1(5):30-42.
5. Buchanan S and Robertson P. 1987. Calculus removal by scaling/root planing with and without surgical access. *J Periodontol* 58:163.
6. Jones WA and O'Leary TJ. 1978. The effectiveness of root planing in removing bacterial endotoxin from the roots of periodontally involved teeth. *J Periodontol* 49:337-342.
7. Rabbani GM, Ash MM, and Caffesse RG. 1981. The effectiveness of subgingival scaling and root planing in calculus removal. *J Periodontol* 52:119-123.
8. Caffesse R, Sweeney PL, and Smith BA. 1986. Scaling and root planing with and without periodontal flap surgery. *J Clin Periodontol* 13:205-210.
9. Tabita PV, Bissada NF, and Maybury JE. 1981. Effectiveness of supragingival plaque control on the development of subgingival plaque and gingival inflammation in patients with moderate pocket depth. *J Periodontol* 52:88-93.
10. Waerhaug J and Steen E. 1952. The presence or absence of bacteria in the gingival pocket and the reaction in healthy pockets to certain pure cultures. *Odontol Tidskr* 60:1-24.
11. Stahl SS. 1965. Healing of gingival tissues following various therapeutic regimens—a review of histologic studies. *J Oral Ther Pharmacol* 2:145-160.
12. Morris M. 1954. The removal of pocket and attachment epithelium in humans: A histologic study. *J Periodontol* 25:7-11.
13. Smukler HM and Landsberg J. 1984. The toothbrush and gingival traumatic injury. *J Periodontol* 55:713-719.
14. Nevins M. 1982. Interproximal periodontal disease—the embrasure as an etiologic factor. *Int J Periodontics Restorative Dent* 2(6):9-27.
15. Fugazzotto PA. 1989. *Preparation of the Periodontium for Restorative Dentistry*. St Louis: Ishiyaku EuroAmerica, pp. 44-54.
16. Ochsenein C. 1986. A primer for osseous surgery. *Int J Periodontics Restorative Dent* 6(1):8-46.
17. Johnson RL. 1979. Osseous surgery in periodontal therapy. In: Prichard JF (ed), *The Diagnosis and Treatment of Periodontal Disease in General Dental Practice*. Philadelphia: The W.B. Saunders.
18. Fugazzotto PA and Parma-Benfenati S. 1984. Preprosthetic periodontal considerations. Crown length and biologic width. *Quintessence Int* 15:1247-1256.
19. Gelfand HB, Tencate AR, and Freeman E. 1978. The keratinization potential of crevicular epithelium: An experimental study. *J Periodontol* 49:113-118.
20. Caffesse RG, Karring T, and Nasjleti CE. 1977. Keratinizing potential of sulcular epithelium. *J Periodontol* 48:140-146.
21. Caffesse RG and Nasjleti CE. 1976. Enzymatic penetration through intact sulcular epithelium. *J Periodontol* 47:391-397.
22. Thilander H. 1963. The effect of leukocytic enzymes activity on the structure of the gingival pocket epithelium in man. *Acta Odontol Scand* 21:431-451.
23. Rosenberg MM, Kay HB, Keough BE, and Holt RL. 1988. *Periodontal and Prosthetic Management for Advanced Cases*. Chicago: Quintessence, pp. 148-156.
24. Matherson DG. 1988. An evaluation of healing following periodontal osseous surgery in Rhesus monkeys. *Int J Periodontics Restorative Dent* 8(5):9-34.
25. Squier CA. 1981. Keratinization of the sulcular epithelium—a pointless pursuit. *J Periodontol* 52:426-429.
26. Schroeder HE and Munzel-Pedrazzoli S. 1970. Morphometric analysis comparing junctional and oral epithelium of normal human gingiva. *Helv Odontol Acta* 14:53-66.
27. Barnett ML. 1979. Letter to the editor. *J Periodontol* 50:154.
28. Saito I, Watanabe O, Kawahara H, Igarashi Y, Yamamura T, Shimono M. 1981. Intercellular junctions and the permeability barrier in the junctional epithelium. A study with freeze-fracture and thin sectioning. *J Periodontol Res* 16:467-480.
29. Shimono M and Clementi F. 1976. Intercellular junctions of oral epithelium. I. Studies with freeze-fracture and tracing methods of normal rat keratinized oral epithelium. *J Ultrastruct Res* 56:121-136.
30. Claude P and Goodenough DA. 1973. Fracture faces of zonulae occludentes from "tight" and "leaky" epithelia. *J Cell Biol* 58:390-400.
31. Shimono M, Yamamura Y, and Fumagalli G. 1980. Intercellular junctions in salivary glands: Freeze-fracture and tracer studies of normal rat sublingual gland. *J Ultrastruct Res* 72:286-299.
32. McDougall WA. 1971. Penetration pathways of a topically applied foreign protein into rat gingiva. *J Periodontol Res* 6:89-99.
33. McDougall WA. 1970. Pathways of penetration and effects of horseradish peroxidase in rat molar gingiva. *Arch Oral Biol* 15:621-633.
34. Tolo KJ. 1971. A study of permeability of gingival pocket epithelium to albumin in guinea pigs and Norwegian pigs. *Arch Oral Biol* 16:881-888.
35. Romanowski AW, Squier CA, and Lesch CA. 1988. Permeability of rodent junctional epithelium to exogenous protein. *J Periodontol Res* 23:81-86.
36. Neiders ME. 1972. Contact phenomena of epithelial cells. *Sci Rev* 1:69-101.
37. Listgarten MA. 1975. Similarity of epithelial relationships in the gingiva of rat and man. *J Periodontol* 46:677-680.

38. Listgarten MA. 1980. Periodontal probing: What does it mean? *J Clin Periodontol* 7:165-176.
39. Saglie R, Johansen JR, and Flotra L. 1975. The zone of completely and partially destructed periodontal fibers in pathological pockets. *J Clin Periodontol* 2: 198-202.
40. Spray JR, Garnick JJ, Doles LR, and Klawitter JJ. 1978. Microscopic demonstration of the position of periodontal probes. *J Periodontol* 49:148-152.
41. Silvertson JF and Burgett FG. 1976. Probing of pockets related to the attachment level. *J Periodontol* 47:281-286.
42. Powell B and Garnick JJ. 1978. The use of extracted teeth to evaluate clinical measurements of periodontal disease. *J Periodontol* 49:621-624.
43. Spray R and Garnick JJ. 1979. Position of probes in human periodontal pockets. *J Dent Res* 58(Special Issue A):176. Abstract No. 331.
44. Hancock EB, Wirthlin MR, and Ellingson J. 1978. Histologic assessment of periodontal probes in normal gingiva. *J Dent Res* 57(Special Issue A):239. Abstract No. 584.
45. Ezis I and Burgett F. 1978. Probing related to attachment levels on recently erupted teeth. *J Dent Res* 57(Special Issue A):307. Abstract No. 932.
46. Page RC and Schroeder HE. 1976. Pathogenesis of inflammatory periodontal disease. A summary of current work. *Lab Invest* 33:235-249.
47. Ruben MP, Schulman SM, and Kon S. 1973. Healing of periodontal surgical wounds. In: Goldman HM and Cohen DW (eds), *Periodontal Therapy*, 5th edn. St Louis: CV Mosby Company.
48. Parma-Benfenati S, Fugazzotto PA, and Ruben MP. 1985. The effect of restorative margins on the postsurgical development and nature of the periodontium. *Int J Periodontics Restorative Dent* 5(6):31-51.
49. Carnevale G, Sterrantino SF, and DiFebo G. 1983. Soft and hard tissue wound healing following tooth preparation to the alveolar crest. *Int J Periodontics Restorative Dent* 3(6):37-53.
50. Vieira E, O'Leary T, and Kafrawy A. 1982. The effect of sodium hypochlorite and citric acid solutions on healing of periodontal pockets. *J Periodontol* 53:71-80.
51. Caton J, Nyman S, and Zander H. 1980. Histometric evaluation of periodontal surgery. II. Connective tissue attachment levels after four regenerative procedures. *J Clin Periodontol* 7:224-231.
52. Kalkwarf K, Tussing G, and Davis M. 1982. Histologic evaluation of gingival curettage facilitated by sodium hypochlorite solution. *J Periodontol* 53:63-70.
53. Yukna R. 1976. A clinical and histologic study of healing following the excisional new attachment procedure in Rhesus monkeys. *J Periodontol* 47:701-709.
54. Yukna R and Lawrence J. 1980. Gingival surgery for soft tissue new attachment. *Dent Clin North Am* 24:705-718.
55. Bowen W, Bowers G, Bergquist J, and Organ R. 1981. Removal of pocket epithelium in humans utilizing an internally beveled incision. *Int J Periodontics Restorative Dent* 1(5):9-19.
56. Froum W, Coran J, Thaller B, et al. 1982. Periodontal healing following open debridement flap procedures. I. Clinical assessment of soft tissue and osseous repair. *J Periodontol* 53:8-14.
57. Caton J and Zander H. 1976. Osseous repair of an infrabony pocket without new attachment of connective tissue. *J Clin Periodontol* 47:54-62.
58. Hiatt W, Schallhorn R, and Aaronian A. 1978. The induction of new bone and cementum formation. IV. Microscopic examination of the periodontium following human bone and marrow allograft, autograft, and nongraft periodontal regeneration procedures. *J Periodontol* 49:495-512.
59. Egelberg J. 1987. Regeneration and repair of periodontal tissues. *J Periodontol Res* 22:233-242.
60. Wirthlin MR. 1981. The current status of new attachment therapy. *J Periodontol* 52:529-544.
61. Listgarten M and Rosenberg M. 1979. Histological study of repair following new attachment procedures in human periodontal lesions. *J Periodontol* 50:333-344.
62. Ellagaard B, Karring T, Listgarten M, and Loe H. 1973. New attachment after treatment of interradicular lesions. *J Periodontol* 44:209-217.
63. Stahl S, Froum S, and Kushner L. 1982. Periodontal healing following open debridement flap procedures. II. Histologic observations. *J Periodontol* 53: 15-21.
64. Frank R, Fiore-Donno G, Cimasoni G, and Matter J. 1974. Ultrastructural study of epithelial and connective tissue gingival reattachment in man. *J Periodontol* 45:626-635.
65. Stahl S. 1979. Repair or regeneration. *J Clin Periodontol* 6:389-396.
66. Magnusson I, Ronstad L, Nyman S, and Lindhe J. 1983. A long junctional epithelium—a locus minoris resistentiae in plaque infection? *J Clin Periodontol* 10:333-340.
67. Beaumont R, O'Leary T, and Kafrawy A. 1984. Relative resistance of long junctional epithelial adhesions and connective tissue attachments to plaque-induced inflammation. *J Periodontol* 55:213-225.
68. Caton J and Nyman S. 1980. Histometric evaluation of periodontal surgery. I. The modified Widman flap procedure. *J Clin Periodontol* 7:212-223.
69. Steiner S, Crigger M, and Egelberg J. 1981. Connective tissue regeneration to periodontally diseased teeth. II. Histologic observations of cases following replaced flap surgery. *J Periodontol Res* 16:109-116.
70. Ross I and Thompson R. 1980. Furcation involvement in maxillary and mandibular molars. *J Periodontol* 51:450-454.

### 38 Tooth Retention and Implant Placement

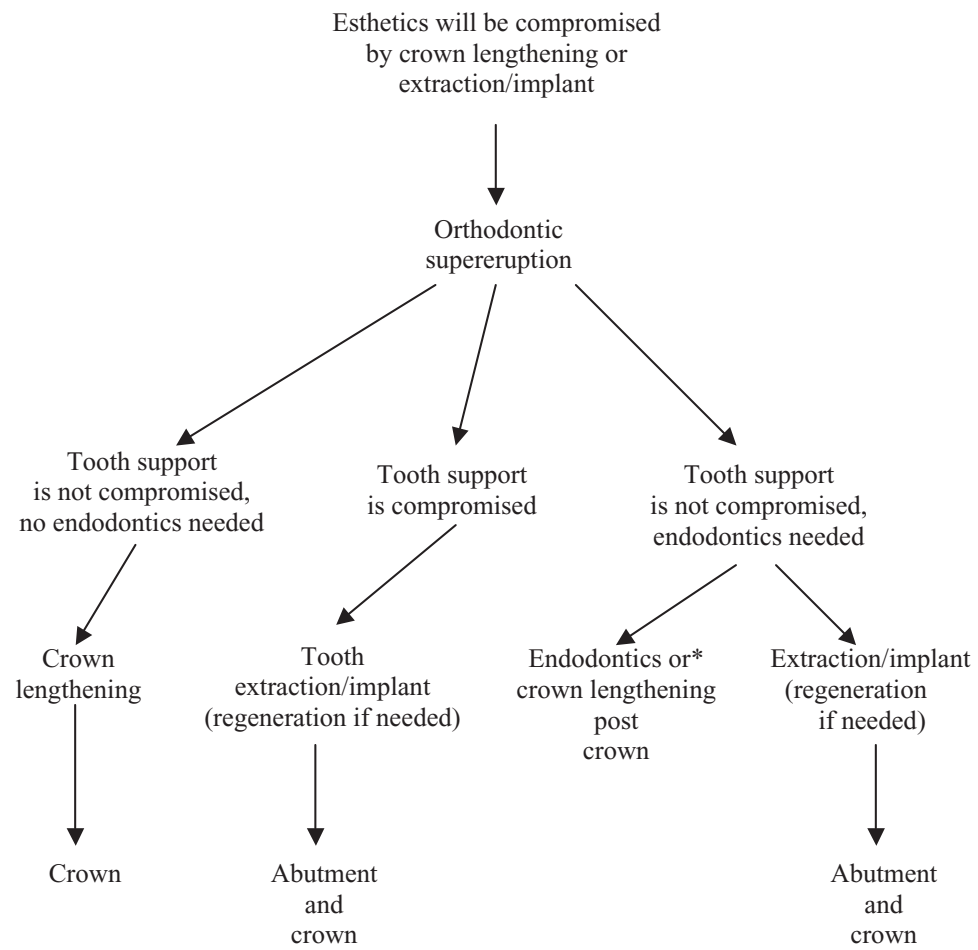
71. Larato DC. 1975. Some anatomical factors related to furcation involvements. *J Periodontol* 46:608-609.
72. Ricchetti PA. 1982. A furcation classification based on pulp chamber-furcation relationships and vertical radiographic bone loss. *Int J Periodontics Restorative Dent* 2(5):51-59.
73. Sternlicht HC. 1963. New approach to the management of multicrooked teeth with advanced periodontal disease. *J Periodontol* 34:150-158.
74. Karlsen K. 1970. Gingival reaction to dental restorations. *Acta Odontol Scand* 28:895-899.
75. Waerhaug J. 1953. Tissue reactions around artificial crowns. *J Periodontol* 24:172-185.
76. Newcomb GM. 1974. The relationship between the location of subgingival crown margins and gingival inflammation. *J Periodontol* 45:151-154.
77. Renggli HH and Regolati B. 1972. Gingival inflammation and plaque accumulation by well adapted supragingival and subgingival proximal restorations. *Helv Odontol Acta* 16:99-101.
78. Waerhaug J. 1956. Effect of rough surfaces upon gingival tissues. *J Dent Res* 35:323-325.
79. Silness J. 1970. Periodontal conditions in patients treated with dental bridges. II. The influence of full and partial crowns on plaque accumulation, development of gingivitis and pocket formation. *J Periodontol Res* 5:219-224.
80. Mormann W, Regolatti B, and Renggli HH. 1974. Gingival reaction to well fitted subgingival gold inlays. *J Clin Periodontol* 1:120-125.
81. Gilmore N and Sheiham A. 1971. Overhanging dental restorations and periodontal disease. *J Periodontol* 42:8-12.
82. Fugazzotto PA. 1985. Periodontal restorative interrelationships: The isolated restoration. *J Am Dent Assoc* 110:915-917.
83. Ramfjord S, Nissle R, Shick R, and Cooper H. 1968. Subgingival curettage vs. surgical elimination of periodontal pockets. *J Periodontol* 39:167-175.
84. Ramfjord S, Knowles J, Nissle R, Shick R, and Burgett F. 1973. Longitudinal study of periodontal therapy. *J Periodontol* 44:66-77.
85. Ramfjord S, Knowles J, Nissle R, Burgett F, and Shick R. 1975. Results following three modalities of periodontal therapy. *J Periodontol* 46:522-526.
86. Knowles J, Burgett F, Nissle R, Shick R, Morrison E, and Ramfjord S. 1979. Results of periodontal treatment related to pocket depth and attachment level. Eight years. *J Periodontol* 50:225-233.
87. Ramfjord S, Knowles J, Morrison E, et al. 1980. Results of periodontal therapy related to tooth type. *J Periodontol* 51:270-273.
88. Knowles J, Burgett F, Morrison E, Nissle R, and Ramfjord S. 1980. Comparison of results following three modalities of periodontal therapy related to tooth type and initial pocket depth. *J Clin Periodontol* 7:32-47.
89. Burgett F, Knowles J, Nissle R, Shick R, and Ramfjord S. 1977. Short term results of three modalities of periodontal treatment. *J Periodontol* 48:131-135.
90. Hill R, Ramfjord S, Morrison E, et al. 1981. Four types of periodontal treatment compared over two years. *J Periodontol* 52:655-662.
91. Ramfjord S, Caffesse R, Morrison E, et al. 1987. Four modalities of periodontal treatment compared over five years. *J Clin Periodontol* 14:445-452.
92. Becker W, Becker B, Ochsenein C, et al. 1988. A longitudinal study comparing scaling and osseous surgery and modified Widman procedures—results after one year. *J Periodontol* 59:351-358.
93. Stahl S. 1974. Lecture given at Boston University School of Graduate Dentistry, Fall, 1981.
94. Ramfjord S and Nissle R. 1974. The modified Widman flap. *J Periodontol* 45:601-607.
95. Lindhe J, Socransky S, Nyman S, and Westfelt E. 1987. Dimensional alterations of the periodontal tissues following therapy. *Int J Periodontics Restorative Dent* 7: 9-21.
96. Attstrom A and Lindhe J. 1983. Pathogenesis of plaque associated periodontal disease. In: Lindhe J (ed), *Textbook of Clinical Periodontology*. Copenhagen: Munksgaard.
97. Egelberg J. 1989. The impact of regression towards the mean on probing changes in studies on the effect of periodontal therapy. *J Clin Periodontol* 16: 120-123.
98. Aeppli DM and Pihlstrom BL. 1989. Detection of longitudinal change in periodontitis. *J Periodontol Res* 24:329-334.
99. Sterne JAC, Johnson NW, Wilton JMA, Joyston-Bechal S, and Smales FC. 1988. Variance components analysis of data from periodontal research. *J Periodontol Res* 23:148-153.
100. Lindhe J and Nyman S. 1984. Long term maintenance of patients treated for advanced periodontal disease. *J Clin Periodontol* 11:504-514.
101. Saadoun A. 1985. Management of furcation involvement. *J West Soc Periodontol Abstr* 33:91-125.
102. Basaraba N. 1969. Root amputation and tooth hemisection. *Dent Clin North Am* 13:121-132.
103. Newell D. 1981. Current status of the management of teeth with furcation invasion. *J Periodontol* 52:559-568.
104. Rosenberg MM, Kay HB, Keough BE, and Holt RL. 1988. *Periodontal and Prosthetic Management for Advanced Cases*. Chicago: Quintessence, pp. 247-298.
105. Waerhaug J. 1980. The furcation problem. Etiology, pathogenesis, diagnosis, therapy and prognosis. *J Clin Periodontol* 7:73-95.
106. Everett F, Jump E, Holder T, and Wilson G. 1958. The intermediate bifurcational ridge: A study of the morphology of the bifurcation of the lower first molar. *J Dent Res* 37:162-169.

107. Gher M and Vernino A. 1981. Root anatomy: A local factor in inflammatory periodontal disease. *Int J Periodontics Restorative Dent* 1(5):53-63.
108. Bower R. 1979. Morphology relative to periodontal treatment: Furcation entrance architecture. *J Periodontol* 50:23-27.
109. Matia J, Bissada NF, and Maybury JE. 1986. Efficacy of scaling of the molar furcation area with and without surgical access. *Int J Periodontics Restorative Dent* 6(6):25-35.
110. Fleischer HC, Mellonig JT, Brayer WK, Gray JL, and Barnett JD. 1989. Scaling and root planing efficacy in multirooted teeth. *J Periodontol* 60:402-409.
111. Pihlstrom BL, Oritz-Campos C, and McHugh RB. 1981. A randomized four year study of periodontal therapy. *J Periodontol* 52:227-242.
112. Pihlstrom BL, McHugh RB, Oliphant TH, and Oritz-Campos C. 1983. Comparison of surgical and non surgical treatment of periodontal disease. A review of current studies and additional results after six and one half years. *J Clin Periodontol* 10:524-541.
113. Stern T, Everett F, and Robicsek KS. 1965. Robicsek: A pioneer in the surgical treatment of periodontal disease. *J Periodontol* 36:265-268.
114. Yukna R and Williams JE. 1980. Five year evaluation of the excisional new attachment procedure. *J Periodontol* 51:382-385.
115. Smith DH, Ammons WF, and van Belle G. 1980. A longitudinal study of periodontal status comparing flap curettage and osseous recontouring. I. Six month results. *J Periodontol* 51:367-375.
116. Olsen CT, Ammons WF, and van Belle G. 1985. A longitudinal study comparing apically repositioned flaps, with and without osseous surgery. *Int J Periodontics Restorative Dent* 5:11-33.
117. Lindhe J and Nyman S. 1980. Alterations of the position of the marginal soft tissue following periodontal surgery. *J Clin Periodontol* 7:525-530.
118. Lindhe J, Haffajee AD, and Socransky SS. 1983. Progression of periodontal disease in adult subjects in the absence of periodontal therapy. *J Clin Periodontol* 10:433-442.
119. Nabers CL, Stalker WH, Esparza D, et al. 1988. Tooth loss in 1535 treated periodontal patients. *J Periodontol* 59:297-300.
120. McFall WT. 1982. Tooth loss in 100 treated patients with periodontal disease—a long term study. *J Periodontol* 53:539-549.
121. Goldman MJ, Ross IF, and Goteiner D. 1986. Effect of periodontal therapy on patients maintained for 15 years or longer. A retrospective study. *J Periodontol* 57:347-353.
122. Kaldahl WB, Kalkwarf KL, Kashinath DP, Molvar MP, and Dyer JK. 1996. Long term evaluation of periodontal therapy: I. Response to four therapeutic modalities. *J Periodontol* 67:93-102.
123. Kaldahl WB, Kalkwarf KL, Kashinath DP, Molvar MP, and Dyer JK. 1996. Long term evaluation of periodontal therapy: II. Incidence of sites breaking down. *J Periodontol* 67:103-108.
124. Lindhe J, Okamoto H, Yoneyama T, Haffajee A, and Socransky SS. 1989. Longitudinal changes in periodontal disease in untreated subjects. *J Clin Periodontol* 16:662-670.
125. Badersten A, Nilveus R, and Egelberg J. 1984. Effect of nonsurgical periodontal therapy. II. Severely advanced periodontitis. *J Clin Periodontol* 11:63-76.
126. Badersten A, Nilveus R, and Egelberg J. 1984. Effect of nonsurgical periodontal therapy. III. Single versus repeated instrumentation. *J Clin Periodontol* 11:114-124.
127. Waite IM. 1976. A comparison between conventional gingivectomy and a nonsurgical regime in the treatment of periodontitis. *J Clin Periodontol* 3:173-185.
128. Buchanan SA and Robertson PB. 1987. Calculus removal by scaling/root planing with and without surgical access. *J Periodontol* 58:159-163.
129. Rabbani GM, Ash MM, and Caffesse RG. 1981. The effectiveness of subgingival scaling and root planing in calculus removal. *J Periodontol* 52:119-123.
130. Jeffcoat MK and Reddy MS. 1991. Progression of probing attachment loss in adult periodontitis. *J Periodontol* 62:185-189.
131. Badersten A, Nilveus R, and Egelberg J. 1985. Effect of non-surgical periodontal therapy. VII. Bleeding, suppuration and probing depth in sites with probing attachment loss. *J Clin Periodontol* 12:432-440.
132. Grbic JT and Lamstr IB. 1992. Risk indicators for future clinical attachment loss in adult periodontitis. Tooth and site variables. *J Periodontol* 63:262-269.
133. Haffajee AD, Socransky SS, Smith C, and Divart S. 1991. Microbial risk indicators for periodontal attachment loss. *J Periodontol Res* 26:293-296.
134. Vanooteghem R, Hutchenes LH, Garrett S, Kiger R, and Egelberg J. 1987. Bleeding on probing and probing depth as indicators of the response to plaque control and root debridement. *J Clin Periodontol* 14:226-230.
135. Nyman S, Gottlow J, Karring T, et al. 1982. The regenerative potential of the periodontal ligament. An experimental study in the monkey. *J Clin Periodontol* 9:257-265.
136. Gottlow J, Nyman S, Karring T, et al. 1984. New attachment formation as the result of controlled tissue regeneration. *J Clin Periodontol* 11:494-503.
137. Nyman S, Lindhe J, Karring T, et al. 1982. New attachment following surgical treatment of human periodontal disease. *J Clin Periodontol* 9:290-296.
138. Gottlow J, Nyman S, Lindhe J, et al. 1986. New attachment formation in the human periodontium by guided

## 40 Tooth Retention and Implant Placement

- tissue regeneration. Case reports. *J Clin Periodontol* 13:604–616.
139. Schallhorn RG, Hiatt WH, and Boyce W. 1970. Iliac transplants in periodontal therapy. *J Periodontol* 41:566–580.
  140. Hiatt WH and Schallhorn RG. 1973. Intraoral transplants of cancellous bone and marrow in periodontal lesions. *J Periodontol* 44:194–208.
  141. Froum SJ, Ortiz M, Witkin RT, et al. 1976. Osseous autografts. III. Comparison osseous coagulum-bone blend implants with open curettage. *J Periodontol* 47:287–294.
  142. Renvert S, Garrett S, Schallhorn RG, et al. 1985. Healing after treatment of periodontal intraosseous defects. III. Effect of osseous grafting and citric acid conditioning. *J Clin Periodontol* 12:441–455.
  143. Quintero G, Mellonig JT, Gambil VM, et al. 1982. A six month clinical evaluation of decalcified freeze-dried bone allografts in periodontal osseous defects. *J Periodontol* 53:726–730.
  144. Fugazzotto PA. 2001. A comparison of the success of root resected molars and molar position implants in function in private practice: Results of up to 15-plus years. *J Periodontol* 72:1113–1123.
  145. Fugazzotto PA and Hains F. 2009. The effect of therapeutic costs on development of treatment algorithms in the partial edentulous patient. A comparative fee survey and treatment planning philosophy. *J Acad Gen Dent* (submitted for publication).
  146. Fugazzotto PA. 1985. *Preparation of the Periodontium for Restorative Dentistry*. St Louis: Ishiyaku Euro America.
  147. Fugazzotto PA. 1998. Comprehensive surgical management of the embrasure space in the prosthetic patient. *J Mass Dent Soc* 46:18–22.
  148. Bakhshandeh S, Murtomaa H, Mofid R, Behkalahti MM, and Suomalainen K. 2007. Periodontal treatment needs of diabetic adults. *J Clin Periodontol* 1:53–57.
  149. Marx R, Sawatari Y, Fortin M, and Broumand V. 2005. Bisphosphonate induced exposed bone of the jaws: Risk factors, recognition, prevention, and treatment. *J Oral Maxillofac Surg* 63:1567–1575.
  150. Migliorati CA, Schubert MM, Peterson DE, and Seneda LM. 2005. Bisphosphonate associated osteonecrosis of mandibular and maxillary bone: An emerging complication of supportive cancer therapy. *Cancer* 104:89–93.
  151. Jeffcoat MK. 2006. Safety of oral bisphosphonates: Controlled studies on alveolar bone. *Int J Oral Maxillofac Implants* 21:349–353.
  152. Fugazzotto PA, Lightfoot WS, Jaffin R, and Kumar A. 2007. Implant placement with or without simultaneous tooth extraction in patients taking oral bisphosphonates: Post-operative healing, early follow-up, and the incidence of complications of two private practices. *J Periodontol* 78:1664–1669.
  153. Levin A, Aviv A, and Schwartz-Arad D. 2007. Denture related osteonecrosis of the maxilla associated with oral bisphosphonate treatment. *J Am ENT Assoc* 138:1218–1220.
  154. Marx RE, Cillo JE, Jr, and Uloa JJ. 2007. Oral bisphosphonate induced osteonecrosis: Risk factors, prediction of risk using serum CTX testing, prevention, and treatment. *J Oral Maxillofac Surg* 65:2397–2410.

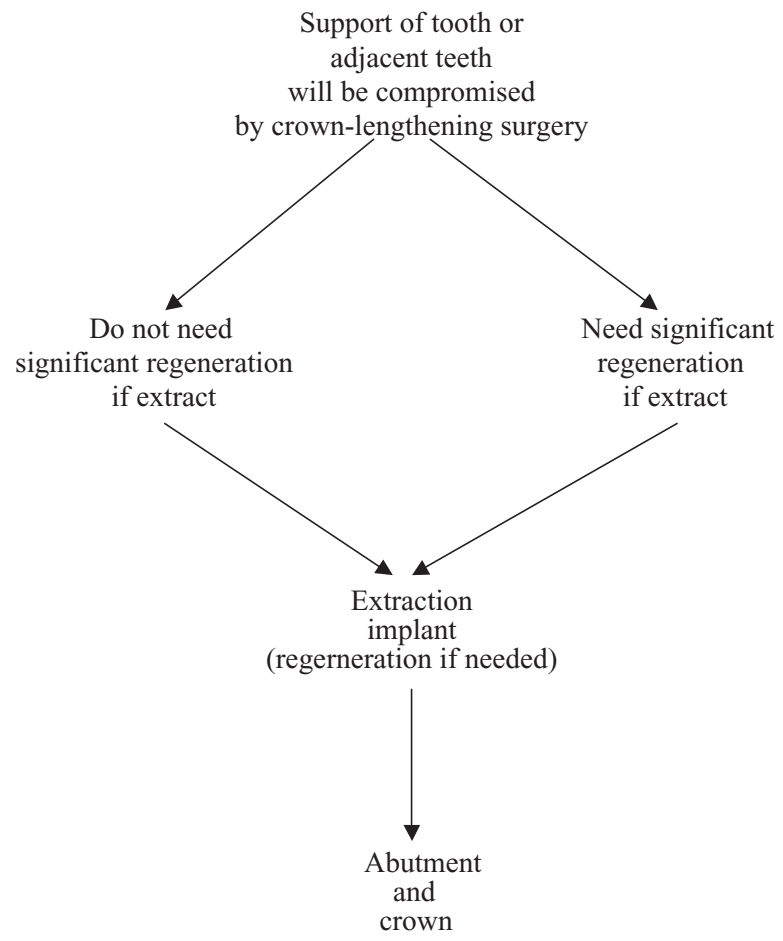




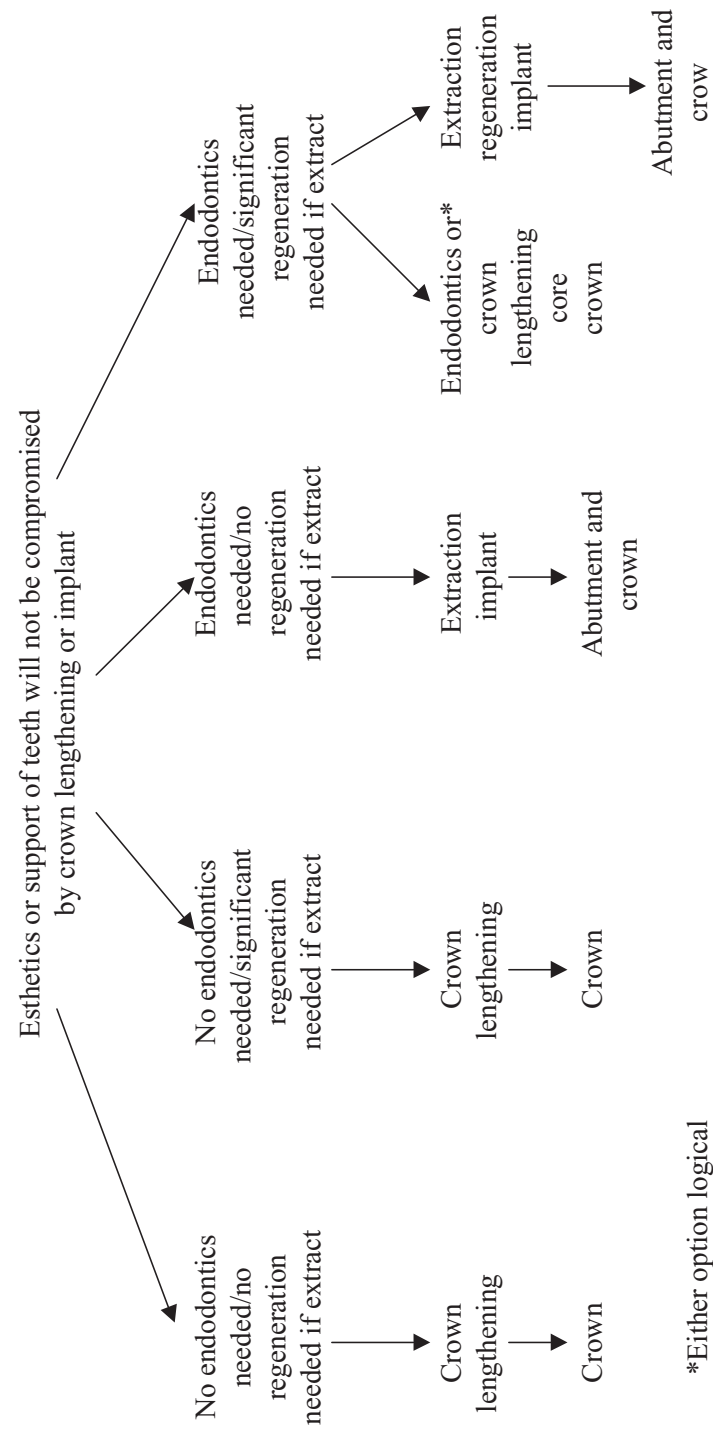
\*Either option logical

Flow chart 1.1 Treating a decayed single tooth (Part 1 of 3).

**42** Tooth Retention and Implant Placement

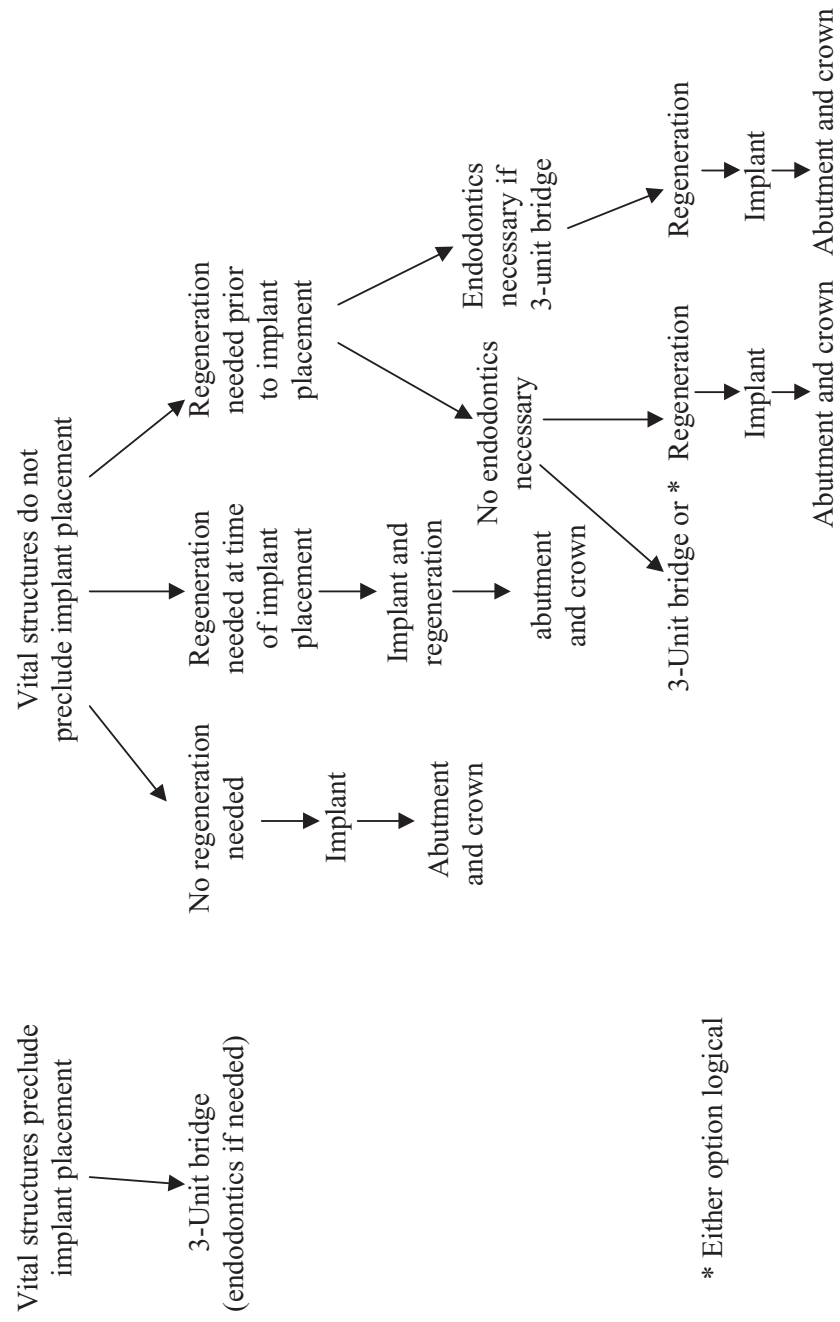


**Flow chart 1.1** Treating a decayed single tooth (Part 2 of 3).

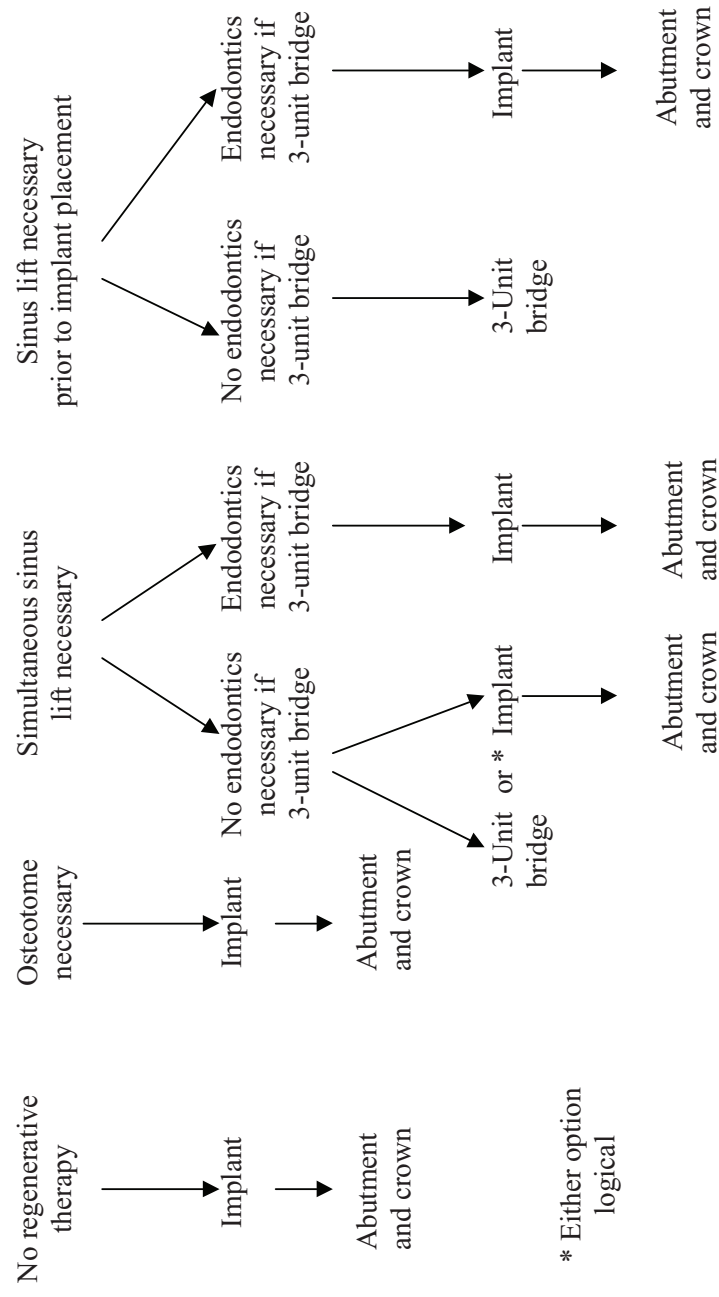


\*Either option logical

Flow chart 1.1 Treating a decayed single tooth (Part 3 of 3).



**Flow chart 1.2** Replacing a single missing tooth in a tooth-bounded space.



Flow chart 1.3 Replacing a maxillary first molar when the second molar is present.