Preface

When the groundwork for the fifth edition of *Clinical Periodontology and Implant Dentistry* began in early 2007, it became clear that we had reached a fork in the road. It has always been my intention that each successive edition of this work should reflect the state of the art of clinical periodontology and, in doing such, should run the gamut of topics within this subject area. However, thorough coverage of an already large and now rapidly expanding specialty has resulted in a book of commensurate size and therefore for the fifth edition, the decision was taken to divide the book into two volumes: basic concepts and clinical concepts. The decision to make the split a purely physical one, and not an intellectual one, reflects the realization that over the past decade, implant dentistry has become a basic part of periodontology. The integrated structure of this latest edition of the textbook mirrors this merger.

In order for the student of dentistry, whatever his or her level, to learn how teeth and implants may function together as separate or connected units in the same dentition, a sound knowledge of the tissues that surround the natural tooth and the dental implant, as well as an understanding of the various lesions that may occur in the supporting tissues, is imperative. Hence, in both volumes of the textbook, chapters dealing with traditional periodontal issues, such as anatomy, pathology and treatment, are followed by similar topics related to tissues surrounding dental implants. In the first volume of the fifth edition, "basic concepts" as they relate to anatomy, microbiology and pathology, for example, are presented, while in the second volume ("clinical concepts"), various aspects of often evidence-based periodontal and restorative examination and treatment procedures are outlined.

It is my hope that the fifth edition of *Clinical Periodontology and Implant Dentistry* will challenge the reader intellectually, provide elucidation and clarity of information, and also impart an understanding of how the information presented in the text can, and should, be used in the practice of contemporary dentistry.

Jan Lindhe

Chapter 5

Osseointegration

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The edentulous site

The fully healed, edentulous site of the alveolar ridge (see Fig. 2-23 and Chapter 2) is most often covered by a masticatory mucosa that is about 2–3 mm thick. This type of mucosa is covered by a keratinized epithelium and includes a connective tissue, rich in collagen fibers and fibroblasts, that is firmly attached to the bone via the periosteum. The outer walls of the alveolar process, the cortical plates, are comprised of lamellar bone and enclose the spongy or cancellous bone that contains bone trabeculae (lamellar bone) embedded in marrow. The bone marrow contains numerous vascular structures as well as adipocytes and pluripotent mesenchymal cells.

Osseointegration

Different types of implant systems have been used to replace missing teeth, including subperiosteal implants, endosseous implants with fibrous encapsulation, and endosseous implants with direct bone contact (*osseointegrated*). One definition of *osseointegration* (a term originally proposed by Brånemark *et al*. 1969) was provided by Albrektsson *et al*. (1981) who suggested that this was "a direct functional and structural connection between living bone and the surface of a load carrying implant". Another, clinical definition was provided by Zarb and Albrektsson (1991) who proposed that *osseointegration* was "a process whereby clinically asymptomatic rigid fixation of alloplastic materials is achieved and maintained in bone during functional loading".

Schroeder *et al*. (1976, 1981, 1995) used the term *"functional ankylosis"* to describe the rigid fixation of the implant to the jaw bone, and stated that "new bone is laid down directly upon the implant surface, provided that the rules for atraumatic implant placement are followed (rotation of the cutting instrument and less than 800 rpm, cooling with sterile physiologic saline solution) and the implant exhibits primary stability".

Thus, in order to acquire proper conditions for osseointegration (or functional ankylosis), the implant must exhibit proper initial fixation (stability) following installation in the recipient site. This initial (primary) stability is the result of the contact relationship or friction that is established following insertion of the implant, between mineralized bone (often the cortical bone) at the recipient site and the metal device.

Implant installation

Tissue injury

Basic rule: the less traumatic the surgical procedure is and the smaller the tissue injury (the damage) becomes in the recipient site during implant installation, the more expeditious is the process through which new bone is formed and laid down on the implant surface.

The various steps used in the implant installation procedure, such as (1) *incision* of the mucosa, often but not always followed by (2) the elevation of *mucosal flaps* and the separation of the periosteum from the cortical plates, (3) the preparation of the *canal* in the cortical and spongy bone of the recipient site, and (4) the insertion of the titanium device (the implant) in this canal, bring to bear a series of mechanical insults and injury to both the mucosa and the bone tissue. The host responds to this injury with an inflammatory reaction, the main objective of which is to eliminate the damaged portions of the tissues and prepare the site for regeneration or repair. To the above described hard tissue injury must be added the effect of the so-called "press fit", i.e. when the inserted implant is slightly wider than the canal prepared in the host bone at the recipient site. In such situations, (1) the mineralized bone tissue in the periphery of the

implant is compressed, (2) the blood vessels particularly in the cortical portion of the canal are collapsed, (3) the nutrition to this portion of the bone compromised, and (4) the affected tissues most often become non-vital.

The damage or injury to the soft and hard tissues of the recipient site initiates the process of wound healing that ultimately ensures that (1) the implant becomes "ankylotic" with the bone, i.e. osseointegrated, and (2) a delicate mucosal attachment (see Chapter 3) is established and a soft tissue seal formed that protects the bone tissue from substances in the oral cavity.

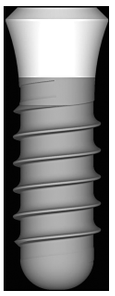

Fig. 5-1 A "non-cutting" implant (solid screw: Straumann® Implant System).

Wound healing

The healing of the severed bone following implant installation is a complex process that apparently involves different events in the cortical and in the spongy (cancellous) compartments of the surgical site.

In the *cortical bone compartment*, the non-vital mineralized tissue must first be removed (resorbed) before new bone can form. In the *spongy compartment* of the recipient site, on the other hand, the surgically inflicted damage (preparation of the canal and the installation of the implant) results mainly in soft tissue (marrow) injury that initially is characterized by localized bleeding and clot (coagulum) formation. The coagulum is gradually resorbed and the compartment thus becomes occupied by proliferating blood vessels and mesenchymal cells; granulation tissue. As result of the continuous migration of mesenchymal cells from the surrounding marrow, the young granulation tissue becomes replaced with provisional connective tissue and eventually with osteoid. In the osteoid, deposition of hydroxyapatite will occur around the newly formed vascular structures. Hereby, immature bone, most often woven bone, is formed (for detail see Chapter 2) and sequentially osseointegration, a direct connection between the newly formed bone and the metal device, takes place.

In summary: in the initial phase of the process that results in osseointegration, the non-vital lamellar bone in the cortical compartment is of importance for the initial fixation of the implant. Osseointegration, however, is often first established in areas occupied by cancellous bone.

Cutting and non-cutting implants

In this chapter only screw-shaped implants made of c.p. titanium will be discussed. The design of the metal device and the installation protocol followed may influence the speed of the process that leads to osseointegration.

"*Non-cutting*" implants (Fig. 5-1) require meticulous handling of the recipient site including the preparation of a standardized track (thread) on the inside

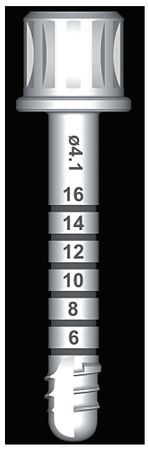

Fig. 5-2 A thread-tap (Straumann® Implant System) that is used to cut a track in the walls of the hard tissue canal. Following this preparation the cavity in the host tissue and the implant are congruent.

of the hard tissue canal. This preparation (precutting) of the track (thread) is made by the use of a thread-tap that is fitted with cutting edges (Fig. 5-2).

Figure 5.1 illustrates a "non-cutting" implant (solid screw, 4.1 mm: Straumann® implant system) that is designed as a cylinder with a rounded "apical" base. The diameter of the cylinder is 3.5 mm. Pilot and twist drills of gradually increasing dimension are used to prepare the hard tissue canal of the recipient site to a final diameter of 3.5 mm. On the surface of the cylinder the implant is designed with a helix-shaped pitch that is 0.3 mm high. The diameter of the entire screw shaped device therefore becomes 4.1 mm.

In sites with a high bone density a thread-tap (Fig. 5-2) is used to cut a 4.1 mm wide helix-shaped track in the walls of the hard tissue canal. The implant and the cavity prepared in the hard tissues of the recipient site are now congruent. When the implant is installed, the pitch on the device will capture and follow the helix-shaped track on the walls of the hard tissue canal and hereby guide the implant with a minimum of force into the pre-prepared position (Fig. 5-3).

Fig. 5-3 Ground section with a "non-cutting" implant and surrounding tissues obtained from a biopsy performed 24 hours after implant installation.

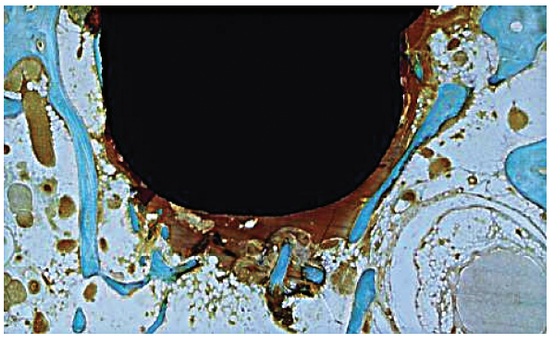

Fig. 5-4 Detail from the apical region of the implant described in Fig. 5-3. Note the presence of a coagulum in the bone marrow.

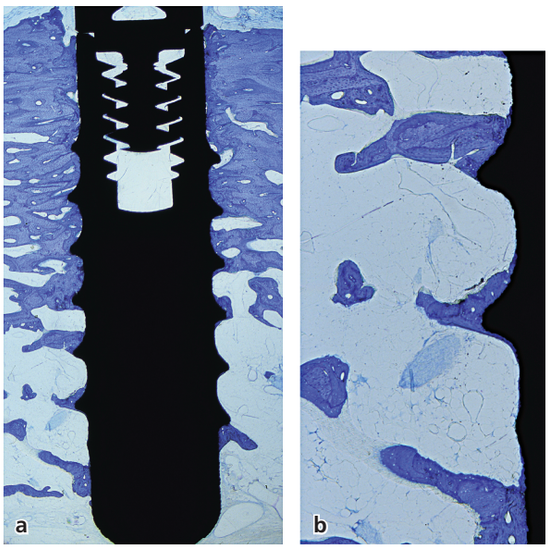

Fig. 5-5 (a) Ground section illustrating a "non-cutting" implant and surrounding bone after 16 weeks of healing. In the cortical portion of the recipient site, the bone density is high. (b) Detail of (a). In more apical areas a thin coat of bone is present on the implant surface. Note also the presence of trabeculae of lamellar bone that extend from the implant into the bone marrow.

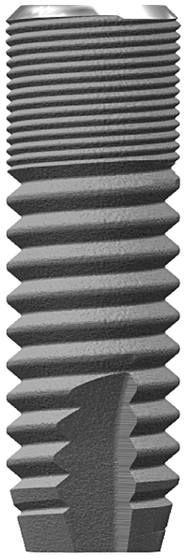

Fig. 5-6 A "cutting" implant (Astra Tech® Implant System). Note the presence of cutting edges in the "apical" portion of the implant. During insertion this implant will cut a 0.3 mm wide chip from the lateral border of the canal prepared in the recipient site.

Figure 5-3 illustrates a "non-cutting" Straumann® solid screw with surrounding tissues in a biopsy sampled 24 hours after implant installation. The implant had proper initial fixation (stability) obtained by the large contact area that was achieved between the metal screw and the buccal and lingual bone walls in the cortical compartment of the recipient site. During site preparation and placement of the implant, bone trabeculae in the spongy compartment of the site were obviously dislocated into the bone marrow. Blood vessels in the marrow compartment were severed, bleeding provoked and a coagulum formed (Fig. 5-4).

After 16 weeks of healing (Fig. 5-5) the marginal portions of the "non-cutting" implant are surrounded by dense lamellar bone that is in direct contact with the rough surface of the metal device. Also in the apical portion of the implant, a thin coat of mature bone can be seen to contact the implant surface and to separate the titanium screw from the bone marrow.

Cutting or self-tapping implants (e.g. Astra Tech® implants, diameter 4.0 mm) (Fig. 5-6) are designed with cutting edges placed in the "apical" portion of the screw-shaped device. The threads of the screw are prepared during manufacturing by cutting a continuous groove into the body of the titanium

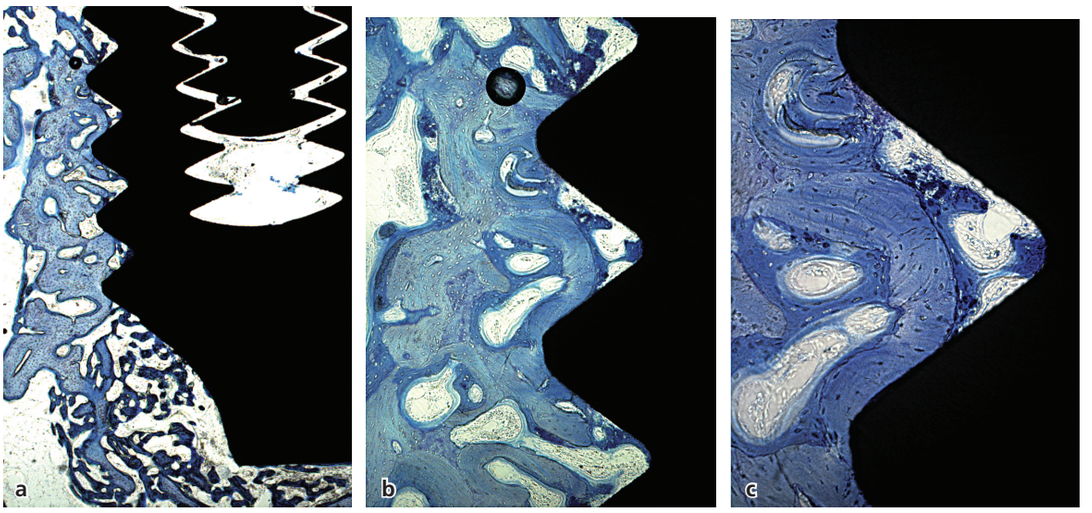

Fig. 5-7 (a) Ground section of an implant (Astra Tech®) site from a biopsy sampled after 2 weeks of healing. In the apical area large amounts of woven bone has formed. (b) Detail of (a). In the threaded region, newly formed bone can be seen to reach contact with the implant surface. (c) Higher magnification of (b). Newly formed bone extends from the old bone and reaches the titanium surface in the invagination between two consecutive "threads".

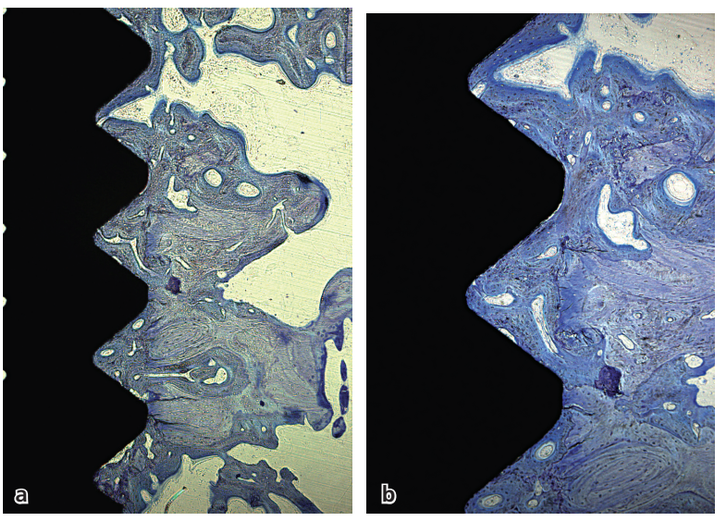

Fig. 5-8 Ground section of an implant site (Astra Tech® self-tapping implant) from a biopsy specimen obtained after 6 weeks of healing. (a) In the marginal area a continuous layer of bone covers most of the TiOblast® surface. (b) Higher magnification. Note the zone of newly formed (darker stained) bone that is in direct contact with the implant surface.

cylinder. When a self-tapping 4.0 mm wide implant is to be placed, the recipient site is first prepared with pilot and twist drills to establish a hard tissue canal that most often has a final diameter of 3.7 mm. During the insertion the cutting edges in the "apical" portion of the implant create a 0.15 mm wide track in the walls of the canal and thereby establish the final 4.0 mm dimension. When the implant has reached its insertion depth, contact has been established between the outer portions of the threads and the mineralized bone in the cortical compartment (initial or primary fixation is hereby secured) and with the severed bone marrow tissue in the spongy compartment.

Figure 5-7 illustrates a recipient site with a self-tapping implant (Astra Tech® implant). This implant is designed with a TiOblast® surface modification. The biopsy was harvested 2 weeks after installation surgery. The outer portion of the thread is in contact with the parent "old" bone, while bone formation is the dominant feature in the invaginations between the threads and in areas lateral to the "apical" portions of the implant. Thus, discrete areas of newly formed bone can be seen also in direct contact with the implant surface. In sections representing 6 weeks of healing (Fig. 5-8), it was observed that a continuous layer of newly formed bone covers most of the TiOblast® surface. This newly formed bone is also in contact with the old, mature bone that is present in the periphery of the recipient site. After 16 months of healing (Fig. 5-9), the bone tissue in the zone of osseointegration has remodeled and the entire hard tissue bed for the implant is comprised of lamellar bone including both concentric and interstitial lamella.

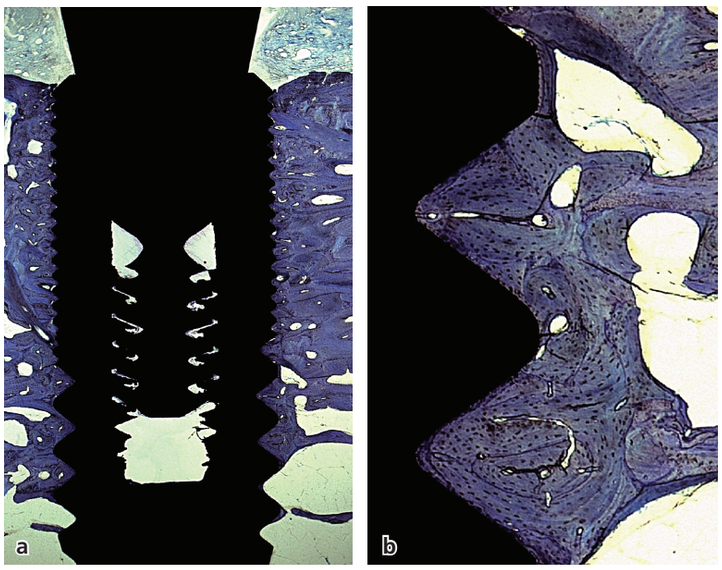

Fig. 5-9 Ground section of an implant site representing 16 months of healing. (a) The implant is surrounded by dense lamellar bone. (b) Higher magnification.

Fig. 5-10 The device used in the dog experiment. The implant is a modification of a solid screw (Straumann® Implant System). The distance between two consecutive threads is 1.25 mm. The depth of the trough is 0.4 mm.

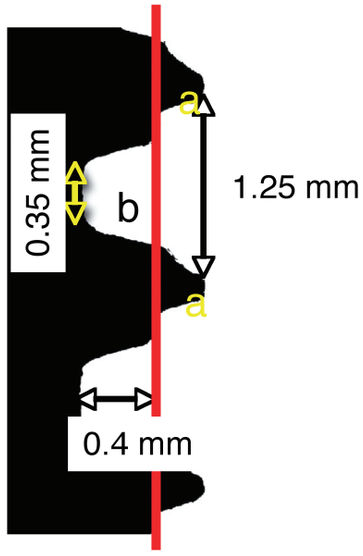

Fig. 5-11 The device. Schematic drawing illustrating the dimensions of the "wound chamber".

The process of osseointegration

De novo bone formation in the severed alveolar ridge following implant placement was studied in experiments in various experimental animal models (for review see Schroeder *et al*. 1995).

Recently Berglundh *et al*. (2003) and Abrahamsson *et al*. (2004) described various steps involved in bone formation and osseointegration to implants placed in the mandible of dogs.

The device: Custom-made implants that had the shape of a solid screw (Straumann® implant), that were made of c.p. titanium and configured with a rough surface topography (SLA®; Straumann) were utilized (Fig. 5-10). In the implant device the distance between two consecutive profiles of the pitch (i.e. the threads in a vertical cross section) was 1.25 mm. A 0.4 mm deep U-shaped circumferential trough had been prepared within the thread region during manufacturing (Fig. 5-11). The tip of the pitch was left untouched. Following the installation of the non-cutting device (Fig. 5-12) the pitch was engaged in the hard tissue walls prepared by the cutting tapping device. This provided intitial or primary fixation of the device. The void between the pitch and the body of the implant established a geometrically well defined wound chamber (Fig. 5-13). Biopsies were performed to provide healing periods extending from 2 hours following implant insertion to 12 weeks of healing. The biopsy specimens were prepared for ground sectioning as well as for decalcification and embedding in epon.

The wound chamber: Figure 5.13 illustrates a cross section (ground section) of an implant with surrounding soft and hard tissues from a biopsy specimen sampled 2 hours after installation of the metal device. The peripheral portions of the pitch were in

contact with the invaginations of the track prepared by the tap in the cortical bone. The wound chambers (Fig. 5-14a) were occupied with a blood clot in which erythrocytes, neutrophils, and monocytes/macrophages occurred in a network of fibrin (Fig. 5-14b). The leukocytes were apparently engaged in the wound cleansing process.

Fibroplasia: Figure 5-15a illustrates a device with surrounding tissues after 4 days of healing. The coagulum had in part been replaced with granulation tissue that contained numerous mesenchymal cells, matrix components, and newly formed vascular structures (angiogenesis) (Fig. 5-15b). A *provisional connective tissue* had been established.

Bone modeling: After 1 week of healing the wound chambers were occupied by a provisional connective tissue that was rich in vascular structures and contained numerous mesenchymal cells (Fig 5-16a). The number of remaining inflammatory cells was relatively small. In large compartments of the chamber, a cell-rich immature bone (woven bone) was seen in the mesenchymal tissues that surrounded the blood vessels. Such areas of woven bone formation occurred in the center of the chamber as well as in discrete locations that apparently were in direct contact with the surface of the titanium device (Fig. 5-16b). This

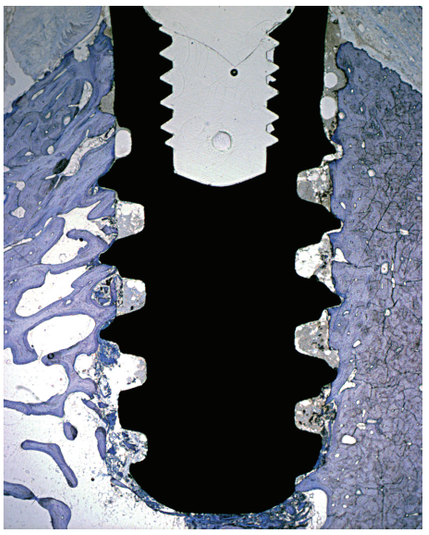

Fig. 5-12 Ground section showing the implant and adjacent tissues immediately after implant installation. The pitch region is engaged in the hard tissue walls. The void between two consecutive pitch profiles includes the wound chamber.

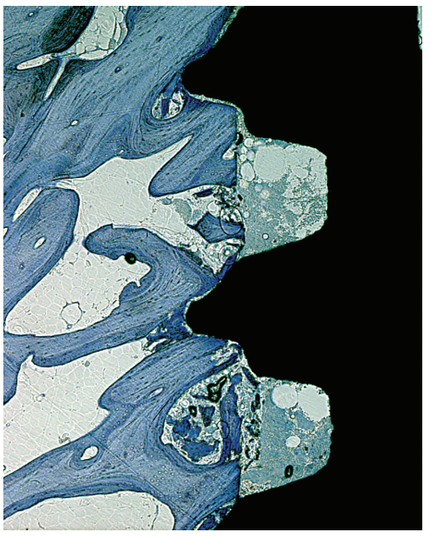

Fig. 5-13 Detail of Fig. 5-12. The wound chamber was filled with blood and a coagulum has formed.

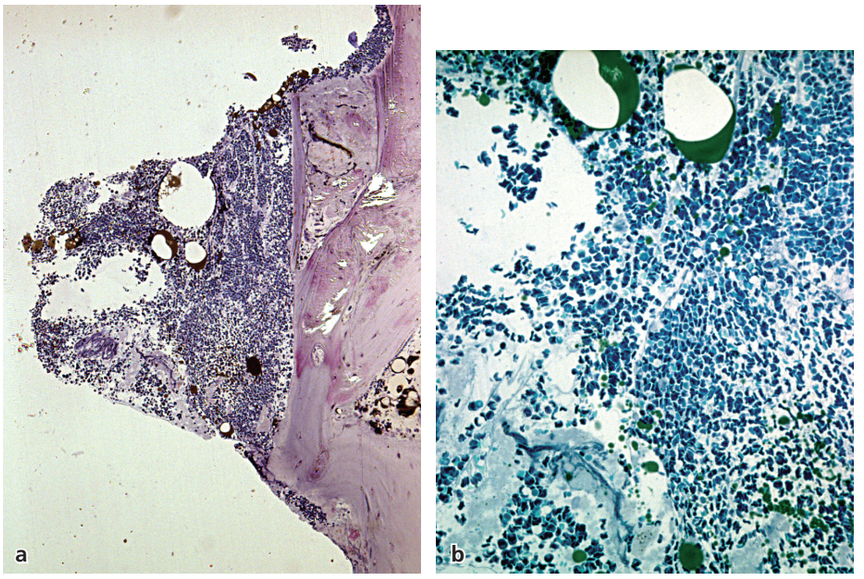

Fig. 5-14 The wound chamber 2 hours after implant installation. Decalcified sections. (a) The wound chamber is filled with blood. (b) Erythrocytes, neutrophils, and macrophages are trapped in a fibrin network.

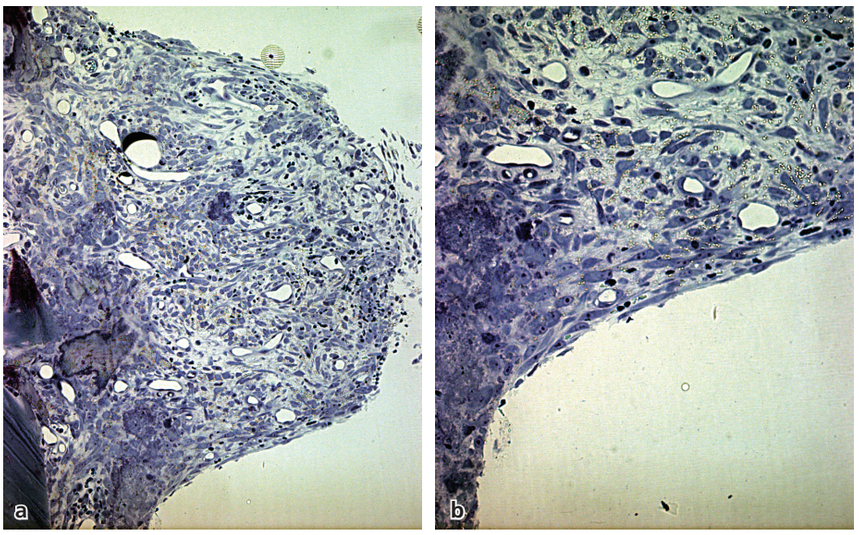

Fig. 5-15 The wound chamber after 4 days of healing (decalcified sctions). (a) Most portions of the wound chamber are occupied by granulation tissue (fibroplasia). (b) In some areas of the chamber provisional connective tissue (matrix) is present. This tissue includes large numbers of mesenchymal cells.

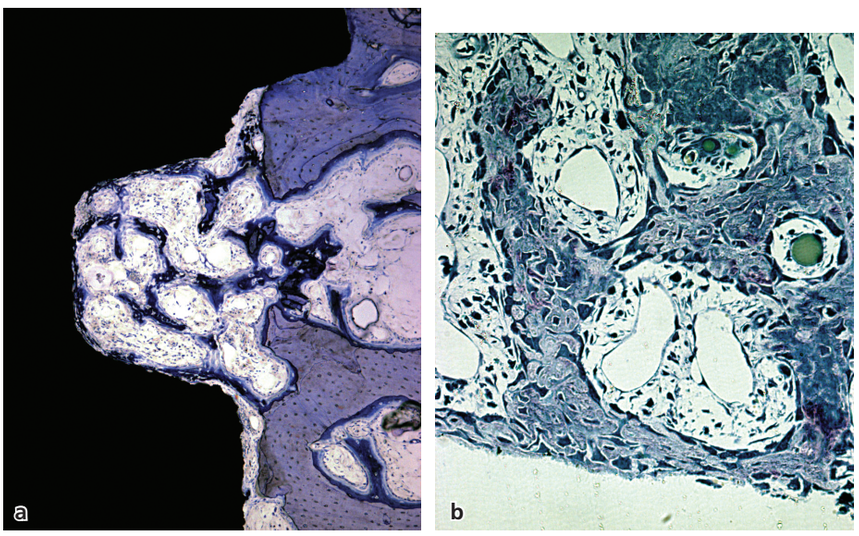

Fig. 5-16 (a) Ground section representing 1 week of healing. Note the presence of newly formed woven bone in the wound chamber. (b) Decalcified section. The woven bone is in direct contact with the implant surface.

was considered to represent the very first phase of osseointegration; contact between the implant surface and newly formed woven bone.

After 2 weeks of healing, woven bone formation appeared to be pronounced in all compartments, apical as well as lateral, surrounding the implant (Fig. 5-17a). Large areas of woven bone were found in the bone marrow regions "apical" of the implant. In the wound chamber, portions of the newly formed woven bone apparently extended from the parent bone into the provisional connective tissue (Fig. 5-17b) and had in many regions reached the surface of the titanium device. At this interval most of the implant surface was occupied by newly formed bone and a more comprehensive and mature osseointegration had been established (Fig. 5-17c). In the pitch regions there were signs of ongoing new bone formation (Fig. 5-17d). Thus, areas of the recipient site located lateral to the device, that were in direct contact with the host bone immediately following installation surgery and provided initial fixation for the implant, had undergone tissue resorption and were also involved in new bone formation after 2 weeks of healing.

At 4 weeks (Fig. 5-18a), the newly formed mineralized bone extended from the cut bone surface into the chamber and a continuous layer of cell-rich, woven bone covered most of the titanium wall of the

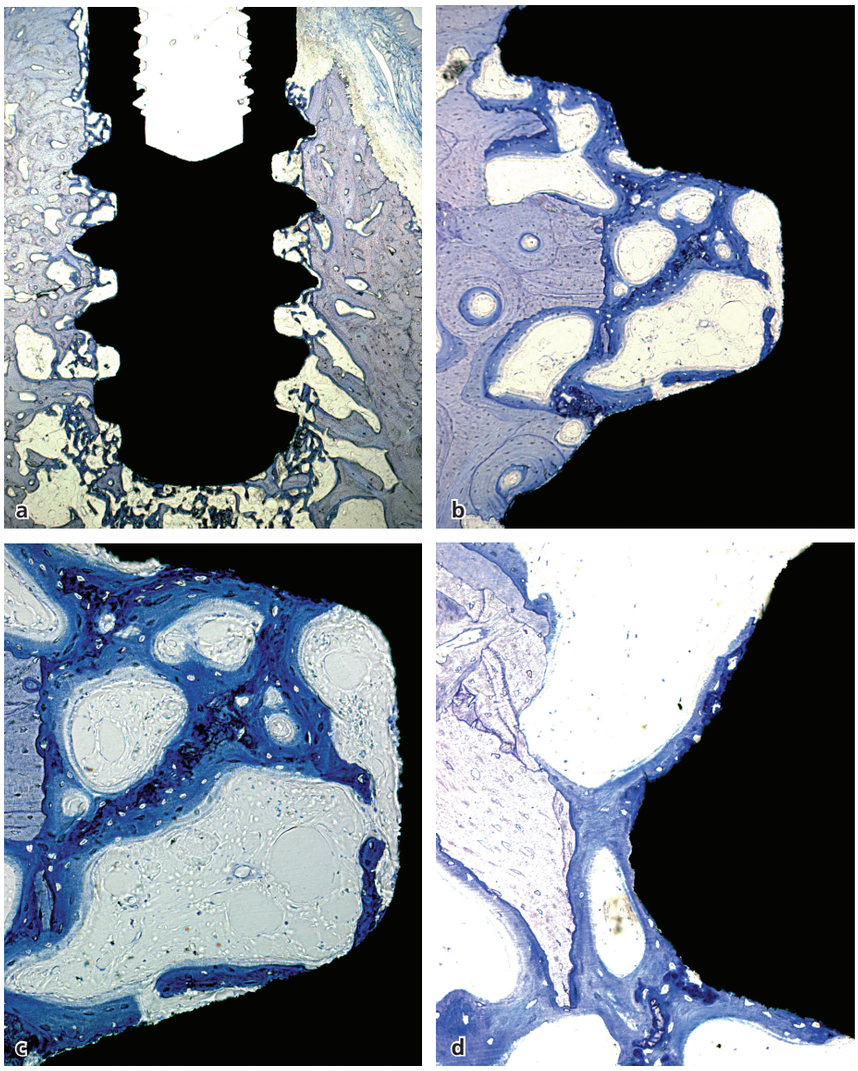

Fig. 5-17 Ground sections illustrating, in various magnifications, the tissues in the wound chamber after 2 weeks of healing. (a) Darker stained woven bone is observed in the apical area of the metal device. (b, c, d) Most portions of the implant surface are coated with bone.

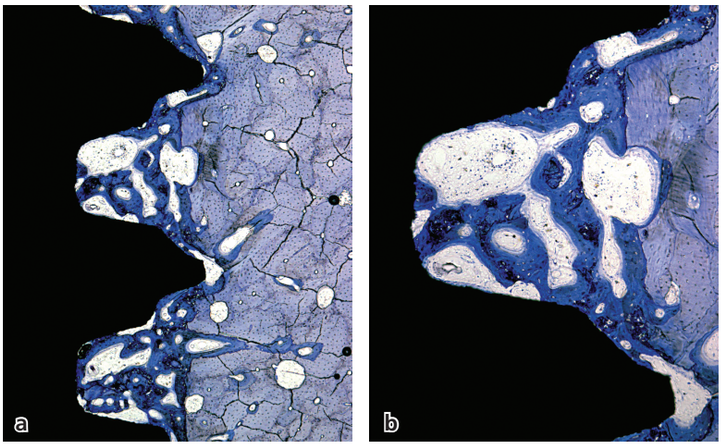

Fig. 5-18 Ground sections representing 4 weeks of healing. (a) The newly formed bone (dark blue) extends from the "old" bone into the wound chamber. (b) Appositional growth. Note the presence of primary osteons.

chamber. The central portion of the chamber was filled with a primary spongiosa (Fig. 5-18b), rich in vascular structures and a multitude of mesenchymal cells.

Remodeling: After 6–12 weeks of healing most of the wound chambers were filled with mineralized bone (Fig. 5-19). Bone tissue, including primary and secondary osteons, could be seen in the newly formed tissue and in the mineralized bone that made contact with the implant surface. Bone marrow that contained blood vessels, adipocytes, and mesenchymal cells was observed to surround the trabeculae of mineralized bone.

Summary: The wound chambers were first occupied with a coagulum. With the ingrowth of vessels and migration of leukocytes and mesenchymal cells, the coagulum was replaced with granulation tissue. The migration of mesenchymal cells continued and the granulation tissue was replaced with a provisional matrix, rich in vessels, mesenchymal cells, and fibers. The process of *fibroplasia* and angiogenesis had started. Formations of newly formed bone could be recognized already during the first week of healing; the newly formed woven bone projected from the lateral wall of the cut bony bed (appositional bone formation; distance osteogenesis) (Davies 1998) but *de novo* formation of new bone could also be seen on the implant surface, i.e. at a distance from the parent bone (contact osteogenesis) (Davies 1998). During subsequent weeks the trabeculae of woven bone were replaced with mature bone, i.e. lamellar bone and marrow (bone remodeling).

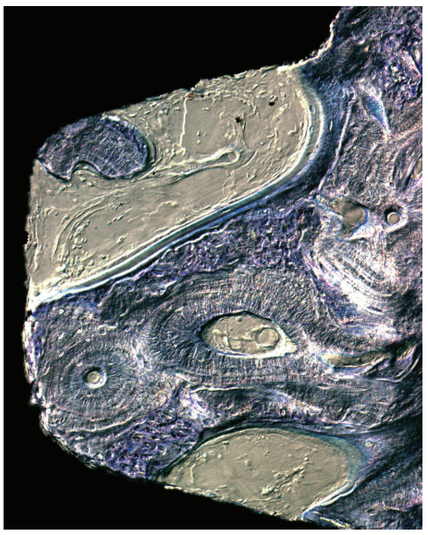

Fig. 5-19 Ground section representing 12 weeks of healing. The woven bone is being replaced with lamellar bone and marrow. Note the formation of secondary osteons.

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Chapter 18

Chronic Periodontitis

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Chronic periodontitis is considered to start as *plaque-induced gingivitis* (see Chapter 17), a reversible condition that, left untreated, may develop into *chronic periodontitis*. Chronic periodontitis lesions include loss of attachment and bone and are regarded as irreversible. In this chapter, various aspects of chronic periodontitis will be described, including its links to plaque-induced gingivitis.

Clinical features of chronic periodontitis

The clinical features of chronic periodontitis include symptoms such as (1) color, texture and volume alterations of the marginal gingiva, (2) bleeding on probing (BoP) from the gingival pocket area, (3) reduced resistance of the soft marginal tissues to probing (increased pocket depth or periodontal pocketing), (4) loss of probing attachment level, (5) recession of the gingival margin, (6) loss of alveolar bone (even or angular pattern), (7) root furcation exposure, (8) increased tooth mobility, (9) drifting and eventually exfoliation of teeth.

Figure 18-1 illustrates the clinical status of a 30-year-old male with severe chronic periodontitis. The clinical examination revealed that (1) most approximal and lingual/palatal sites exhibited BoP, (2) most teeth showed increased mobility, and (3) gingival recession had occurred at a large number of buccal and interproximal sites. Tooth 16 had erupted beyond the occlusal plane. Teeth 37 and 38 had tilted mesially. The altered position of the molars had evidently compromised the occlusion. Forces elicited during function may have caused the maxillary incisors to tilt in a buccal direction and multiple open spaces, diastemata, had developed in the front tooth segment of the maxilla.

Figure 18-2 presents the radiographic status of the same patient. In the radiographs it can be observed that a large number of teeth have lost substantial amounts of bone support. At teeth 17, 16, 27, 37, 36, and 47 the furcation areas have lost their periodontal tissue support and are open for "through and through" probing.

Overall characteristics of chronic periodontitis

- Chronic periodontitis is prevalent in adults but may occur in children.
- The amount of destruction of the periodontal tissues seen in a given patient is commensurate with oral hygiene and plaque levels, local predisposing factors, smoking, stress, and systemic risk factors.
- The subgingival biofilm harbors a variety of bacterial species; the composition of the biofilm may vary between subjects and sites.
- Subgingival calculus is invariably present at diseased sites.
- Chronic periodontitis is classified as localized when <30% of sites are affected and generalized when this level is exceeded.
- Severity of chronic periodontitis at the site level may be classified based on the degree of probing attachment loss (PAL) as *mild* (PAL = 1–2 mm), *moderate* (PAL = 3–4 mm), and *severe* (PAL ≥ 5 mm).
- Although chronic periodontitis is initiated and sustained by microbial plaque, host factors deter-

Fig. 18-1 A 30-year-old male patient with chronic periodontitis, clinical status prior to treatment.

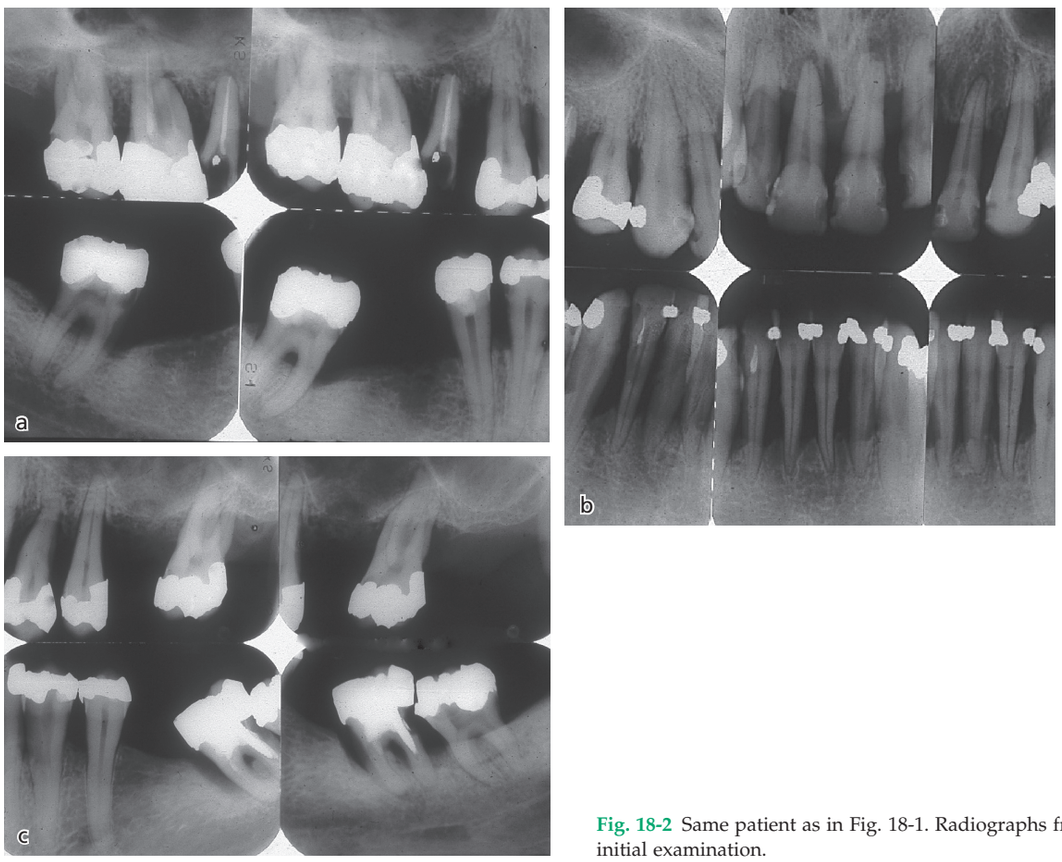

Fig. 18-2 Same patient as in Fig. 18-1. Radiographs from the initial examination.

mine the pathogenesis and (rate of) progression of the disease.
- The rate of progression of chronic periodontitis is in most cases slow to moderate; periods of rapid tissue destruction may, however, occur.
- Additional periodontal tissue breakdown is likely to occur in diseased sites that are left untreated.

Gingivitis as a risk for chronic periodontitis

Findings from epidemiologic studies (cross-sectional as well as longitudinal) indicate that gingival inflammation is invariably a component of chronic periodontitis and that gingivitis precedes the onset of periodontitis (see Chapter 11). The interpretation of data from early cross-sectional studies led to the belief that untreated gingivitis always progressed to chronic periodontitis. More recent studies have demonstrated, however, that this is not the case. Gingivitis lesions may remain stable for many years, and may never progress to become periodontitis lesions that include features such as attachment and bone loss. The two conditions have been considered, therefore, as separate disease entities with the explanation being that the bacterial plaque challenge will induce overt gingivitis but that the degree of response of the host (the susceptibility) will determine whether or not chronic periodontitis will develop. In a review paper, Kinane and Attström (2005) evaluated epidemiologic and experimental data on gingivitis and chronic periodontitis. The independence of these two conditions was called into question. It was proposed that gingivitis and periodontitis most likely represented different aspects of the same disease, namely chronic periodontitis.

Gingivitis becomes manifest after only days or weeks of plaque accumulation (Löe *et al*. 1965) while destructive chronic periodontitis is a condition that in the majority of cases requires far longer periods (years) of plaque and calculus exposure to develop (Lindhe *et al*. 1975; Löe *et al*. 1978). The proportion of untreated gingival lesions in a given subject or in a population that converts to destructive periodontitis lesions is at present unknown. Furthermore, the factors that cause the conversion are not well understood (Schätzle *et al*. 2003).

Findings from epidemiologic studies and prospective clinical trials have indicated that the presence of gingivitis may be regarded as a risk factor for chronic periodontitis. In a 2-year longitudinal study of 15–24-year-old Chinese adolescents from a rural district, it was observed that the percentage of sites that bled on probing at a baseline examination was related to overall attachment loss after 2 years of monitoring (Suda *et al*. 2000). This suggests that gingival inflammation was a risk indicator for additional attachment loss in this cohort. The role of gingivitis in the pathogenesis of chronic periodontitis was further elucidated by Schätzle *et al*. (2004) in longitudinal studies on the initiation and progression of periodontal disease in a Norwegian population. The results demonstrated that gingival sites, which during a 20-year interval never showed signs of inflammation, experienced modest loss of attachment (1.86 mm). For sites which presented with mild inflammation at each examination, the corresponding attachment loss was 2.25 mm, while at sites with severe gingival inflammation, the mean loss of attachment was 3.23 mm. Moreover, while teeth surrounded with healthy gingival tissues were maintained during the study period, teeth with gingivitis lesions were 46 times more likely to be lost.

The above data indicate that gingival inflammation may represent a relevant risk factor not only for destructive chronic periodontitis but also for tooth loss. This conclusion is in agreement with results documenting the absence of gingivitis as a good indicator for long-term maintenance of periodontal health in a subject (Joss *et al*. 1994) as well as at a site (Lang *et al*. 1990) level.

Susceptibility to chronic periodontitis

As stated above plaque-induced gingivitis and chronic periodontitis represent different aspects of the same disease (Kinane & Attström 2005). An important question is whether both gingivitis and chronic periodontitis are affected by the same subject response (the host response) to plaque. If this is the case, the corollary is that susceptibility to gingivitis will reflect susceptibility to chronic periodontitis and may have prognostic utility.

Even in the very first reports from studies called "Experimental gingivitis in man" (Löe *et al*. 1965; Theilade *et al*. 1966) (see Chapters 11 and 17), evidence was presented that suggested that the onset and severity of the inflammatory response of the gingiva to plaque accumulation differed markedly among participants. The differences were, however, at that time attributed to differences in plaque accumulation rates (quantitative plaque differences) and/or differences in bacterial species present in plaque (qualitative plaque differences). More recent studies utilizing the "Experimental gingivitis" model have documented that significant differences in the inflammatory response occurred in different subjects although their plaque accumulation was quantitatively and/or qualitatively similar (Trombelli *et al*. 2004, 2005). It was suggested that the intensity of the inflammatory response to the plaque challenge may represent an individual trait (Tatakis & Trombelli 2004). Thus, an individual's susceptibility to gingivitis may be dependent on host-related factors, possibly of genetic origin (Shapira *et al*. 2005; Scapoli *et al*. 2005).

With the use of the "Experimental gingivitis" model it was also demonstrated that the susceptibility to gingivitis differed between two groups of patients consistent with different susceptibilities to

periodontitis (Abbas *et al*. 1986; Winkel *et al*. 1987). Thus, the group with greater periodontitis susceptibility had also a greater susceptibility to gingivitis. Furthermore, in more recent studies it was documented that subjects with a history of aggressive periodontitis presented significantly more gingivitis in response to *de novo* plaque accumulation when compared to periodontally healthy subjects matched for extent and rate of supragingival plaque accumulation (Trombelli *et al*. 2006).

Prevalence of chronic periodontitis

From epidemiologic studies (see Chapter 7) it was concluded that chronic periodontitis is the most commonly occurring form of periodontal disease. While most subjects over 50 years of age have suffered moderate amounts of periodontal tissue destruction, advanced forms of chronic periodontitis are seen in only a small (<10%) subset of the population. Both age of onset of chronic periodontitis and subsequent rate of progression of the disease vary between individuals and are probably influenced by genetics (see Chapter 13) and environmental risk factors (see Chapters 7 and 12). Findings from examination of dizygotic and monozygotic twins (Michalowicz *et al*. 1991, 2000) indicated that (1) between 38% (regarding probing attachment loss) and 82% (regarding gingivitis) of the population variance could be attributed to genetic factors (Michalowicz *et al*. 1991), and (2) chronic periodontitis has about 50% heritability (Michalowicz *et al*. 2000).

On a population basis chronic periodontitis is often classified according to number (prevalence) of diseased sites (extent) and severity of tissue breakdown (probing attachment loss) at such sites. For the extent of chronic periodontitis, the low category would involve one to ten diseased (probing attachment loss) sites, the medium category would involve 11–20 sites, while the high category would involve more than 20 diseased sites. The amount of PAL at a given site may be used to describe the severity of chronic periodontitis. The severity may be considered as mild (PAL = 1–2 mm), moderate (PAL= 3–4 mm) or severe (PAL ≥ 5 mm). It has been documented that the extent and severity of chronic periodontitis are useful predictors of future disease progression.

Clinical (probing) attachment loss of 1–2 mm at one or several sites can be found in nearly all members of an adult population. The prevalence of subjects with one or more sites with PAL ≥ 3 mm increases with age. Furthermore the number of diseased sites in any one individual increases with age, as does the population prevalence (extent and severity) of chronic periodontitis with age.

Progression of chronic periodontitis

Chronic periodontitis is generally a slowly progressing form of periodontal disease that at any stage may undergo exacerbation resulting in additional loss of attachment and bone.

Tissue destruction in chronic periodontitis does not affect all teeth evenly, but has site predilection. In other words, in the same dentition some teeth may be severely affected with periodontal tissue destruction while other teeth are almost free of signs of attachment and bone loss. Figure 18-3 illustrates the clinical condition of a subject with chronic periodontitis. Clinically (Fig. 18-3a) it can be observed that most teeth exhibit advanced recession of the soft tissue margin. In the corresponding radiographs (Fig. 18-3b) it is noted that the mesial surface of tooth 16 has a normal periodontal tissue support, while the neighboring tooth, i.e. the first premolar (tooth 14), has lost several millimeters of bone support on the distal aspect. The mesial surface of tooth 14, on the

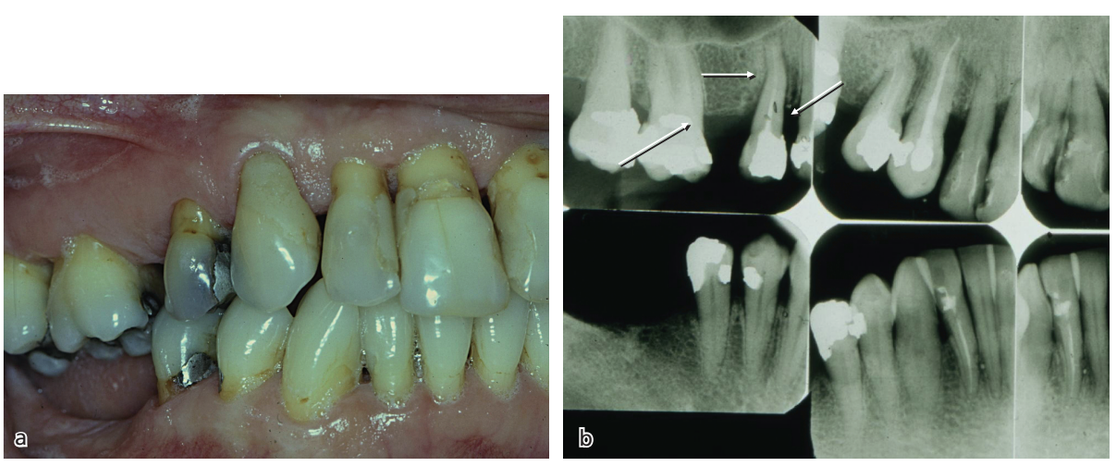

Fig. 18-3 (a) The clinical condition in quadrants 1 and 3 of a subject with chronic periodontitis. Most teeth in both quadrants exhibit advanced recession of the soft tissue margin. (b) Corresponding radiographs: the mesial surface of tooth 16 (arrow) has a normal periodontal tissue support, while the neighboring tooth, tooth 14, has lost several millimeters of bone support on the distal aspect (arrow). The mesial surface of tooth 14 has a comparatively normal tissue support (arrow).

other hand, has a comparatively normal tissue support.

When considering changes in attachment level over time, it is also peculiar that only relatively few sites in a subject with chronic periodontitis undergo marked, additional tissue destruction during any given observation period. Based on data from a series of longitudinal studies, Socransky *et al*. (1984) proposed that chronic periodontitis progressed in episodes of exacerbation and remission. They termed this the "burst hypothesis" of disease progression. Findings from other similar studies, however, indicated that the progression of chronic periodontitis may be a continuous, slowly destructive process rather than exhibiting a "burst" pattern. The current consensus is that the progression of chronic periodontitis in most subjects and at most sites is a continuous process but that periods of exacerbation occasionally may occur. Clinically, the progressive nature of the disease can only be confirmed by repeated examinations over time but it is a safe assumption that untreated lesions of chronic periodontitis will progress and cause additional attachment and bone loss. Flemmig (1999) reported a mean additional PAL of ≥3 mm in up to 27% of subjects in untreated populations during a 1-year period. When progression was studied on a site basis, the overall annualized incidence ranged from 0.3–4.2% (Flemmig 1999). This indicates that the number of sites that actually exhibited progression within a given time varied considerably between subjects.

It is important to realize that factors associated with the initiation of chronic periodontitis may also influence disease progression. Furthermore, the extent and severity of disease within an individual, i.e. number of sites with attachment loss, bone loss, and/or deep pockets, are good predictors of future disease occurrence. In fact the best predictor of disease progression is previous disease experience.

Risk factors for chronic periodontitis

The term "risk factor" means an aspect of lifestyle, an environmental exposure, or an inborn or inherited characteristic, which on the basis of epidemiologic evidence is known to be associated with a given disease. Risk factors may be part of the causal chain of a disease and/or may predispose the host to develop a disease. An individual presenting with one or more risk factors has an increased probability of contracting the disease or of the disease being made worse.

Bacterial plaque

Bacterial aspects of periodontal disease are dealt with in Chapters 8 and 9. From the data presented it is obvious that a cumulative risk for a given microbiota can be estimated. It is not clear, however, if the specific microbiota is the principal disease-causing factor or whether it reflects the disease process. Specific microorganisms have been considered as potential periodontal pathogens but it is clear that, although pathogens are necessary, their mere presence may not be enough for the progressive disease to occur. Microbial plaque (biofilm) is a crucial factor in inflammation of the periodontal tissues, but the progression of gingivitis to periodontitis is largely governed by host-based risk factors (Michalowicz 1994; Shapira *et al*. 2005). Microbial biofilms of particular compositions will initiate chronic periodontitis (Marsh 2005) in certain individuals whose host response and cumulative risk factors predispose them to periodontal tissue destruction and attachment loss.

Age

Although the prevalence of periodontal disease increases with age it is unlikely that becoming older in itself greatly increases susceptibility to periodontal disease. It is more likely that the cumulative effects of disease over a lifetime, i.e. deposits of plaque and calculus, and the increased number of sites capable of harboring such deposits, as well as attachment and bone loss experience, explain the increased prevalence of disease in older people.

Smoking

The association between periodontal disease and smoking is dealt with in detail in Chapter 12. Only a brief discussion of smoking as a risk factor for chronic periodontitis is thus given here. The literature consistently indicates a positive association between smoking and chronic periodontitis across the many cross-sectional and longitudinal studies performed over the years (Kinane & Chestnutt 2000) and the risk attributable to tobacco for chronic periodontitis is between 2.5 and 7.0. It is not only the risk of developing the disease that is enhanced by smoking, but also the response to periodontal therapy is impaired in smokers. A further feature in smokers is that their signs and symptoms of both gingivitis and chronic periodontitis, mainly gingival redness and bleeding on probing (BOP), are masked by the dampening of inflammation seen for smokers as compared to non-smokers.

Systemic disease

It is difficult to determine the precise role any systemic disease may play in the pathogenesis of chronic periodontitis. There are several reasons for this. Firstly, in epidemiologic studies attempting to evaluate the effect of systemic disease, control groups should be carefully matched in respect of age, sex, oral hygiene, and socioeconomic status. Many studies, particularly before the etiologic importance of dental plaque was recognized, failed to include such controls. Secondly, because of the chronic nature of periodontal disease, longitudinal studies spanning several years are preferable in individuals both with

and without systemic disease. Unfortunately, most of the available data are derived from cross-sectional studies (Kinane 1999).

A reduction in number or function of polymorphonuclear leukocytes (PMNs) generally results in increased rate and severity of periodontal tissue destruction (Wilton *et al*. 1988). Many drugs, such as phenytoin, nifedipine, and cyclosporine, predispose to gingival overgrowth in response to plaque and thus may modify pre-existing chronic periodontitis (Ellis *et al*. 1999). Changes in circulating hormone levels may increase severity of plaque-induced gingival inflammation but typically do not result in any increased susceptibility to periodontitis. Hormonal changes following menopause have been associated with osteoporosis but studies are lacking to link this disease or an estrogen deficient state to a higher susceptibility to periodontal disease. Immunosuppressive drug therapy and any disease resulting in suppression of inflammatory and immune processes (such as HIV infection) may predispose the individual to exaggerated periodontal tissue destruction (Barr *et al*. 1992).

Nutritional deficiencies in animals have been shown to affect the periodontal tissues. Epidemiologic data do not support the suggestion that such deficiencies play an important role in chronic periodontal disease although nutritional influences on inflammation are now accepted and are now actively being researched (Ritchie & Kinane 2005). Gingival bleeding is the most consistent oral feature of vitamin C deficiency or scurvy but there is also some evidence to suggest that avitaminosis-C may aggravate established chronic periodontitis.

The periodontal features of *histiocytosis X* and other conditions in the rare histiocytoses disease group may present as necrotizing ulcerative periodontitis (Kinane 1999). Diabetes appears to be one of the most fascinating systemic diseases that interacts with periodontitis. On the one hand periodontitis severity and prevalence are increased in subjects with long-duration diabetes and more so in poorly controlled diabetics, than non-diabetics. On the other hand, periodontitis may also exacerbate diabetes as it may decrease glycemic control (Thorstenson 1995).

Despite the paucity of high quality data on individuals both with and without systemic disease the following general conclusions can be drawn (Kinane 1999):

- The *blood cells* have a vital role in supplying oxygen, hemostasis and protection to the tissues of the periodontium. Systemic hematological disorders can thus have profound effects on the periodontium by denying any of these functions necessary for the integrity of the periodontium.
- The *polymorphonuclear leukocyte* (PMN cell) is undoubtedly crucial to the defense of the periodontium. To exert this protective function several activities of PMNs must be integrated, namely chemotaxis, phagocytosis, and killing or neutralization of the ingested organism or substance. Individuals with either quantitative (neutropenia) or qualitative (chemotactic or phagocytic) PMN deficiencies, exhibit severe destruction of the periodontal tissues, which is strong evidence that PMNs are an important component of the host's protective response to the subgingival biofilm. Quantitative deficiencies are generally accompanied by destruction of the periodontium of all teeth, whereas qualitative defects are often associated with localized destruction affecting only the periodontium of certain teeth (i.e. chronic periodontitis may be modified).
- *Leukemias* which give excessive numbers of leukocytes in the blood and tissues also cause a greatly depleted bone marrow function with concomitant anemia, thrombocytopenia, neutropenia, and reduced range of specific immune cells which give some characteristic periodontal features: anemic gingival pallor; gingival bleeding; gingival ulceration. Leukemic features are further complicated by the potential for the proliferating leukocytes to infiltrate the gingiva and result in gingival enlargement. In broad terms leukemias result in gingival pathologies, whereas periodontal bone loss is the consequence of neutrophil functional defects or deficient numbers and other severe functional defects such as deficiency of leukocyte adhesion receptors.
- *Diabetes mellitus:* there are numerous confounding variables which must be considered in determining the true relationship between periodontitis and diabetes. The current consensus is that diabetics are at increased risk of periodontal disease, and whilst periodontitis can be successfully treated, both disease susceptibility and the outcome of therapy are influenced by poor metabolic control. Thus, it may be of benefit to the dentist to have knowledge of the control status of diabetes in an individual patient, as in the longer term metabolic control could indicate the probable outcome of periodontal therapy. In addition, it is now accepted that periodontal therapy can improve metabolic control in diabetics, meaning that the relationship is two-way and periodontal therapy is beneficial to the control of both diseases.
- *Medications* such as phenytoin, cyclosporine, and nifedipine may predispose to gingival overgrowth in patients with gingivitis.
- *Genetic traits*, which result in diseases that modify the periodontal structures or change the immune or inflammatory responses, can result in gross periodontal destruction in the affected individual; although the destruction seen may imitate periodontitis, this is not etiopathologically chronic periodontitis.

Stress

Stressful life events and negative emotions have been shown to modulate several physiologic systems,

including the endocrine and the immune system, leading to health changes (Kiecolt-Glaser *et al*. 2002; LeResche & Dworkin 2002). The association between stress and disease is particularly strong for infectious diseases, inflammatory conditions, and impaired wound healing (Kiecolt-Glaser *et al*. 2002; LeResche & Dworkin 2002; Broadbent *et al*. 2003). Specific periodontal conditions have been associated with psychosocial variables, including chronic periodontitis (Green *et al*. 1986; Linden *et al*. 1996; Genco *et al*. 1999; Wimmer *et al*. 2002; Pistorius *et al*. 2002), necrotizing ulcerative gingivitis (Shields 1977; Cohen-Cole *et al*. 1983; Horning & Cohen 1995), chronic and experimental gingivitis (Minneman *et al*. 1995; Deinzer *et al*. 1998; Waschul *et al*. 2003). In adults, the reported contribution of psychosocial factors to enhanced gingivitis expression (Deinzer *et al*. 1998) may relate to the stress-associated increase in plaque accumulation (Deinzer *et al*. 2001). However, the possible association of other psychosocial variables, such as personality traits and coping behavior, which are associated with either susceptibility or resistance to stress, with changes in the inflammatory response of the gingiva to *de novo* plaque accumulation, remains uncertain (Trombelli *et al*. 2005).

Most of the literature on stress and periodontal conditions is quite old, and reports of acute necrotizing ulcerative gingivitis (or trench mouth) were made on stressed soldiers on the front line during World War I. It is understood that stress may be immunosuppressive and that acute necrotizing ulcerative gingivitis may occur in the immunosuppressed (also in HIV patients), but there is insufficient data as yet to substantiate the assumption that psychosocial factors are indeed of etiologic importance in chronic periodontitis.

Genetics

There is convincing evidence from twin studies for a genetic predisposition to the periodontal diseases. The twin studies have indicated that risk of chronic periodontitis has a high inherited component. A great deal of research is underway attempting to identify the genes and polymorphisms associated with all forms of periodontitis. It is likely that chronic periodontitis involves many genes, the composition of which may vary across individuals and races. Much attention has focused on polymorphisms associated with the genes involved in cytokine production (Shapira *et al*. 2005). Such polymorphisms have been linked to an increased risk for chronic periodontitis but these findings have yet to be corroborated (Kinane & Hart 2003; Kinane *et al*. 2005).

Scientific basis for treatment of chronic periodontitis

Chronic periodontitis is initiated and sustained by microorganisms living in biofilm communities which are present in supra- and subgingival plaque in the form of uncalcified and calcified biofilms. Prevention of initiation or primary prevention of periodontitis is clearly related to preventing formation and/or eradication of the microbial biofilm and it follows that prevention of gingivitis is a primary preventive measure for chronic periodontitis. Initial periodontal therapy or basic treatment of periodontitis involves the removal of both sub- and supragingival plaque. The clinical outcome is largely dependent on the skill of the operator in removing subgingival plaque and the skill and motivation of the patient in practicing adequate home care. A further variable is the innate susceptibility of the patient which is related to the way in which their innate, inflammatory, and immune systems operate in response to the microbial challenge. In addition, local and systemic risk factors can influence the quantity and quality of both the microbial challenge and the host response to these pathogens. The relative contribution of these risk factors has yet to be fully determined but their influence would be limited if the periodontium were kept free of microbial plaque. Thus, sub- and supragingival debridement and the quality of the patient's home care are of vital importance in preventing inflammation that manifests as both gingivitis and periodontitis.

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Chapter 31

Treatment Planning of Patients with Periodontal Diseases

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Caries and periodontal diseases represent opportunistic infections associated with biofilm formation on the surfaces of teeth. Factors such as bacterial specificity and pathogenicity as well as the disposition of the individual for disease, e.g. local and general resistance, may influence the onset, the rate of progression, and clinical characteristics of plaque-associated dental disorders. Findings from animal experiments and longitudinal studies in humans, however, have demonstrated that treatment, including the elimination or the control of the biofilm infection and the introduction of careful plaque control measures, in most, if not all, cases results in dental and periodontal health. Even if health cannot always be achieved and maintained, the arrest of disease progression following treatment must be the goal of modern dental care.

The treatment of patients affected by caries and periodontal disease, including symptoms of associated pathologic conditions such as pulpitis, periapical periodontitis, marginal abscesses, tooth migration, etc., may from a didactic point of view be divided into four different phases:

1. Systemic phase of therapy including smoking counseling
2. Initial (or hygiene) phase of periodontal therapy, i.e. cause-related therapy
3. Corrective phase of therapy, i.e. additional measures such as periodontal surgery, and/or endodontic therapy, implant surgery, restorative, orthodontic and/or prosthetic treatment
4. Maintenance phase (care), i.e. supportive periodontal therapy (SPT).

Treatment goals

In every patient diagnosed with periodontitis, a treatment strategy, including the elimination of the opportunistic infection, must be defined and followed. This treatment strategy must also define the clinical outcome parameters to be reached through therapy. Such clinical parameters include:

- Reduction or resolution of gingivitis (bleeding on probing; BoP). A patient full mouth mean BoP ≤25% should be reached.
- Reduction in probing pocket depth (PPD). No residual pockets with PPD >5 mm should be present.
- Elimination of (through-and-through) open furcations in multi-rooted teeth. Initial furcation involvement should not exceed 3 mm.
- Absence of pain.
- Individually satisfactory esthetics and function.

In this context it must be emphasized that risk factors for periodontitis that can be controlled must be addressed as well. The three main risk factors for chronic periodontitis are (1) improper plaque control, (2) cigarette smoking, and (3) uncontrolled diabetes mellitus (Kinane *et al.* 2006).

Systemic phase

The goal of this phase is to eliminate or decrease the influence of systemic conditions on the outcomes of therapy and to protect the patient and the dental care

providers against infectious hazards. Contact with a physician or specialist should enable appropriate preventive measures to be taken, if necessary. Efforts must be undertaken to stimulate a smoker to enroll in a smoking cessation program. Additional aspects are discussed in Chapter 33.

Initial (hygiene) phase

This phase represents the cause-related therapy. The objective of this phase is the achievement of clean and infection-free conditions in the oral cavity through complete removal of all soft and hard deposits and their retentive factors. Furthermore, this phase should aim at motivating the patient to perform optimal plaque control. The initial phase of periodontal therapy is concluded by re-evaluation and planning of both additional and supportive therapies.

Corrective phase (additional therapeutic measures)

This phase addresses the sequelae of the oportunistic infections and includes therapeutic measures, such as periodontal and implant surgery, endodontic therapy, restorative and/or prosthetic treatment. The amount of corrective therapy required and the selection of the type of restorative and prosthetic therapy can be determined only when the degree of success of the cause-related therapy can be properly evaluated. The patient's willingness and ability to cooperate in the overall therapy must determine the type of corrective treatment. If this cooperation is inadequate, it may not be worth initiating treatment procedures: permanent improvement of oral health, function and esthetics may therefore not be achieved. The validity of this statement can be exemplified by the results of studies aimed at assessing the relative value of different types of surgical methods in the treatment of periodontal disease. A number of clinical trials (Lindhe & Nyman 1975; Nyman *et al.* 1975, 1977; Rosling *et al.* 1976a,b; Nyman & Lindhe 1979) have demonstrated that gingivectomy and flap procedures performed in patients with proper plaque control levels often result in gain of alveolar bone and clinical attachment, while surgery in plaque-contaminated dentitions may cause additional destruction of the periodontium.

Maintenance phase (supportive periodontal therapy)

The aim of this treatment is the prevention of reinfection and disease recurrence. For each individual patient a recall system must be designed that includes (1) assessment of deepened sites with bleeding on probing, (2) instrumentation of such sites, and (3) fluoride application for the prevention of dental caries. In addition, this treatment involves the regular control of prosthetic restorations incorporated during the corrective phase of therapy. Tooth sensitivity testing should be applied to abutment teeth as loss of vitality is a frequently encountered complication (Bergenholtz & Nyman 1984; Lang *et al.* 2004; Lulic *et al.* 2007). Based upon the individual caries activity, bitewing radiographs should be incorporated into SPT at regular intervals.

Screening for periodontal disease

A patient seeking dental care is usually screened for the presence of carious lesions by means of clinical and radiographic tools. Likewise, it is imperative that such a patient is screened for the presence of periodontitis as well, using a procedure termed the basic periodontal examination (BPE) (or periodontal screening record; PSR).

Basic periodontal examination

The goal of the BPE is to screen the periodontal conditions of a new patient and to facilitate treatment planning. BPE scoring will allow the therapist to identify:

- A patient with reasonably healthy periodontal conditions, but in need of long-term preventive measures
- A patient with periodontitis and in need of periodontal therapy.

In the BPE the screening of each tooth or implant is evaluated. For this purpose, the use of a thin graduated periodontal probe is recommended. At least two sites per tooth/implant (i.e. mesio-buccal and disto-buccal) should be probed using a light force (i.e. 0.2 N). Each dentate sextant within the dentition is given a BPE code or score, whereby the *highest* individual site score is used.

BPE system code

- Code 0 = probing pocket depth (PPD) ≤3 mm, BoP negative, no calculus or overhanging fillings (Fig. 31-1a)
- Code 1 = PPD ≤3 mm, BoP positive, no calculus or overhanging fillings (Fig. 31-1b)
- Code 2 = PPD ≤3 mm, BoP positive, presence of supra- and/or subgingival calculus and/or overhanging fillings (Fig. 31-1c)
- Code 3 = PPD >3 mm but ≤5 mm, BoP positive (Fig. 31-1d)
- Code 4 = PPD >5 mm (Fig. 31-1e).

If an examiner identifies a single site with a PPD >5 mm within a sextant, the sextant will receive a code of 4, and no further assessments are needed in this particular sextant. Patients with sextants given codes of 0, 1 or 2 belong to the relatively periodontally healthy category. A patient exhibiting a sextant with codes of 3 or 4 must undergo a more comprehensive periodontal examination (for details see Chapter 26).

Fig. 31-1 Clinical illustration of the basic periodontal examination scores. (a) BPE code 0. (b) BPE code 1. (c) BPE code 2. (d) BPE code 3. (e) BPE code 4.

Fig. 31-2 (a–d) Clinical status of a 27-year-old female patient (S.B.) diagnosed with generalized aggressive periodontitis with furcation involvement.

The aim of the following text is to explain the overall objectives of the treatment planning of patients with BPE codes of 3 and 4 undergoing a comprehensive diagnostic process.

Diagnosis

The basis for the treatment planning described in this chapter is established by the clinical data collected from the patient's examination (see Chapter 26). This patient (Ms. S.B., 27 years of age) was systemically healthy and a non-smoker. She was examined with respect to her periodontal conditions, i.e. gingival sites displaying signs of *bleeding on probing* were identified, *probing pocket depths* were measured, the *periodontal attachment level* was calculated, *furcation involvements* were graded, *tooth mobility* was assessed, and the radiographs were analyzed to determine the *height* and *outline* of the *alveolar bone crest*.

The clinical characteristics of the dentition of this patient are shown in Fig. 31-2. The periodontal chart

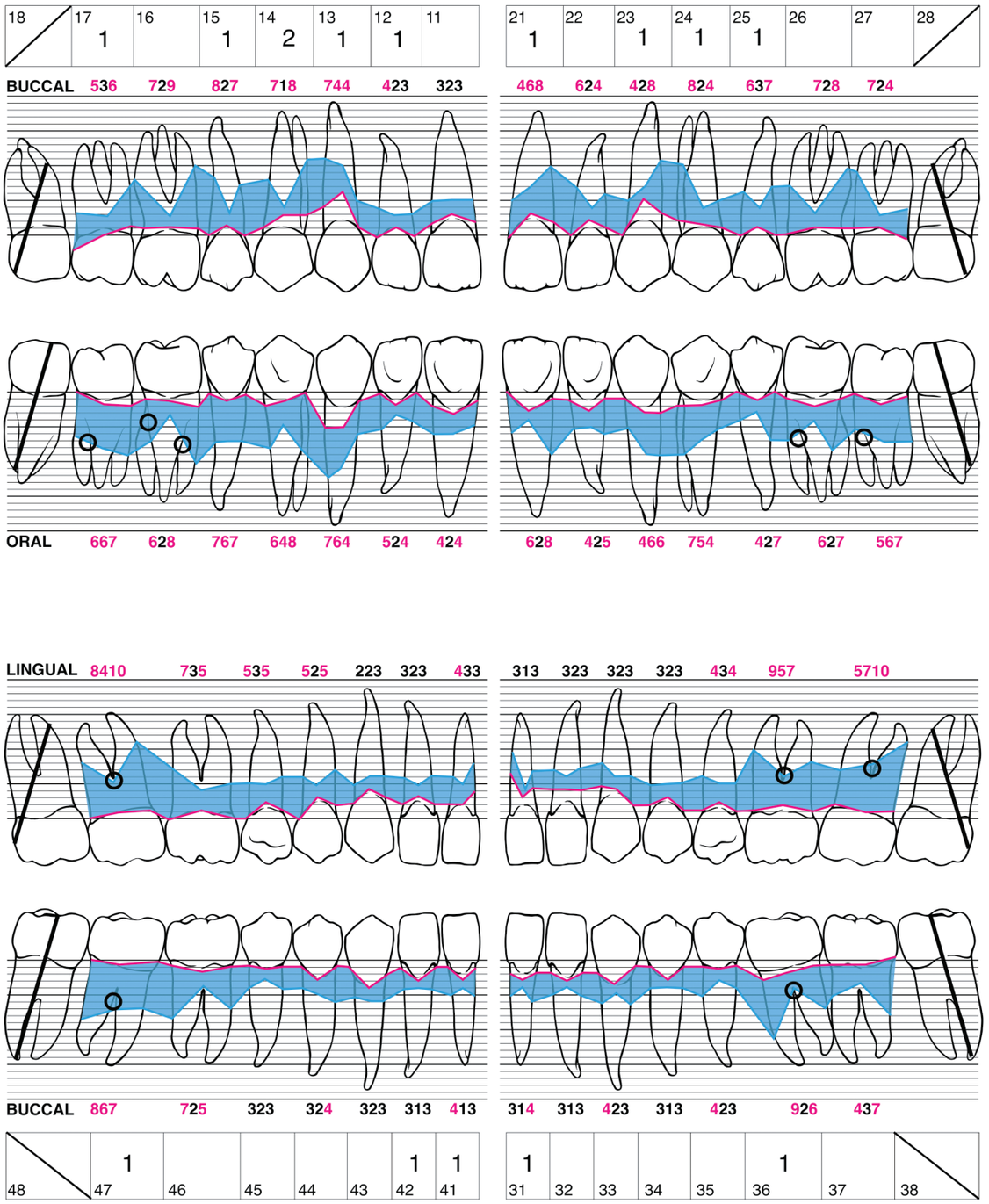

Fig. 31-3 Periodontal chart of the patient presented in Fig. 31-2.

and the radiographs are presented in Figs. 31-3 and 31-4, respectively. Based on these findings, each tooth in the dentition was given a diagnosis (Fig. 31-5) and a pre-therapeutic prognosis (Fig. 31-6). In addition to the examination of the periodontal condition, detailed assessments of primary and recurrent caries were made for all tooth surfaces in the dentition. Furthermore, the patient was examined with respect to endodontic and occlusal problems as well as temporomandibular joint dysfunction.

Treatment planning

Initial treatment plan

Provided that the patient's examination has been completed (see Chapter 26) and a diagnosis of all pathologic conditions has been made, an initial treatment plan can been established. At this early stage in the management of a patient, it is, in most instances,

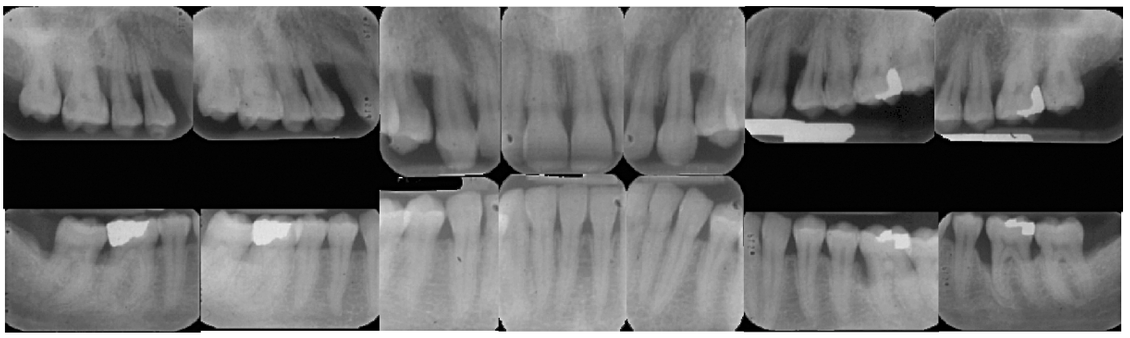

Fig. 31-4 Radiographs of the patient presented in Fig. 31-2.

	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
Gingivitis																
Parodontitis superficialis							X	X								
Parodontitis profunda		X	X	X	X	X			X	X	X	X	X	X	X	
Parodontitis interradicularis		X	X											X	X	
Parodontitis interradicularis		X	X											X	X	
Parodontitis profunda		X	X											X	X	
Parodontitis superficialis				X	X			X	X		X		X			
Gingivitis						X	X			X		X				
	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Fig. 31-5 Single tooth diagnosis of the patient presented in Fig. 31-2.

	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
Irrational to treat																
Doubtful (unsecure)			X	X	X	X					X	X		X	X	
Good (secure)							X	X	X	X			X			
Good (secure)				X	X	X	X	X	X	X	X	X	X			
Doubtful (unsecure)		X	X											X	X	
Irrational to treat																
	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Fig. 31-6 Pre-therapeutic single tooth prognosis of the patient presented in Fig. 31-2.

impossible to make definite decisions regarding all aspects of the treatment sequence, because:

1. *The degree of success of initial therapy is unknown*. The re-evaluation after initial, cause-related therapy forms the basis for the selection of means for additional therapy. The degree of disease elimination that can be reached depends on the outcome of subgingival instrumentation, but also on the patient's ability and willingness to exercise proper plaque control and to adopt adequate dietary habits.
2. *The patient's "subjective" need for additional (periodontal and/or restorative) therapy is unknown*. When the dentist has completed the examination of the patient and an inventory has been made regarding periodontal disease, caries, pulpal disease, and temporomandibular joint disorders, the observations are presented to the patient (i.e. "the case presentation"). During the case presentation

session it is important to find out if the patient's subjective need for dental therapy coincides with the dentist's professional appreciation of the type and amount of therapy that is required. It is important that the dentist understands that the main objective of dental therapy, besides *elimination of pain*, is *to satisfy the patient's demands regarding chewing function (comfort) and esthetics*, demands that certainly vary considerably from one individual to another.

3. *The result of some treatment steps cannot be predicted.* In patients exhibiting advanced forms of caries and periodontal disease it is often impossible to anticipate whether or not all teeth that are present at the initial examination can be successfully treated, or to predict the result of certain parts of the intended therapy. In other words, critical and difficult parts of the treatment must be performed first, and the outcome of this treatment must be evaluated before all aspects of the definitive corrective treatment can be properly anticipated and described.

Pre-therapeutic single tooth prognosis

Based on the results of the comprehensive examination, including assessments of periodontitis, caries, tooth sensitivity, and the resulting diagnosis, as well as considering the patient's needs regarding esthetics and function, a pre-therapeutic prognosis for each individual tooth (root) is made. Three major questions are addressed:

1. Which tooth/root has a "*good*" (secure) prognosis?
2. Which tooth/root is "*irrational-to-treat*"?
3. Which tooth/root has a "*doubtful*" (unsecure) prognosis?

Teeth with a *good* prognosis will require relatively simple therapy and may be regarded as secure abutments for function.

Teeth that are considered "irrational-to-treat" should be extracted during initial, cause-related therapy. Such teeth may be identified on the basis of the following criteria:

- Periodontal:
  - Recurrent periodontal abscesses
  - Combined periodontal–endodontic lesions
  - Attachment loss to the apex
- Endodontal:
  - Root perforation in the apical half of the root
- Dental:
  - Vertical fracture of the root
  - Oblique fracture in the middle third of the root
  - Caries lesions that extend into the root canal
- Functional:
  - Third molars without antagonists and with periodontitis/caries.

Teeth with a *doubtful* prognosis are usually in need of comprehensive therapy and must be brought into the category of teeth with a *good* prognosis by means of additional therapy. Such teeth may be identified on the basis of the following criteria:

- Periodontal:
  - Furcation involvement
  - Angular (i.e. vertical) bony defects
  - "Horizontal" bone loss involving more than two thirds of the root
- Endodontal:
  - Incomplete root canal therapy
  - Periapical pathology
  - Presence of voluminous posts/screws
- Dental:
  - Extensive root caries.

Case presentation

The "case presentation" is an essential component of the initial treatment plan and must include a description for the patient of different therapeutic goals and the modalities by which these may be reached. At the case presentation for Ms. S.B. the following treatment plan was described:

- The teeth in the dentition from 12 to 22 and from 45 to 35 will probably not confront the dentist with any major therapeutic challenges. For the remaining teeth in the dentition, however, the treatment plan may involve several additional measures.

Expected benefits inherent to a certain treatment plan versus obvious disadvantages should always be explained to and discussed with the patient. His/her attitude to the alternatives presented must guide the dentist in the design of the overall treatment plan.

Based on the pre-therapeutic single tooth prognosis (Fig. 31-6), the following detailed treatment plan was presented to the patient.

Systemic phase

Owing to the fact that the patient was systemically healthy and a non-smoker, no medical examination and smoking cessation counseling were required.

Initial phase (cause-related therapy)

The treatment was initiated and included the following measures to eliminate or control the bacterial infection:

1. *Motivation* of the patient and *instruction* in oral hygiene measures with subsequent check-ups and re-instruction
2. *Scaling and root planing* under local anesthesia in combination with removal of plaque-retentive factors

Fig. 31-7 (a–c) Clinical front and lateral views of the patient presented in Fig. 31-2 at re-evaluation after initial periodontal therapy.

3. *Excavation and restoration* of carious lesions (16 and 26)
4. *Endodontic treatment* of tooth 46.

Re-evaluation after initial phase

The initial phase of therapy is completed with a thorough analysis of the results obtained with respect to the elimination or degree of control of the dental infections. This implies that a re-evaluation of the patient's periodontal conditions and caries activity must be performed. The results of this re-evaluation (Figs. 31-7 and 31-8) form the basis for the selection, if necessary, of additional corrective measures to be performed in the phase of definitive treatment (i.e. corrective phase). In order to provide time for the tissues to heal, the re-evaluation should be performed no earlier than 6–8 weeks following the last session of instrumentation.

Planning of the corrective phase (i.e. additional definitive therapy)

If the results from the re-evaluation, made 6–8 weeks after the termination of the initial treatment phase, show that periodontal disease and caries have been brought under control, the additional treatment may be carried out. The main goal of this phase is to correct the sequelae caused by oral infections (i.e. periodontal disease and caries). The following procedures may be performed:

- *Additional endodontic treatment with/without post-and-core build-ups*
- *Periodontal surgery*. The type (i.e. open-flap debridement, regenerative or resective surgery) and extent of surgical treatment should be based on probing depth measurements, degree of furcation involvement and BoP score assessed at re-evaluation. Periodontal surgery is often confined to those areas of the dentition where the inflammatory lesions were not resolved by root instrumentation and in areas with angular bony defects or in furcation-involved molars.
- *Installation of oral implants*. In regions of the dentition where tooth abutments are missing, implant therapy for esthetic and functional reasons may be considered. It is essential to realize that implant therapy must be initiated once all dental infections are under control, i.e. after successful periodontal therapy.
- *Definitive restorative and prosthetic treatment* including fixed or removable dental prostheses.

Corrective phase (additional therapy)

After initial therapy the patient (Ms. S.B.) exhibited low plaque and gingivitis scores (i.e. 5–10%) and no active carious lesions. The corrective phase therefore included the following components:

1. *Periodontal surgery* (i.e. open-flap debridement) in the maxillary left and right quadrants as well as in the mandibular molar regions (Fig. 31-9)
2. *Guided tissue regeneration* (GTR) for tooth 36
3. *Re-evaluation* after periodontal surgery (Figs. 31-10 and 31-11)

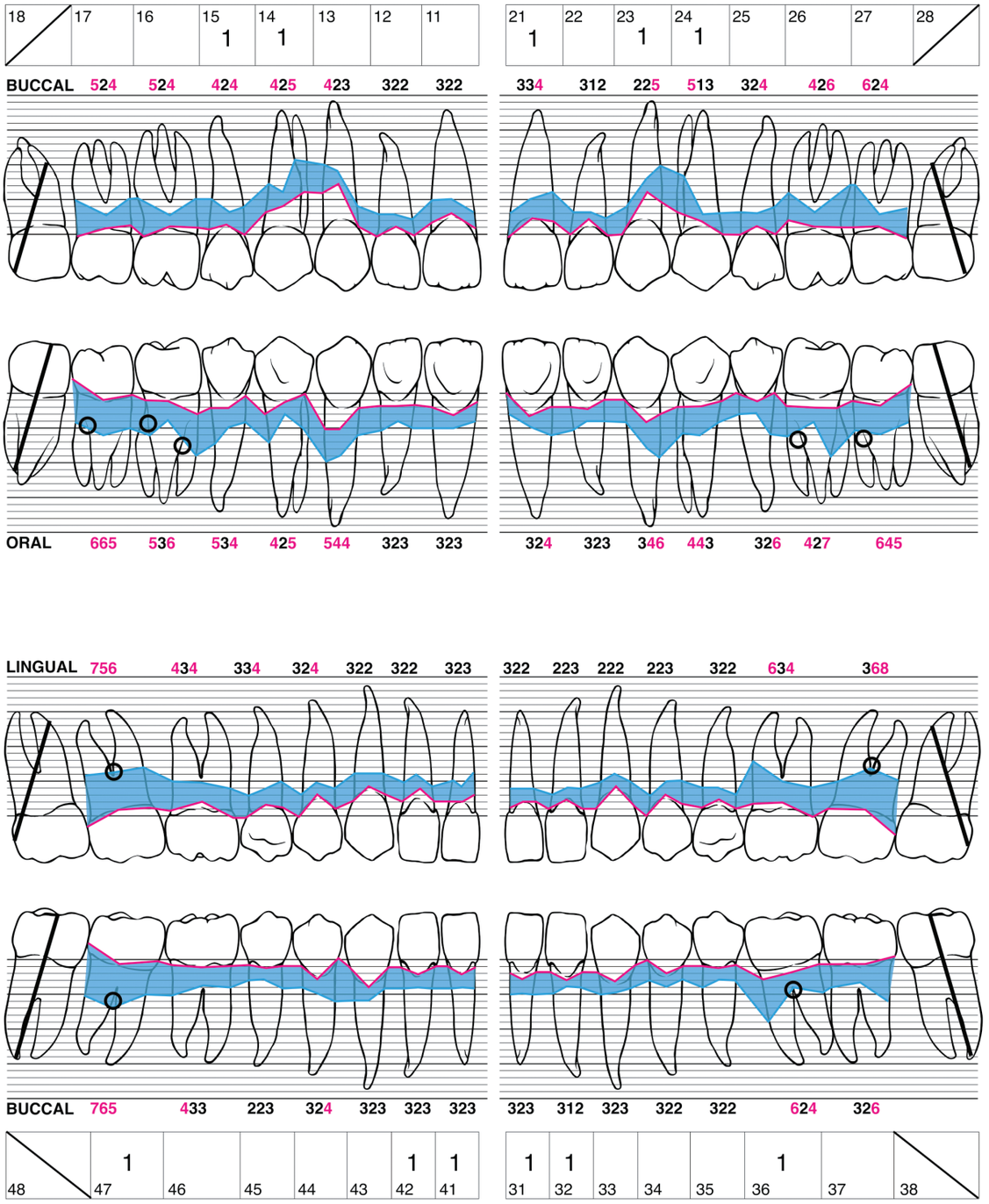

Fig. 31-8 Periodontal chart of the patient presented in Fig. 31-2 at re-evaluation after initial periodontal therapy.

4. *Orthodontic therapy* in the maxillary front area (Fig. 31-12)
5. *Restorative therapy* in the maxillary front area for esthetic reasons (Fig. 31-13).

Re-evaluation after corrective phase

The corrective phase of therapy is completed with a thorough analysis of the results obtained with respect to the elimination of the sequelae of periodontal tissue destruction (Figs. 31-14, 31-15 and 31-16). This implies that a re-evaluation of the patient's periodontal and peri-implant conditions must be performed. The results of this re-evaluation form the basis for the assessment of the residual periodontal risk. The outcomes of the periodontal risk assessment (PRA), in turn, will determine the recall frequency of the patient during maintenance phase.

Maintenance phase (care)

Following completion of cause-related therapy, the patient must be enrolled in a recall system aiming at

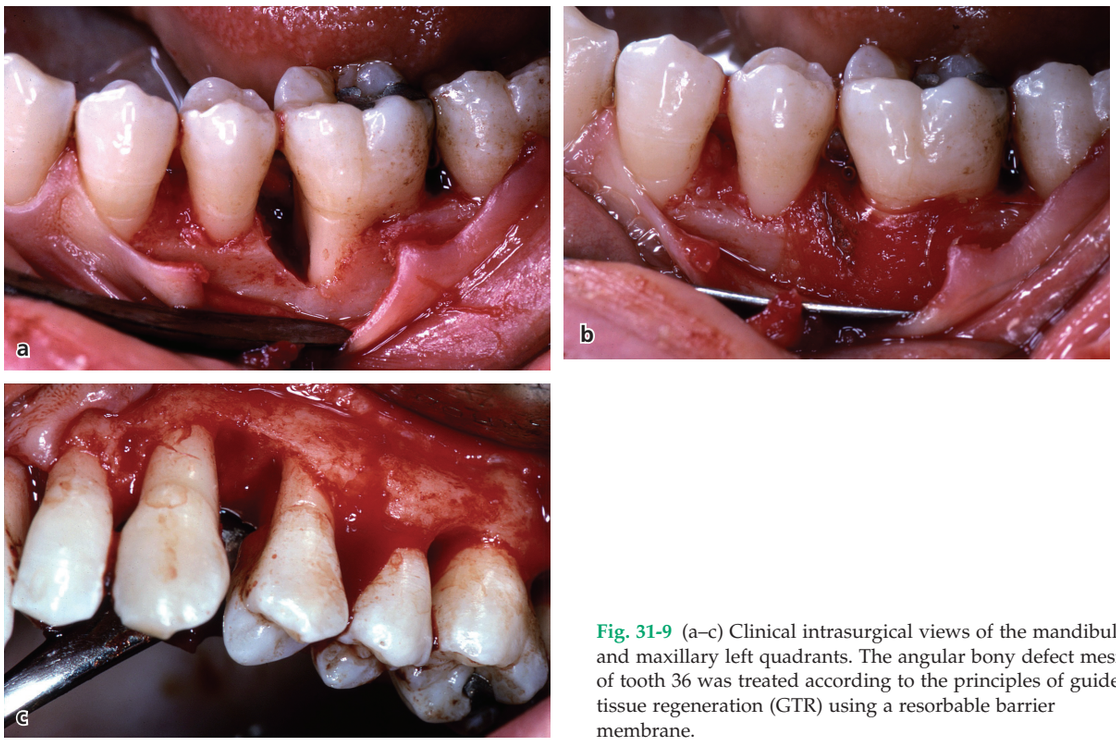

Fig. 31-9 (a–c) Clinical intrasurgical views of the mandibular and maxillary left quadrants. The angular bony defect mesial of tooth 36 was treated according to the principles of guided tissue regeneration (GTR) using a resorbable barrier membrane.

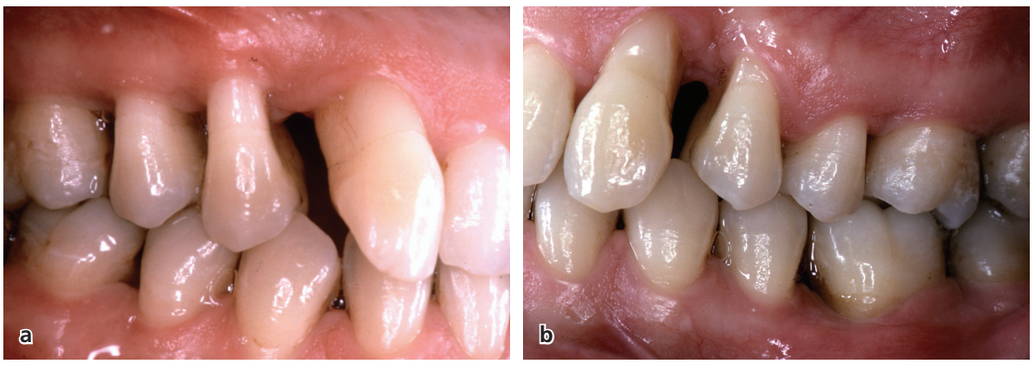

Fig. 31-10 (a,b) Clinical lateral views of the patient presented in Fig. 31-2 at re-evaluation after periodontal surgery.

preventing the recurrence of oral infections (i.e. periodontitis, caries, and peri-implantitis). Supportive periodontal therapy (SPT) should be scheduled at the re-evaluation after initial therapy and independently of the need for additional therapy. The time interval between the recall appointments should be based on a periodontal risk assessment established at the re-evaluation after the corrective phase. It is well established that self-performed plaque control combined with regular maintenance care visits following active periodontal treatment represents an effective means of controlling gingivitis and periodontitis and limiting tooth mortality over a 30-year period (Axelsson *et al.* 2004). It is important to emphasize, however, that the recall program must be designed to meet the individual needs of the patient. According to a PRA performed after completion of active therapy, some patients should be recalled every 3 months, while others may have to be checked only once a year (Lang & Tonetti 2003).

At the various recall visits the following procedures should be carried out:

1. Update of the medical and smoking history
2. Soft tissue examination as cancer screening
3. Recording of the full-mouth PPD ≥5 mm with concomitant BoP
4. Re-instrumentation of bleeding sites with PPD ≥5 mm
5. Polishing and fluoridation for the prevention of dental caries.

The patient (Ms. S.B.), presented to describe the guiding principles of treatment planning, was recalled twice during the first 6 months after active treatment (i.e. every 3 months) and subsequently only once every 6 months based on the individual PRA.

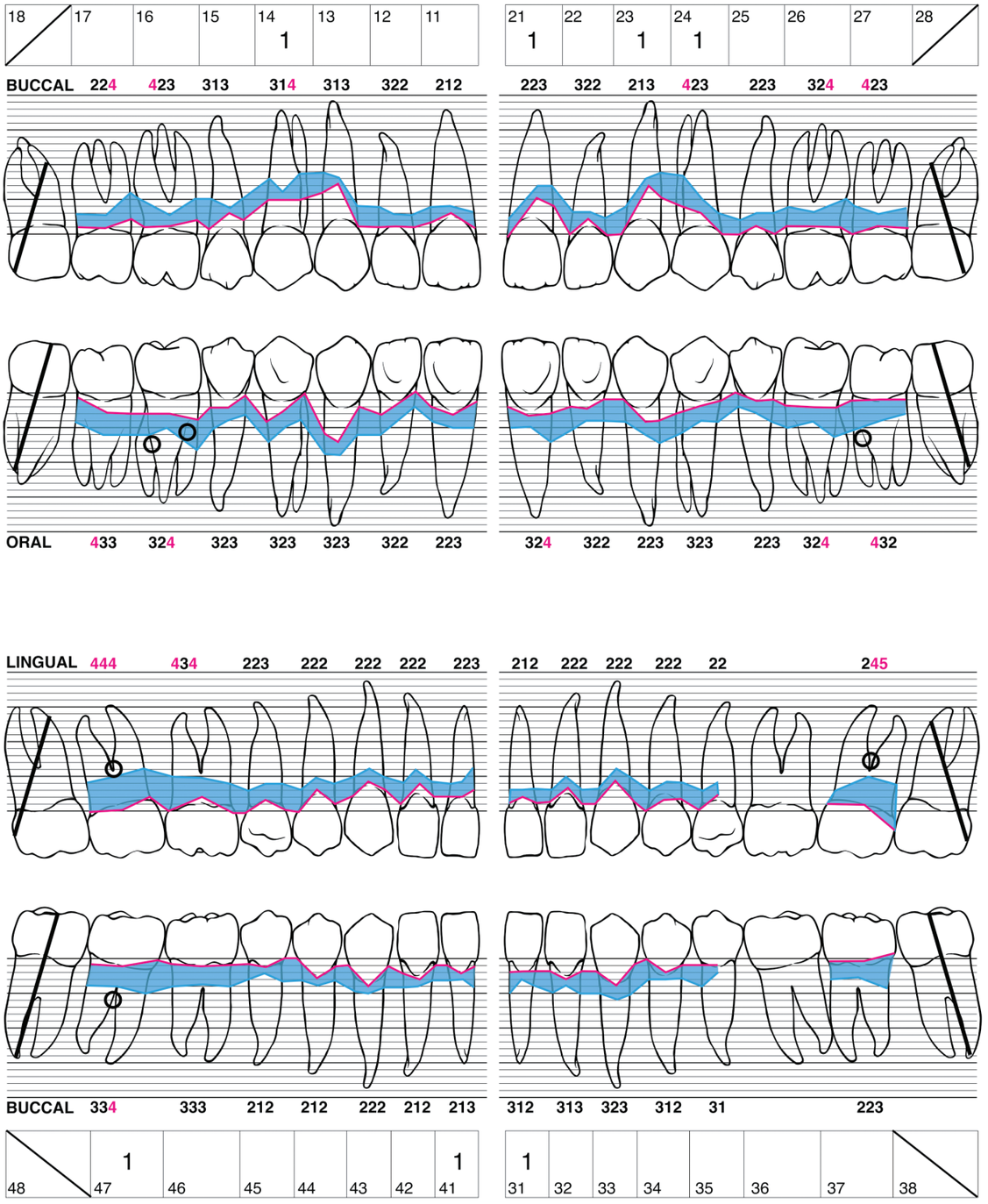

Fig. 31-11 Periodontal chart of the patient presented in Fig. 31-2 at re-evaluation after periodontal surgery.

Concluding remarks

The overall treatment plan and the sequence of the different treatment procedures used in this case were selected for presentation in order to illustrate the following principle: *in patients exhibiting a generalized advanced breakdown of the periodontal tissues, but with an intact number of teeth, considerable efforts should be made to maintain all teeth*. Extraction of a single tooth in such a dentition will frequently also call for the extraction of several others for "prosthetic reasons". The end result of such an approach thus includes a prosthetic rehabilitation that, if the treatment planning had been properly done, would have been unnecessary.

The large variety of treatment problems that different patients present may obviously require deviations from the sequence of treatment phases (i.e. systemic phase, initial cause-related therapy, corrective therapy, and maintenance care) discussed above.

Fig. 31-12 (a–c) Clinical front and lateral views of the patient presented in Fig. 31-2 during orthodontic therapy of the maxillary front teeth.

Fig. 31-13 (a–c) Clinical front and lateral views of the patient presented in Fig. 31-2 at the final re-evaluation. To improve the esthetic outcome, the maxillary front teeth were restored with composite fillings.

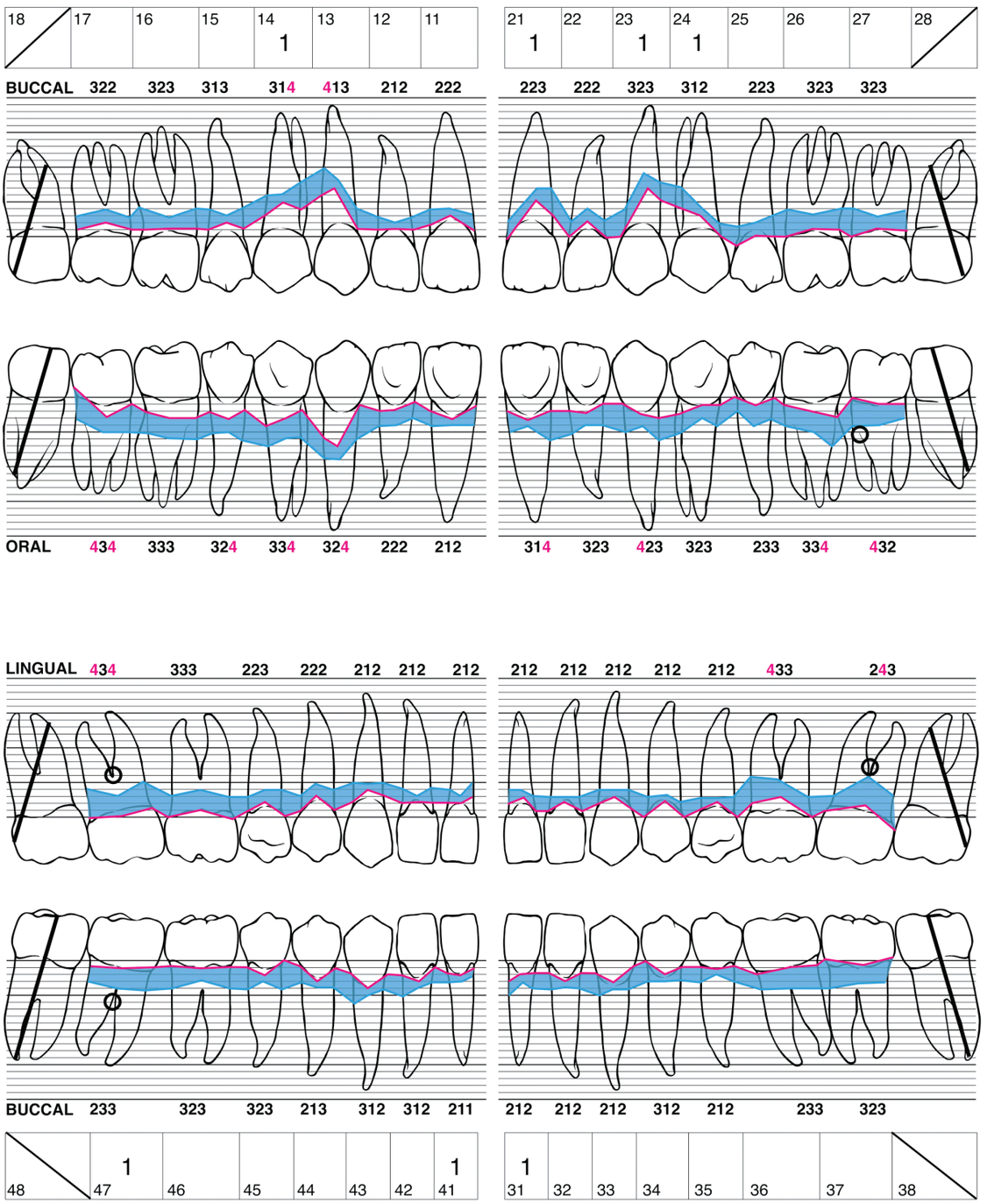

Fig. 31-14 Periodontal chart of the patient presented in Fig. 31-2 at the final re-evaluation.

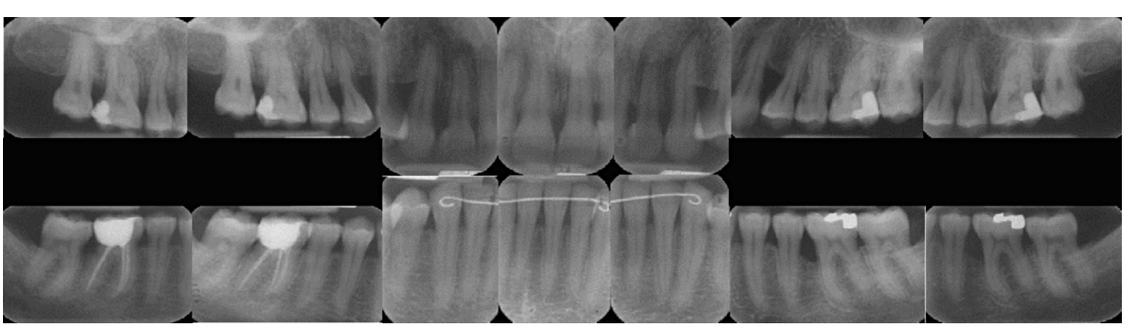

Fig. 31-15 Radiographs of the patient presented in Fig. 31-2 at the final re-evaluation.

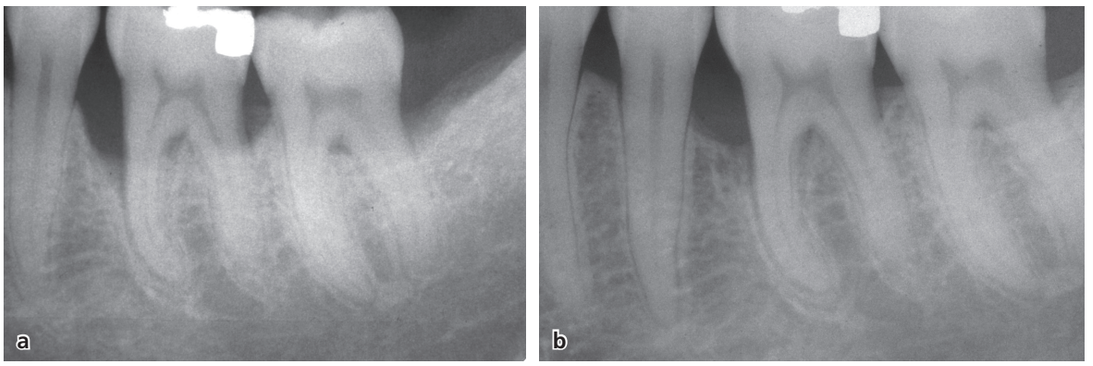

Fig. 31-16 (a,b) Radiographs of tooth 36 of the patient presented in Fig. 31-2 before and after regenerative periodontal therapy according to the principles of GTR.

Fig. 31-17 (a–c) Clinical front and lateral views of patient S.K. at initial examination.

Such deviations may be accepted as long as the fundamental principles characterizing the treatment phases are understood.

Case report

A patient will be presented below together with a brief description of his specific dental problems and the treatment delivered in order to demonstrate the rationale behind such treatment phases.

Patient S.K. (male, 35 years old)

Initial examination

The chief complaint of the patient was the slightly increased mobility of tooth 21. The periodontal conditions (i.e. probing pocket depths, furcation involvements, tooth mobility, and periapical radiographs) from the initial examination are shown in Figs. 31-17, 31-18, and 31-19.

The data obtained from the initial examination disclosed the presence of an advanced destruction of the supporting tissues in most parts of the dentition (Fig. 31-18) and the presence of several angular bony defects (Fig. 31-19). The full-mouth plaque score (FMPS) and full-mouth bleeding score (FMBS) were 32% and 86%, respectively. The patient was systemically healthy and a former smoker.

Diagnosis

The patient was diagnosed with generalized chronic periodontitis with furcation involvement.

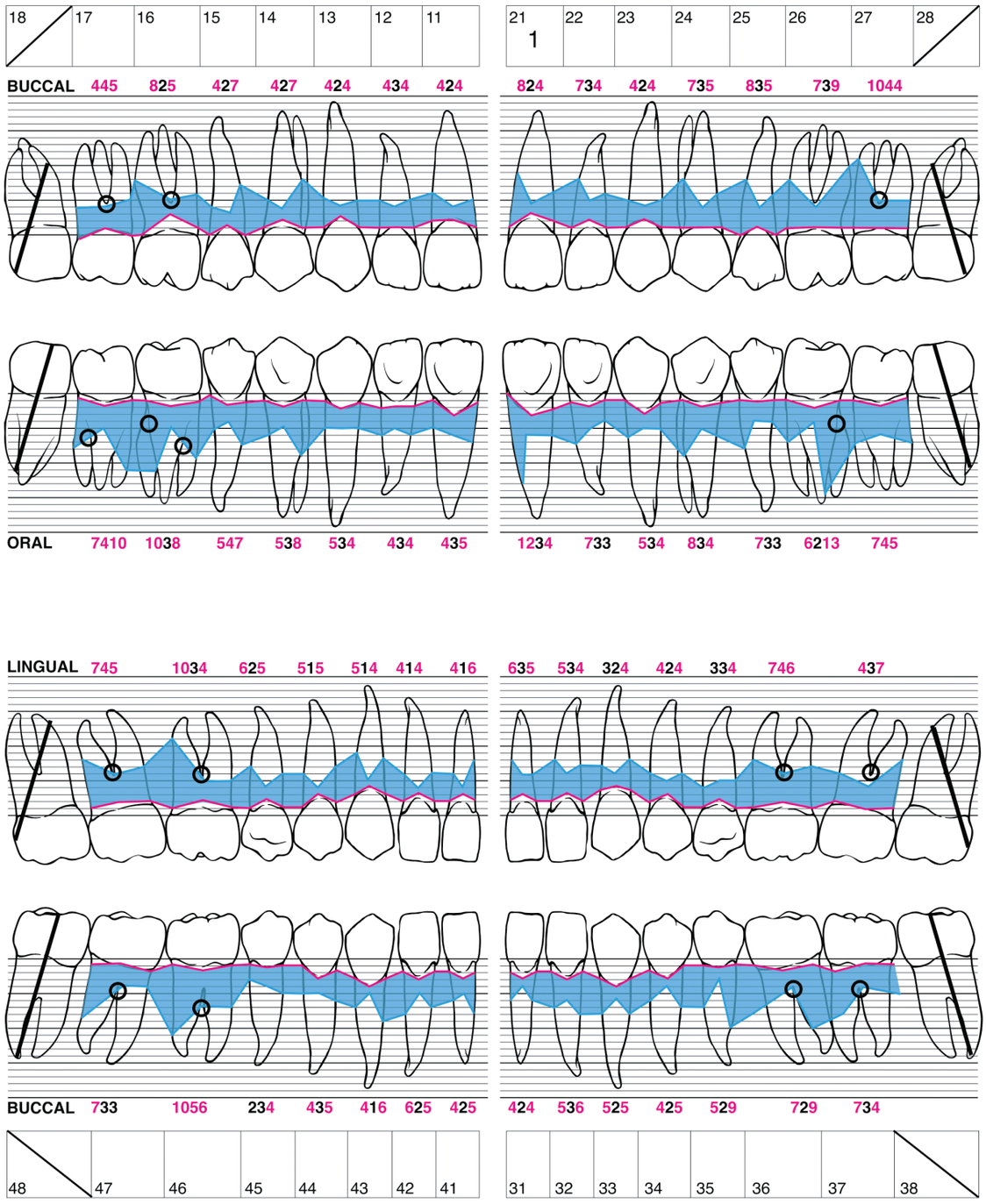

Fig. 31-18 Periodontal chart of the patient presented in Fig. 31-17.

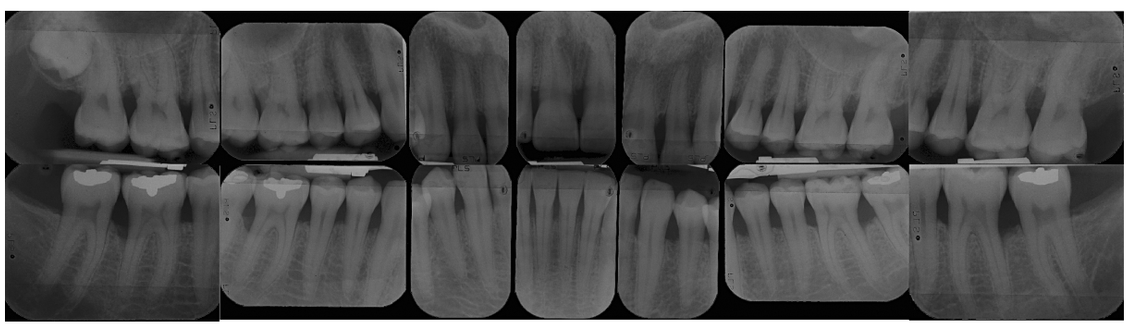

Fig. 31-19 Radiographs of the patient presented in Fig. 31-17.

Etiology

The supra- and subgingival bacterial deposits were identified as the main etiologic factors. Past cigarette smoking was considered a modifying factor.

Pre-therapeutic single tooth prognosis

Teeth 28, 38, and 48 were missing. Tooth 18 was impacted and considered irrational-to-treat. Teeth 13, 12, 11, and 23 in the maxilla and from 45 to 35 in the mandible were classified as secure. A doubtful prognosis was assigned to 17, 16, 15, 14, 21, 22, 24, 25, 26, and 27 in the maxilla and to 36, 37, 46, and 47 in the mandible (Fig. 31-20).

Treatment planning

In the treatment planning of this young patient, it seemed reasonable to anticipate the retention of all teeth of his periodontally compromised dentition. The prerequisites for a good long-term prognosis after therapy included (1) optimal self-performed plaque control, (2) proper healing of the periodontal tissues following non-surgical and surgical therapy, and (3) a carefully monitored maintenance care program. As stated above, tooth 21 displayed increased mobility. This mobility, however, did not disturb the chewing comfort of the patient.

In such a young patient, extensive efforts were made to treat inflammatory periodontal disease pro-

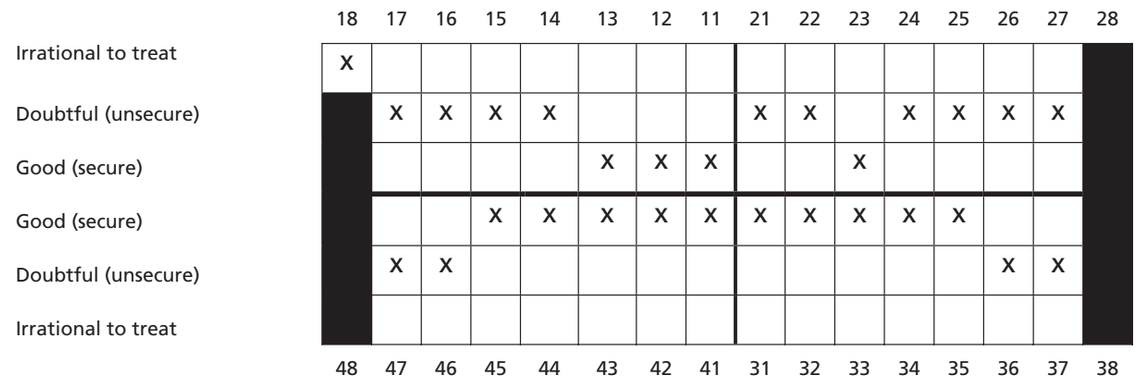

Fig. 31-20 Pre-therapeutic single tooth prognosis of the patient presented in Fig. 31-17.

Fig. 31-21 (a–c) Clinical front and lateral views of the patient presented in Fig. 31-17 at re-evaluation after initial therapy.

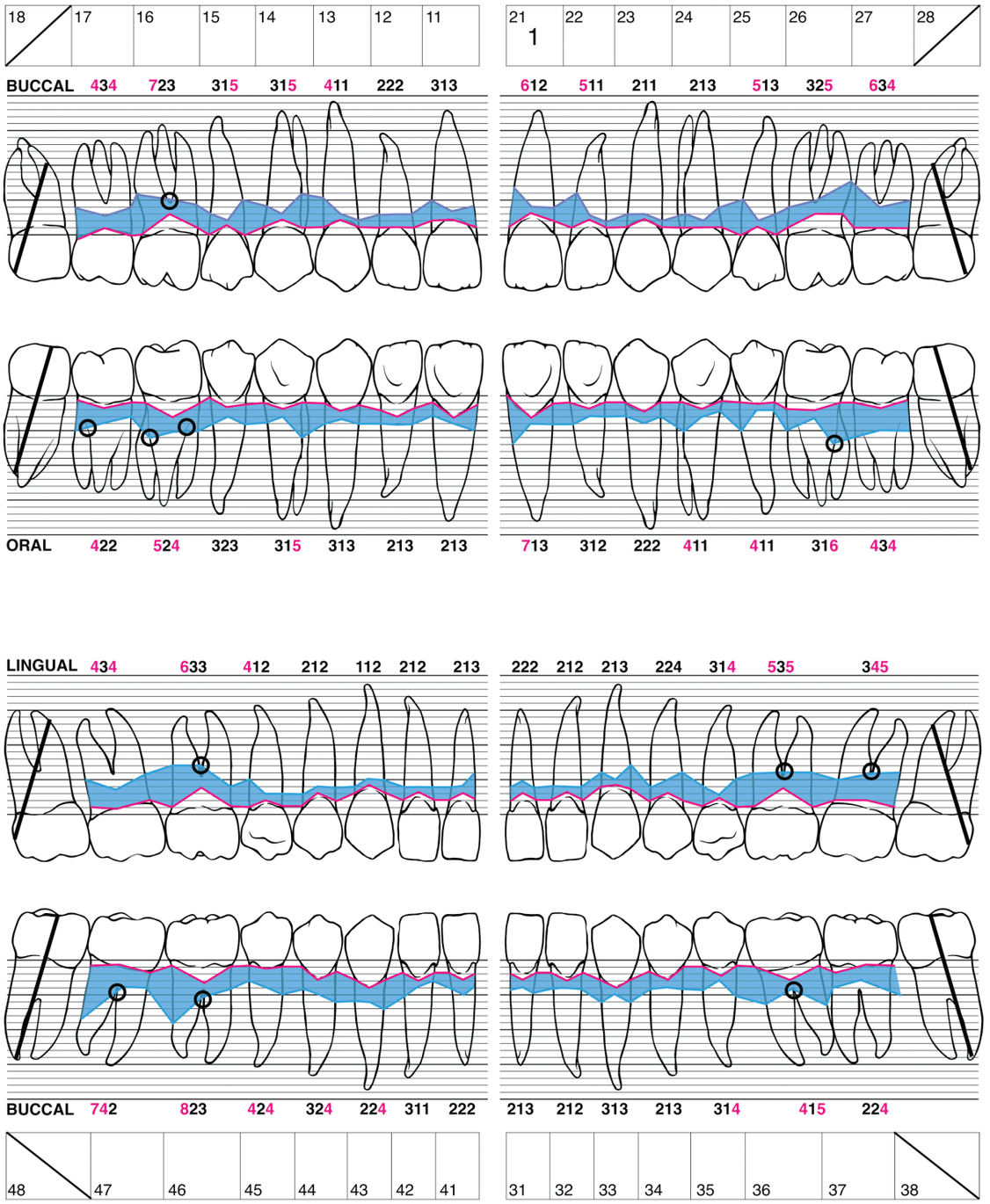

Fig. 31-22 Periodontal chart of the patient presented in Fig. 31-17 at re-evaluation after initial therapy.

perly in the entire dentition, in order to avoid tooth extraction and subsequent prosthetic rehabilitation.

Treatment

Subsequent to initial examination, the patient was given a detailed "case presentation" and information regarding alternative goals of and prerequisites for the overall treatment. This information included a description of the role of dental biofilms in the etiology of periodontal disease and the significance of optimal plaque control for a successful outcome of therapy. A treatment program was subsequently planned which aimed at maintaining all teeth. The overall treatment was performed in the sequence described below.

Initial cause-related therapy

The patient was counseled not to start smoking again. After thorough motivation, the patient was instructed

Fig. 31-23 (a-c) Intra- and postsurgical views of the upper front area of the patient presented in Fig. 31-17.

Fig. 31-24 (a–c) Clinical front and lateral views of the patient presented in Fig. 31-17 at the final examination.

in the toothbrushing technique according to Bass and in the use of interdental brushes. Scaling and root planing of all teeth were performed under local anesthesia. The front and lateral views as well as the periodontal chart at re-evaluation after initial therapy are presented in Figs. 31-21 and 31-22, respectively.

Additional therapy

The need for additional therapy was based on the re-evaluation after initial therapy (Fig. 31-22). Periodontal surgery in conjunction with regenerative procedures was deemed necessary in all quadrants. During access flap surgery in the first quadrant extending from 13 to 17, tooth 18 was extracted.

Between the upper front teeth 11 and 21, the modified papilla preservation technique (Cortellini *et al.* 1995) was incorporated in the surgical procedure to gain access to the angular bony defect of tooth 21 (Fig. 31-23). In this area, the application of enamel matrix derivatives (i.e. Emdogain®) aimed at regenerating the lost periodontal tissues on the mesial aspect of 21.

In the third quadrant, the surgical access flap extended from 35 to 37. In the fourth quadrant, flap surgery in conjunction with the simplified papilla preservation technique (Cortellini *et al.* 1999) was used to gain access to the angular bony defect on the distal aspect of 46. In this area, the application of enamel matrix derivatives (i.e. Emdogain®) aimed at regenerating the lost periodontal tissues. Six months after completion of the corrective phase (Fig. 31-24), a re-evaluation of the periodontal conditions (Fig. 31-25), including radiographs (Figs. 31-26 and 31-27) followed by a PRA, were performed.

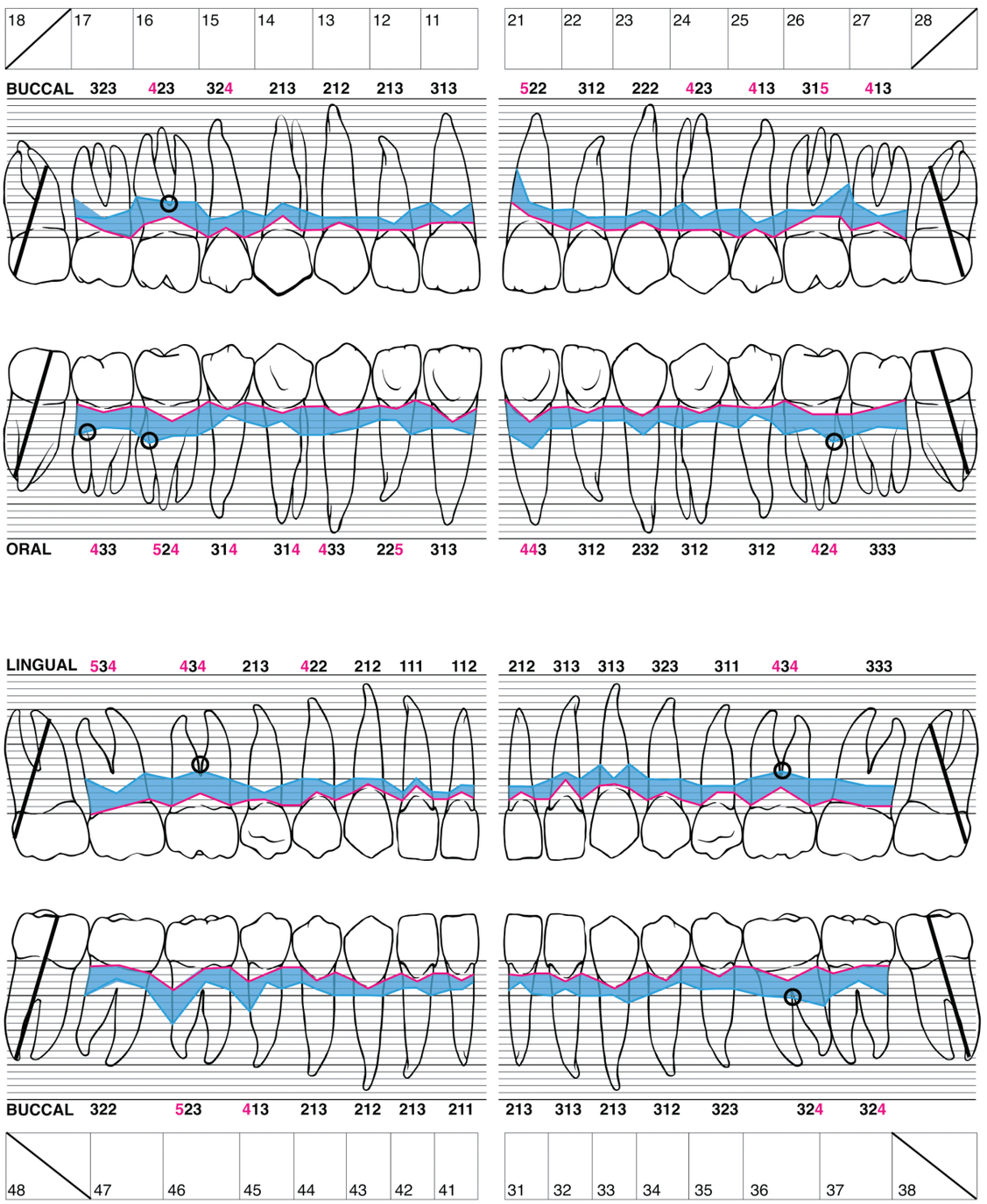

Fig. 31-25 Periodontal chart of the patient presented in Fig. 31-17 at the final examination.

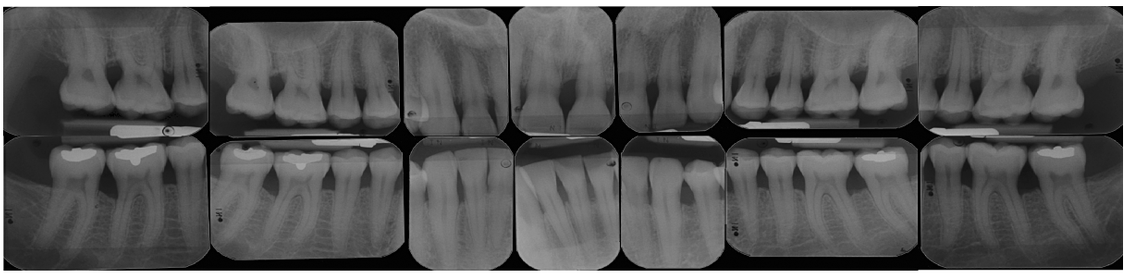

Fig. 31-26 Radiographs of the patient presented in Fig. 31-17 at the final examination.

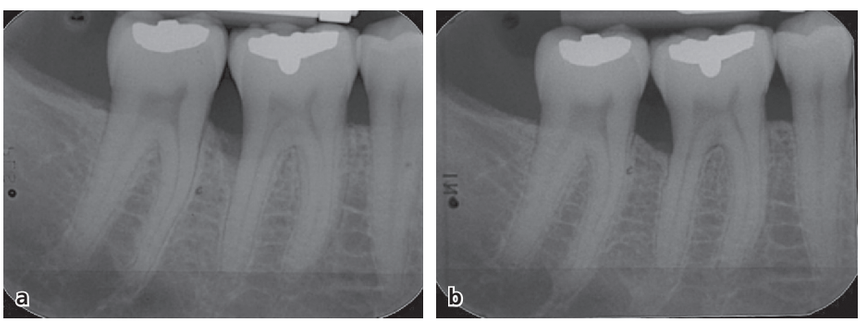

Fig. 31-27 Radiographs before (a) and after (b) periodontal regeneration of the angular bony defect on the distal aspect of tooth 46.

Supportive periodontal therapy

After completion of initial and corrective therapy, the patient was recalled for maintenance care every 3 months. During recall appointments, sites bleeding on probing and with a PPD ≥5 mm were re-instrumented. If necessary, the patient was remotivated and re-instructed in oral hygiene procedures. Fluoride was regularly applied in order to prevent the onset of dental caries.

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