MODERN IMPLANT DENTISTRY

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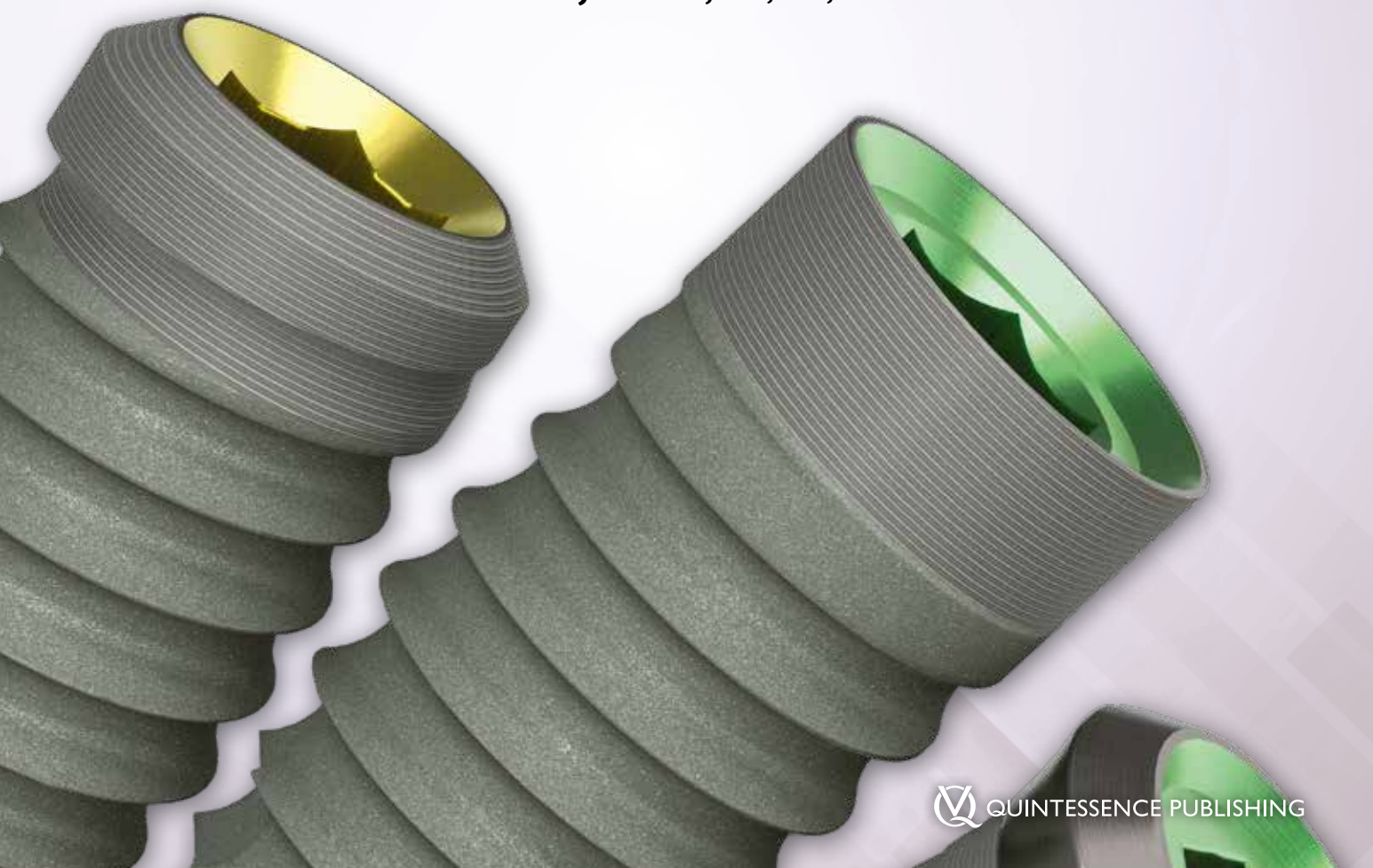

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PREFACE

The use of dental implants has become so widespread over the past few decades that scores of textbooks have been dedicated to this topic. As little as 50 years ago, the practice of dentistry did not include the modern implants, bone grafts, barrier membranes, or growth factors currently available in today's market. It wasn't until the late 1970s that Brånemark and his colleagues presented the two-stage threaded titanium implant procedure that we know today, following a decade of testing in various animal and human models. While the original Brånemark implant was a cylindrical fixture, over the years the shape has shifted to tapered, and many new implant types, body designs, and surface morphologies have been adopted. Entire companies have been formed with the goal of delivering faster, more predictable results to our patients. Today, the field has been trending, as rapidly as ever, into the world of digital implant dentistry. This book highlights all these groundbreaking discoveries, many of which are credited to the book's contributors—some of the most talented clinicians working in the profession today.

Over 50 authors from around the world have contributed to this textbook, all of them with different backgrounds and expertise in various subspecialties of implant dentistry. Chapters 1 to 4 focus on the biologic background for dental implants as well as medical considerations required prior to placing them, and chapters 5 and 6 are dedicated to documentation, including photography and CBCT imaging. Chapters 7 and 8 focus on treatment planning and digital workflow, and chapters 9 to 18 cover many surgical concepts, from single-tooth extraction to guided All-on-X treatment. Chapters 19 to 22 focus on the prosthetic options available to us in implant dentistry, while chapters 23 to 31 hit upon additional topics related to the field. These include soft tissue management, treatment of peri-implant disease, the socket shield technique, and marketing of dental implant therapy.

Preparing this textbook has been a massive undertaking. The editors wanted to bring you THE book on implant dentistry so every clinician, whether a beginner or a seasoned pro, can find what they need to practice implant dentistry well and continue to improve their skills and patient outcomes. To accomplish this goal, the book was written using only evidence-based approaches performed on a daily basis by experienced clinicians. As such, the techniques and advice given are based on real-world experiences in the clinic. As an added learning tool, over 60 surgical and clinical videos are included within the book (linked via QR codes) to SHOW you what these procedures and techniques and products look like in real life, not in photographs taken in ideal conditions.

We are very proud to present this book to you, and we hope to keep it updated over time as new developments, products, techniques, and research become available. The goal will always be to provide new and experienced clinicians with the most up-to-date and evidence-based textbook written in implant dentistry. As such, it is our hope that this book will benefit all implant surgeons by adding to their current knowledge base and improving their ability to make rational, evidence-based decisions in implant dentistry.

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BONE METABOLISM AROUND DENTAL IMPLANTS

RICHARD J. MIRON
ANGEL INSUA

SUMMARY

Despite the increasing number of studies in the field of implant dentistry investigating novel dental implant surfaces, biomaterials, and growth factors, comparatively very few have studied the biology and metabolism of bone healing and its implication in peri-implant tissue health. The aim of this chapter is to provide a thorough understanding of the biologic properties that impact bone formation and osseointegration, including the coupling mechanisms between immune cells and bone. This chapter focuses on the various bone cells in the body—osteocytes, bone lining cells (BLCs), osteoblasts, and osteoclasts—and their bone remodeling cycle. Furthermore, the importance of immune cells and their impact on biomaterial integration during bone formation and implant osseointegration is also discussed. Finally, the putative effects of cholesterol, hyperlipidemia, and vitamin D deficiency are addressed. Such factors should be monitored during patient care, and ultimately future research should focus on these avenues as well as meticulous maintenance programs to favor both early and long-term maintenance and stability of dental implants.

OBJECTIVES

- Understand how overall patient health directly affects dental implant osseointegration
- Understand the key cells involved in bone formation, maturation, and maintenance
- Understand the direct role of immune cells on biomaterial and dental implant integration
- Understand the essential role of optimizing the immune system prior to dental implant placement
- Investigate the relationship between vitamin D deficiency and early implant failure and how to avoid such pitfalls

Bone regeneration requires bone grafting materials that possess excellent biocompatibility and osteoinductivity without eliciting an antigenic effect. While companies that manufacture replacement biomaterials intended to mimic autogenous bone grafts often report on their osteoconductive, osteoinductive, or osteogenic potential, autogenous bone still favors the greatest bone regeneration compared to allografts, xenografts, and synthetic alternatives because it combines all three of these properties. Thus, despite the increasing number of

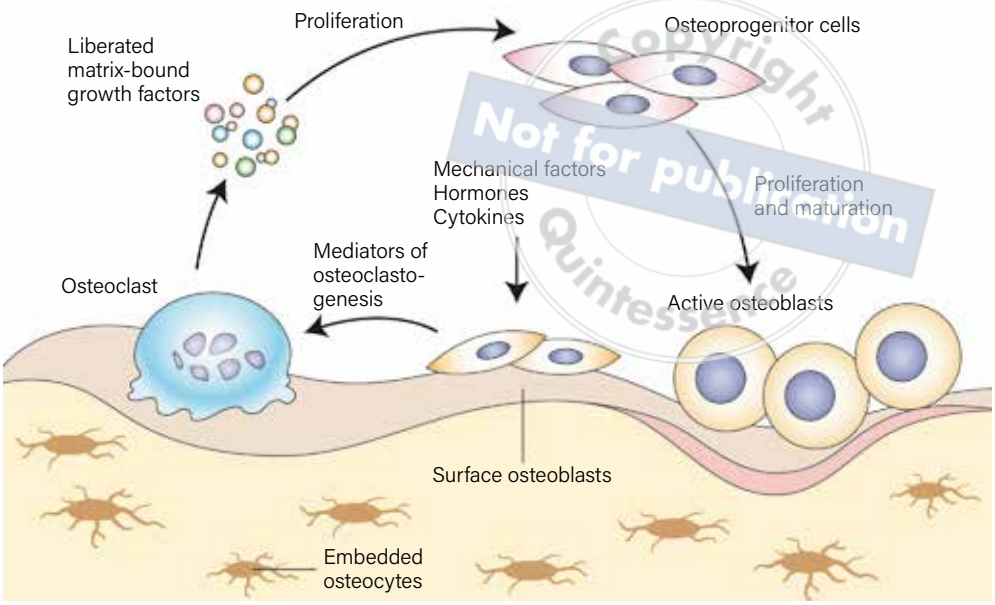

FIG 1-1 The bone remodeling cycle. Osteoclasts—cells that destroy and resorb bone—are present on the bone matrix surface and liberate growth factors and cytokines to the local microenvironment. These growth factors act on mesenchymal stem cells/progenitor cells to rapidly stimulate their proliferation and differentiation toward bone-forming osteoblasts. Osteoblasts lay new bone matrix and, once embedded within the bone matrix, become osteocytes.

new bone grafting materials brought to market as substitute replacement grafts, to date there is no true replacement for autogenous bone grafts.[1] Autografts carry no risk of immunologic reaction or disease transmission and provide optimal conditions for the penetration of new blood vessels and migration of osteoprogenitor cells. In contrast, many bone grafting substitutes are osteoconductive but have limited osteoinductive potential.[2]

For bone regeneration to take place, especially with foreign-body biomaterials such as allografts and xenografts or dental implants, there is a great need to better understand the regulatory properties and integration process of these biomaterials. After all, no matter the biomaterial placed, bone formation relies on immune-related factors working at the cellular level. The aim of this chapter is therefore to provide the biologic background on the cells involved in graft consolidation and give a brief overview of fracture healing. This chapter focuses on the bone cells involved in bone formation and dental implant osseointegration, including osteocytes, BLCs, osteoblasts, and osteoclasts, and their bone remodeling cycle. The chapter also addresses the importance of immune cells and their impact on biomaterial integration, as well as the putative effects of cholesterol, hyperlipidemia, and low vitamin D levels.

Bone Cells: Osteoclasts, Osteoblasts, and Osteocytes

There are three main cell types in bone tissue involved in the bone remodeling cycle: osteoclasts, osteoblasts, and osteocytes[3] (Fig 1-1). Osteoclasts are the bone-resorbing cells that degrade bone tissues. They are derived from hematopoietic stem cells following their differentiation from monocytes in response to two key factors: receptor activator of nuclear factor kappa-B ligand (RANKL)[4] and macrophage colony-stimulating factor (M-CSF).[5] Osteoclasts can be characterized histologically based on their multinucleated morphology and expression of tartrate-resistant acid phosphatase (TRAP), cathepsin k (CatK), and the calcitonin receptor (CTR). Their formation, activity, and survival are also regulated by various hormones (such as calcitonin and estrogen) that regulate several downstream cytokines and cellular pathways.[6] Activated osteoclasts form distinct and unique membrane domains, including the sealing zone, the ruffled border, and the functional secretory domain, which facilitate resorption of bone or bone graft particles.[7] Rearrangement of their F-actin fibers from the cytoskeleton forms a ring shape consisting of a dense continuous zone of highly dynamic podosomes.[8] These podosomes allow for mineralized bone to be gradually resorbed, creating grooves and tunnels on the bone surface. This process is also quite important for bone remodeling because the resorbed bone liberates calcium phosphates and growth factors contained within the bone matrix that attract bone-forming osteoblasts to the local environment.[9]

Osteoblasts perform the opposite role of osteoclasts and are responsible for bone formation (see Fig 1-1). They are derived from cells of the mesenchymal lineage, and their formation and development are controlled locally and systemically by several growth factors, including bone

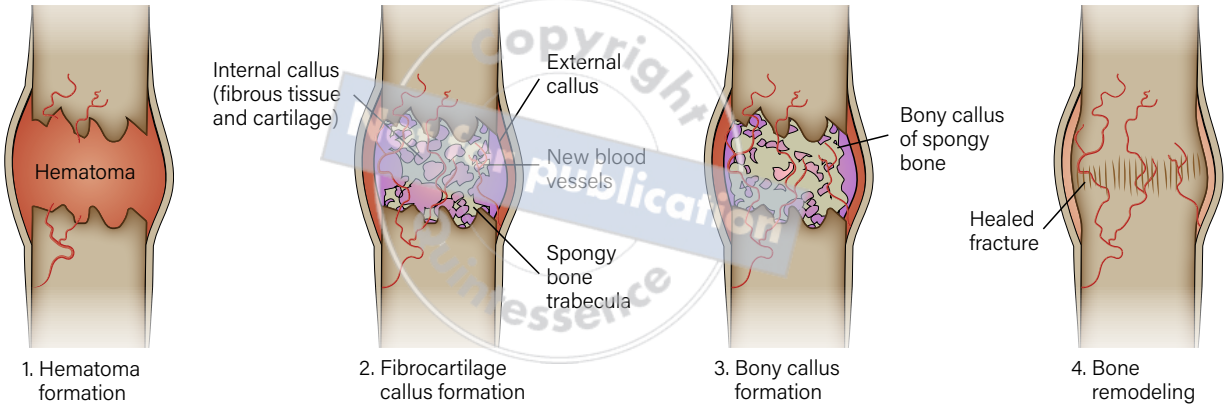

FIG 1-2 The fracture healing process is divided into four phases: *(1)* hematoma formation, *(2)* fibrocartilage callus formation, *(3)* bony callus formation, and *(4)* bone remodeling.

morphogenetic proteins (BMPs).[10,11] Osteoblasts secrete a range of molecules including growth factors, cell adhesion proteins, and other extracellular matrix molecules that support new bone formation.[6] While osteoblasts are forming bone, they become embedded within bone tissues and transform into osteocytes.

Contrary to the short lifespan of osteoblasts and osteoclasts, osteocytes can live for decades within the bone matrix. They no longer produce new bone and instead undergo morphologic changes, losing cytoplasm organelles and acquiring a stellar shape morphology with numerous extensions that connect to other osteocytes through the canalicular network.[12] Osteocytes can then transmit signals through this network similar to neuron communication; this communication has a profound impact on neighboring osteoblasts, osteoclasts, and osteocytes. While it was initially believed that osteocytes served only a mechanical transduction function,[13] more recently their role has been deemed one of the most important within the bone tissues because they release numerous paracrine signals to their environment, thereby influencing both osteoblasts and osteoclasts.[12,14,15]

Fracture Healing and Graft Consolidation

Fracture healing is an important process that involves various cell types and a variety of signaling pathways.[3,16,17] Unlike other tissues in the body, bone tends to regenerate and repair itself quite rapidly. Natural fracture healing is a four-stage process (Fig 1-2). The first step is hematoma formation. Following injury, a blood clot forms, creating a fibrin matrix with an abundance of infiltrating inflammatory and immune cells that take place with an activation of platelets.[16,18] These cells secrete an array of growth factors that initiate the second and third phases of fracture repair.[16,18] At the terminal end of the first phase, osteoclast formation occurs from precursor monocytes, and these osteoclasts invade the bone surface, commencing bone resorption.[16]

The second phase of fracture healing is the repair phase. During this phase, a bone callus is formed. Initially, blood vessels begin to form with infiltration of mesenchymal progenitor cells; this process is responsible for creating a fibrocartilagenous tissue that matures into bone. This woven bone is gradually replaced with dense lamellar bone to form a dense bony callus in phase 3.[19]

The final phase is the remodeling phase, during which the callus is gradually resorbed. In this stage, the bone is replaced by native bone lacking scar tissue.[20] It has been shown that during this phase, resident macrophages play a predominant role in orchestrating host cells.[21,22] While these four phases are not described in great detail within the present chapter, it is important to note that graft consolidation is tightly regulated by secretion of growth factors and cytokines (also secreted from autogenous bone particles and blocks). This is all tightly regulated in very distinct cell-to-cell communication events that take place during bone regeneration.[3]

Role of Osteocytes and Bone Lining Cells in Bone Remodeling

Osteocytes

Osteocytes are the pivotal cells in the regulation of bone mass and structure, along with osteoblasts and osteoclasts.[23] Osteoblasts are derived from mesenchymal stem cells and

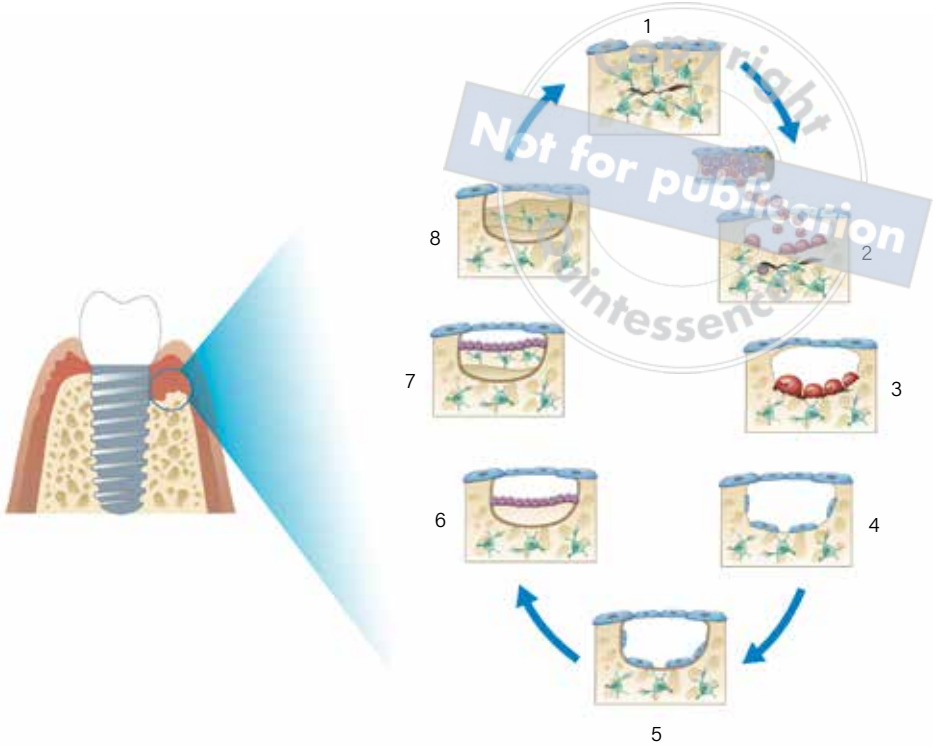

FIG 1-3 Bone remodeling after excessive implant torque. *(1)* Excessive torque promotes bone damage, including the osteocyte network. *(2)* Osteoblasts and osteoclasts are recruited from the blood, from the marrow, or from BLCs to populate the bone remodeling compartment. *(3)* Osteoclasts remove the damaged bone. *(4)* BLCs clean the debris after osteoclast resorption. *(5)* BLCs secrete fibrillar collagen. *(6)* This collagen layer allows osteoblasts to attach. *(7)* Osteoblasts deposit osteoid to fill the compartment. *(8)* Osteoblasts trapped in the osteoid become osteocytes or BLCs, after which most undergo apoptosis. (Reprinted with permission from Seeman.[34])

synthesize new bone matrix.[24] Osteoclasts are terminally differentiated multinucleated cells from the monocyte-macrophage lineage; beyond their resorbing bone function, these cells are also a source of cytokines that play an important role in bone homeostasis.[25] Osteocytes are terminally differentiated osteoblasts, and their primary function is to support bone structure and mechanosensation.[24] Osteocytes may act as regulators of bone remodeling by modulating osteoclast and osteoblast activities.[25] These stellate-shaped cells are located within lacunae surrounded by mineralized bone matrix and present with connections through cytoplasmic prolongations with surface BLCs but also with bone marrow.[15,25]

Bone lining cells

BLCs are cells involved in bone formation much like preosteoblasts, osteoblasts, and osteocytes.[26] They are characterized by a flat-shaped architecture along bony surfaces[25] and may be considered latent osteoblasts.[27] In human cancellous bone, around 65% of osteoblasts undergo apoptosis, with approximately 30% differentiating into osteocytes[28]; the reduced remnants become BLCs and chondroid-like cells.[26,28] BLCs maintain their proliferative capability and often differentiate into other osteogenic cells.[29,30] Various studies have shown that some factors can induce their proliferation prior to bone formation,[31] while mature osteoblasts are unable to divide.[26] Osteoblasts may also undergo a quiescent stage when there is no bone resorption or remodeling,[29] but the function of BLCs might be more complex than a simple latent state,[32] including catabolic and anabolic bone processes[31] and rapid bone formation under osteogenic signaling.[32]

In the complex process of bone remodeling,[33,34] external factors such as mechanical loading, irradiation, parathyroid hormone (PTH), fibroblast growth factor-2 (FGF2), sclerostin inhibition, or inflammation may lead BLCs to exit the quiescent stage and enter into an active function phase by re-forming their cuboidal appearance and their secretory capability.[25,31,35] The presence of BLCs observed histologically indicates a strong sign of osteogenic potential[29] and is often regarded as a major source of osteoblasts and proliferating preosteoblasts in the adult population.[31] This prominent role in new bone formation was previously highlighted[28,32] when rapid bone formation was observed after mechanical loading without previous bone resorption. Early peak bone formation after 3 days was only possible if BLCs underwent reactivation and reaquired their secretory capacities.[28,32]

Moreover, BLCs exert a prominent function during bone resorption,[36] demonstrated by their ability to express key ostoclastogenesis markers including M-CSF receptor (M-CSFR), and after the modulation of bone resorption, BLCs play another important role in the early stages of bone formation by entering the resorption lacunae to remove collagen fibers and debris left by osteoclasts (Fig 1-3). Subsequent to this cleaning function, BLCs secrete a layer of fibrillar collagen, allowing osteoblasts to attach and deposit new osteoid.[36]

One novel replacement option that has shown promising results both before and after surgery is StellaLife's recovery kit, a homeopathic wound healing rinse (Fig 1-5). It favors better wound healing of the defect site as well as improved pain management with microbial resistance.[141] A split-mouth study revealed that the StellaLife VEGA Oral Care Recovery Kit performed better than many frequently utilized materials including Peridex, plasma rich in growth factors (PRGF), platelet-rich plasma (PRP), Emdogain, and PerioSciences products. The following conclusions were reported in the StellaLife test group[141]:

- The product achieved anesthetic pain relief.
- Healing was faster than with the control products.
- At 1 week, the sites treated with StellaLife resembled the other sites at 1 month (those treated with Peridex, PRGF, PerioSciences, PRP, or Emdogain).
- Less pain was associated with the sites treated with StellaLife.
- Fewer problems were reported for the sites treated with StellaLife.
- The pain level (rated as 5 or less the first day followed by less than 3 the consecutive days) was lower than anticipated (7 on a scale of 1 to 10).

StellaLife was initially developed to limit the need for narcotic drugs during healing and thereby battle the growing problem of narcotic dependence and addiction in the United States. In a study of 150 patients, all patients unanimously reported having fewer problems and recovering more quickly while utilizing StellaLife's VEGA system compared to other products and therefore required fewer narcotic pain medications.

Conclusion

While implants have been highly researched over the years, it remains equally important to better understand how loading and implant bed preparation affect BLCs and osteocyte viability and signaling both at early and late time points. Furthermore, the effects of systemic levels of cholesterol, fatty acids, and vitamin D are important factors that may affect implant survival. In addition, the prominent role of immune cells (eg, OsteoMacs and MNGCs) on bone formation, bone remodeling, and implant osseointegration and maintenance must be researched further to discover how immune cells can be controlled to favor long-term stability and prevent peri-implant disease. Future research investigating these various topics is ongoing.

FIG 1-5 StellaLife Recovery Kit used both before and after implant surgery.

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