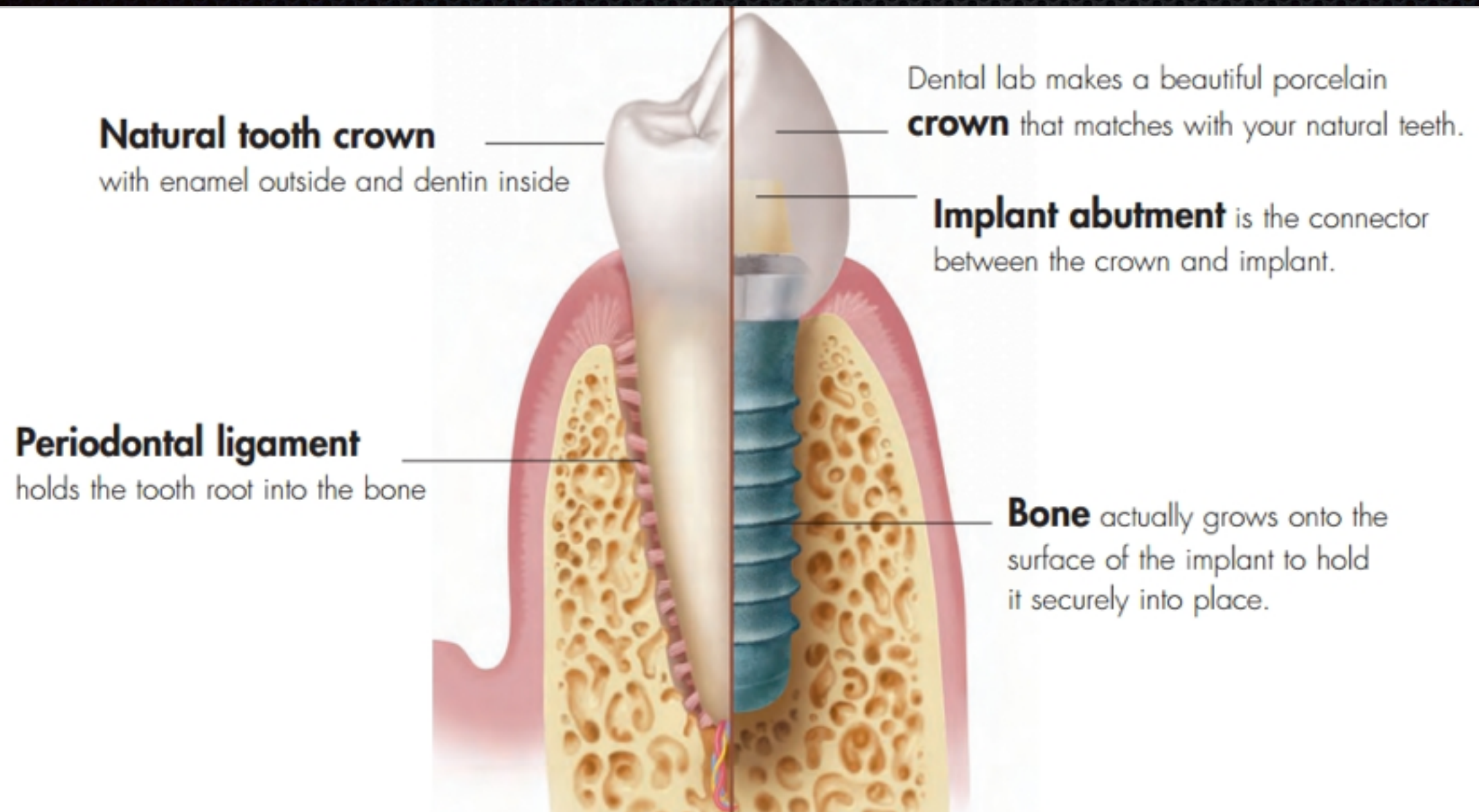


In The Name of GOD

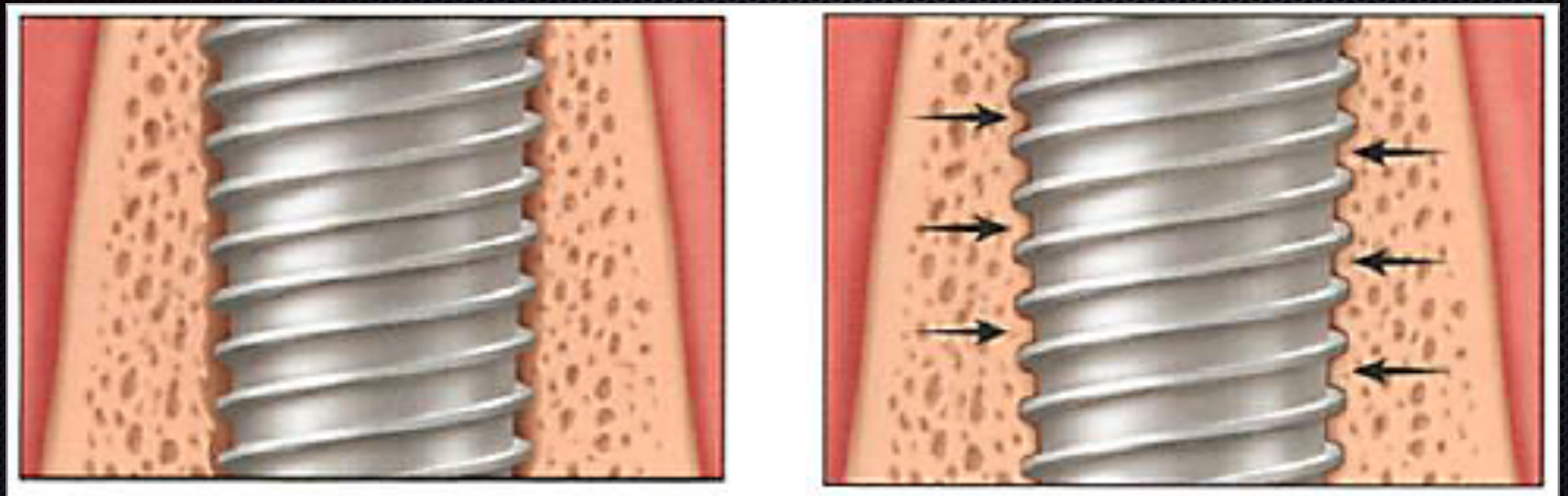
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*Osseointegration and soft tissue
healing around the dental
implants*



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 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 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** (primary stability) following installation in the recipient site.

Tissue injury

Basic rule: The **less traumatic** the surgical procedure and the smaller the tissue injury (the damage) 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 healing of the severed bone following implant installation is a complex process that apparently involves **different events in different compartments** of the surgical site.

In the *cortical bone compartment*, the nonvital mineralized tissue must *first* be removed (*resorbed*) before new bone can form.

In the *spongy (cancellous) 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 involves *localized bleeding and clot (coagulum)* formation. The coagulum is gradually resorbed and becomes replaced with *granulation* tissue; in-growth (from the walls of the prepared canal) of *blood vessels, leukocytes, and mesenchymal cells.*

As a result of the continuous migration of mesenchymal cells from the surrounding marrow, the granulation tissue in turn is replaced with **provisional connective tissue** (provisional matrix) and eventually with **osteoid**. In the osteoid, deposition of **hydroxyapatite crystals** will occur in the collagen network **around the newly formed vascular structures**. Hereby, **immature woven bone** is formed and sequentially osseointegration occurs.

Process of osseointegration

The void between the pitch and the body of the implant established a geometrically well-defined **wound chamber**. were occupied with a **blood clot** in which **erythrocytes, neutrophils, and monocytes/macrophages** occurred in a **network of fibrin**.

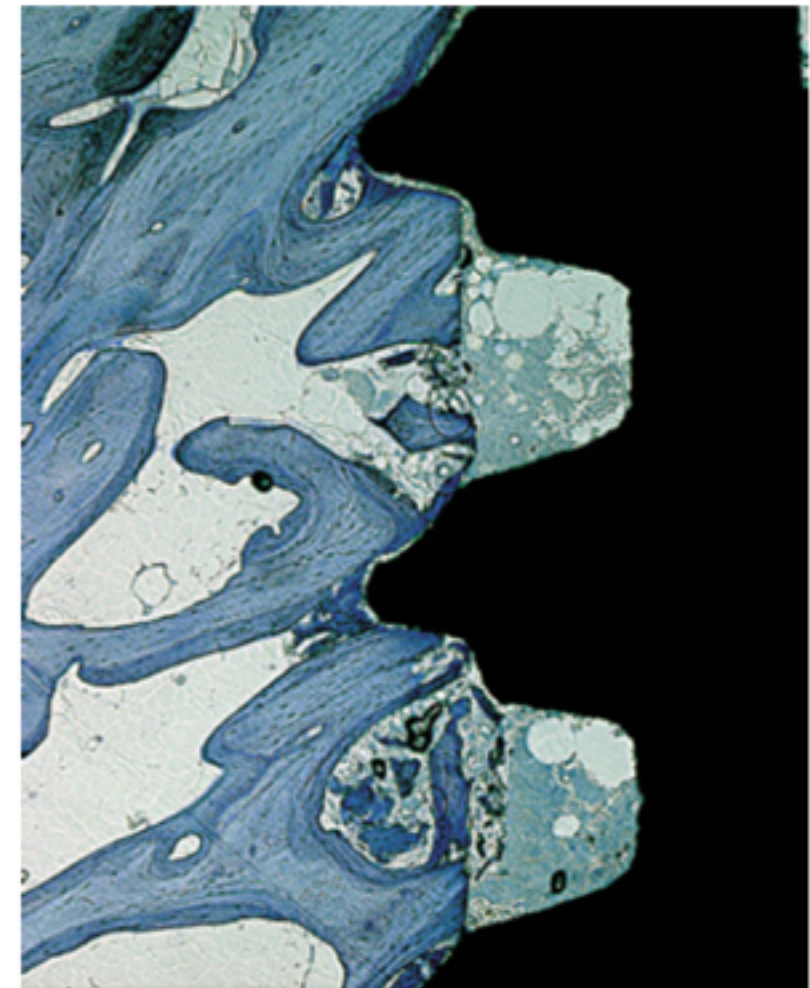
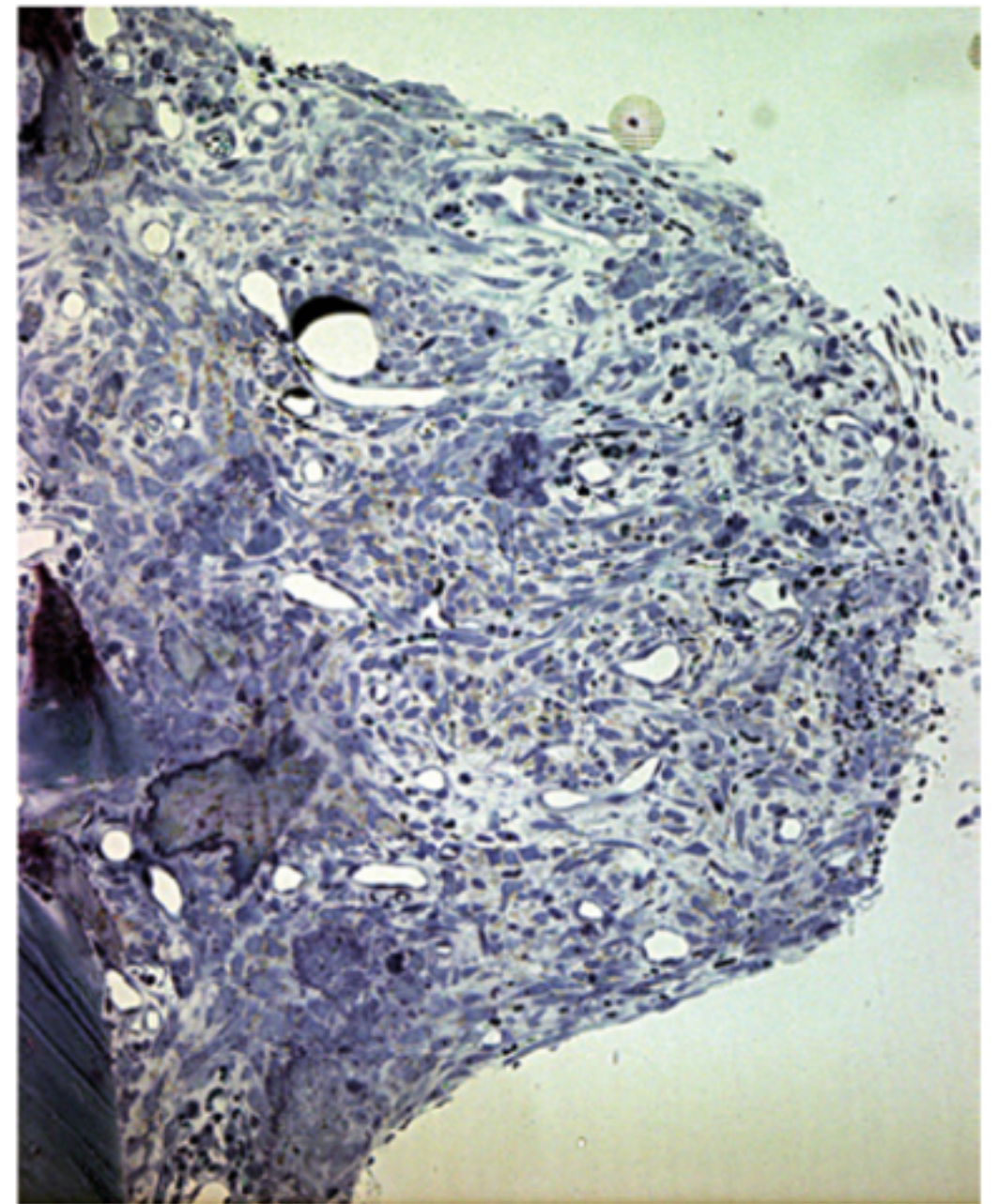


Fig. 5-13 Detail of Fig. 5-12. The wound chamber was filled with blood and a coagulum has formed.

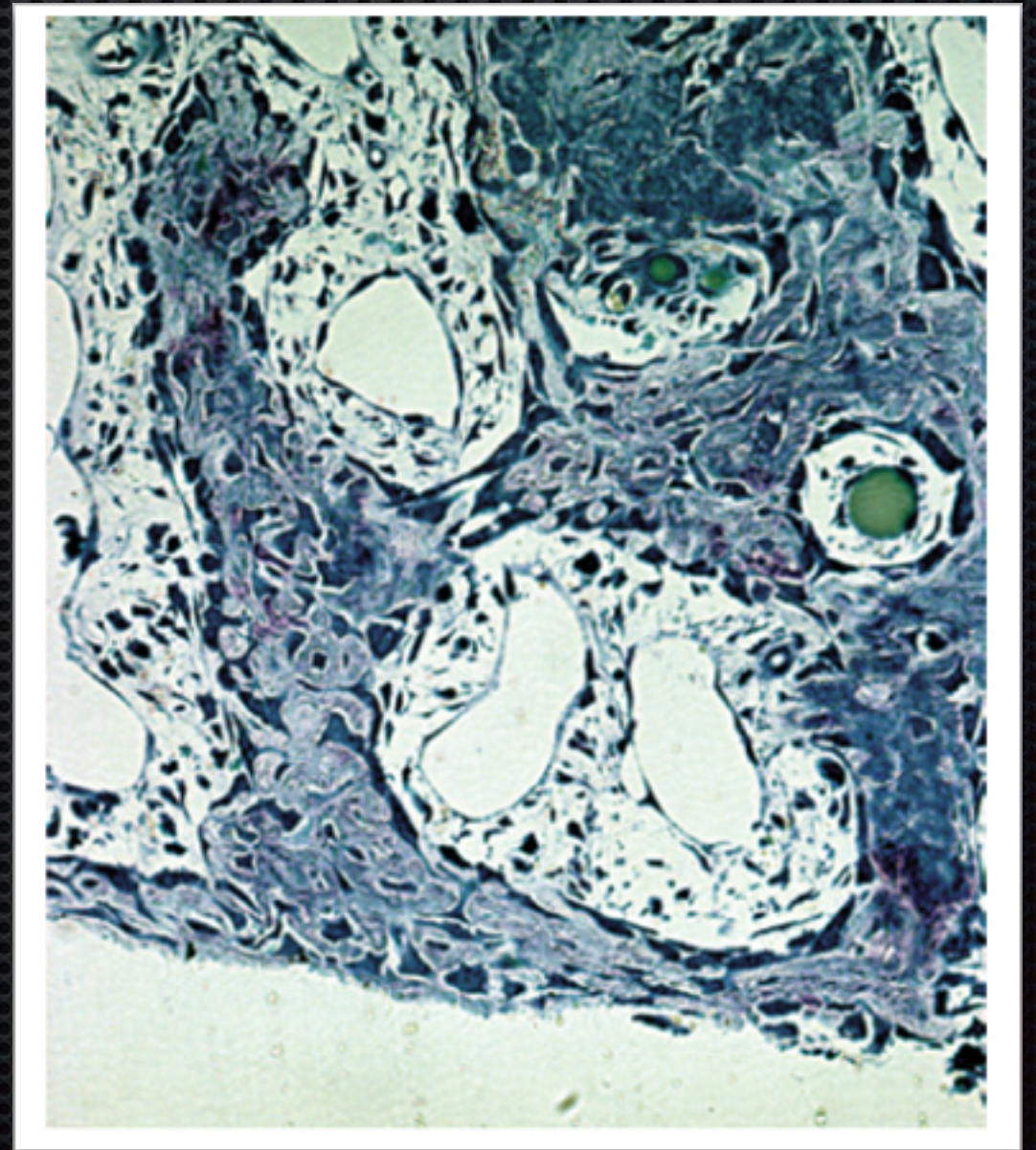
Fibroplasia: Figure illustrates a device with surrounding tissues after 4 days of healing. The coagulum had in part been replaced with granulation tissue that contained numerous mesenchymal cells, matrix components, and newly formed vascular structures (*angiogenesis*).

A provisional connective tissue (matrix) had been established.

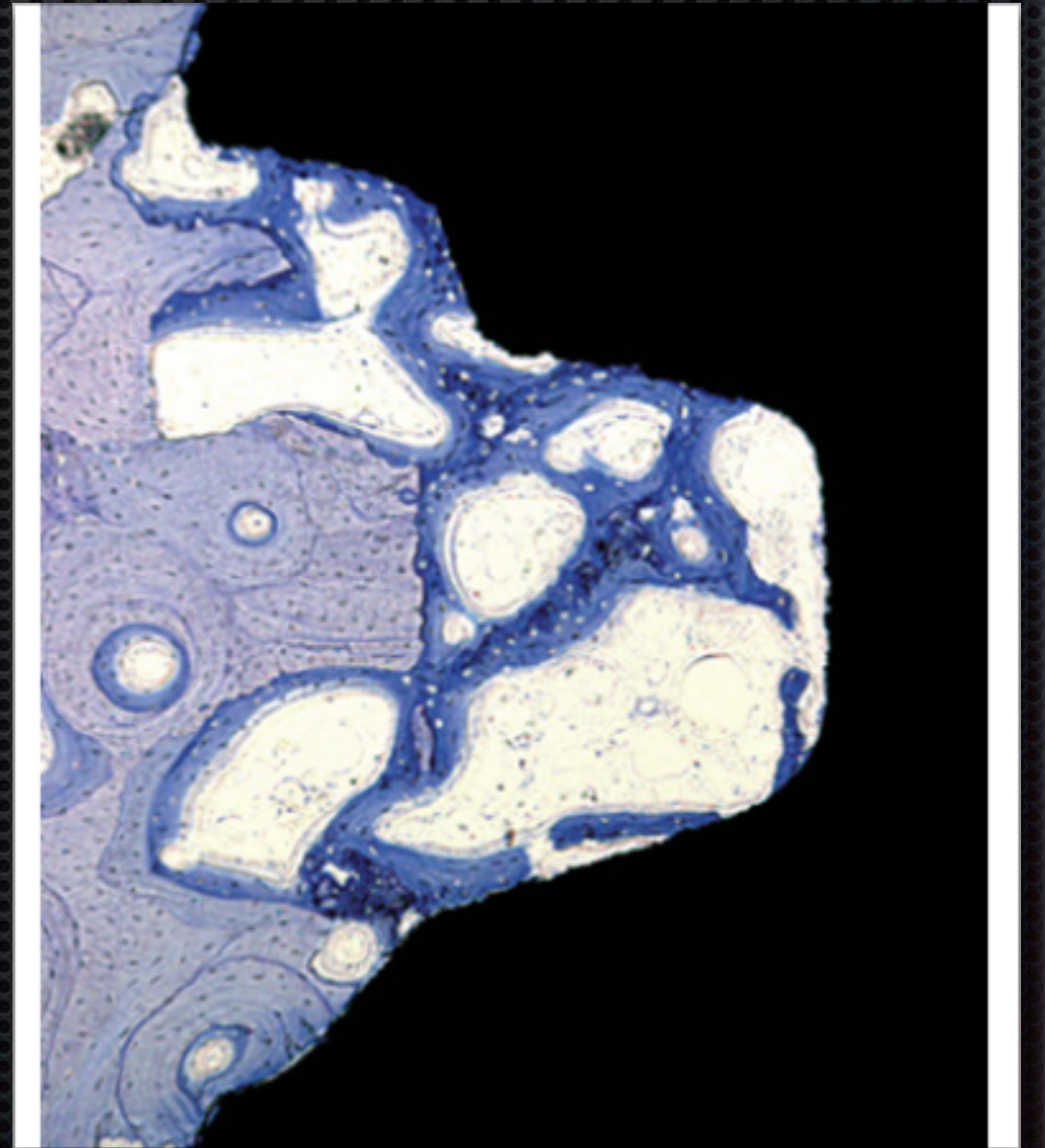
(a)



Bone modeling: After **1 week** of healing, the provisional connective tissue in the wound chambers was rich in **vascular structures** and contained numerous **mesenchymal cells**. The number of remaining inflammatory cells was relatively small. In several compartments of the chamber, a cell-rich immature bone (**woven bone**) was seen in the provisional connective tissue that **surrounded the blood vessels**. 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. This 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. 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 old bone into the provisional connective tissue 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



Remodeling: After 6–12 weeks of healing, most of the wound chambers were filled with mineralized bone. 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.

Overall pattern of implant integration

After 1 week of healing, about 40% of the interface region was made up of soft tissue (granulation tissue, provisional connective tissue) and an additional 45% of bone debris and old bone.

After 2 weeks, the amount of newly formed bone was still small, but the amount of soft tissue was markedly reduced.

In the interval between 2 and 4 weeks, new bone formation was apparently pronounced in the interface zone. Thus, in this interval, newly formed bone increased from about 10% to about 30%, while the amount of hard tissue debris was markedly reduced.

Also, in the period between 4 and 6 weeks, new bone formation was pronounced (from 30% to about 60%) and the diminution of old bone and bone debris markedly decreased. In other words,

In humans the process of osseointegration appears to be most active in the interval between 2 and 6 weeks.



Wound healing biology

The process of wound healing is the body's primary mechanism to restore tissue integrity upon injury. If wound healing does not occur properly, chronic disruption of the protective barrier may lead to severe physiologic, immunologic, and metabolic abnormalities. Wound healing basically represents a dynamic process that involves several cell types and biologic mediators. Within the active system of the periodontal wound, cell populations migrate, differentiate, and proliferate; epithelial and connective tissues interact; and a vast array of cytokines and extracellular matrix (ECM) molecules orchestrates the whole process that takes place in overlapping phases.

Phases of wound healing

The general principles of healing, and the cellular and molecular events observed in extraoral sites, also apply to the healing processes that take place following periodontal surgery. Traumatic injury causes capillary damage and hemorrhage, and, as a result, a blood clot is formed. **The formation of a clot is the immediate response to any trauma.**

The clot has two functions:

It temporarily protects the denuded tissues AND it serves as a provisional matrix for cell migration.

The blood clot consists of all cellular components of blood (including red and white blood cells and platelets) in a matrix of fibrin, plasma fibronectin, vitronectin, and thrombosporin.

Beyond this, the process has been divided into three stages:

1. Inflammation phase
2. Granulation phase
3. Matrix formation and remodeling (maturation) phase (Wikesjo *et al.* 1992).

Each of the steps of wound healing is essential to success, **but the initial healing process often determines the outcome.**

Inflammatory phase

The **growth factors** present in the clot recruit inflammatory cells, and then serve to regulate the granulation process. Within **hours** of injury, **inflammatory cells** (predominantly neutrophils and monocytes) populate the clot. These cells cleanse the wound of bacteria and necrotic tissue through **phagocytosis** and release of **enzymes** and toxic oxygen products.

Within 3 days, the inflammatory reaction moves into its late phase.

Macrophages >>>> cleansing process by phagocytosis of used polymorphonuclear leukocytes and erythrocytes.&

>>>>>biologically active molecules such as inflammatory cytokines and tissue growth factors, which recruit further inflammatory cells as well as fibroblastic and endothelial cells,

thus playing an essential role in the transition of the wound from the inflammatory into the granulation tissue formation phase.

Granulation phase

Macrophages

Granulation tissue formation begins on approximately day 4.

Macrophages constitutively release growth factors that promote the healing process.

Growth factors and cytokines secreted by macrophages are involved in the proliferation and migration of fibroblasts, endothelial cells, and smooth muscle cells into the wound area.

At 7 days after initiation of wound healing, granulation dominates the wound site and the initial collagen fibers are being formed. Eventually, cells and matrix form cell-to-cell and cell-to-matrix links that generate a concerted tension resulting in tissue contraction. The phase of granulation tissue formation gradually develops into the final phase of healing in which the reformed, more cell-rich tissue undergoes maturation and sequenced remodeling to meet functional needs

Maturation phase

Fibroblasts responsible for the replacement of the provisional ECM produce a new collagen-rich matrix. Approximately **1 week following wounding**, and once the collagen matrix has been synthesized, some **fibroblasts undergo transformation** into **myofibroblasts** and express

α -smooth muscle actin.

This transformation and synthesis is responsible for **wound contraction**.

Endothelial cells, responsible for angiogenesis, migrate into the provisional wound matrix to form vascular tubes and loops, and as the provisional matrix matures, the endothelial cells undergo programmed cell death (**apoptosis**) and the number of **vascular units** is reduced.

Maturation of the granulation tissue will lead to the **regeneration or repair** (scar formation) of the injured tissues.

Whether the damaged tissues heal by regeneration or repair depends upon two crucial factors:

The availability of the necessary **cell** type(s) and the presence or absence of cues and **signals** necessary to recruit and stimulate these cells.

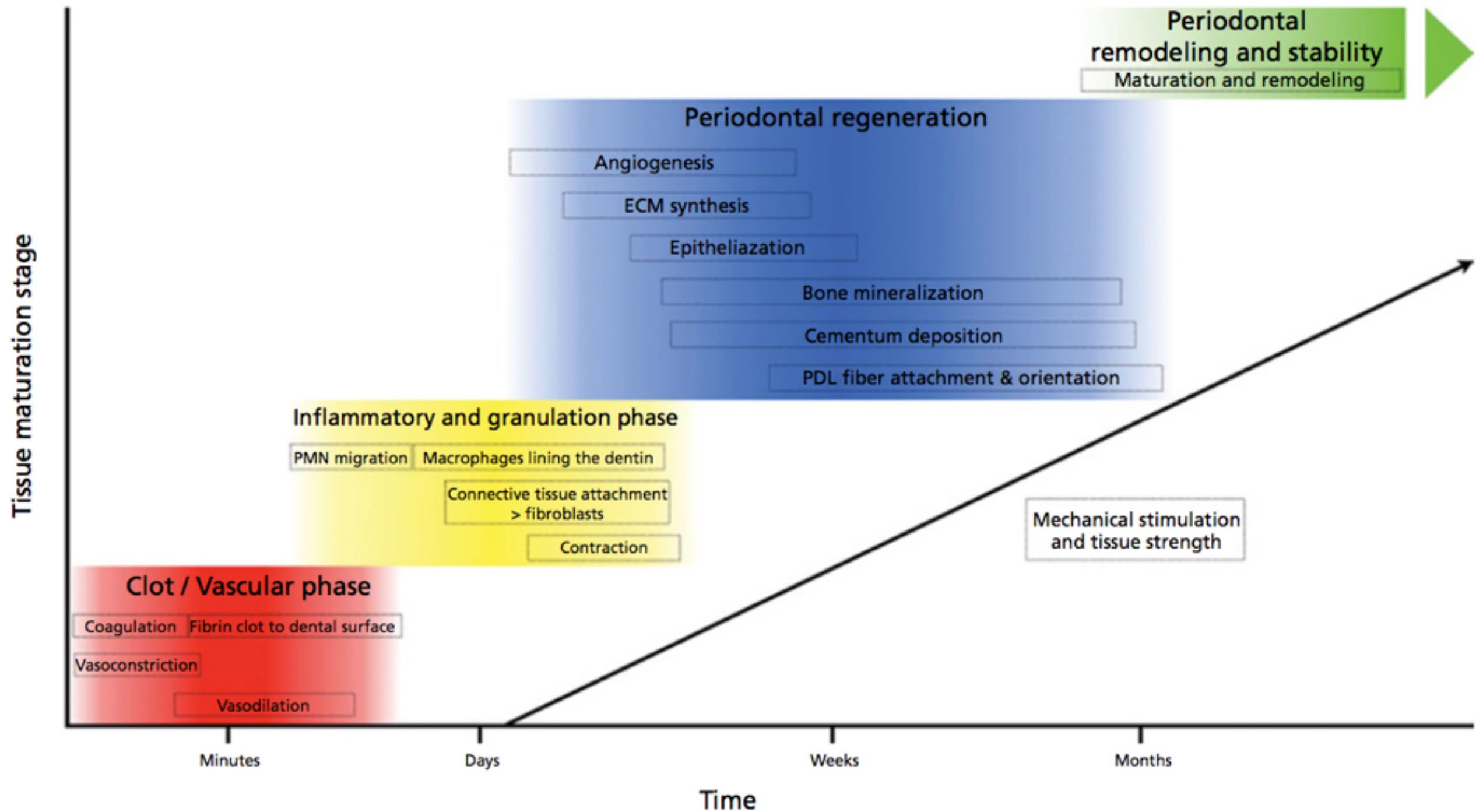


Fig. 27-2 Stages of periodontal wound healing. Optimal periodontal healing requires different processes in a sequential manner. After the initial coagulation phase, inflammatory reaction, and granulation tissue formation events, progenitor cells involved in multitissue regeneration are locally recruited and mediate the bioavailability of important growth factors. As the healing progresses, mechanical stimuli increase and promote an organized extracellular matrix (ECM) synthesis as well as cementum and bone formation and maturation. Once those structures are established, periodontal ligament (PDL) fibers are organized and oriented. Progressively, the tissues mature and ultimately increase in mechanical strength. Remodeling processes continue in the regenerated periodontium as an essential mechanism that monitors the adaptation potential to the challenging local and systemic environment.

Factors that affect healing

Local factors

Plaque **microorganisms**

Excessive tissue **manipulation** during treatment

Trauma to the tissues

Presence of **foreign bodies**

Repetitive treatment procedures that disrupt the orderly cellular activity during the healing process

Inappropriate **vascular perfusion** to the surrounding area.

Healing is therefore improved by debridement (removal of degenerated and necrotic tissue), immobilization of the healing area, and pressure on the wound.

The cellular activity in healing entails an increase in **oxygen consumption**. However, healing of the gingival tissue is not accelerated by artificially increasing the oxygen supply beyond the normal requirements (Glickman *et al.* 1950).

Systemic factors

It is clearly reported that healing capacity diminishes with **age** (Holm–Pedersen & Löe 1971). Healing is also impaired by **insufficient food intake**; systemic **disorders** that interfere with the use of nutrients; and deficiencies in **vitamin C** (Barr 1965), **proteins** (Stahl 1962), and other nutrients. **Hormones** also have an impact on healing.



Systemically administered **glucocorticoids** such as cortisone hinder repair by depressing the inflammatory reaction or by inhibiting the growth of fibroblasts, the production of collagen, and the formation of endothelial cells.

Systemic stress, thyroidectomy, testosterone, adrenocorticotrophic hormone, and large doses of estrogen suppress the formation of granulation tissue and impair healing (Butcher & Klingsberg 1963).

Progesterone increases and accelerates the vascularization of immature granulation tissue (Lindhe & Brånemark 1968) and appears to increase the susceptibility of the gingival tissue to mechanical injury by causing dilation of the marginal vessels (Hugoson 1970).

A close-up photograph of a mossy forest floor. In the foreground, there is a thick layer of vibrant green moss. To the right, a tree trunk with deeply textured, peeling bark is visible. The background is a soft-focus view of a forest with more trees and foliage. The entire image is framed by a dark, textured border at the top and bottom.

Thanks!