International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

A Comprehensive Review on Periodontal Wound Healing: Mechanisms, Factors Affecting and Therapeutic Approaches

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Abstract: ***This review explores the complex process of periodontal wound healing. It delves into the stages of healing, the role of various cells and proteins, and the factors affecting healing. The review also discusses healing patterns following different periodontal procedures, providing a comprehensive understanding of the subject. This knowledge is crucial for developing new therapies to optimize periodontal structure and function restoration after wounding.***

Keywords: Dental scaling, gingivectomy, wound healing

1. Introduction

The native periodontium includes cementum, a functionally oriented periodontal ligament, alveolar bone and gingiva. Pathologic and/or traumatic events may lead to the loss or damage of this anatomical structure. Conventional periodontal therapy, be it surgical or nonsurgical in nature, usually involves instrumentation in the inflamed dentogingival complex. Such therapies result in wounding of the already inflamed periodontal tissues. Thus, the consequence of such therapeutic procedures depends largely on the cellular and molecular events associated with wound healing. Wound healing is a complex and dynamic process of replacing devitalized and missing cellular structures and tissue layers. Periodontal wound healing follows well - acknowledged biological principles (Clark 1996). The cells of five or more tissue types—epithelium, gingival and periodontal connective tissue, bone, root cementum—are essentially expected to create a new connection to the nonvascular and non - vital hard tissue of the root surface. Histological studies have shown that various surgical periodontal procedures can lead to different patterns of healing. More recently, a great deal of research has been directed at understanding the critical factors that influence poorly healing wounds. An improved understanding of the physiology and regulation of periodontal healing will be critical for the development of new therapies to optimize the restoration of periodontal structure and function after wounding.

Stages of periodontal wound healing

The wound - healing process consists of four highly integrated and overlapping phases: hemostasis, inflammation, proliferation, and tissue remodeling or resolution (Gosain and DiPietro, 2004).

1) Hemostasis and Inflammation –

- **Fibrin Clot Formation**

Injury to the blood vessels during surgery/periodontal therapeutic procedure causes extravasation of blood. A fibrin rich clot is formed by blood coagulation and platelet aggregation. It has following functions: 1. It temporarily protects the denuded tissues, 2. It serves as a provisional matrix for cell migration.3. The clot also act as a reservoir of growth factors and cytokines that are released by the de - granulation of activated platelets.

- **Inflammatory Phase**

Clot formation is followed by an early stage of inflammation. The growth factors and cytokines present in the fibrin clot in fact might be providing the start signals for wound repair. Neutrophils and monocytes are usually recruited initially by the present in the clot.

a) **Role of Neutrophils:** 1. Neutrophils cleanse the wound of foreign particles and debris and bacteria by releasing enzymes and toxic oxygen products.2. Neutrophils also serve as a source of pro - inflammatory cytokines providing signals that activate adjacent fibroblasts and keratinocytes. Neutrophil infiltration ceases after a few days, and they are eventually phagocytosed either by macrophages or fibroblasts.

b) **Role of Macrophages:** Peripheral blood monocytes that continue to be recruited into the wound site become macrophages upon activation.1. Macrophages continue the task of phagocytosing bacterial, cellular and matrix debris in the wound.2. Macrophages continually synthesize and secrete growth factors and cytokines into the wound environment. Thus, the wound repair signals initiated by degranulating platelets and neutrophils are maintained by macrophages.

2) Re - Epithelialization of Wounds

a) Role of Integrins

Integrins are a family of cell adhesion receptors that mediate cell surface interactions with extracellular matrix and in some cases with other cells. In the normal gingival tissues, the basal layer of epithelium is attached to the basal lamina via receptors on their surface (integrins), to bind to laminin in the basal lamina. In preparation for migration, the keratinocytes at the edge of the surgical wound have to dissolve the hemidesmosome attachment and begin to express other integrins that are more suitable for the wound environment. This mobilization and expression of new

integrins facilitates adhesion of keratinocytes to matrix molecules in the provisional matrix as well as the adjacent wound debris. Once re - epithelialization is complete, the components of basal lamina are deposited in a sequential manner starting from wound margin and the epithelial cells revert to their normal phenotype.

3) Matrix Degradation and the Wound - Cleaning Process

a) Role of plasmin and MMPs

Migration of epithelial cells through the fibrin clot or along the junction between the clot and underlying connective tissue (mucoperiosteal flap surface in the case of periodontal surgery) requires the creation of a migrating path. This is achieved by the dissolution of the fibrin barrier by the enzyme plasmin that is derived from the activation of plasminogen in the clot. The two activators, tissue - type plasminogen activator and urokinase - type plasminogen activator along with its receptor, are upregulated in the migrating keratinocytes. Similarly, several other proteases mainlyMatrix metalloproteinases (MMP) are also expressed to clear the path for cell migration.

Classification of Matrix Metalloproteinases

Group	Enzyme	Name
Collagenases	MMP-1	Collagenase 1, fibroblast collagenase
	MMP-8	Collagenase 2, neutrophil collagenase
	MMP-13	Collagenase 3
Gelatinases	MMP-2	Gelatinase A
	MMP-9	Gelatinase B
Stromelysins	MMP-3	Stromelysin 1
	MMP-10	Stromelysin 2
	MMP-11	Stromelysin 3
Matrilysins	MMP-7	Matrilysin 1, pump-1
	MMP-26	Matrilysin 2
Membrane-type MMPs	MMP-14	MT1-MMP
	MMP-15	MT2-MMP
	MMP-16	MT3-MMP
	MMP-17	MT4-MMP
	MMP-24	MT5-MMP
	MMP-25	MT6-MMP
Others	MMP-12	Macrophage elastase
	MMP-19	—
	MMP-20	Enamelysin

MMPs, Matrix metalloproteinases; *MT*, membrane type.

4) Granulation Tissue Formation, Contraction and Angiogenesis of the Wound

Granulation tissue formation usually can begin around day 4 after the wounding. Granulation tissue is a complex reservoir of cytokines possessing chemoattractive, mitogenic and other regulatory activities. Growth factors are also present in granulation tissue and, at this stage of healing, are derived mostly from macrophages. During the formation of granulation tissue, macrophages, fibroblasts and new blood vessels grow into the wound space in a coordinated manner. Their interdependence is illustrated by the release of cytokines by macrophages that stimulate fibroblasts to synthesize an extracellular matrix. This extracellular matrix serves to support cell and vascular in - growth carrying nutrients to sustain the cellular functions. In addition to macrophages, fibroblasts themselves express many cytokines to which they can also respond in an autocrine manner. Components of the extracellular matrix such as fibronectin and collagen facilitate the adhesion and migration of fibroblasts in the granulation tissue. Fibroblasts adhere to fibronectin, collagen and vitronectin via the integrin receptors. As wound healing progresses, the provisional matrix is replaced by a new, collagen - rich matrix synthesized by fibroblasts migrating into the wound. The synthesis of specific extracellular matrix molecules by fibroblasts in the wound is regulated by growth factors.

Role of Myofibroblasts

Around 7–10 days after wounding, some of the fibroblasts in the wound transform into myofibroblasts and express α - smooth muscle actin. Such transformation allows these myofibroblasts to generate strong contractile forces that are responsible for wound contraction. In the final stages of healing the number of fibroblasts and myofibroblasts in the healing wound are decreased by programmed cell death.

Angiogenesis of Wound

Angiogenesis refers to the formation of new blood vessels and is a crucial event during the healing of wounds. The term granulation tissue in wounds was coined in reference to the red granular appearance of new blood vessels that invade the healing connective tissues. Several growth factors are important in the induction of angiogenesis in healing wounds primarily - 1. Fibroblast growth factor - 2 is synthesized by macrophages and damaged endothelial cells 2. Vascular endothelial growth factor is induced in wound - edge keratinocytes and macrophages. As in the case of fibroblasts, endothelial cells involved with angiogenesis in wounds also undergo programmed cell death during the

ultimate maturation of the matrix characterized by regression of capillaries.

Distinct characteristics of Periodontal Wound Healing

- The oral cavity, wound healing occurs in warm mouth fluids containing millions of oral microorganisms that might be perceived as detrimental to the healing process
- When a mucoperiosteal flap is apposed to an instrumented root surface deprived of its periodontal attachment, the wound margins are not two opposing vascular gingival margins. Rather the opposing surfaces comprise of the rigid nonvascular mineralized tooth surface on one hand, and the connective tissue and epithelium of the gingival flap, on the other.
- In the oral cavity wounds heal much faster compared to skin wounds, with rapid reepithelialization and re - modeling resulting in minimal scar formation. This advantage could be explained by the presence of growth factors or cytokines in the saliva.
- The periodontal wound also includes tissue resources from the alveolar bone and the periodontal ligament.

Biological concepts of periodontal regeneration by melcher

Melcher postulated biological concepts at the base of periodontal regeneration. He stated that the nature of new atachment following periodontal surgery is determined by the cells repopulating the root surface.

During the healing stages of periodontal pocket, the area is invaded by cells from four different sources –

1) If the epithelium proliferates along the tooth surface before any other tissue reach the are the result will be a long junctional epithelium.
2) If the cells from gingival connective tissue are the first to populate the area, the result will be fibers parallel to the tooth surface and remodelling of alveolar bone with no attachment to the cementum.
3) If bone cells arrive first, root resorption and ankylosis may occurs.
4) Finally, only when cells from the periodontal ligament proliferate coronally is there new formation of cementum and periodontal ligament.

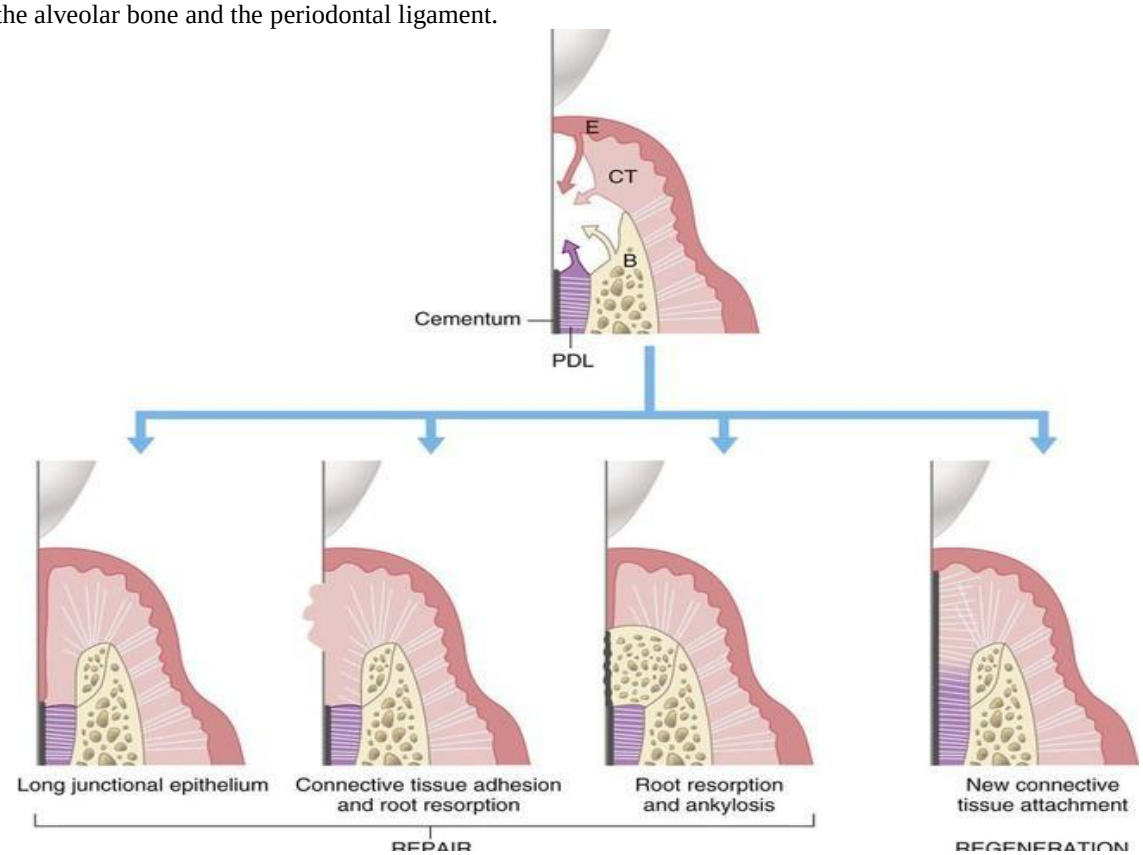

Figure 1: Possible healing patterns for a periodontal wound.

Factors affecting wound healing

Multiple factors can lead to impaired wound healing. In general terms, the factors that influence repair can be categorized into local and systemic.

Local Factors

1) Plaque micro - organisms are the most common deterrents to healing after periodontal treatment.
2) Iatrogenic causes include - Excessive tissue manipulation, trauma to tissues, presence of foreign body, repetitive treatment procedures that disrupt the orderly cellular activity in the healing process.
3) Impaired or insufficient blood supply results in development of areas of necrosis and delay the healing process.

Systemic Factors

1) **Age:** Delayed wound healing in the aged is associated with an altered inflammatory response, such as delayed T - cell infiltration into the wound area with alterations in chemokine production and reduced macrophage phagocytic capacity. Healing capacity diminishes with age probably due to atherosclerotic vascular changes common in aging and the resulting reduction in blood circulation.

2) **Sex Hormones**: Testosterone and large doses of Estrogen supress the formation of granulation tissue and impair healing. Estrogen affects wound healing by regulating a variety of genes associated with regeneration, matrix production, protease inhibition and. Progesterone increases and accelerates the vascularization of immature granulation tissue and appears to increase the susceptibility of the gingiva to mechanical injury by causing dilation of the marginal vessels.
3) **Stress**: The pathophysiology of stress results in the deregulation of the immune system, mediated primarily through the hypothalamic - pituitary adrenal (HPA) and sympathetic - adrenal medullary axes or sympathetic nervous system (SNS) that regulates the release of various pituitary and adrenal hormones. Stress up - regulates glucocorticoids (GCs) and reduces the levels of the pro - inflammatory cytokines IL - 1β, IL - 6, and TNF - α at the wound site.
4) **Diabetes:** Diabetic individuals exhibit a documented impairment in the healing of wounds. The reasons for impaired healing include - 1. Hyperglycaemia – It adds to the oxidative stress when the production of ROS exceeds the anti - oxidant capacity 2. The formation of advanced glycation end - products (AGEs) 3. High levels of metalloproteases that supports tissue destruction and inhibits normal repair processes.4. Several dysregulated cellular functions that are involved in diabetic wounds, such as defective T - cell immunity, defects in leukocyte chemotaxis, phagocytosis, and bactericidal capacity, and dysfunctions of fibroblasts and epidermal cells.5. Diabetic wounds also exhibit inadequate angiogenesis.
5) **Medications:**
   a) **Glucocorticoids:** Systemic glucocorticoids (GC) are frequently used as anti - inflammatory agents and are well - known to inhibit wound repair via global anti - inflammatory effects and suppression of cellular wound responses, including fibroblast proliferation and collagen synthesis. Systemic steroids cause wounds to heal with incomplete granulation tissue and reduced wound contraction.
   b) **Non - Steroidal Anti - Inflammatory Drugs (NSAIDs)** - Non - steroidal anti - inflammatory drugs have analgesic, anti - inflammatory, and anti - pyretic properties, which are used to aid recovery by decreasing the inflammatory phase of healing. NSAIDS are associated with significant side effects and rebound effect. The effects of low - dose aspirin on healing are not completely clear. Clinical recommendations suggest that, to avoid anti - platelet effects, individuals should discontinue NSAIDs for a time period equal to 4 to 5 times the half life of drugs before surgery. Thus, the majority of surgical patients do not have significant NSAID activity at the time of wound repair. (Exception - Those cardiac patients who must be maintained on low - dose aspirin due to severe risk of cardiovascular events) (Pieringer et al., 2007).
   c) **Chemotherapeutic Drugs:** Most chemotherapeutic drugs are designed to inhibit cellular metabolism, rapid cell division, and angiogenesis and thus inhibit many of the pathways that are critical to appropriate wound repair.
6) **Smoking:** Post - operatively, patients who smoke show a delay in wound healing and an increase in a variety of complications such as infection, wound rupture, anastomotic leakage, wound and flap necrosis, epidermolysis, and a decrease in the tensile strength of Studies have shown that smoking cessation leads to improved repair and reduces wound infection (Sorensen et al., 2003; Lauerman, 2008).

Healing Following Individual Periodontal Procedures

1) Healing After Phase One Periodontal Therapy (Scaling And Curettage)

Immediately after curettage, a blood clot fills the pocket area, which is totally or partially devoid of epithelial lining. Haemorrhage is also present in the tissues with dilated capillaries, and abundant of polymorphonuclear leukocytes (PMNs) appear shortly thereafter on the wound surface. This is followed by a rapid proliferation of granulation tissue, with a decrease in the number of small blood vessels as the tissues matures. Restoration and epithelialization of the sulcus generally requires 2 to 7 days, and restoration of junctional epithelium occurs in animals as early as 5 days after treatment. Immature collagen fibers appear within 21 days. Several investigators have reported that in monkeys and in humans treated by scaling procedures and curettage, healing results in formation of a long thin junctional epithelium with no new connective tissue attachment.

2) Healing Following Gingivectomy

Following excision of the gingival soft tissues coronal to the base of the periodontal pocket, clot formation occurs with the fibrin clot covering the exposed connective tissue. Within hours the connective tissue begins to produce granulation tissue that buds from the surface. This is soon covered and infiltrated by a great number of neutrophils. The healing wound surface consists of a moderately inflamed connective tissue covered with granulation tissue, a layered zone of neutrophils and a clot in that order. Epithelium begins to proliferate from the margins of the wound and migrates cell by cell (at about 0.5mm per day) under the clot through the neutrophil zone and over the granulation tissue. The epithelialization of the gingivectomy wound is usually complete within 7–14 days following surgery (Engler et al.1966; Stahl et al.1968). During the following weeks, a new dentogingival unit is formed. The fibroblasts in the supra- alveolar tissue adjacent to the tooth surface proliferate (Waerhaug 1955) and new connective tissue is laid down.

Complete healing of the gingivectomy wound takes 4–5 weeks, although from clinical inspection of the surface of the gingiva, it may appear to be healed after approximately 14 days (Ramfjord et al.1966).

Healing after gingivectomy by electrosurgery

Some investigators report no significant differences on gingival healing after resection by electrosurgery and resection by periodontal knives. On the contrary, other researchers have reported delayed healing, greater reduction in gingival height and a more bone injury after electrosurgery. If deep resections are used close to the bone, electrosurgery can produce gingival recession, bone necrosis, and sequestration, loss of one height, furcation

involvement, and tooth mobility that do not occur with the use of periodontal knives

3) Healing Following Flap Surgery

Many surgical procedures involve the use of flaps of various designs. The union of approximated granulating surfaces, results in healing by primary intention. The better the wound approximation and smaller the clot, the more rapid the epithelial bridging between the cut soft tissue surfaces occurs, thereby sealing off the slower maturing connective tissue from the oral environment. In gaping wounds, the epithelium will migrate farther into the depths along the cut surfaces, in fact it may cover all exposed maturing granulation tissue and line the wound. Thus a relatively large mass of clot is soughed. This type of healing is termed as healing by secondary intention.

Stages of Healing Following Flap Surgery

a) **Immediately after Suturing** (≤24 HOURS): A connection between the flap and the tooth or bone surface is established by a blood clot, which consists of a fibrin reticulum with many polymorphonuclear leukocytes, erythrocytes, debris of injured cells, and capillaries at the edge of the wound. Bacteria and an exudate or transudate also result from tissue injury.

b) **One to Three Days after Flap Surgery:** The space between the flap and the tooth or bone is thinner. Epithelial cells migrate over the border of the flap, and they usually contact the tooth at this time. When the flap is closely adapted to the alveolar process, the inflammatory response is minimal.

c) **One Week after Surgery:** An epithelial attachment to the root has been established by means of hemidesmosomes and a basal lamina. The blood clot is replaced by granulation tissue derived from the gingival connective tissue, the bone marrow, and the periodontal ligament.

d) **Two Weeks after Surgery:** Collagen fibers begin to appear parallel to the tooth surface. Union of the flap to the tooth is still weak because of the presence of immature collagen fibers, although the clinical aspect may be almost normal.

e) **One Month after Surgery:** A fully epithelialized gingival crevice with a well - defined epithelial attachment is present. A functional arrangement of the supracrestalfibers is beginning.

4) Healing of Soft Tissue Grafts –

a) Healing of pedicle soft tissue grafts

Healing in the Areas Surrounding The Recession Defect

It is the area where the recipient bed consists of bone covered by connective tissue. The pattern of healing is similar to that observed following a traditional flap operation. Cells and blood vessels from the recipient bed as well as from the tissue graft invade the fibrin layer, which is gradually replaced by connective tissue. After 1 week, a fibrous reunion is already established between the graft and the underlying tissue.

Healing in the area of denuded root surface

It is the area where the pedicle graft is in contact with the denuded root surface. It was studied by Wilderman and Wentz (1965) in dogs who suggested that the healing process can be divided into four different stages:

1) **Adaptation Stage (From 0 to 4 Days):** The laterally repositioned flap is separated from the exposed root surface by a thin fibrin layer. The epithelium covering the transplanted tissue flap starts to proliferate and reaches the tooth surface at the coronal edge of the flap after a few days.

2) **Proliferation Stage (From 4 to 21 Days):** In the early phase of this stage, the fibrin layer between the root surface and the flap is invaded by connective tissue proliferating from the subsurface of the flap. After 6–10 days, a layer of fibroblasts is seen in apposition to the root surface. These cells are believed to differentiate into cementoblasts at a later stage of healing. At the end of the proliferation stage, thin collagen fibers are formed adjacent to the root surface, but a fibrous union between the connective tissue and the root has not been observed. From the coronal edge of the wound, epithelium proliferates apically along the root surface.

3) **Attachment Stage (From 27 to 28 Days):** During this stage of healing, thin collagen fibers become inserted into a layer of new cementum formed at the root surface in the apical portion of the recession.

4) **Maturation Stage:** This last stage of healing is characterized by continuous formation of collagen fibers. After 2–3 months, bundles of collagen fibers insert into the cementum layer on the curetted root surface in the apical portion of the recession.

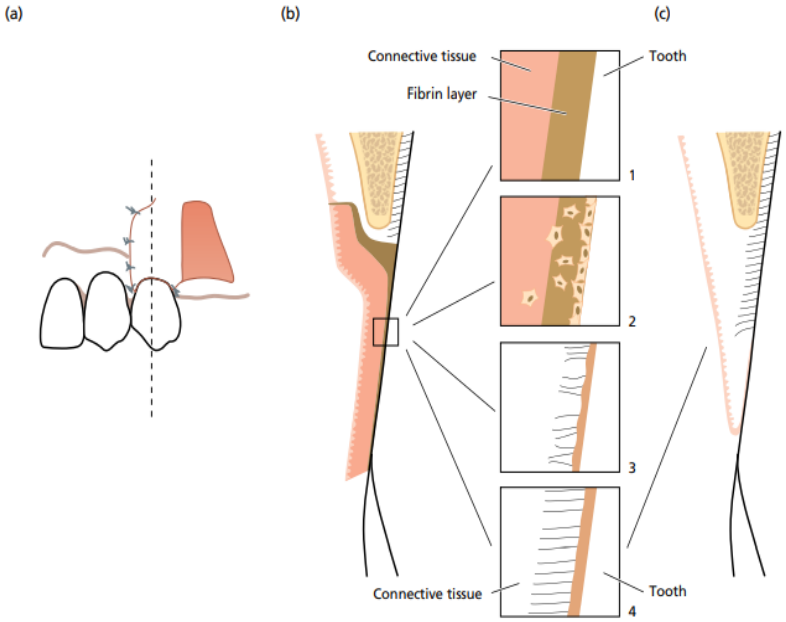

Figure 2: (a) Schematic drawing illustrating healing following treatment of a localized soft tissue recession with a pedicle graft. (b) Cross-section through the area immediately after operation. The framed areas (1–4) illustrate the four stages into which the healing process can be divided. (c) Area after healing. Approximately 50% of the successfully covered defect may show new connective tissue attachment.

B. Healing of Free Soft Tissue Grafts

Survival of a free soft tissue graft placed over a denuded root surface depends on diffusion of plasma and subsequent revascularization from those parts of the graft that are resting on the connective tissue bed surrounding the dehiscence.

Healing of Recipient Site: Healing of free soft tissue grafts placed entirely on a connective tissue recipient bed were studied in monkeys by Oliver et al. (1968) and Nobuto et al. (1988). According to these authors, healing can be divided into three phases:

1) Initial phase (from 0 to 3 days). During these first days of healing, a thin layer of exudate is present between the graft and the recipient bed. During this period the grafted tissue survives with an avascular ―plasmatic circulation from the recipient bed. Therefore, it is essential for the survival of the graft that a close contact is established to the underlying recipient bed at the time of operation. A thick layer of exudate or a blood clot may hamper the ―plasmatic circulation and result in rejection of the graft. The epithelium of the free graft degenerates early in the initial healing phase, and subsequently is desquamated. The area of the graft over the avascular root surface must receive nutrients from the connective tissue bed that surrounds the recession. Thus, the amount of tissue that can be maintained over the root surface is limited by the size of the avascular area.
2) Revascularization phase (from 2 to 11 days). After 4–5 days of healing, anastomoses are established between the blood vessels of the recipient bed and those in the grafted tissue. Thus, the circulation of blood is re-established in the pre- existing blood vessels of the graft. The subsequent time period is characterized by capillary proliferation, which gradually results in a dense network of blood vessels in the graft. At the same time, a fibrous union is established between the graft and the underlying connective tissue bed. The re-epithelialization of the graft occurs mainly by proliferation of epithelium from the adjacent tissues. If a free graft is placed over the denuded root surface, apical migration of epithelium along the tooth- facing surface of the graft may take place at this stage of healing.
3) Tissue maturation phase (from 11 to 42 days). During this period, the number of blood vessels in the transplant is gradually reduced, and after approximately 14 days the vascular system of the graft appears normal. Also, the epithelium gradually matures with the formation of a keratin layer during this stage of healing. The establishment and maintenance of a plasmatic circulationbetween the recipient bed and the graft during the initial phase of healing is critical in this kind of therapy.

Therefore, in order to ensure ideal conditions for healing, blood between the graft and the recipient site must be removed by exerting pressure against the graft following suturing.

In later stages of healing – CREEPING ATTACHMENT PHENOMENON occurs which is the coronal migration of the soft tissue margin. This occurs as a consequence of tissue maturation over a period of about 1 year post treatment.

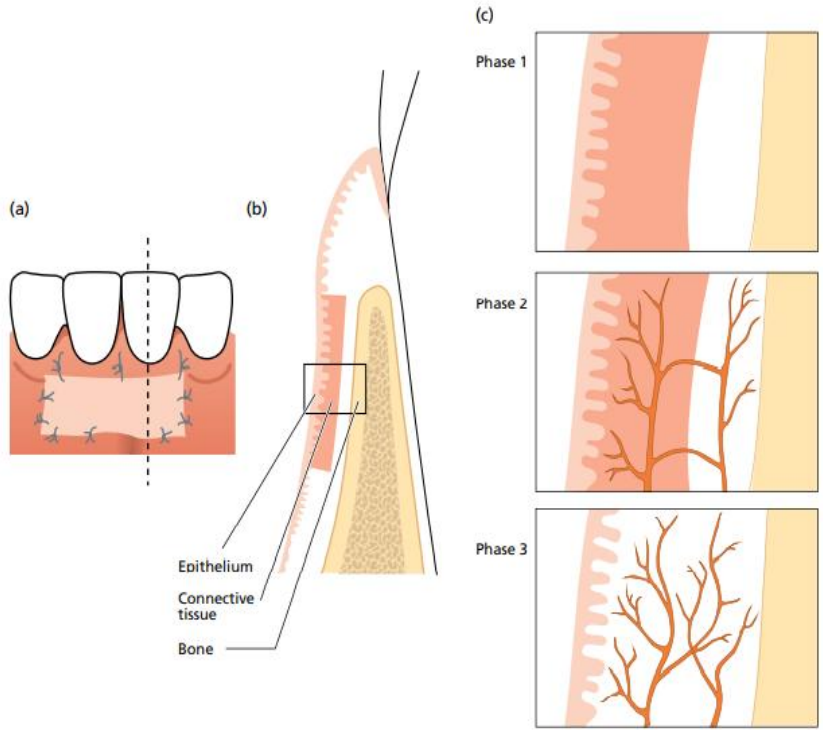

Figure 2: Schematic drawings illustrating healing of a free gingival graft placed entirely on a connective tissue recipient bed (a). (b) Cross-section through the area. The framed areas (c) illustrate the three phases into which the healing process can be divided.

Healing of the donor site

The donor site for a free gingival graft is an open wound that cannot be sutured and for which primary closure is not possible. Hence, the healing occurs by fourth intention. The 3 phases of fourth intention healing that occurs at donor site

1) During the first phase, which was shown to be delayed by stress events, the wound area is progressively (1 - 3 days) covered by an exudate or/and by a boot cot layer which acts as a protection mechanism from external stimuli.
2) In second phase (4 - 10 days), epithelial cells migrate from the adjacent tissues to completely cover the denuded area.
3) Finally, during the maturation phase (11 - 42 days), the epithelial layer becomes normally keratinized.

HEALING OF SUB - EPITHELIZED CONNECTIVE TISSUE GRAFT The subepithelial connective tissue graft (i. e., Langer procedure) is indicated for larger and multiple defects with good vestibular depth and gingival thickness to allow a split - thickness flap to be elevated. Adjacent to the denuded root surface, the donor connective tissue is sandwiched between the split flaps. This technique was described by Langer and Langer in 1985.

HEALING AT THE DONOR SITE During the sub - epithelized connective tissue grafting procedure, the donor tissue is obtained from the undersurface of the palatal flap, which is sutured back in primary closure, hence the healing occurs by primary intention.

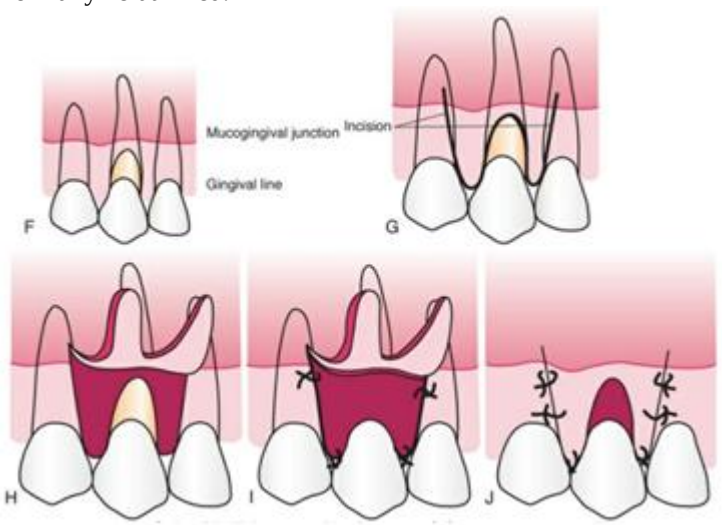

Figure 8: Subepithelial connective tissue graft for root coverage. (A through E) Sagittal views. (A) Preoperative view of facial recession on a maxillary central incisor. (B) Split - thickness incision for the recipient site. (C) Split - thickness flap is reflected. (D) Connective tissue is placed over the denuded root surface. The apical portion of the donor tissue is placed

between the split - thickness flaps. (E) Recipient flap is closed. Subepithelial connective tissue graft is used for root coverage. (F) Gingival recession. (G) Vertical incisions to prepare the recipient site. (H) Split - thickness flap is reflected. (I) Connective tissue is sutured over the denuded root surface. (J) Split - thickness flap is sutured over the donor connective tissue.

Complications during wound healing

Complication is a disease or disorder arising as a consequence of another disease. some complications are avoidable whereas some are inevitable under certain circumstances. The clinician should be able to diagnose the aetiology and provide the proper management of these complications without causing much of discomfort to the patients.

A. General Complications Arising after Periodontal Surgery

1) **Bleeding:** Following periodontal surgery, haemorrhage occurs which ranges from a minor leakage or oozing at the site, to extensive bleeding at the surgical site. Though some amount of bleeding is considered normal post operatively within 24 hours. Bleeding in a surgical patient can be classified as following:

- **Primary Bleeding**: in this the bleeding occurs during the intraoperative period. This is mostly resolved during the surgery, but if any major haemorrhages are recorded, then the patient is monitored closely post - operatively.
- **Reactive Bleeding:** occurs within 24 hours of surgery. Mostly it occurs when a ligature slips.
- **Secondary Bleeding:** occurs 7 - 10 days post - surgery. Secondary bleeding is often due to erosion of a vessel from a spreading infection due to contaminated wound.

Management - 1. Find the source of bleeding and 2. Then the approach for its management should be planned.3. In case of mild bleeding a pressure pack can be applied for 15 - 20 minutes.4. Still if bleeding is persistent then haemostatic agents like surgicel, gelfoam, microfibrillar collagen (Avitene) etc. can be used.5. If the bleeding is arterial, then ligating the vessel is considered as the best option

2) **Swelling:** Swelling is considered as the body's normal reaction to surgery and repair process. The swelling becomes apparent after the day following surgery and will reach its maximum within 2 - 3 days post - operatively. Swelling after an injury or surgery occurs as a result of increased blood supply to the affected body part, thus bringing extra nutrients to promote healing. Although swelling is considered normal until it interferes with healing.
   Management: Though the swelling subsides within 4 - 5 days in case if it doesn't then use of antibiotics, corticosteroids, surgical approach to manipulate soft and hard tissues should be considered and lastly considering alternative surgical approaches like piezo surgery, cryosurgery that are less traumatic to tissues should be done.
3) **Postoperative Pain:** Postoperative pain experienced within the first 3 days after surgery is considered normal and should progressively diminish throughout the healing phase. Postoperative pain can occur as a result of - Extensive and long surgical procedures; Poor tissue handling; Poor infection control; Patients who underwent the procedures that involved mucogingival / bone or surgeries with large wounds; etc.
   Management For relieving pain initially certain medications like nonsteroidal anti - inflammatory drugs (NSAIDs), such as Diclofenac (1 mg/kg) and Ibuprofen, Paracetamol (15 mg/kg) can be prescribed.
4) **Root Hypersensitivity**: A minimal root hypersensitivity is considered normal post periodontal surgery, as it gradually reduces in about 2 weeks.
   Causes
   - During periodontal therapy, scaling and root planing removes the outer layer of hyper mineralized dentine and thus leaves the surface expose to the effect of hydrodynamic phenomenon.
   - Surgical periodontal treatment, usually involves complete debridement of root surface.
   - Post - operative recession of soft tissue further exposes the dentinal tubules.
   - Patient inability to maintain plaque control in the healing phase further complicates the problem.
   - Management - Though the sensitivity decreases around 2 weeks but if it doesn't then it is recommended to use desensitizing agents like sodium fluoride, stannous fluoride, calcium sodium phosphosilicate bioactive glass (NovaMin); resins, varnishes, toothpastes (occlusion of dentinal tubules); lasers, etc.
5) **Increased Tooth Mobility:** Excisional procedures, particularly with flap retraction and the accompanying removal of interdental tissues, actually devoid a tooth of gingival and periosteal support on a temporary basis. Initial reattachment may be evident in the first 10 - 14 days after surgery which may be the cause of transient mobility following which more advanced collage nation and renewal of the gingival attachment to tooth and bone occurs which may require 30 - 45 days or more days.
   Management - After 30 - 45 days if mobility persists then the etiological factor for mobility should be identified and corrected through occlusal adjustment and finally splinting should be done to stabilize the teeth. Although if the mobility is still progressive then extraction can be considered as an option.
6) **Postoperative Bacteremia:** There is huge microbial challenge to the patient during periodontal surgery. The occurrence of post - surgical bacteraemia depends on amount of trauma imposed during surgery. It is documented that 88% of all blood cultures are positive after periodontal therapy.
   Transient bacteremia can be effectively treated by giving antibiotic prophylaxis before surgery. Amoxicillin is considered to be highly effective in reducing post - operative bacteremia in periodontal flap surgery as well as in preventing the possible sequelae (infective endocarditis and other systemic maladies) in susceptible patient.
7) **Delayed Wound Healing:** Wound healing, as a normal biological process in the human body, is achieved through four precisely and highly programmed phases: haemostasis, inflammation, proliferation, and remodelling. By 7 days surface epithelisation gets completed following periodontal surgery. CAUSES - A.

The most probable cause of delayed wound healing is infection which results in dead necrotic tissue which promotes bacterial growth.
Other causes include–

- Wound dehiscence (unapproximated flap margins),
- Hematoma,
- Stitch abscess (infection of suture track),
- Foreign substances (like calculus, tooth fragments, periodontal pack),
- Allergic reactions to graft material, suture material, periodontal pack,
- Tight closure via suturing.

Thorough debridement and irrigation followed by prescription of antibiotics and analgesics usually lowers down the symptoms and accentuates wound healing.

B. Complications arising during each step of the procedure employed

1) **Local Anaesthesia Related:** The most common complications arising from local anaesthesia via needle insertion or is attributed to solution include toxicity, syncope, allergy, trismus, paraesthesia etc.

- ***Local Anaesthetic Toxicity:*** is due to systemic absorption of an excessive amount of the drug. Because local anaesthetics block conduction in many tissues in addition to the peripheral nerve, toxicity could result if sufficient amounts of the anaesthetic reach these other tissues, such as the heart or brain. Signs and symptoms – Loos of consciousness, talkativeness, and agitation, along with increased heart rate, blood pressure, and respiratory rate. ManagementAdequate oxygen supply should be ensured, cardiovascular status should be assessed throughout and medical assistance should be provided.
- ***Syncope:*** most often occurs when the blood pressure is too low (hypotension) and the heart doesn't pump a normal supply of oxygen to the brain. Signs and symptoms – Pallor, cold, sweaty, dizzy, nausea, loss of consciousness, dilated pupils. Management - 1. Place the patient in supine position with slight head down or elevate the legs (to increase cerebral circulation). Recovery is almost instantaneous if the patient has simply fainted.2. Then maintain airway, check pulse (if absent, indicates cardiac arrest), and start CPR immediately.3. To regain consciousness aromatic ammonia ampoules can be administered.4. If pulse is palpable and the patient has not completely lost consciousness, four sugar lumps may be given orally or intravenous 20 ml of 20 - 50% sterile glucose in case of hypoglycaemia.
- ***Allergy:*** is a hypersensitive reaction that occurs through exposure to an antigen (Ag) such as a drug (as L. A. agent) which the patient has been previously exposed to it, resulting in an Ag - Ab reaction. Management Allergic reactions can be effectively managed by the administration of anti - histaminic (benadryl 20 - 40 mg IV or IM.), epinephrine 1: 1000 concentration 0.3 mg SC. or IM. Bronchodilator via inhaler, corticosteroid 100mg IV. Hydrocortisone hemisuccinate.
- ***Paraesthesia:*** occurs when patient reports feeling numb many hours or days after a local anaesthetic injection. Trauma to the nerve is the most common cause of this. It could be transient occurring for hours, days, or months. Management Discomfort to patient can be minimised by the use of medications which include the immunosuppressant prednisone, intravenous gamma globulin (IVIG), anticonvulsants such as gabapentin depending on the underlying cause.
- ***Hematoma:*** can occur due to injury of the blood vessel by penetration of needle to far distally during Posterior superior alveolar nerve block. Hematoma may or may not result in the formation of puncture of vein by needle but perforation of artery subsequently result in hematoma which rapidly increases in size until the treatment is instituted, due to significantly greater blood pressure within the artery.

Emergency management begins by gently cleaning the mouth and locating the source of bleeding and the application of cold compress, pressure packs, or styptics. Tranexamic acid - 500 mg in 5 ml by slow IV injection is the drug of choice.

2) **Flap Related**- Most commonly occurs due to improper incisions which if it is not made up to the bone/root surface could result in inappropriate visibility and access of operative area or could cause overexposure of bone leading to bone resorption; improper debridement which may be considered as crucial factor in the success of periodontal therapy; also, improper suturing which affects the flap approximation and can lead to reoccurrence of disease.
3) **Graft Related-** Loosened sutures could lead to displacement of grafts or contamination of graft. Inadequate size of the graft or improper root preparation for graft may lead to failure of graft. Also, allergic reaction to the grafts are rare but can occur in a hypersensitive patient. Commonest failure associated with root coverage procedures is recipient bed is too small to provide adequate blood supply.

Monitoring of Healing

Post - surgical wound healing monitoring is mainly performed by wound inspection after careful food and plaque debridement.

1) **Suture Removal-** Suture monitoring and removal after proper evaluation of soft tissue healing progression is also an integral part of wound healing monitoring. Since sutures have been shown to exert both an adverse influence on flap blood circulation and an inflammatory reaction in surrounding tissues, they should be removed according to each individual situation and not after a routine 7 - 10 day period. Early suture removal may lead to dehiscence of the wound. However, loose sutures do not play a role in wound healing and can be pulled off during function causing tissue lacerations which can interfere with the healing process, so they should be removed early. Lastly, when a muco - periosteal flap is replaced in its pre - surgical position rather than an apical one, sutures should be removed later than 7 - 10 days since flap adherence to the root surface is impeded by early gingival epithelial cell apical migration.
2) **Probing-** Probing of periodontal and peri - implant soft tissue is another important tool in post - surgical clinical

monitoring but it should not be performed before tissue healing is complete, usually 2 weeks after sub - gingival scaling and root planing and 2 months after both gingivectomy and implant prosthesis application. However, probing re - evaluation after scaling and root planing should be performed after 4 weeks, when soft tissues reach complete maturation and the patient has had sufficient time to acquire practice with oral hygiene techniques.

3) **Follow Up Visits**- Scheduling post - operative visits is somewhat different, depending on the type and complexity of surgery, occurrence of intra - operative accidents, risk of post - surgical complications, surgeon experience, patient compliance, and possible application of periodontal dressing.

If no surgical accidents occur and no dressing is applied, the first follow - up visit can be scheduled 1 week after surgery, when the suture is usually removed, and at least another post - operative visit is recommended at the second or third week.

Patients undergoing regenerative therapies with membranes should be seen more frequently during the first 2 - 3 weeks for professional tooth cleaning and to allow early discovery of any flap dehiscence with possible membrane or graft material exposures. In these patients suture removal is usually postponed from 10 to 14 days after surgery, until complete tissue healing occurs, although at 14 days some sutures can be lost, at which point they should be seen for monitoring every 1 - 2 weeks. Since regenerative procedures require that sutures be left in for a longer period, a careful choice of suture materials is of paramount importance. However, an acute inflammatory reaction is common for all suture materials. For example, expanded polytetrafluoroethylene (e - PTFE) seems to induce a weaker inflammatory response and more rapid tissue repair. Moreover, monofilament sutures seem less able to conduct bacteria than braided sutures. The choice of appropriate suture material and the correct timing for its removal are therefore crucial in reducing wound inflammation and improving tissue healing.

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