The use of CO_2 Laser In Correction of Ankyloglossia

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TABLE OF CONTENTS

List of Figures

Introduction

Laser technology is developing very quickly. New Lasers with a wide range of characteristics are available today and are being used in the various fields of dentistry. The search for new devices and technologies for dental procedures was always challenging and in the last two decades much experience and knowledge has been gained.[1] The History of laser began with the name of Albert Einstein, who in 1917 was the first to describe the physical principles of the stimulated emission of radiation.[2]

The carbon dioxide laser was developed in 1964 by Patel,[3] while Polanyi [4] was the first to perform a surgical procedure with a CO_2 Laser. The first reported application of a laser for maxillofacial surgery was by Lenz[5] who used an argon Laser to create a naso antral window. Many researches have been directed to evaluate the healing process of laser surgery, in comparison to the conventional scalpel surgery using experimental animals.[6]

Many areas of routine CO_2 Laser use for soft – tissue surgery have developed during the past 40 years, including orofacial surgery and periodontal applications.[7] During surgical procedures, lasers have specific advantages over other instrumentation options. The laser energy is bactericidal and viricidal, creating a sterile surgical site. Wound contraction by cicatrix formation is reduced, especially when dealing with lesions of the oral mucosa at the vestibule, oral commisures or vermillion. Access to difficult to reach anatomic sites, hemostasis and ablation of diseased tissue as well as a decreased risk to adjacent anatomic structures have directed surgeons to select this device.[6, 8-10]

However, as with any type of surgical instrumentation, Lasers have limitations and disadvantages. Operation of this technology requires didactic and clinically oriented instruction, as well as trained ancillary assistants.[8]

The advantage of Laser beams over the scalpel or thermocautery, however lies in the precise excision and ablation of tissue in areas where surgical access is limited. Yet thermal effects may lead to extended zones of thermal damage similar to those known from thermocautery and may cause complications in wound healing and re - epithelization.[11]

In this study we are going to evaluate the healing process of laser in comparison to the conventional scalpel for the treatment of Ankyloglossia. The following clinical criteria will be assessed, edema, swelling, pain and healing. Our results will depend on the clinical evaluation and follow up of the wound healing process in both applied techniques.

Review of Literature

Healing of surgical wounds.

Healing of a wound is a dynamic process involving soluble mediators, a variety of cells, and extracellular matrix. These components are involved in a number of different processes or steps in healing, including coagulation, inflammation, fibroplasia, collagen deposition, epithelization and scar contraction with remodeling.[12]

Healing terminology

A Wound: It represents an anatomic or functional interruption in the continuity of a tissue that is accompanied by cellular damage and death. It may be inflicted by physical, chemical or biologic injury.[13]

Healing: It is the restoration of the integrity to an injured tissue.[13]

Regeneration : When the end product of the healing process is a tissue which is structurally and functionally indistinguishable from its preinjury state, regeneration has taken place.[13] Based on their ability to regenerate, cells are classified as *Labile cells,* which divide throughout their life span. *Stable cells*, they exhibit a low rate of duplication but can undergo rapid proliferation in response to injury. *Permanent cells*, do not divide in postnatal life.[14]

Repair : When the tissue integrity is reestablished through the formation of a fibrous connective tissue scar, healing has occurred through repair.[2]

Wound contraction : Is a reduction in the size of a wound mediated principally by myofibroblasts. It reduces the size of an open defect by as much as 70% and results in faster healing.[13]

Types of wound healing

Wound healing was classified into two types, either by primary or secondary intention.

Healing by primary intention:

It occurs when the wound edges are in good apposition. It is the least complicated example of wound repair, as the incision causes death of a limited number of epithelial cells, connective tissue cells as well as minimum disruption of the basement membrane continuity.[15]

Healing by secondary intention.

It involves filling a tissue defect through formation of connective tissue. The large tissue defect leads to the presence of more fibrin, necrotic debris and exudates. This consequently makes the inflammatory reaction more intense.[6]

The paradigm of the healing process is divided to five cellular phases [16]:

Inflammatory phase

Inflammation is the initial response to injury.[16] The function of inflammation is to mobilize all the defenses of the body and bring them to the site of battle with the purpose of overwhelming the source of injury[17]

The cardinal signs of inflammation are caused by changes in the blood vessels i.e. its dilatation leading to erythema and separation of the endothelial cells allowing plasma extravasation and so produces localized edema.[18] The appearance of one type of inflammation or the other depends on the type and intensity of the irritant and the nature of the host (eg. Species, age, nutritional, hormonal and immunologic status).[14]

Inflammation is commonly divided into acute and chronic types:

Acute inflammation: It is of relatively short duration, and its main characteristics are the exudation of fluid (protein rich exudate) and the emigration of leucocytes, predominantly neutrophils.[18,19]

Chronic inflammation: It is of larger duration and is associated histologically with the presence of lymphocytes and macrophages and with the proliferation of blood vessels and connective tissue.[18]

The tissue trauma and local bleeding results in the activation of the kinin system. For example factor X II (inactive kinin precursor) is activated by vessel injury and collagen exposure. This activates a series of intravascular events that ultimately results in bradykinin production in the area of necrosis. It is a short – term vasodilator that results in the initiation of biochemical reactions such as activation of clotting factors, and complement fixation.[20]

On the cellular basis, platelets come in direct contact with the exposed endothelial wall leading to their activation. Activated platelets are sticky and aggregate to form a plug in order to occlude the small vessels and amplify the clotting mechanism. Degranulation of these platelets releases serotonin and histamine. Histamine is a short acting vasodilator responsible for reversible separation of the vascular endothelial cells to allow the migration of leukocytes, plasma proteins and erythrocytes to the interstitial space.[16]

The Neutrophils are the first cells to populate the wound site during the 2nd and 3rd days post- injury. They serve to control infection by phagocytosing bacteria and lysing devitalized tissue.[20] The migration of the neutrophils to the site of injury first starts by adhesion to the endothelial wall, then its passage to the extravascular space through the

junctions between the endothelial cells. They then migrate to the site of injury by the concentration gradient of the chemotatic factors and produce oxygen metabolites to kill the phagocytosed cells. The neutrophils are unable to regenerate their enzymes and so they decay after Phagocytosis.[21]

By day 5 or 6, macrophages become the predominant phagocytic cells in the inflammatory tissue. They come from macrophages resident in the tissue at the site of injury or from circulating blood monocytes that reach the site of injury. Attracted to the wound by chemo attractants like fibrin degradation products, the actively phagocytic macrophages continue the process of wound microdebridement initiated by the neutrophils.In addition to killing bacteria and producing vaso active mediators, macrophages also release a series of monokines like fibroblast stimulation factors and fibroblast chemotactic factors that are believed to direct subsequent healing activity.[16]

In contrast to the neutrophils, the macrophages continue to synthesize their enzymes, thus they persist for a longer time and phagocytose the decayed neutrophils. They produce growth factors as transforming growth factor-B (TGF-B) that regulates the macrophage function by the production of macrophage chemotactic factor, inhibiting factor, and activating factor. They also secrete platelet Derived growth factor (PDGF), and basic fibroblast growth factor (b FGF) necessary for initiation and propagation of granulation tissue.The (PDGF) and (b FGF), serve as stimulants for fibroplasias, neovascularization and endothetial cell participation in connective tissue repair.[21] The (bFGF) significantly accelerates granular tissue formation, reepithelization and collagen

maturation. It simulates cell proliferation of the basal cell layer in the regenerating epithelium.

The defence mechanism against bacteria is maintained, however by lymphocytes that do not migrate to the area of acute inflammation except if the injurious agent is antigenic. They are peaked by the 8^{th} to 14^{th} day post-injury. The sensitized cells cause antibody, production by the plasma cells and lymphokine production by the T-cells. More importantly, the sensitized lymphocytes become memory cells that will help to eleminate a similar antigen bearing injurious agent on the next exposure.[22] Those cells participate in the secretion of Transforming growth factors (TGF-B), which has unique and potent immunoregulatory properties.[21] It is also involved in the regulation of all phases of wound healing and tissue remodeling.[23]

Connective tissue Restitution

Repair begins early, sometimes 24 hours after injury, proliferation of fibroblast and vascular endothelial cells form a specialized type of tissue called granulation tissue. Connective tissue Restitution takes place in four steps as follows:[19]

A- Angiogenesis :

Angiogenesis (neovascularization) are the capillary buds, sent out from the pre existing vessels to produce a new one.[24] It occurs within the first few days after injury as it is stimulated by local tissue hypoxia due to the oxygen consumed by the phagocytic cells.[6] Recently, it was found that vacuum–assisted closure (VAC) also stimulates angiogenesis.[25] Revascularization supplies nutrition necessary for healing.[26]

A series of steps are needed for the development of new capillary vessels. First, formation of a capillary sprout through proteolytic degeneration of the basement membrane of the parent vessel. Second, migration of the endothedial cells towards the angiogenic stimulus. Third, proliferation of the endothelial cells just behind the leading front of the migrating cells.Fourth, maturation of the endothelial cells which includes inhibition of the growth and maturation of the capillary tubes. Fifth, recruitment of the periendothelial cells e.g vascular smooth muscle cells to support the endothelial tubes providing maintenance for the vessels.[19]Once new vascular anastomoses are established, rising oxygen tensions depress further release of these angiogenic factors.[16]

B- Fibroblastic proliferation and migration

Fibroblasts begin to proliferate in the wound area the 2nd day after injury. They appear to originate primarily from the perivascular connective tissue. Fibroblastic growth is aided by fibrin strands of the blood clot, which serves as a scaffold for invading fibroblasts and by the new vasculature supplying fresh nutrients to the cell. The aim of fibroblastic invasion is the bridging of the wound edges by collagen strands and the production of Extra cellular Matrix (ECM) components that provides support for the developing vessels.[16,27]Activated fibroblasts secrete extracellular matrix components, including fibronection proteoglycans and collagen types I and III.[13]

C- Deposition of Extracellular matrix (ECM) and cell matrix interactions :

Fibroblasts secrete components of the extracellular matrix. Fibronectin and hyaluronic acid are the first fibroblast glycoproteins to be deposited in

the healing wound,and proteoglycans appear later. Because proteoglycans are very hydrophilic, their accumulation contributes to the edematous appearance of the wound.[13]

Production of collagen by the fibroblasts is stimulated by the growth factors and the high tissue lactate levels produced by the inflammatory cells.[16] Significant collagen deposition in the wound is apparent by 4th day after injury. Initially type III collagen dominates, but after a week, type I is abundant and eventually becomes the major collagen of mature scar tissue.[16]

Collagen is the most common protein. Its synthesis by fibroblasts begins 4 days post injury and continues for several weeks depending on the wound size. The net collagen accumulation however depends not only on synthesis but also on collagen degradation.[19] The increasing strength of the wound over time is a manifestation of continued cross-linking and remodeling of the newly formed collagen. Elastin was located in tissues like that of the blood vessels as they required elasticity for their function.[16]

D- Remodeling of connective tissue :

Specific enzymes essential for debridment of the injured site undergo tissue remodeling in order to facilitate repair of tissue defects. These enzymes are called metalloproteinases that serve to degrade the extra cellular matrix (ECM) components. They are dependant on zinc ions for their activity.[28]The net result of ECM synthesis versus degradation is remodeling of the connective tissue frame work. Several cell types produce these enzymes as fibroblasts, macrophages, neutrophils and some