Ministry of Higher Education and Scientific Research University of Baghdad Institute of Laser for Postgraduate Studies



## Diode laser 940nm assisted coagulation and healing in extraction socket of diabetic patients

A Thesis Submitted to the Institute of Laser for Postgraduate Studies, University of Baghdad in Partial Fulfillment of the Requirements for the Degree of Master of Science in Laser / Dentistry

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2017AD

1439AH

بسم الله الرَّحْمَن الرَّحيم

نَرْفَعُ حَرَبَاتٍ مَّن نَّشَاءُ وَفَوْقَ كُلِّ خِي عِلْمِ عَلِيمَ ﴿ 76 حدق اللهُ العظيم

سورة يوسخه: الآية 76

# **Dedication**

To the memory of my Grandmother, may Allah bless your soul.

To my parents, thanks for your love, care and support.

To my husband, thanks for being in my life you are the source of my strength.

To my aunt, thanks for your unconditioned love and believe in me.

To my children, I love you my angels.

## Acknowledgements

First I would like to thank **Allah** for every boon in my life, thanks for supporting me to finish this work.

I wish to express my admiration and respect to Prof. **Dr. Abd-Alhadi Al-Janabi**, **Dean** of Institute of Laser for Postgraduate Studies for his full support and kind attention to all the students.

I wish to express my sincere appreciation to my supervisor, **Dr. Ali S. Mahmood** he has been actively interested in my work and has always been available to advise me. I am very grateful for his patience, motivation, enthusiasm, and immense support.

My thanks go to **Dr. Mohammed Karim Dhahir,** associate dean of Institute of Laser for Postgraduate Studies for his continuous support to the students.

Many Thanks are to Head of Department **Dr. Layla Mohammed** as well as all the teaching staff of Institute of Laser for Postgraduate Studies for their continuous support and efforts during the study.

Thanks to **Dr. Hussein Ali. Jawad, Lutfi Ghulam Awazli and Dr. Mohammed Al-Maliky** (Institute of Laser for Postgraduate Studies / Baghdad University) for their support, you have been a tremendous advisors for me. I would like to thank you for encouraging my research and for every information you gave to me.

I must express my gratitude to **Dr. Tamara Al- Karadaghi**. I am indebted to her for the help and scientific advice for my work.

Much gratitude for the staff in Institute of Laser lab (Institute of Laser for Postgraduate Studies / Baghdad University) for helping me in my patients investigations.

I am very grateful to the staff of Al- Elam Sector for supplying me with diabetic patients.

My thanks to **Miss Sameera Saleem Al- Wardi** (Center of Urban & Regional Planning for Postgraduate Studies / Baghdad University) for providing me with blood samples from diabetes patients.

Many thanks to **Dr. Israa Salman Al- Amiri** (collage of Dentistry /Baghdad University) for the information for paper publication.

Finally I want to thank my colleagues in the MSc period for every help and information they've given to me.

Thanks to all of them.

#### Abstract

**Background :** Diode lasers are widely used in oral soft tissue procedures they provide hemostatic effect due to their high absorption by hemoglobin, they cause no harmful effects to the surrounding hard tissue of teeth and alveolar bone due to the poor absorption of these lasers by hydroxyapatite and water which are the main components on these tissues. **Objectives:** The purpose of this work was the study the effect of 940 nm continuous diode laser in fastening clotting, prevention of dry socket and fastening healing after tooth extraction for diabetes patients. Materials and methods: Fresh blood samples were obtained from 12 diabetes patients and distributed in eppendrofs tubes as 0.5cc of blood in each one after storage in EDTA tubes to prevent their coagulation. Then the power was selected after many tests and applied to blood samples to calculate the laser assisted clotting time and compare it with the clotting time that obtained in the lab, then blood temperature was measured before, during and after laser exposure to know the thermal effect of laser on two areas 4 and 14mm deep to the blood surface for single and double laser exposures, then the laser was applied on dental sockets of 11 diabetes patients aged between 44-55 years after extraction of their teeth and follow up was in day 3, 10 and 21 after extraction and laser radiation. **Results:** according to in vitro study the distance of 12 mm between the laser fiber optic tip and blood surface with the use of 3W for 10 s diode laser exposure was the least power setting that coagulated the blood with simple elevation in temperature that wouldn't harm the periodontal ligaments and this power setting reduced the mean of clotting time to 8 s

while it was 202.5 s, also it was obvious that after patients examination in follow up appointments there were no complications and the wounds were closed in 21 days after extraction. **Conclusions:** Diode laser is safe and effective as a hemostatic agent, it also stimulates soft tissue healing in the dental socket.

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## List of Abbreviations

Abbreviations	Term
DM	Diabetes Miletus
mg	milligram (unit of weight) = $10^{-3}$ gram
dl	deciliter (unit of volume) =0.1 liter
g	gram (unit of weight) = $10^{-3}$ Kilogram
vWF	Von Willebrand factor
ADP	Adenosine diphosphate
HT	Serotonin
EC.	endothelial cells
Lum.	lumen
Mac.	Macrophage
Mat.	Matrix
Plt.	Platelet
SE	Subendothelial matrix
SM	smooth muscle
ECM	extracellular matrix
ММр	M atrix metalloproteinases
COX	cyclooxygenase
ТхА	thromboxane A
RBCs	red blood cells
РТ	Prothrombin Time

APTT	Active Partial Thromboplastin
PDGF	platelets derivative growth factors
AGEs	advanced glycation end-products
WHO	World Health Organization
EDTA	Ethylenediamine tetra-acetic acid
°C	Degree Celsius (unit of temperature)
W	Watt (unit of power)
PDT	photodynamic therapy
HpD	hematoporphyrin derivative
J	Joule (Energy unit)
LLLT	Low level laser therapy
ArF	argon fluoride
KrF	krypton fluoride laser
XeCl	Xenon monochloride
UV	Ultraviolet
CO2	Carbon dioxide
Nd-YAG	Neodymium doped Yttrium – Aluminum Garnet
Nd-YLF	Neodymium-doped yttrium lithium fluoride
۸	Wavelength
IR	infrared
PRR	Pulse repetition rate
Hz	Hertz (unit of frequency)

8	Second (unit of time)
Р	Power
ANSI	American National Standard Institute
m	meter
mm	millimeter
μm	micrometer
nm	nanometer
СТ	Clotting time
BT	Bleeding time
сс	Cubic square (unit of volume)
Hb	Haemoglobin
RBS	Random blood sugar

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**Chapter One** 

**Introduction and Basic Concepts** 

#### **Chapter One**

#### **Introduction and Basic Concepts**

#### **1.1 Introduction**

Diabetes Miletus (DM) is a systemic disease that characterized by chronic elevation in blood glucose level. The patient is considered to be diabetic when the fasting glucose level in blood is 126 mg/ dl or higher, the glycosylated hemoglobin is 6.5% or higher, random glucose level is 200mg/dl or higher (Alamo et al., 2011).

Oral disturbances associated with DM include destructive periodontal disease, salivary gland dysfunction, various types of stomatitis and delayed wound healing. Also the relationship between metabolic control of DM and oral health status is another risk for impaired wound healing in diabetic patients (Radović et al., 2016).

DM patients may develop many complications and infections after dental extraction especially if the glucose level is uncontrolled, inflammation and poor wounds healing is very common in those patients (Huang et al., 2013).

Lasers application increased in dentistry especially oral surgeries, the specific advantages of lasers are incision and excision of tissues, coagulation during operation and postoperative wounds healing. Also they produce minimal swelling and edema, no need for suturing, and less or no post- operative pain and infections (Azma and Safavi, 2013).

Low-level laser therapy (LLLT) is effective for some applications in dentistry it has been used to stimulate wound healing, enhancement of osteoblastic proliferation, collagen synthesis, lymphatic system activation, epithelial cells and fibroblast proliferation and revascularization of the surgical area (Hamad et al., 2016). LLLT is also known as laser phototherapy (LPT), biostimulative therapy (BT), Low intensity laser therapy (LILT) (Surendranath and Arjunkumar, 2013).

In this study 940 nm diode laser will be used after tooth extraction for diabetes patient to fasten socket healing and prevent post-operative complications.

#### **1.2** Oral manifestations of DM

DM affects the oral cavity especially in uncontrolled patients these effects are caused by diminishing of collagen and glycosaminoglycan formation in the gingiva this can stimulate the collagenolytic activity in the crevicular fluid and lead to destruction of the periodontal fibers so that slackening of the teeth, also DM alters the immunity function of monocytes, polymorph nuclear leukocytes and macrophages which lead to inflammation, rapid destruction and slow repairing of the tissue (Sanjeeta, 2014).

#### **1.2.1 Gingivitis and periodontitis**

Gingivitis is an inflammation of the gingival tissue only that doesn't extend to the supporting structures of the teeth, while periodontitis is an inflammation that extends to the periodontium (alveolar bone and periodontal ligaments) figures (1-1) and (1-2), this can cause teeth mobility and loss (Bissett el al., 2015).

Periodontal diseases are more common in diabetic patients than in nondiabetics. They are caused by some alterations in the immunity cells function, changes in vascularity and microflora of the gingiva and periodontium, some diabetes medications and/or destruction of collagen fibers in the supporting structures of teeth (Thayumanavan et al., 2015). These diseases are painless, progress slowly and cannot be noticed in the early stages, in advanced stages there will be bleeding, redness and recession of gingiva, bone destruction, mobility of teeth and festers of teeth pockets (Akhila and Malaiappan 2014).



Figure (1-1) Periodontal abscess in poorly controlled diabetic patient (al-Maskari et al., 2011).



Figure (1-2) Radiograph of diabetic patient demonstrating rapid and aggressive periodontitis-associated alveolar bone loss (Al-Maskari et al., 2011).

#### 1.2.2 Salivary dysfunction

Dysfunction of salivary glands can cause Xerostomia (dryness of the mouth) or Hyposalivation (deficiency in saliva secretion). Saliva plays an important role in maintaining good oral hygiene through its buffering action and antibacterial effect.

Xerostomia and hyposalivation can be seen in association with diabetes type1 and type 2(Khovidhunkit et al., 2009).

A study made by Shrimali et al in 2011 shows that hyposalivation is the most common oral manifestation of diabetes and it is seen more in uncontrolled patients. This can be caused by either alteration in salivary gland structure, reduction in microcirculation in oral cavity that may affects salivary glands or polyuria that cause reduction in body fluids. Reduction or absence of saliva causes inflammation and irritation in oral soft tissues and increases the possibility of oral infections (Al-Maskari et al., 2011).

#### **1.2.3 Dental caries**

Dental caries is an irreversible tooth destruction caused by bacterial acids that dissolve minerals in the enamel and dentine (Featherstone, 2008). Figure (1-3) shows, xerostomia and dental caries in a patient with diabetes.

The mutans streptococci and the lactobacilli species are responsible for dental caries in the presence of carbohydrates and the host which is the tooth (Featherstone, 2008).

People with diabetes develop dental caries due to reduction in salivary flow, impaired metabolism, poor oral hygiene and the presence of cariogenic bacteria (Ship, 2003).

Root caries is often seen in association with gingival recession (Marín et al., 2008).



Figure (1-3) Salivary hypofunction, xerostomia and dental caries in a patient with longstanding type 1 diabetes (Ship, 2003).

#### **1.2.4 Fungal infections**

Candida albicans species cause oral candidiasis, this is an opportunistic infection due to decrease the salivary flow, some medications, smoking, and some systemic diseases, there are two types of oral candidiasis: primary and secondary, the primary is subdivided into acute and chronic, the acute candidiasis which is pseudomembranous and erythematous, while chronic candidiasis is pseudomembranous, erythematous and hyperplastic (Al-Maskari et al., 2011).

Pseudomembranous or oral thrush is seen as a white patch that covers red or bleeding oral mucosa. The most common sites are soft palate, cheek, tongue and gingiva, the erythematous is mostly seen on the tongue; these two types can be acute or chronic, figure (1-4) shows oral thrush.

Hyperplastic or leukoplakia candidiasis is chronic white color irregular lesion seen mostly on the buccal mucosa near the commissures (Al-Maskari et al., 2011). Candidiasis is more prevalent in diabetes patients than in non-diabetic this is due to the changes in the oral environment and immunity function also the increase of glucose level in the oral fluids (Jafari et al., 2013).



Figure (1-4) Pseudomembranous candidiasis (Krishnan, 2012).

#### 1.2.5 Taste disturbances

Taste dysfunction or hypogeusia is widespread in diabetes patients. According to one study more than one third of diabetes patients have taste disturbance (Thayumanavan et al., 2015).

Taste threshold increased in diabetics who have neuropathy this can result in hyperphagia and poor glycemic regulation (Al-Maskari et al., 2011).

#### 1.2.6 Neurosensory disorder

Neurosensory disorder can cause burning mouth syndrome. This may be results from secondary candidiasis or mouth dryness (Wilson et al., 2010).

Uncontrolled diabetics are at danger of developing peripheral neuropathy and tongue pain (glossodynia) more than those with controlled glucose levels (Al-Maskari et al., 2011).

#### 1.2.7 Diseases of oral mucosa

Lichen planus (figure 1-5) is a lesion that occurs in many sites in the oral cavity, buccal mucosa is the most common site. Lesions can be found on the dorsum and lateral borders of tongue, hard palate and vermilion (Ahmed et al., 2012).

Recurrent aphthous stomatitis is seen as single or multiple vary in size (from 8 to 10 mm in diameter), painfull and shallow lesions. The most common sites are floor of the mouth, labial and buccal mucosa (preeti et al., 2011). These lesions occur in diabetes patients because of their immunity suppression that caused by glucose elevated levels (Thayumanavan et al., 2015).



Figure (1-5) Lichen planus (Thayumanavan et al., 2015).

#### **1.3** Tooth extraction

Tooth extraction or Exodontia is a combination of two principles surgical and physical, these principles if applied correctly no adverse forces will be needed to remove the tooth (Hupp, 2008).

Tooth extraction is a minor surgery that performed in the dental clinics (Soodan et al., 2015).

Tooth socket is dilated either by elevator that applied between the tooth and the socket wall or by forceps, when the blades of forceps hold the tooth a vertical force will be applied then the tooth is removed by various movements according to its morphology (Mitchell et al., 2006).

According to Kundi et al. the main cause of tooth extraction in diabetes patients is dental caries (more than 60% of extractions) followed by periodontal diseases (more than 20%) in both male and female patients (Kundi et al., 2015).

#### **1.3.1** Indications for teeth extraction

There are many indications for teeth extraction.

#### **1.3.1.1** Unrestorable dental caries

Dental caries according to some studies is the main cause of teeth extraction (Kundi et al., 2015; Al Qudah et al., 2012).

When the caries extended in a manner that cannot be restored or the treatment cost is more than replacement by prostheses tooth extraction is indicated (Hupp, 2008).

#### **1.3.1.2 Periodontal diseases**

Sever periodontal diseases are one of the main indications of tooth extraction, bone loss, tooth mobility (grade III) and deep pocket >5 mm extraction must be done (Hupp, 2008; Mohammed, 2008).

#### **1.3.1.3** Orthodontic reasons

The orthodontist will decide which and how many teeth to be extracted to get the best results (Yagi et al., 2009).

Case relapse may occur if orthodontic treatment is done without extraction (Ruellas et al., 2010).

#### **1.3.1.4** Malposed teeth

Malposed teeth must be extracted if they cause trauma to the oral soft tissues and can't be repositioned by dentist, also the over erupted teeth that have no opposing in the other jaw must be extracted if they interfered with the desired prostheses (Hupp, 2008).

#### **1.3.1.5** Impacted teeth

Impacted tooth is the tooth that doesn't erupt in the position in the age of eruption, sometimes they cause pain, inflammation and infections (Krishnan et al., 2009).

It is better to remove the impacted teeth before root formation is completed and the late teenager ages are the best ages for tooth removal because the healing is faster and better in these ages (Hupp, 2008).

#### **1.3.1.6** Supernumerary teeth

Supernumerary teeth are those teeth that exceed the normal dentition number, this case is also called hyperdontia which can be single (as shown in figure 1-6) or multiple, unilateral or bilateral and may cause many complications such as impaction, delayed eruption, malposition, space anomalies and follicular cysts (Ali et al., 2014).



Figure (1-6) Clinical and radiographic picture of supernumerary tooth (Karayilmaz et al., 2013).

#### 1.3.1.7 Cracked teeth

The crack is incomplete fracture of the teeth that can involve crows, roots or both. Cracks can be vertical or horizontal and their symptoms vary from discomfort to sever pain especially on biting (Kahler, 2008), figure (1-7). The cracked tooth must be extracted when the crack is under the alveolar bone, vertically cracked teeth must be extracted and if the crack is passing along the pulpal floor (Lynch and McConnell, 2002).



Figure (1-7) Cracked teeth (Kahler, 2008).

#### 1.3.1.8 Trauma and pathology of teeth

Trauma of teeth can cause pain and infection, if the traumatized tooth can't be treated extraction must be done (Hupp, 2008).

Pathological lesions that can't be treated by surgical removal or endodontic treatment force the dentist to remove the tooth (Hupp, 2008).

#### **1.3.1.9 Primary teeth removal**

Primary teeth can be removed if they are mobile and cause pain and discomfort to the child. Also primary teeth must be removed if they didn't fall and interfere with the eruption of permanent dentition (Mohammed, 2008).

#### **1.3.2** Limitations for teeth extraction

Tooth or teeth extraction can cause some problems, many factors or contraindications can delay or stop extraction these limitations are systemic and local (Hupp, 2008).

#### **1.3.2.1 Systemic limitations**

Tooth extraction is contraindicated because of the patient condition. Extraction can cause serious problems or complications (Hupp, 2008).

#### 1.3.2.1.1 Systemic diseases

Tooth extraction for patients with severe metabolic disorder such as uncontrolled diabetes and sever renal failure is contraindicated, diabetes patients are at high risk of infection and delayed wound healing (Alamo et al., 2011), in diabetic patients with renal failure bleeding and infection take place (Cerveró, 2008).

Patients with malignant diseases such as leukemia and Lymphoma may suffer from infections due to immunity alteration and bleeding because of platelets deficiency (Hupp, 2008).

Heart diseases can cause many complications, tooth extraction for such patients must be done in hospital with antibiotic coverage (Hupp, 2008).

Hypertension if uncontrolled > 160 systolic, > 90 diastolic for more than three months can cause cardiac and renal complications, bleeding is common when extraction is done for such patients(Hupp, 2008).

#### 1.3.2.1.2 Bleeding disorders

Bleeding disorders can be caused by deficiency in coagulating factor, platelets defects, vascular diseases and fibrinolytic disorders (Gupta et al., 2007).

The most common bleeding disorder diseases are hemophilia and von Willebrand's disease, the main cause of these diseases is factor VIII deficiency.

Hemostatic agents must be provided to assist in clot formation and stop bleeding after tooth extraction (Gupta et al., 2007).

#### 1.3.2.1.3 Pregnancy

Tooth extraction during pregnancy is safe in the interval between the endings of the  $1^{st}$  to the  $1^{st}$  month of the  $3^{rd}$  trimester, at the end of  $3^{rd}$ trimester extraction must be postponed after delivery (Hupp, 2008).

Local anesthesia is safe for pregnant woman, some antibiotics such as penicillin and cephalosporin are safe, corticosteroids are contraindicated for pregnant woman (Kanotra et al., 2010).

#### **1.3.2.1.4 Patients on medications**

Consultation with physician must be made before tooth extraction for patients on some drugs such as anticoagulants, corticosteroids, immunosuppressive agents, bisphosphonates, and chemotherapy (Hupp, 2008).

#### **1.3.2.2 Local limitations**

Complications in the extraction area will occur if the condition or disease is not controlled these include

#### **1.3.2.2.1** Area of tumor or radiotherapy

Tooth extraction in tumor site especially malignant tumor can spread malignant cells and metastasis will occur (Hupp, 2008).

Osteoradionecrosis will result if the tooth in the radiation site is extracted (Hupp, 2008).

#### **1.3.2.2.2 Pericoronitis**

Inflammation of soft tissue around the partially erupted teeth is called pericoronitis mostly seen in lower partially impacted 3<sup>rd</sup> molars (Moloney and Stassen, 2009), figure (1-8).

Extraction of tooth with sever pericoronitis is contraindicated because it can cause many complications.

Inflamed tissue must be irrigated, antibiotics must be taken by the patient and in some cases removal of upper 3<sup>rd</sup> molar is indicated to reduce the pressure on the inflamed tissue, extraction can be done when inflammation is reduced or treated (Hupp, 2008).



Figure (1-8) Pericoronitis of mandibular 3<sup>rd</sup> molar (Moloney and Stassen, 2009).

#### 1.3.2.2.3 Dentoalveolar abscess

Pulpal necrosis is the main cause of dentoalveolar abscess, pus may interfere with anesthetic solution action therefore the patient may not be anesthetized (Hupp, 2008). Pus can cause swelling in the infected area and limitation of mouth opening. The usage of antibiotics is indicated before extraction (Hupp, 2008).

#### **1.3.3** Complications of exodontia for diabetes patients

Teeth extraction sometimes causes complications which are unexpected results that worsen the patient condition and need to be treated before serious problems occur (Venkateshwar et al., 2011).

Dry socket or Alveolar osteitis is common after tooth extraction especially mandibular 3<sup>rd</sup> molar, it is painful inflammation of the alveolar bone surrounding the extracted tooth socket, clinically it is characterized by bad odor, sever pain, redness and swelling of gingiva. The bone of the socket is exposed to oral environment due to clot dislodgement (Akinbami and Godspower, 2014).

A study made by Karbassi et al. showed that 30.4% of DM patients had abnormal hemorrhage after teeth extraction due to destruction of vascular epithelium. Also 17.4% of DM patients complained from dry socket (figure 1-9) which occurs in only 1.2% of non-diabetics extracted teeth sites (Karbassi et al., 2015).

Infections, fever, swelling and pain are complications of teeth extraction of uncontrolled diabetes patients, these complications occur due to immunity alteration and defects in vascularity and healing mechanism of the tissue (Karbassi et al., 2015).



Figure (1-9) Clinical presentation of dry socket (Sharif et al., 2014).

#### **1.4** Dental managements of DM patients

Controlled diabetes patients can be treated as healthy but uncontrolled DM patients, treatment needs special care (Wilson et al., 2010). Patient medications and glucose level measurement must be known by the dentist, for type I DM patients if blood glucose level is between 100-200 mg/dl tooth extraction can be done, for patients with glucose level higher than 200 mg/dl appointment can be postponed if the case was not urgent, on prolonged treatment glucose level must be measured and insulin must be administrated when glucose level> 200 mg/dl. Patient must take half dose of his daily insulin on treatment day (Alamo et al., 2011).

There is no specific glucose level to do treatment for type II DM, patient must take his medications and breakfast before attending his appointment (Alamo et al., 2011).

Morning appointments are better because late visits and waiting time can cause stress and discomfort to the patients (Wilson et al., 2010).

Anesthesia must be used with cautions, some studies assumed that epinephrine which is a vasoconstrictor in dental anesthesia is an antagonist of insulin thus can elevate the level of glucose in blood (Balakrishnan and Ebenezer, 2013), another study said that vasoconstrictor enhanced hypoglycemia can result when high anesthetic dose is given to uncontrolled DM patients (Budenz, 2000), while a study made by Paul et al. concluded that there is no relation between anesthesia vasoconstrictor and blood glucose level (Paul et al., 2015).

During or after dental procedure the most common event to occur is hypoglycemia which is a drop in blood glucose level below the normal. The main cause is the fast metabolism of carbohydrate that enhanced by stress (Alamo et al., 2011).

The main signs, symptoms and treatment of hypoglycemia are mentioned in table (1-1).

IDENTIFICATION AND TREATMENT OF HYPOGLYCEMIA			
IDENTIFICATION			
Symptoms	Signs		
• Shakiness	• Tremors		
• Anxiety	• Tachycardia		
<ul> <li>Increased sweating</li> </ul>	<ul> <li>Altered consciousness (lethargy and</li> </ul>		
• Hunger	obtundation or personality change)		
	• Blood glucose level: < 60 mg/dl		
TREATMENT			
Conscious patient	Unconscious patient		
• Administer 15 mg of simp	ple With intravenous access:		
carbohydrates	• Administer 5 to 25 g of 50% dextrose		
• Repeat finger- stick glucose test in	immediately		
minutes:	<ul> <li>Notify the patient's physician</li> </ul>		
• Blood glucose level > 60 mg/dl: patie	Without intravenous access:		
should be asked to eat or drink (f	• Apply glucose gel inside the mouth in a		
example, a sugar-sweetened beverage)	semi obtund patient or treat with 1 mg of		
• Blood glucose level < 60 mg/dl: repe	· ·		
treatment of 15 g of simple carbohydrat	•		
and check blood glucose in 15 minute			
Continue until achieving a blood gluco			
level $> 60 \text{ mg/ dl}$	• Establish intravenous access and		
• Ask the patient to notify his/ h	her notify the patient's physician		
physician			

Table (1-1) signs, symptoms and treatment of hypoglycemia (Alamo et al., 2011).

Prophylactic antibiotics are indicated for uncontrolled DM patients, these drugs cannot replace dental treatment but they help to prevent or stop systemic or local infections (Ramu and Padmanabhan, 2012).

Wide spectrum antibiotics in association with NSAID can control many kinds of bacterial infections (Roda et al., 2007), infections must be controlled because they disturb glucose level by their effect on insulin resistance this can cause delay in wounds healing (Lalla and D'ambrosio, 2001).

#### **1.5** Wounds healing after teeth extraction

Healing after tooth extraction occurs in a complex pattern.

# **1.5.1** Normal healing processes

Wounds healing are complex reparative mechanisms by which recovery occurs (Politis et al., 2016).

Healing occurs by two main mechanisms regenerative which is the formation of the same tissue but this mechanism is limited in some tissues for example liver, neural and epithelial tissues, while reparative mechanism is the main healing process in the body, it is a replacement of the damaged parts with connective tissue (Flanagan, 2000).

# **1.5.1.1 Soft tissue healing**

Wound healing is divided in to three phases: inflammatory, proliferative and remodeling (Broughton et al., 2006).

#### **1.5.1.1.1 Inflammatory phase**

Blood coagulation is the 1<sup>st</sup> stage in the inflammatory phase, coagulation is the process by which clot is formed, it's a combination of many events which are: vasculature, platelets, coagulation factors and fibrinolytic system, these compartments interact together to achieve hemostasis when bleeding occurs (Chee, 2014).

Blood vessels are lined with endothelium this prevents the passage of blood but allowed gas exchanging and nutrition passage to the surrounding tissues, damage to the vessels cause interactions between blood and tissue factors and stimulation of series of processes which are the coagulation cascade(figure 1-10) that lead to hemostasis with specific enzymes assistance (Israels et al., 2006).

Tissue factors change Factor VII to the active factor VIIa and form a complex interaction to activate more factor VII in addition to factors IX and X, the activated factor X which is Xa converts prothrombin which is factor II to thrombin which in turn activate factors V to Va, VIII to VIIIa and XI to XIa (Israels et al., 2006).

Thrombin also converts fibrinogen to fibrin monomer and forms the active XIIIa by the activation of factor XIII as shown in figure (1-10), these active forms are very important to start hemostasis (Israels et al., 2006).

Prothrombin also produces platelets activator which is important for platelets aggregation (Israels et al., 2006).

Platelets are small, anucleate, disk shape and very numerous blood cells, their life cycle is 10 days, their shape and size make them able to support vascular endothelium when injury occurs (Harrison, 2005).

Platelets are activated when vascular damage occurs (figure 1-11) they change their shape, release their granules and aggregate together to reduce blood loss (Ghoshal and Bhattacharyya, 2014).

The granules of platelets are two types: alpha granules that contain proteins such as Von Willebrand factor (vWF) and fibrinogen which are adhesive materials, in addition there are some coagulation and growth factors, dense granules contain platelets activating materials for example ADP and serotonin (5-HT), on the platelets surfaces there are receptors that arbitrate the activation and adhesion of them (Israels et al., 2006).

Endothelial linings of blood vessels store materials that assist in platelets adhesion (figure 1-11). These materials are secreted to the vessels lumen when injury occurs, (vWF) is the protein that secreted in the small vessels, in large vessels the main protein is collagen.Von Willebrand factor in small vessels binds to the platelets by the glycoprotein Ib-V-IX complex on their surface and stimulates them, while collagen activates platelets by  $\alpha 2\beta 1$ platelets surface receptors (Israels et al., 2006).

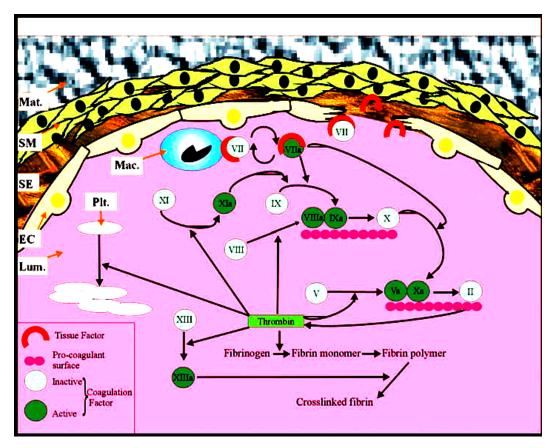


Figure (1-10) Coagulation cascade initiated by vascular damage leads to the expression of tissue factor and culminates in the generation of thrombin and cross-linked fibrin. EC = endothelial cells; Lum. = lumen; Mac. = macrophage; Mat. = matrix; Plt. = platelet; SE = subendothelial matrix; SM = smooth muscle (Israels et al., 2006).

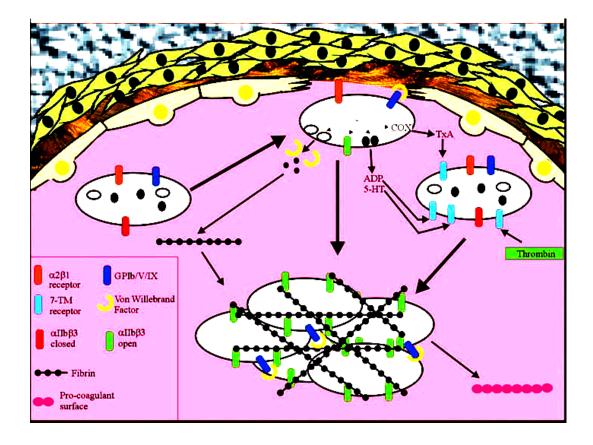


Figure (1-11) Platelet activation, initiated by vascular damage exposing subendothelial von Willebrand factor and collagen, culminates in platelet aggregation. ADP = adenosine diphosphate; COX = cyclooxygenase; 5-HT = serotonin; TxA = thromboxane A. (Israels et al., 2006).

COX-1 and TXA2 increased platelets response and act as vasoconstrictors. When platelets are activated they start to release their granules, change their structure and adhere to each other to form an interaction via the activated receptors on their surfaces, these receptors bind to each other through cross linked fibrin and few other proteins.

The connected platelets aggregation on the injury site is increasing gradually by the addition of more platelets that linked to each other by fibrin and a stable clot is formed (Israels et al., 2006). After clot formation growth factors and cytokines which are synthesized by platelets are released, cytokines mediate the inflammatory reaction to remove debris, dead cells and microorganisms, by stimulating the inflammatory cells (Politis et al., 2016); (Broughton et al., 2006).

Neutrophils are the 1<sup>st</sup> inflammatory cells that infiltrate to the injury site in addition to their debridement action they attraction of monocytes to the defected tissue, monocytes are differentiated in to macrophages, they are also assist in healing and repairing of the wound by removing the infective organisms and dead cells, also they stimulate fibroblasts to produce collagen and to start the second healing phase which is the proliferative phase (Broughton et al., 2006).

# **1.5.1.1.2** Proliferative phase

In this phase dental socket is filled with granulation tissue, new vessels formation (angiogenesis), epithelialization and collagen deposition (Cohen and Levy, 2014).

Epithelialization occurs when the basal stratum cells around the extraction wound start to divide and migrate in 12 hours after extraction, regeneration rapidly if the area is uninfected, some studies said that healing of the oral mucosa is faster than that of skin, healing time is differ between individuals it's also depends on tissue situation before surgery (Cohen and Levy, 2014).

Granulation tissue is finally formed which is the new connective tissue that contains the new vessels and a tissue matrix that supports many types of cells (Broughton et al., 2006).

Fibroblasts infiltrate to the wound site, activated by the stimulation of growth factors and form collagen then they convert to myofibroblasts which are responsible for wound contraction by making a matrix that composed of collagen type III, glycosaminoglycans and fibronectin by the stimulation of platelets derivative growth factors (PDGF) (Broughton et al., 2006).

# **1.5.1.1.3 Remodeling phase**

Remodeling or maturation phase is the final phase of wounds healing, in this phase collagen organized in a regular form and definite amount, if any condition lead to increase in collagen deposition scar will occur, if collagen deposition is less than normal the healing area will be weak, with time thicker collagen will be deposit in the area and the tensile strength of wound will increase (Broughton et al., 2006).

Clinically the wound appeared to be healed no scar is seen, histologically there is a connective under the newly formed epithelial layer (Broughton et al., 2006); (Politis et al., 2016).

Healing time varies between individuals in normal condition after tooth extraction dental socket will be closed by blood clot within minutes, in 24 hours epithelialization occurs and one week is needed to replace the clot with granulation tissue.

Interdental papilla must be intact, papillary destruction cause black triangle in the area of extraction and affect the esthetic and the health of the area (Politis et al., 2016). Figure (1-12) shows three phases of a typical wound healing response.

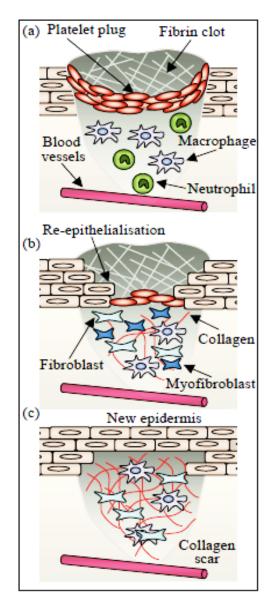


Figure (1-12) Three phases of a typical wound healing response (Rajan and Murray, 2008).

(a) Inflammation – a fibrin clot forms and platelets plug the wound; neutrophils then macrophages migrate into the wound and are responsible for bacterial destruction and removal of foreign material and cell debris.

(b) Proliferation – mediators secreted by macrophages and surrounding cells initiate proliferation and migration of keratinocytes and fibroblasts into the wound; collagen deposition and contraction.

(c) Remodeling – matrix remodeling by macrophages, fibroblasts, endothelial and epithelial cells.

#### 1.5.1.2 Healing processes in bone

Bone loss after tooth extraction differs from one patient to another, vertical and horizontal remodeling takes place, dimensional changes in the alveolar bone are clinically obvious also resorption of buccal and shifting of lingual alveolar bone of the extraction socket takes places (Politis et al., 2016).

At the apical part of the socket osteoid is started in the form of un-calcified fragments that start to calcified after three weeks and with the help of a vascular network a connective tissue is formed and filled the socket, three weeks later trabecular bone is formed and deposited, bone formation is slow down after four to six months but keep on formation and specialization for several months (Kubilius et al., 2012).

Most of the bone resorption occurs in the 1<sup>st</sup> 12 months after tooth extraction (figure 1-13) and it continue to occur and fastened by pressure force on the edentulous area (Kubilius et al., 2012).

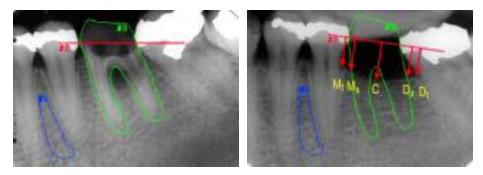


Figure (1-13a) Linear radiographi measurements from reference line to crestal bone levels: image taken before tooth extraction (Schropp et al., 2003).

Figure (1-13b) Image taken 12 months after tooth extraction (Schropp et al., 2003).

### 1.5.2 Wounds healing in diabetes patients

Healing of wounds in DM patients is delayed in compare to healthy patients of the same age due to many factors (Abiko and Selimovic, 2010).

Vascularity impairment in diabetes occurs due to vascular sclerosis which leads to reduction in the circulated blood and in oxygen (Hypoxia) which stimulates the formation of oxidant free radicals that in turn inhibit the formation of new blood vessels and destroy the junction between them (Abiko and Selimovic, 2010).

Hemostasis is impaired in DM patients, elevated glucose level affects red blood cells (RBCs) and some proteins such as hemoglobin, prothrombin, fibrinogen and other proteins to form glycation end products of these coagulation proteins (Ismail et al., 2015).

One study declared that there is an increase in Prothrombin Time (PT) and Active Partial Thromboplastin Time (APTT) in patients with long diabetes periods (Ismail et al., 2015), another study said that in addition to prolonged APTT in DM patients there is an increase in the weight of fibrinogen therefore special care is needed for DM patients to avert the excessive bleeding during teeth extraction (Ifeany et al., 2014).

Studies made on animals show that there is impaired wound healing due to reduction of collagen in granulation tissue, in human studies shows that there are defects in growth factors cause immature wound healing (Broughton et al., 2006).

DM patients with renal complications have defects in the mechanism of platelets aggregation and adhesion (Gupta et al., 2007).

Figure (1-14) shows the differences in healing mechanism between normal and DM patients.

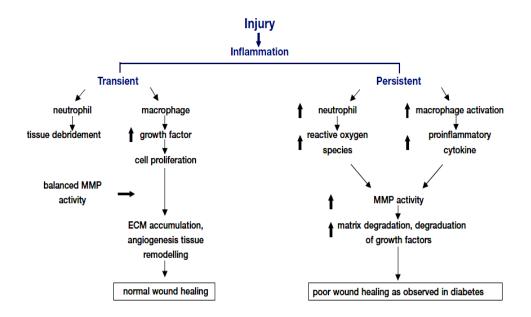


Figure (1-14) A schematic representation the steps of the wound healing process in normally healing and chronic wounds (McLennan et al., 2006).

Reduction in number of neutrophils and monocytes in the wound area due to the presence of advanced glycation end-products (AGEs), increasing the level of blood glucose lead to many defects in some immune amino acids and proteins this lead to immunity reduction then infections which lead to delay wounds healing (figure 1-15), also DM affects salivary quantity which affect the buffering action, and quality because of defects in the immunity factors of saliva therefore healing process is affected (Abiko and Selimovic, 2010). Psychological stress of DM patients causes many alteration in immunity and endocrine systems, also stress develops bad nutritional habits all these factors

affect wound healing (Abiko and Selimovic, 2010).

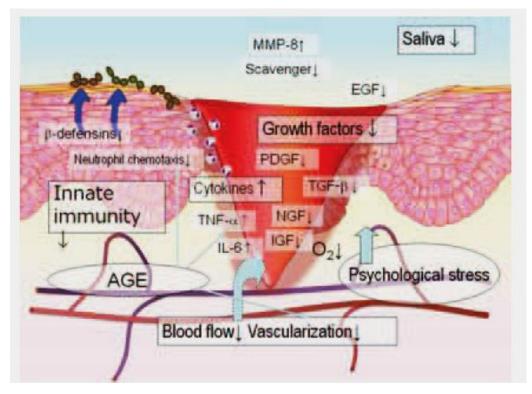


Figure (1-15) Factors that affecting wound healing in diabetes patients (Abiko and Selimovic, 2010).

#### **1.6** Laser basics

The word Laser came from 'Light Amplification by Stimulated Emission of Radiation', light is an electromagnetic energy travels as waves (Coluzzi, 2008).

Lasers are different from ordinary light by that laser light is coherent (the difference between phases is fixed), nearly monochromatic (single color), collimated (beams are travelling in parallel waves) but sometimes they diverge to an angle after certain distance (Coluzzi, 2008).

#### **1.6.1 Elements of laser system**

Laser devise composed of many components that work together to deliver the radiation, these components are:

#### 1.6.1.1 Active medium

It's the material by which laser is named, active medium can be an element, a molecule or a compound. It can be gas as in CO2 lasers, solid crystal like that in Nd-YAG, solid state semiconductor like that in diode lasers or can be a liquid such as some medical lasers (Convissar, 2015).

#### **1.6.1.2 Pumping mechanism**

Pumping mechanism is done by sources that excite the laser active medium, it can be a flash lamp or electrical source (Convissar, 2015).

#### **1.6.1.3 Optical resonators**

Optical resonators are two mirrors or polished surfaces parallel to each other at the ends of the laser cavity, they reflect the waves to produce an amplified laser beam. Cooling system is used to control the temperature inside the cavity (Convissar, 2015).

# 1.6.2 Laser tissue interaction

When laser beam hit the tissue one of these four effects will occur, as shown in figure (1-16).

# • Reflection

Reflection is the return of the laser radiation away from the incidence surface, the reflecting surface is a physical boundary between two materials of different indices of refraction (Niemz, 2007).

Reflection is called Specular when the surface irregularities are smaller than the light wavelength, incidence and reflection angles are equal, Diffuse Reflection occurs when the irregularities of the reflecting surface equal to or larger than the light wavelength, incident and reflected beams are not in the same plane and more than one beam are reflected in many directions (Niemz, 2007).

Reflection of the beam can be in any direction and may be harmful especially to the eyes, therefore protective glasses must be worn. Reflection has no effects on the surface (Convissar, 2015).

An example on this is the reflection of CO2 laser from titanium dental implants (Convissar, 2015).

#### • Scattering

Light scattered and change its direction inside the tissue, sometimes it interacts with another chromophores and absorbed by them leading to heat generation and sometimes thermal damage, in some cases scattering of light is useful and can be used for many application such as fastening the curing of composite resin (Convissar, 2015).

#### Transmission

Light is transmitted through the tissue without any effect, the transmission of laser depends on the laser wavelength and the tissue chromophores. An example of transmission is diode lasers in water because water doesn't absorb those wavelengths (David and Gupta, 2015).

The penetration of laser in the tissue depends on laser parameters and optical properties of the tissue, therefore each laser has different transmissions in different tissues (Ansari and Mohajerani, 2011).

#### • Absorption

When laser absorbed by the target tissue chromophores, desired effect will be obtained. The amount of absorbed energy depends on laser wavelength and the concentration of tissue components (water, pigments, hemoglobin and hydroxyapatite).

Laser tissue interactions occur after laser absorption by the tissue (Convissar, 2015), figure (1-17) shows the absorption of laser in oral tissues chromophores.

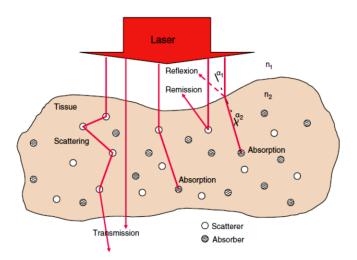


Figure (1-16) Laser effect on tissue (Steiner, 2011).

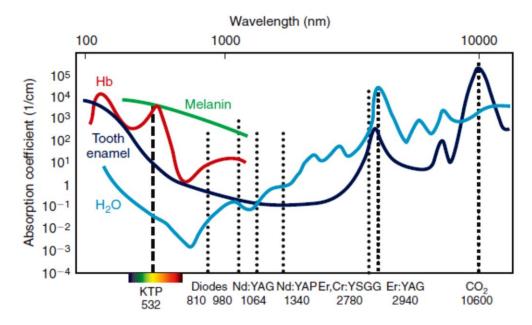


Figure (1-17) Approximate absorption curves of the prime oral chromophores (Convissar, 2015).

Laser tissue interaction after absorption can be either wavelength dependent or wavelength independent, the type of interaction depends on laser parameters such as: wavelength, exposure time, applied energy, focal spot area, energy density, and power density. Also tissue properties play an important role in the mechanism of interaction, tissue properties can be optical such as: the coefficients of reflection, absorption, and scattering or thermal properties such as: heat conduction and heat capacity (Niemz, 2007). Figure (1-18) shows a plot of laser tissue interaction.

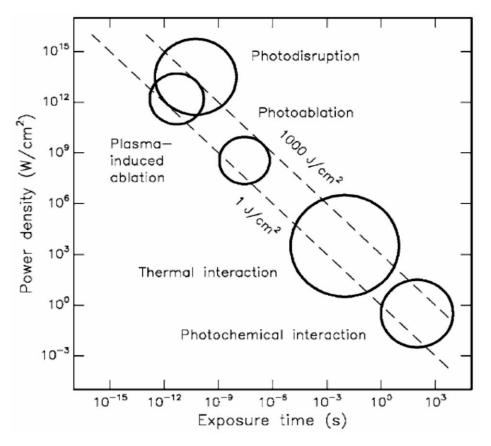


Figure (1-18) a plot of laser tissue interaction (Niemz, 2007).

## 1.6.2.1 Wavelength dependent interaction

Many interactions can occur due to laser absorption by tissue chromophore.

#### **1.6.2.1.1 Photochemical interaction**

Photochemical interaction occurs at low intensity, this interaction can be used in Photodynamic and in Biostimulation therapies (Niemz, 2007). In photodynamic therapy (PDT) a suitable chromophore (photosensitizers) e.g. hematoporphyrin derivative (HpD) 2.5–5mg per kg body weight, is injected in the body then irradiated by monochromatic radiation to trigger a photochemical reaction lead to biological transformation (Niemz, 2007). Photosensitizer helps to produce reaction in the non-absorbing tissue when it irradiated by laser, leading to produce toxic components causing an irreversible oxidation of essential cell structures (Niemz, 2007).

Photodynamic therapy application is the destruction of tumor tissues when HPD is injected into the vein a period of few days will be needed to declare the healthy tissue from the chromophore while in tumer cell chromophore concentration remains high, laser then is applied after three to seven days this lead to selective necrosis of tumer cells. Figure (1-19) shows the PDT for tumer cells (Niemz, 2007).

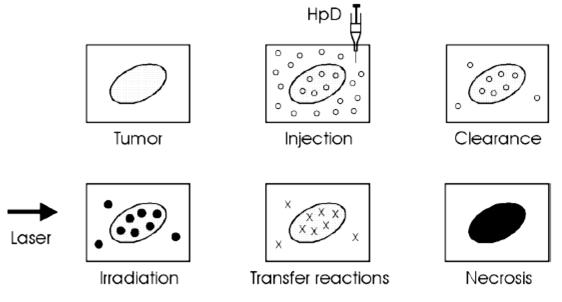


Figure (1-19) Scheme of photodynamic therapy (Niemz, 2007).

Another form of photochemical interaction is Biostimulation which is the usage of laser for wounds healing and for anti-inflammatory properties by using low level laser therapy (LLLT) which dose is between 0.001-10J/cm<sup>2</sup> to increase adenine triphosphate ATP levels to fasten cellular and oxidative processes to accelerate healing, growth, vascularity and other mechanisms (Pesevska et al., 2006).

A study showed that LLLT is safe and effective method to enhance healing, pain relief and decrease inflammation in the oral cavity with the benefit of reduction the need to post-operative medications (Elson and Foran, 2015).

#### **1.6.2.1.2 Photothermal interaction**

Photothermal interaction means that laser energy will transfer to heat when it interacts with the tissue, elevation of tissue temperature leads to many changes starting from hyperthermia which is an increase in tissue temperature above the normal to 50°C to tissue charring or carbonization which occurs at temperature elevation to more than 200°C as shown in table (1-2), (Convissar, 2015).

Tissue temperature °C	Observed effect
>37-50	Hyperthermia; bacterial inactivation
>60	Coagulation; protein denaturation
70-90	Welding of soft tissue wound edges
100-150	Vaporization
>200	Carbonization; tissue charring

Table (1-2) laser energy and thermal effects on dental soft tissue (Convissar, 2015).

Photothermal interaction is the mechanism by which laser can do many actions by varying some parameters such as spot area, energy and exposure time (Convissar, 2015).

#### 1.6.2.1.3 Photoablation

The principles of photoablation are summarized in figure (1-20), photoablation is obtained by UV lasers only, the most common application of photoablation effect is refractive corneal surgery by using excimer lasers e.g. ArF, KrF, XeCl, XeF (Niemz, 2007). High laser intensity  $10^7$ – $10^8$  W/cm2, with nanoseconds pulse duration are good to ablate tissue without causing thermal damage to the adjacent area (Niemz, 2007).

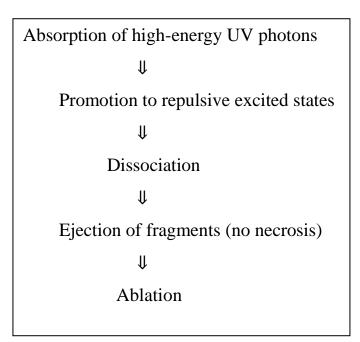


Figure (1-20) Scheme of the principles of photoablation (Niemz, 2007).

# 1.6.2.2 Wavelength independent interactions

Two types of interaction can occur.

# 1.6.2.2.1 Plasma induced ablation

In power density of about  $10^{11}$  W/cm<sup>2</sup> in solid and fluid or  $10^{14}$  in air optical breakdown (sparkle and noise) occurs. Plasma induced ablation produces a very clean removal of tissue without thermal or mechanical damage (Niemz, 2007).

Q switch which provide pulses of Nanosecond duration or mode locked laser pulses in Pico or Femtoseconds, both Q switch and mode locked can produce a localized microplasma, in Q switch the release of electrons is due to thermal ionization or thermal emission. While in mode locked pulses multiphoton ionization occurs this means several ions are absorbed and provide the ionizing energy, this is provided by high peak intensities due to ultrashort pulse duration. Figure (1-21) shows plasma induced ablation by Q switch and mode locked laser pulses (Niemz, 2007).

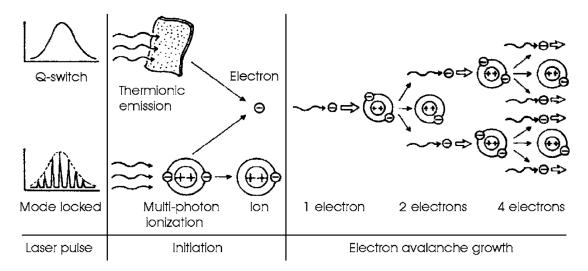


Figure (1-21) Initiation of ionization with subsequent electron avalanche (Niemz, 2007).

Some electrons initiate an avalanche effect when they absorb photons and accelerated they will collide with another atoms ionize them and release their electrons to absorb another photons and collide with other atoms and repeat the process (Niemz, 2007).

Plasma induced ablation can be used for refractive corneal surgery and caries therapy, many lasers can be used in this type of interaction such as Nd-YAG, Nd-YLF and Ti-Sapphire (Niemz, 2007).

#### **1.6.2.2.2 Photodisruption**

This type of interaction is associated with optical breakdown, plasma formation and shock wave generation, this lead to the formation of cavitation when laser beam is focused into the tissue (not on tissue), this cavitation bubbles contain gases mainly water vapor and CO2 which then distribute to the surrounding tissue (Niemz, 2007).

Photodisruption is a mechanical effect caused by laser pulses of duration of pico and femtoseconds this lead to formation of pulses with a very high peak power even if the pulse energy is low (Niemz, 2007).

Photodisruption starts with optical breakdown this mechanical effect mechanism is shock wave generation and cavitation which lead to jet formation if the cavitation is collapsed near a solid boundary but in fluid, plasma formation, shock wave generation, cavitation and jet formation occur in different timescale (Niemz, 2007).

The power density to produce photodisruption range between  $10^{11}$  -  $10^{16}$  W/Cm<sup>2</sup>, the main lasers are solid state lasers e.g. Nd-YAG, Nd-YLF and Ti:Sapphire the main applications of photodisruption are lens fragmentation and lithotripsy. Figure (1-22) shows a scheme of optical breakdown that lead to tissue ablation (Niemz, 2007).

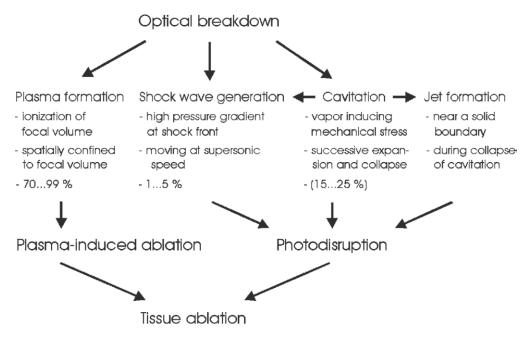


Figure (1-22) Scheme of the physical processes associated with optical breakdown. Percentages given are rough estimates of the approximate energy transferred to each effect (incident pulse energy: 100 %). Cavitation occurs in soft tissues and fluids only. In fluids, part of the cavitation energy might be converted to jet formation (Niemz, 2007).

The summery of laser tissue interaction is shown in figure (1-23).

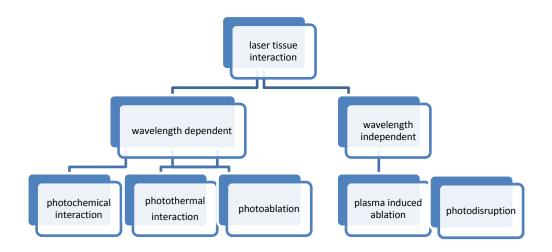


Figure (1-23) Scheme of laser tissue interaction.

#### 1.6.3 Laser modes

Lasers modes can be either as continuous waves. A CW laser is one whose power output undergoes a little or no fluctuation with time, pulsed mode when the output beams that undergo marked fluctuations that is the beams power changes with time in a very noticeable fashion, or can be gated which can be obtained by the opening and closing of a mechanical shutter in front of the beam path of a continuous-wave emission (Convissar, 2015).

#### **1.6.4 Laser parameters**

Lasers parameters control their action these parameters are:

- 1- Wavelength (Λ) which is spatial period of the wave—the distance over which the wave's shape repeats, wavelength depends on the material of active medium the unit on measuring WL is the nanometer nm which is 10<sup>-9</sup> of the meter, wave length can be in Ultraviolet (UV), visible, or Infrared (IR) ranges of the electromagnetic spectrum (Jawad et al., 2011).
- 2- Energy and Energy density: Energy is measured by joules (J), the amount of energy that radiated by optical source can be modified according to application (Jawad et al., 2011).

Energy density is the single pulse energy (E) deposited on certain area (A), (Jawad et al., 2011). and can be obtained by:

Energy density= $\frac{\text{pulse energy}}{\text{area}}$  it's measured by J/Cm<sup>2</sup>

- 3- Pulse repetition rate (PRR): it is the number of pulses per one second, it can be measured by Hertz (Hz) or S<sup>-1</sup> (Jawad et al., 2011).
- 4- Pulse duration or width (t) is the full width at half-maximum (FWHM) of the optical power versus time, measured in seconds s, (Jawad et al., 2011).

- 5- Duty cycle: it is the ratio of the pulse duration (t) to the period T (pulse repetition time which is the time from the beginning of one pulse to the beginning of the next pulse. Duty cycle= $\frac{t}{T}$ , it has no units.
- 6- Power, power density, peak power and average power:

Power (P) is expressed in Watts (Joules per second).  $P = \frac{E}{T}$ .

Power density is the power divided on the area it's measured in W/cm<sup>2</sup>. Power density= $\frac{P}{A}$ .

Peak power is the energy divided by the pulse width, it's measured in J/s or W. peak power= $\frac{E}{t}$ .

Average power is the pulse energy multiplied by PRR, it's measured in J/s or W. Average power=  $E^*$  PRR, (Jawad et al., 2011).

7- Spot diameter which is the diameter of the radiation area on the target the unit of beam diameter is usually in cm (Jawad et al., 2011).

#### **1.6.5 Lasers safety and hazard classification**

(ANSI) according to their power and ability to produce injury to personnel.

**Class 1 laser system** this type of lasers can't cause skin or eye injury when they are operated normally (Benjamin and LeBeau, 2014).

**Class 1M laser systems:** can't cause harm during exposure unless viewed by collecting optics (Benjamin and LeBeau, 2014).

**Class 2 laser systems:** these systems include visible lasers that can't cause injury to eyes or skin if the exposure time is less than 0.25 second (Benjamin and LeBeau, 2014).

**Class 2M laser systems:** visible lasers that are extremely hazard if viewed with collecting optics (Benjamin and LeBeau, 2014).

**Class 3R laser systems:** laser system that causes potential injury to the eyes if they are focused on direct and reflected beams (Benjamin and LeBeau, 2014).

**Class 3B laser systems:** medium powered lasers in visible or invisible spectrum region, these lasers potentially harm the eyes when they are direct or reflected beams, scattered beam can cause skin injury if the laser power is high (Benjamin and LeBeau, 2014).

**Class 4 laser systems:** they are visible and invisible lasers that are potentially hazard to the eyes and skin even when they are scattered, also they can cause fire (ignition) and by products emission hazards, e.g. dental lasers (Benjamin and LeBeau, 2014).

Laser beam can cause injuries to eyes and skin also other hazard due indirect beam effects, laser hazard on eyes can be summarized by table (1-4) protective eyes glasses must be used to prevent eyes injury. (Benjamin and LeBeau, 2014).

Table (1-3) potential ocular (eye) damage from laser light energy (Benjamin and LeBeau,2014).

Wavelengths with the potential of causing	Ocular structure
ocular damage	
400 to 1400 nm (visible and near infrared)	Retina
1400 to 3000 nm (near infrared)	Lens
1400 nm to 1 mm $(10^6 \text{ nm})(\text{near, mid and far infrared})$	Aqueous humor
3000 nm to 1 mm (mid and far infrared)	Cornea

Non beam laser risks or hazards can be **respiratory hazards** that occur when some lasers interact with matters, tissue will be ablated and hazard gas fumes will be created, the compositions of these gases depend on the tissue type and laser irradiance (Benjamin and LeBeau, 2014).

High volume evacuation is good to remove the generated gases, water irrigation can reduce gases generation and special surgical masks must be used (Benjamin and LeBeau, 2014).

**Fire hazards** can also occur beam and laser fiber should never touch a flammable materials or dry gauze, wet gauze must be used to remove tissue debris from the fiber, alcohol should never be used (Benjamin and LeBeau, 2014).

**Electrical hazards** occur if the device cords and cables are not kept in good repair. Laser users must be well-trained on laser systems and have knowledge about lasers and their hazards (Benjamin and LeBeau, 2014).

# **1.7** Literature review of hemostasis and healing acceleration after tooth extraction

Conventional hemostatic agents are used to stop bleeding after tooth extraction such as sutures, some chemical agents (e.g. Tranexamic acid, Ferric sulphate and silver nitrate), hemostatic resorbable gauze, bone wax and electrocautery (McCormick, 2014).

Laser photocoagulation was used first time in 1960 for retina, in 1964 lasers were used by Goldman for oral soft tissue procedures they produced excellent hemostasis (Amid et al., 2012).

In a study made on rabbits in 1983 Eriksson and Albrektsson stated that temperature elevation to more than 10°C caused irreversible periodontal damage when they were studying the root surface temperature elevation of mandibular first molar during root canal filling with high-temperature thermoplasticized Gutta-Percha in those animals (Lipski et al., 2011).

Dimitrov et al. studies of temperature rise on a single rooted tooth during biomechanical tooth preparation indicated that critical temperature for periodontal damage is between 6-7 to 10-11°C, and temperature between 3-4°C cause no thermal damage to the periodontium (Dimitrov et al., 2009).

In 2012 Mirdan used a 980 nm diode laser to produce photocoagulation in rabbits' dental sockets after teeth extraction, with power density of 76 W/cm<sup>2</sup> for 15s of exposure, clot dressed the socket and no tissue charring occurred. In 2013 she used 980 nm diode laser to coagulated EDTA treated blood samples and she declared that blood clot of 0.04 ml volume was formed at power density of 384.61 W/cm<sup>2</sup> and 9 seconds of exposure and no destructive thermal effect took place.

Pandurić et al. stated that diode lasers are good hemostatic agents also diode surgical sites don't need to be dressed even if they are large (Pandurić et al., 2013).

In Russia study of LLLT action began in 1964, immediately after the development of lasers then it was applied in clinical practice in the 80's of the last century in many places in the world (Moskvin, 2017).

LLLT has been used in many medical and dental applications, the modulation of cellular metabolism produced faster healing, also antibacterial and analgesic effects (Nascimento et al., 2004).

In 2014 Spitlera and Berns declared that LLLT produced faster wound closer, cellular migration and proliferation in the wounded area without any elevation in tissue temperature in in vitro study.

Lalabonova and Ilieva studied diode lasers healing effect on soft tissue surgical wounds in the oral cavity, their study showed that diode lasers produced faster wound healing and closer and less post-operative complications such as pain, swelling and discomfort in compare with the surgical areas that healed without laser radiation (Lalabonova and Ilieva, 2013).

LLLT fastening bone healing in the extraction sites by stimulating cellular proliferation and differentiation and acceleration of the healing process (Surendranath and Arjunkumar, 2013), this make it a good c tool to stimulate wound healing in patients with diabetes (Rocha Júnior et al., 2007).

# **1.8** Aims of study

The aim of this study was to obtain complications free healing after tooth extraction in diabetic patients that aim could be very satisfied via:

- 1- Stop the uncontrolled bleeding after tooth extraction for diabetes patients and form a firm clot that doesn't dislodged so that no infection can occur in the area especially DRY SOCKET.
- 2- Fastening the extraction wound healing and preventing the post-operative complications.

# **Chapter Two**

# **Materials and Methods**

# **Chapter Two**

#### **Materials and Methods**

This chapter includes detailed description of all the materials and equipment used in this study, with the in vitro and in vivo methods used to perform the study.

#### 2.1 Materials and equipment

The materials and equipment in this study are:

1- Fresh blood samples size 0.5 ml or cc obtained from 12 diabetes patients in different visits as the following: 240 samples for in vitro study of clot formation, 144 samples for temperature measurement test and 120 samples for clotting time test.

2- Disposable syringes (5 ml/ cc, Abu Dhabi Medical Devices Co. L.L.C, Abu Dhabi-U.A.E), figure (2-1).

3- Anticoagulant tubes EDTA-3K, 2.5 ml/ cc, plastilab, Lebanon, figure (2-1).

4- Eppendrof tubes (2 ml/ cc), figure (2-1).

5- Diagnostic instruments (dental mirror, probe and tweezers).

6- Dental forceps (ADAM SURGICAL, Pakistan) and dental elevators (Aesculp Anatomica, Germany), figure (2-2).

7- Cotton and surgical gauze.

8- X-ray film (DENTAL FILM, ERGONOM.X, ITALY).

9 - Local anesthesia solution (Mepivacaine 3%, New Stetic S.A, Colombia).

11- Dental syringe and needles.

12- Endodontic file size 45 and a mm gauge ruler to measure the tip-blood distance.

# **2.2 Devices**

1- The laser device is Epic 10 diode laser 940 nm with maximum 10W power (Biolase, USA), figure (2-4), with a tip (Biolase E3-7) 7 mm in length and  $300\mu$  in diameter.

2- Vernier caliper (TOPEX Sp. z o.o. S.K., Warsaw, Poland), figure (2-5), the specification are:

a. Resolution: 0.01 mm.

b. Measurement accuracy:  $\pm 0.02$  mm.

3- Thermometer (AMPROBE TMD®-56, Everett, WA, USA), figure (2-6). The specifications are:

a. Highly accurate with 0.1% basic accuracy.

b. Dual input T1, T2

c. K-type thermocouple with range of (-200°C to 1372°C) and head diameter of 0.8 mm.

d. Measures temperature every one second.

e. All the collected data are arranged and processed with system software.

4- Water bath Electrophoresis, BS-11, Korea, figure (2-7).

a. Working Temperature +5C above room temperature to 100C.

b. Temperature Stability  $(\pm C / F)$ : 0.2 / 0.36

c. Dimension

Bath Volume (L / cubic feet): 25 / 0.9

- Bath Opening /Depth (W×L, D) (mm / inch): 229×243, 235/ 9.0×9.6, 9.3
- Overall(W×L×H) (mm / inch): 550×440×355/ 21.7×17.3×14
- d. Net Weight (kg / lbs): 30 / 66
- e. Electrical Requirements: 230V AC, 50 Hz, 4.6 A.
- 5- Spectrophotometer Biotech UV- 9200, figure (2-8), (BIOTECH CO. LTD., UNITED KINGDOM).
- a. Wavelengths range between 190- 1200 nm.
- b. Electrical requirements: 220 V AC, 50 Hz.
- 6- Glucose monitor device ACCU-CHEK active, figure (2-9), Roche Diagnostics GmbH/ Germany.
- 7- X-ray machine (xgenus, OlgiateOlona (VA) ITALY).
- 8- Dental chair (Performer, A- dec, USA).
- 9- Sterilizer statim 5000, figure (2-10), (Pre- owned Inc. dental, USA), this device is:
- a. Cassette dimensions: 15" x 7" x 3"
- b. Outside dimensions 21.75" x 16.25" x 7.57"
- c. Reservoir Capacity: 4 liters.
- d. Highest steam temperature is 138°C.
- 10- Stopwatch, software in mobile phone.



Figure (2-1) Disposable syringe, EDTA and eppendrof tubes.



Figure (2-2) Dental forceps and elevators.



Figure (2-3) laser device with footswitch and protective goggles.



Figure (2-4) Vernier caliper.

Figure (2-5) Digital thermometer.



Figure (2-6) Water bath.





Figure (2-7) Spectrophotometer.



Figure (2-8) Glucose monitor device.



Figure (2-9) Sterilizer.

# 2.3 Laser system

Epic 10 diode laser (Biolase, USA) has the following specifications:

1-The main components, figure (2-4) which are:

- Base console.
- Wireless footswitch.
- Delivery system which consists of:
- Fiber optic assembly (reusable).
- Surgical and whitening handpieces (reusable).
- $\circ$  Disposable tips for surgical handpiece (200, 300 and 400  $\mu m$  in diameter).
- 2- Laser wavelength  $\lambda = 940 \pm 10$  nm, aiming beam 625-670 nm.
- 3- Laser classification: IV (4) and cl 2 for aiming beam.
- 4- Medium: InGaAsP Semi-conductor diode.
- 5- Maximum output power 10 W.
- 6- Power modes: CW mode and gated pulsed modes, pulse modes details are shown in table (2-1)

Table (2-1)	pulse	mode	details
-------------	-------	------	---------

MODE	PULSE DURATION	PULSE INTERVAL	Duty Cycle
CP0	10 microseconds	40 microseconds	20%
CP1	100 microseconds	200 microseconds	33%
CP2	1 millisecond	1 millisecond	50%
P3	20 millisecond	20 millisecond	50%

#### 2.4 Methods

The methods in this study were in vivo and in vitro methods.

#### 2.4.1 In vitro methods

#### **2.4.1.1 Sample preparation**

From diabetes patients blood samples were collected in different visits (10 cc for in vitro study of clot formation 5 cc for clotting time test and 6 cc for temperature test) and stored at room temperature in EDTA tubes for 5 minutes, samples were inverted upside down twice each 2 minutes, then they distributed equally in eppendrof tubes each tube contained 0.5 cc of blood i.e. 20 samples for in vitro study of clot formation, 10 samples for clotting time test and 12 samples for temperature test for each patient.

#### 2.4.1.2 Blood sampling

Sampling of blood is important for vitro studies, when blood is withdrawn from the vessels coagulation factors are activated by the needle injury and clotting of sample will occur, anticoagulant agents are used to prevent blood clotting. It's important to choose an anticoagulant agent that doesn't affect blood component so that the sample can be used for enough time without any changes (WHO, 2002).

Ethylenediamine tetra-acetic acid (EDTA) is one of the anticoagulant factors that are used to keep blood components unchanged if the sample is used in a suitable period (Baffour et al., 2013).

Phlebotomy or blood withdrawal must be done by well-trained persons, needle size selection must be suitable, if the needle size is too small damage of blood cells will occur, too large needle cause patient's discomfort (WHO, 2010).

Storage time of blood sample mustn't be long to prevent damage to blood cells, a study made by Baffour et al. showed that storage time of blood sample with EDTA mustn't exceed four hours after blood withdrawal otherwise changes in blood compositions will occur (Baffour et al., 2013).

If blood samples need to be transported they should be used within only one hour at room temperature (Mackie et al., 2012).

Blood must be transformed immediately to the anticoagulant tubes then the tube must be turned upside down to mix the blood with the anticoagulant material, shaking of the tube destroys blood cells (Mackie et al., 2012).

Before using the blood it's important to put the tubes in water bath for few minutes and adjust the temperature to 37°C (Mackie et al., 2012).

### 2.4.1.3 Clot formation

In this present work pilot study was made to choose the least power that can form a firm, stable and fixed area covering clot.

240 blood samples of 0.5 cc in size were obtained from 12 DM patient as 20 samples for each one, each 5 samples were aliened in water bath of temperature of  $37^{\circ}C \pm 0.5^{\circ}C$  to be exposed to different laser power but same tip surface distance.

Distance between laser tip and blood surface was measured by ruler and the tip position was marked on the eppendrof tube by a marker pen.

The laser tip was perpendicular on the blood samples surfaces, powers from 1- 6 W (as 1,2,3,4,6 W for each sample ) were used for 10 seconds and at distance from 3 to 12 mm ( by increasing the distance by 3 mm for each exposure) between laser tip and blood surface.

Clots which formed by those powers at distance of 3 mm were obvious but didn't cover the desired surface area.

At a distance of 6 mm between laser tip and blood surface the clot was larger in surface area but didn't cover the desire surface.

At 9 and 12 mm distances clot layer that formed by power less than 3 W was corrupted and very thin.

At a power of 3 W clot was firm and covered the surface area of the blood at distance of 12 mm between laser tip and the blood surface for all samples which used in this study, figure (2-10) shows the power measurement of the laser device by the use of a power meter.



Figure (2-10) laser power measurement using a power meter

## 2.4.1.4 Spot area calculation

The spot diameter was measured at 12 mm defocus distance, by using a carbon paper with the CW mode, power 3W, laser spot diameter d was 6.17 mm. Finding spot area by circle area A, r is d/2,  $A = \pi \times r2$ .

 $A=0.29 \text{ cm}^2$  which is the spot area.

### 2.4.1.5 Sample grouping

For clotting time, samples were divideded into two groups: control G1, this group didn't expose to laser and its samples where coagulated by their own clotting factors, and test group G2 this group exposed to 3W powered laser on area of 0.29 cm<sup>2</sup> and laser tip surface distance of 12 mm.

Temperature measurement groups were divided into three groups:

Group 1 exposed to 1.5W, group 2 exposed to 3W and group 3 exposed to 6 W laser power for 10 seconds, each group was subdivided into four subgroups as shown in table (2-2).

Subgroup no.	Times of laser exposures	Depth of thermocouple to blood surface (mm)
1	1	4
2	1	13
3	2	4
4	2	13

Table (2-2): subgroups of temperature groups.

## **2.4.1.6 Clotting time (CT)**

In water bath of temperature  $37^{\circ}C \pm 0.5^{\circ}C$  10 blood samples of 0.5 cc for each patient were put and when the temperature of blood reached to the same of water bath laser was performed.

Laser tip was 12 mm away from the blood surface with a power of 3 W each sample exposed to laser radiation for time ranged from 5 to 14 seconds and the least time that produced the thickest blood clot was recorded.

For control group clotting time was obtained by ordinary CT test in the lab.

# 2.4.1.7 Temperature measurement before and after laser radiation

For each patient 12 blood samples of 0.5 cc were put in water bath of temperature  $37^{\circ}C \pm 0.5^{\circ}C$  to stabilize samples temperature nearly to that of human.

Laser tip was perpendicular to the sample surface and at distance of 12 mm, setup is shown in figure (2-11). Temperature (before, during and after lasing) was measured by thermometer and recorded second by second by computer software.For each subgroup time needed for temperature to return to that before lasing was recorded.

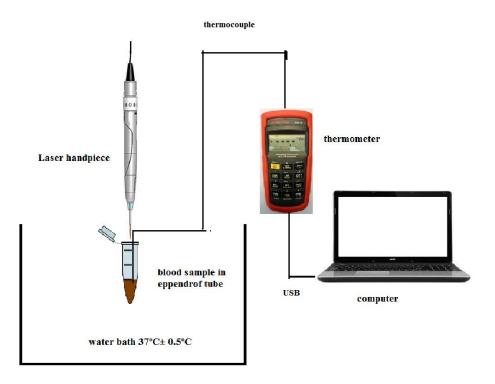


Figure (2-11) Experimental setup for temperature measurement.

# 2.4.2 In vivo methods

Eleven diabetic patients with age ranged between (44-55 years) had teeth extractions 12 teeth extraction sites had laser assisted coagulation by diode laser 940nm, CW mode and power of 3 W for 10 s which was applied immediately after tooth removal. For 2 teeth extraction the interval between the exposures was 60s.

Before extraction investigations were done to each patient these investigations included: random blood sugar (RBS), Hemoglobin A1C test, hemoglobin level (Hb), Packed Cell Volume (PCV) bleeding time and clotting time tests, also blood pressure, medical, dental history and patient's medications were recorded. Flow up was after 3, 10 and 21 days after extraction to examine the extraction sites clinically and radiological investigations were held in day 21, notes were recorded by operator and patient in a questionnaire paper.

Table (2-3) shows the information of patients and the cause of tooth extraction.

Pt. no.	Age	Sex	Accused tooth	Cause of extraction
1 <sup>st</sup>	47	M	Mandibular right 3 <sup>rd</sup> molar	Unrestorable tooth
$2^{nd}$	55	М	Mandibular left 2 <sup>nd</sup> molar	Chronic periodontitis
3 <sup>rd</sup>	51	Μ	Mandibular left 1 <sup>st</sup> molar	Chronic periodontitis
4 <sup>th</sup>	44	F	Mandibular right 3 <sup>rd</sup> molar	Chronic periodontitis
5 <sup>th</sup>	45	F	Mandibular left 2 <sup>nd</sup> premolar	Chronic periodontitis
6 <sup>th</sup>	50	F	Mandibular central incisors	Chronic periodontitis
7 <sup>th</sup>	52	F	Maxillary left 2 <sup>nd</sup> premolar	Pulp necrosis
8 <sup>th</sup>	51	Μ	Mandibular right 3 <sup>rd</sup> molar	Severely fractured
9 <sup>th</sup>	48	Μ	Maxillary left 2 <sup>nd</sup> premolar	Chronic periodontitis
10 <sup>th</sup>	54	F	Mandibular left 1 <sup>st</sup> molar	Pulp necrosis
11 <sup>th</sup>	48	Μ	Mandibular left canine	Pulp necrosis

 Table (2-3): Patient's information.

Male= (M), Female= (F).

# 2.4.3 Statistical analysis

The results were statistically analyzed by SPSS version 20 for windows 7, the analysis including:

1-Descriptive statistics:

Means, Standard deviations (SD), Standard errors (SE), Minimum values and Maximum values.

2- Inferential Statistics:

Shapiro- Wilk's test to study the probability distribution of data (whether or not they were normal)

The following tests were used:

- If probability distribution data is normal: t-test for equality of mean for tow paired samples test.
- If probability distribution data is not normal: Wilcoxon test for equality of mean rank for tow paired samples test,

P > 0.05 NS (Not Significant)

P < 0.05 S (Significant)

P < 0.01 HS (Highly Significant)

# **Chapter Three**

# **Results and Discussions**

# **Chapter Three**

# **Results and Discussions**

This chapter includes the results of this research work, discussion of these results, conclusions and the future work also will be mentioned.

### **3.1 Results**

# **3.1.1 Data distribution**

Shapiro- Wilk test was done to test the normality of data distribution. For blood temperature before and after laser irradiation of 3 W laser power, table (3-1) shows the normality of data distribution.

 Table (3-1):
 Shapiro-wilk test of data distribution normality for samples temperature befor and after laser irradiation.

Subgroup no.	Statistics	df	Р	Sig
1 before laser exposure	0.920	12	0.286	NS
1 after laser exposure	0.894	12	0.134	NS
2 before laser exposure	0.903	12	0.175	NS
2 after laser exposure	0.950	12	0.637	NS
3 before laser exposure	0.910	12	0.213	NS
3 after laser exposure	0.837	12	0.025	S
4 before laser exposure	0.896	12	0.141	NS
4 after laser exposure	0.802	12	0.010	S

When P> 0.05 this means data are normally distributed.

## 3.1.2 Clotting time

Clotting time of control group G1 and test group G2 are shown in table (3-2) and their Descriptive statistics analysis is shown in table (3-3).

Groups				C	lotting	g time	e in s					
	#1	#2	#3	#4	#5	#6	<b>#7</b>	<b>#8</b>	<b>#9</b>	#10	#11	#12
Control group	240	210	150	210	180	180	210	210	210	180	240	210
Test group	9	10	8	8	7	7	6	9	6	9	9	8

Table (3-2): Clotting time for control and test groups.

 Table (3-3): Descriptive statistics for Clotting time of control and test groups.

groups	N	Mean	Min s	Max s	Std Deviation	Std Error Mean
Control group	12	202.5	150	240	25.98076	7.50000
Test group	12	8	6	10	1.27920	0.36927

For clotting time CT of control and test groups, descriptive analysis showed that for control group the mean of CT = 202.5 s minimum CT = 150 s maximum CT = 240 s, while for test group CT mean was 8 s, minimum CT = 6 s while maximum CT = 10 s.

# **3.1.3** Temperature measurements

Temperature was measured before, during and after laser irradiation, changes in temperature were measured second by second by

thermometer connected to computer and recorded by software (AMPROBE).

Table (3-4) shows the highest values of highest temperature change for each subgroup after laser irradiation.

SAU	Subgroup		samples highest temperature change °C										
GROUPS		#1	#2	#3	#4	#5	#6	<b>#7</b>	<b>#8</b>	<b>#9</b>	#10	#11	#12
M	1	1.1	1.4	0.7	0.7	0.8	1.4	1.2	1.1	1.3	1.2	0.7	0.7
1, 1.5 W	2	0.6	0.7	0.4	0.5	0.6	0.8	0.7	1.1	0.5	0.6	0.5	0.6
GROUP	3	1.2	1.6	1.2	1.4	1.6	1.6	1.6	1.3	1.3	1.8	1.1	1.1
GR	4	0.9	1.4	0.9	0.8	1.1	1.4	1.1	1.1	0.9	1.3	0.9	1.1
W	1	2.0	1.9	1.6	1.9	1.8	1.5	1.3	1.5	1.4	1.3	1.1	1.4
GROUP 2, 3 W	2	1.5	1.1	1.5	1.4	1.3	1.1	1.2	1.3	1.0	1.3	1.1	1.0
ROUI	3	2.8	1.4	2.9	2.7	2.5	2.3	2.9	2.6	2.7	2.5	2.5	2.5
3	4	2.5	2.3	2.1	1.8	1.4	1.8	2.0	0.9	2.1	2.1	1.6	1.5
3,6 W	1	3.2	2.1	3.1	2.3	2.8	2.6	3.0	3.0	2.4	2.3	2.2	2.3
P 3, 6	2	3.4	2.4	3.3	2.0	2.8	2.1	2.8	2.2	2.2	1.8	2.1	1.3
GROUP	3	6.2	6.0	5.8	5.4	5.4	5.0	4.8	4.9	4.8	4.4	4.2	4.2
G	4	4.0	3.8	3.7	3.6	3.6	3.3	3.2	3.2	3.3	3.3	3.0	2.2

Table (3-4): samples highest temperature changes for temperature groups.

Paired t-test (for normally distributed data) and Wilcoxon test (when data distribution wasn't normal) was made between samples temperature before and after laser irradiation by 3 W laser power for 10 s.

Subgroup 1:

The relation between temperature before and after laser exposure was analyzed by paired t-test as shown in table (3-5).

 Table (3-5): paired t-test for subgroup1 temperature differences before and after laser exposure.

Groups	Means	SD	Paired t-test	Р	Sig
			value		
Before exposure	36.8583	0.17816	10.007	0.00	IIC
After exposure	38.4167	0.34068	18.987	0.00	HS

Subgroup 2:

The relation between temperature before and after laser exposure was analyzed by paired t-test as shown in table (3-6).

 Table (3-6): paired t-test subgroup2 temperature differences before and after laser exposure.

Groups	Means	SD	Paired t-test	Р	Sig
			value		
Before exposure	36.7667	0.16143			
			24.066	0.000	HS
After exposure	38.0000	0.19069	21.000	0.000	

Subgroup 3:

The relation between temperature before and after laser exposure was analyzed by Wilcoxon test as shown in table (3-7).

Groups	mean	SD	Test statistic		
			Z	Р	Sig
Before exposure	36.8083	0.16765	2.074	0.00	IIC
After exposure	39.3333	0.37009	- 3.074	0.00	HS

 Table (3-7): Wilcoxon test for subgroup3 temperature differences before and after laser exposure.

Subgroup 4:

The relation between temperature before and after laser exposure was analyzed by Wilcoxon test as shown in table (3-8).

Table (3-8): Wilcoxon test for subgroup4 temperature differences before and after
laser exposure.

Groups	mean	SD	Test statistic		
			Z	Р	Sig
Before exposure	36.8500	0.26458			
			3.065	0.02	S
After exposure	38.6917	0.46993			

According to these tests, for subgroups (1, 2 and 3) statistical analysis showed that the value of P was 0.00 < 0.05, which mean that there was high significant different between blood temperature before and after lasing.

For subgroup (4) P value was 0.02 < 0.05, this means that the statistical difference between temperature before and after lasing in this subgroup was significant.

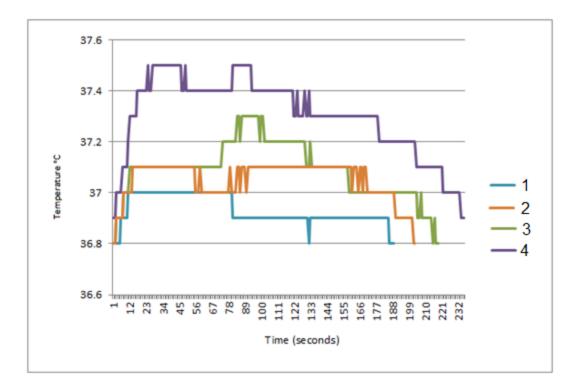


Figure (3-1): Temperature change of group1 with time.

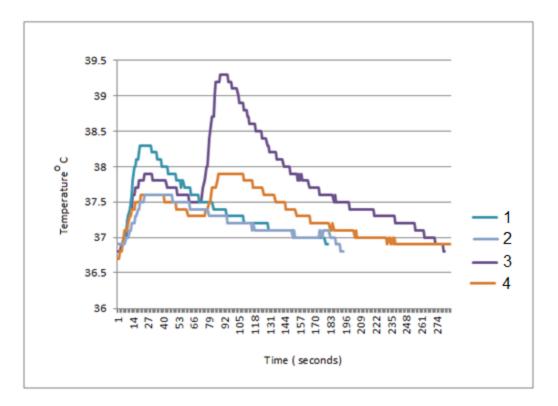


Figure (3-2): temperature change of group2 with time.

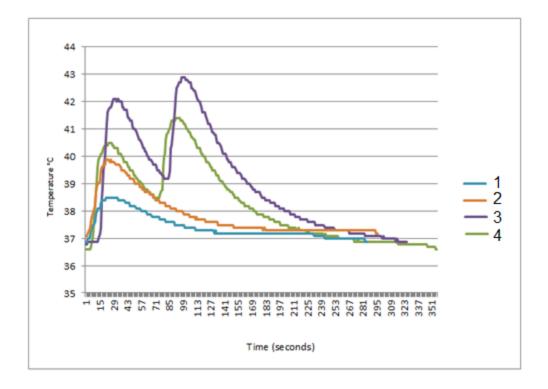


Figure (3-3): Temperature change of group3 with time.

## 3.1.4 Patients' results

Teeth extraction was done for 12 teeth from 11 diabetic patients with post conventional extraction complications of swelling, pain and prolonged bleeding, the extraction sites were exposed to 3 W laser power for 10s and the laser tip was 12 mm defocus distance.

For laser assisted coagulation extraction sites, firm clot was formed and covered the extraction area after 10 s of laser exposure.

In 6 hours after extraction information were obtain as shown in table (3-9).

After 3 and 10 days of extraction patients were examined clinically and information were obtained as shown in table (3-10).

Patient no.	Pain / analgesics	Bleeding	Parasthesia	Swelling
1 <sup>st</sup>	Nil	Oozing	Nil	Nil
2 <sup>nd</sup>	Nil	Nil	Nil	Nil
3 <sup>rd</sup>	Nil	Nil	Nil	Nil
4 <sup>th</sup>	Nil	Nil	Nil	Nil
5 <sup>th</sup>	Nil	Nil	Nil	Nil
6 <sup>th</sup>	Nil	Nil	Nil	Nil
7 <sup>th</sup>	Nil	Nil	Nil	Nil
8 <sup>th</sup>	Mild / paracetamol	Nil	Nil	Nil
9 <sup>th</sup>	Nil	Nil	Nil	Nil
10 <sup>th</sup>	Nil	Nil	Nil	Nil
11 <sup>th</sup>	Nil	Nil	Nil	Nil

 Table (3-9): six hours post-extraction information.

 Table (3-10): three and ten days post-extraction information.

Patient no.	Pain / analgesics	Bleeding	Parasthesia	Swelling	Dry socket
1 <sup>st</sup>	Nil	Nil	Nil	Nil	Nil
2 <sup>nd</sup>	Nil	Nil	Nil	Nil	Nil
3 <sup>rd</sup>	Nil	Nil	Nil	Nil	Nil
4 <sup>th</sup>	Nil	Nil	Nil	Nil	Nil
5 <sup>th</sup>	Nil	Nil	Nil	Nil	Nil
6 <sup>th</sup>	Nil	Nil	Nil	Nil	Nil
7 <sup>th</sup>	Nil	Nil	Nil	Nil	Nil
8 <sup>th</sup>	Nil	Nil	Nil	Nil	Nil
9 <sup>th</sup>	Nil	Nil	Nil	Nil	Nil
10 <sup>th</sup>	Nil	Nil	Nil	Nil	Nil
11 <sup>th</sup>	Nil	Nil	Nil	Nil	Nil

In the 3<sup>rd</sup> week after extraction clinical and radiographical investigations were done.

Figures (3-4), (3-5) and (3-6) show tooth extraction and post-operative follow up clinical results for the laser assisted coagulation.





Figure (3-4): Laser assisted coagulation after mandibular left canine extraction for a male patient. (A) Before extraction, (B) After extraction immediately post laser application, (C) 3<sup>rd</sup> day after extraction, (D) 10<sup>th</sup> day after extraction, (E) 21<sup>st</sup> day after extraction.



Figure (3-5) Laser assisted coagulation after maxillary left 1<sup>st</sup> premolar extraction for a female patient. (A) Before extraction, (B) After extraction immediately post laser application, (C) 3<sup>rd</sup> day after extraction, (D) 10<sup>th</sup> day after extraction, (E) 21<sup>st</sup> day after extraction.

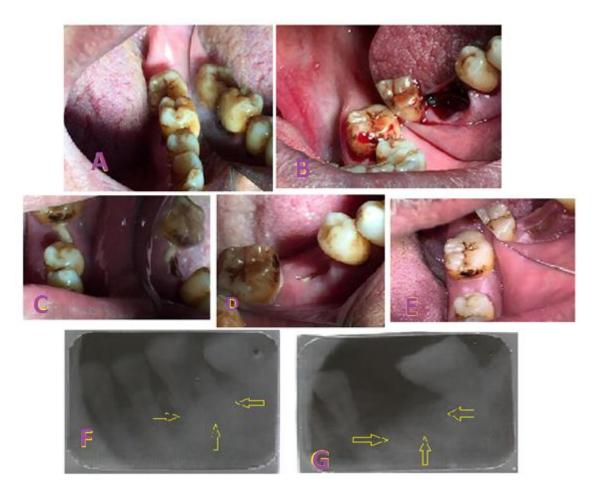


Figure (3-6) Laser assisted coagulation after mandibular left 1st molar extraction for a smoker male patient (dental photography mirror was used). (A) Before extraction, (B) After extraction immediately post laser application, (C) 3rd day after extraction, (D) 10th day after extraction, (E) 21st day after extraction, (F) radiographic image of the tooth before extraction, (G) radiographic image of the tooth 21st day after extraction

#### **3.2 Discussions**

When laser hits biological tissue temperature may be elevated and irreversible damage can occur by altering tissue properties (Jasiński, 2010).

Lasers have many benefits in oral soft tissue applications such as sterilization, bacteremia reduction, reduction of post- operative pain, edema, scar and wound contraction, also they are very good hemostatic agents (Amid et. al., 2012).

Diode lasers had have been used for hemostasis, they are very effective in stop bleeding therefore assist in the management of the wounds, this property enabled lasers to remove vascular lesions by the mechanism of vascular contraction in addition to blood coagulation (Pandurić et.al., 2013).

Absorption coefficient of 940 nm laser in blood is 0.25-0.28mm<sup>-1</sup> and scatter coefficient is 0.6-0.64 mm<sup>-1</sup> which means that clotting of blood after diode laser exposure was occurred due to chromophore absorption (Mirdan, 2012).

LLLT has been used with various power densities to fasten wound healing by stimulating cellular respiratory chain which lead to increase ATP production in the mitochondria, therefore, increasing the energy of the cells which lead to activation of metabolism, proliferation and migration of cells in the wounded area. The outcomes of LLLT is depending on laser's power, power density, wavelength, beam profile, energy, energy density, number and frequency of treatment and duration of treatment. A study made on rabbits dental sockets after teeth extraction and usage of diode lasers on them showed that diode laser application stimulated the healing of dental sockets. The sockets after radiation presented with greater number of bone trabeculae, collagen fibers and blood vessels as compared to sockets without laser radiation. (Hamad et al., 2016).

An in vitro study on various types of human and animals cells showed that LLLT by diode laser accelerated cellular proliferation, migration and closer of the wounds without elevation in the tissue temperature (Spitlera and Berns, 2014).

In this study diode laser 940 nm was used to produce hemostasis after tooth extraction for diabetes patients with previous history of post-extraction complications. An in vitro temperature and clotting study was done before laser radiation inside the patient's mouth.

Hemostasis after tooth extraction took place after exposure to 3 W laser power for 10s and the laser tip was 12 mm defocus distance, no swelling, infection, dry socket or abnormal hemorrhage occurred, while in study done by Karbassi et. al. in 2015 when 23 teeth were extracted and hemostasis took place by conventional method (without laser or any other hemostatic or disinfectant agents) many complication occurred include: abnormal hemorrhage in 30.4%, abnormal pain in 26.1%, fever and infection in 27.1%, swelling in 21.7% and dry socket in 17.4% (Karbassi et. al., 2015).

# **3.3 Conclusions**

1- Diode laser 940 nm CW mode of 3 W power, 10s exposure time and laser tip 12 mm defocus distance reduced blood clotting time and produced acceptable elevation in socket temperature and it is harmless to the periodontium.

2- Post extraction laser assisted radiation produced faster healing and relatively no complications in compare to conventional method.

# **3.4 Suggestions for Future Work**

1- Comparative study of different types of lasers to assist in coagulation after teeth extraction.

2- Laser can be used in patients with coagulation and healing impairment after excessive vitro study on their blood samples.

3- Increase the patients number for more standardized results.

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# Appendices

# **Appendix I**

Patient's case sheet



University of Baghdad

Institute of Laser for postgraduate studies

# Department of biomedical applications

Patient's Name	Age	Sex
Chief Complain / pain		
Swelling		
Others		
History of present illness (HPI)		
Past dental history (PDH) Medical history (MH)/ Diabetes (DM)		
type		
how long diabetes drugs		
Other diseases		
Drugs		
Smoking? (If yes, how many/day?)How lo		

#### **Clinical examination**

E/O
I/O
Investigations
-

# Treatment plan / Extraction of

 8
 7
 6
 5
 4
 3
 2
 1
 1
 2
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 4
 5
 6
 7
 8

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 1
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 7
 8

Date	Start	Treatment
Staff signature		

# **Appendix II**

# استمارة المريض

المريض رقم الهاتف	اسم
وان	المعذ
نس العمر العمر الوزن الطول	الجن
نة التحصيل الدراسي	المهن
ة الاجتماعية	الحالة
مرض السكر مدة الاصابة بالمرض	
شعر بأعراض انخفاض او ارتفاع السكر؟ اذا كانت الاجابة نعم , كم مرة في الاسبوع؟	-
ب الاعراض	ماهي
، تتعامل انت ومن حولك معها؟	كيف
	-

هل يوجد جهاز لقياس السكر في المنزل ...... اذا كانت الاجابة نعم , كم مرة تقوم بقياس السكر خلال الاسبوع ؟ ......

السابع	السادس	الخامس	الرابع	الثالث	الثاني	الاول	الإيام
							مستوى السكر قبل وجبة الفطور
							مستوى السكر العشوائي

هل دخلت المستشفى سابقاً؟..... اذا كانت الاجابة نعم اذكر السبب .....

61. ž
 کم مکثت فیها؟

	بعد قلع السن	
	خروج دم من مكان القلع خلال ال24 ساعة بعد القلع ؟	
نزيف شديد	لايوجد فعدار متوسط مقدار قليل مقدار متوسط	
	هل شعرت بألم بعد زوال مفعول المخدر؟ اذا كانت الاجابة نعم اذكر درجة الالم	
شديد جداً	خفيف جداً متوسط شديد	
	هل تناولت مسكنات بعد زوال مفعول المخدر؟/ نوع المسكن	

# خروج دم من مكان القلع خلال الاسبوع الاول بعد القلع

السابع	السادس	الخامس	الرابع	الثالث	الثاني	الاول	الايام
							كمية الدم

الملاحظات ,ان وجدت


	اي الطرق كانت نتانجها افضل بالنسبة اليك مع ذكر السبب
استخدام الليزر بعد قلع السن لايقاف النزيف	قلع السن و ايقاف النزيف بالطريقة التقليدية
	السبيب

# **Appendix III**

# **Informed consent**

I'm .....

I agree to have tooth extraction followed by laser application to coagulate the dental socket by Dr. Noor Ali Saleem, also I agree to publish the photos of extraction and healing stage in the Journals that interest in this subject.

اني ..... اوافق على قلع سني و استخدام الليزر على مقبس السن لايقاف النزف من قبل د. نور علي سليم و اوافق على نشر الصور الخاصة بقلع السن و مراحل شفاء المقبس السني في المجلات المهتمة بهذا الموضوع.

التوقيع

.....

# Appendix IV

Pt. no.	RBS (mg/dl)	HbA1C %	Hb (mg/dl)	PCV	<b>CT (S)</b>	<b>BT</b> ( <b>S</b> )
					Control	
1 <sup>st</sup>	201.00	11.00	14.00	44.00	240.00	120.00
2 <sup>nd</sup>	195.00	8.60	13.50	42.00	210.00	150.00
3 <sup>rd</sup>	203.00	11.10	14.00	43.00	150.00	150.00
4 <sup>th</sup>	186.00	9.00	13.00	42.00	210.00	150.00
5 <sup>th</sup>	201.00	9.80	13.30	42.00	180.00	150.00
6 <sup>th</sup>	199.00	9.60	14.10	44.00	180.00	120.00
7 <sup>th</sup>	180.00	9.30	13.30	41.00	210.00	90.00
8 <sup>th</sup>	180.00	8.30	13.80	42.00	210.00	120.00
9 <sup>th</sup>	182.00	9.30	14.80	45.00	210.00	90.00
10 <sup>th</sup>	179.00	9.00	12.00	39.00	180.00	180.00
11 <sup>th</sup>	205.00	12.30	14.00	43.00	240.00	150.00
12 <sup>th</sup>	188.00	9.20	13.30	40.00	210.00	190.00

# Patients' investigations.

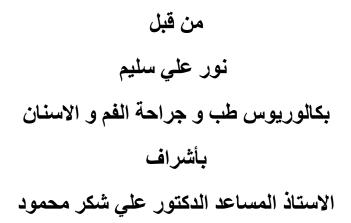
وزارة التعليم العالي والبحث العلمي جامعة بغداد معهد الليزر للدراسات العليا



# دايود ليزر ٤٠ ٤ نانومتر المساعد على التخثر و الالتئام في مقبس السن لمرضى السكري

رسالة مقدمة الى

معهد الليزر للدر اسات العليا /جامعة بغداد /لاستكمال متطلبات نيل شهادة ماجستير علوم في الليزر/ طب الاسنان



2017 م

**A** 1439

# الخلاصة

**مقدمة:** يستخدم الدايود ليزر استخداماً واسعاً في انسجة الفم الرخوة كونه يساعد على تخثر الدم بسبب الامتصاص العالي لاطواله الموجيه المختلفه من قبل الهيمو غلوبين والميلانين الموجودة فيها من دون ان يسبب ضرر للانسجة العظمية والسنية القريبة كون هذه الاطوال الموجية ضعيفة الامتصاص من قبل الماء و الهيدروكسيبتايت والتي تعتبر المكون الرئيس لهذه الانسجة.

**الهدف:** الغرض من هذا العمل هو در اسة دور الدايود ليزر في تسريع زمن تخثر الدم بعد قلع الاسنان لمرضى السكر و تكوين خثرة دموية ثابته لحماية مقبس السن من الاختراق البكتيري و العدوى و بالاخص القليعة الجافة.

**المواد وطريقة العمل:** تم اخذ نماذج دم من 12 شخص مصاب بمرض السكر و تقسيمها في انابيب اختبار يحتوي كل منها على 0.5 ملل<sup>3</sup> بعد خلطها بمادة تمنع تخثر ها, تم بعد ذلك اختيار قياسات الليزر المناسبة بعد عدة اختبارات و تطبيق هذه القياسات على بعض عينات الدم لحساب زمن التخثر بمساعدة الليزر و مقارنته بزمن التخثر الطبيعي الذي تم قياسه في الختبر لكل مريض, بعد ذلك تم قياس درجة حرارة الدم قبل واثناء و بعد تسليط الليزر عليه لمعرفة التأثير الحراري لليزر على منطقتين 4 و 13 ملم تحت سطح الدم للعينة للضربة الواحدة و للضربتين الاشعاعيتين و تم بعد ذلك تسليط الليزر على مقابس اسنان 11 مريض سكر بعمر يتراوح بين 44-55 عام بعد قلع اسانهم و متابعة الحالات بعد 3 و 10 و 12 يوم بعد التشعيع.

النتائج: حسب الدراسة التجريبية تبين انه على مسافة 12 ملم بين رأس الليف الضوئي لليزر و سطح الدم و استخدام 3 واط لمدة 10 ثواني من الدايود ليزر 940 نانومتر هي اقل قياسات قوة لازمة لتخثر الدم مع ارتفاع بسيط في درجات الحرارة لا يسبب اي ضرر للانسجة حول السنية و ان هذه القياسات من الليزر قللت معدل زمن تخثر الدم الى 8 ثانية بعد ان كان 202.5 ثانية و تبين من خلال فحص المرضى خلال فترات المتابعة انه لا توجد اي مضاعفات مؤذية للمرضى و التآم جرح السن بعد 21 يوم من القلع.

**الاستنتاج:** تبين من خلال هذه الدر اسة ان الدايود ليزر هو امن و فعال لايقاف نزف الدم و المساعدة على شفاء انسجة مقابس الاسنان الرخوة.