

# **CLINICAL AND RADIOGRAPHIC EVALUATION OF GUIDED TISSUE REGENERATION IN TREATMENT OF CLASS II FURCATION DEFECTS**

**THESIS SUBMITTED IN PARTIAL  
FULLFILMENT OF THE REQUIRMENT FOR MASTER DEGREE IN ORAL  
MEDICINE AND PERIODONTOLOGY**

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***2009***

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وَقُلْ رَبِّ زِنْنِي عِلْمًا

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## **Dedication**

**I would like to dedicate my effort in this thesis to my generous gift from God; my parents, who were in front of me to light me the way and guided me through my whole life with love & support, my loving angel; my wife, who was behind me pushing me forward and gave me all her love and support and shared me all the hardest moments, without her patience and support, this work would not come to light, my dear sweet sons who were around me giving me hope, and my dear brothers and sister for their cooperation.**

## ***Acknowledgement***

This work came into existence only because of the grace guidance and mercy of God, Allah, The most Merciful, Whose will and help are the actual causes for everything we can do in life.

I would like to express my most sincere gratitude and grateful appreciation to ***Professor Dr. Gamalat Ahmad Hasan***, Professor of Oral Medicine and Periodontology, Faculty of Oral and Dental Medicine, Cairo University, for her great support, encouragement. It was a great honor to work under her experienced supervision, and to accept me as one of her science-seeking candidates.

Great thanks and appreciation are due to my ***Professor. Dr Manal Mohamad Hosny***, Professor of Oral Medicine and Periodontology, Faculty of Oral and Dental Medicine, Cairo University, for her generous infinite help, great support, encouragement, her continuous warm guidance and advice throughout the whole work. It was a great honor to work under her experienced supervision, and to accept me as one of her science-seeking candidates. I will never forget her meticulous efforts and precise criticism. I am extremely indebted and grateful for her generous help, and precise supervision throughout this study.

*I would like to take this opportunity to express my grateful thanks to **Professor. Dr, Mahmoud El-Refaie**, Chairman of Oral Medicine and Periodontology department, Faculty of Oral and Dental Medicine, Cairo University*

I am indebted to ***Dr. Mohamad Hosny Mostafa***, Professor of Oral Medicine and Periodontology, Faculty of Oral and Dental Medicine, Cairo University, for his technical advices he offered to me, and for the hope he gave me when I lost it and was about to sink in despair.

I would like to express my sincere appreciation to all professors and staff members of Oral Medicine and Periodontology Department, Faculty of Oral and Dental Medicine, Cairo University, for their valuable hospitality and generous support.

Finally, I submit my sincere thanks to all the patients who offered maximum cooperation and compliance, without whom this work would not have been possible.

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## **Abbreviations**

AAP	American Academy of Periodontology
β-TCP	β- Tricalcium Phosphate
CAL	Clinical Attachment Level
CPT	Combined Periodontal Therapy
DFDBA	Demineralized Freeze Dried Bone Allograft
ePTFE	Expanded Polytetra Fluoriethylene
FDBA	Freeze Dried Bone Allograft
HCAL	Horizontal Clinical Attachment Level
HEMA	Hydroxyethyl Methacrylate
GI	Gingival Index
GTR	Guided Tissue Regeneration
GBR	Guided Bone Regeneration
MWF	Modified Widman Flap
PGA	Polyglycolic acid
PI	Plaque Index
PLA	Polylactic acid
PMMA	Polymethyl Methacrylate
PPD	Probing Pocket depth
ROI	Region Of Interest
VCAL	Vertical Clinical Attachment Level



CEP	Cervical Enamel Projection
t.d.s	Three times per day
SD	Standard Deviation

## **Key Words**

Guided tissue regeneration

Combined periodontal therapy

Cerasorb

Epi-Guide

Gingival index

Plaque index

Probing pocket depth

Clinical attachment level

Horizontal clinical attachment level

Vertical clinical attachment level

Modified Widman Flap

Region of interest

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## **Introduction**

Periodontitis is a chronic disease affecting the gingiva and the supporting structures of the teeth characterized by the formation of periodontal pockets with loss of connective tissue fiber attachment to the tooth, bone loss and exposure of root cementum. Furcation defects being a frequent sequela of periodontitis have been classified into Class I, Class II, Class III and Class IV according to Glickman classification.

Periodontal therapy of chronic periodontitis should include the following items.

- Alteration or elimination of microbial etiology.
- Alteration or eradication of any contributing risk factor.
- Arresting the progression of periodontal disease.
- Preserving the dentition in a state of health comfort and function.
- Regeneration of the lost part of the periodontium.

Regeneration of the periodontium involves the formation of new cementum, periodontal ligament and alveolar bone (**Melcher 1976**).

Periodontal wound healing indicates that conventional periodontal therapy most likely results in repair rather than regeneration, and is characterized by the development of a long junctional epithelium between the root surface and the gingival connective tissue (**Caton et al 1980**).

A therapy proposed by **Melcher et al (1976)** hypothesized that the type of healing resulting after periodontal therapy is determined by the tissues which first repopulate the root surface.

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**Gottlow et al (1984)** reported that periodontal regeneration can occur if cells arise from the periodontal ligament and/or the alveolar bone are selectively allowed to repopulate the root surface. This information lead to the development of new therapeutic modality called Guided Tissue Regeneration (GTR).

To achieve periodontal regeneration several techniques and materials have been advocated, that were used separately or more than one treatment modality was used. These include open flap debridement, (modified Widman flap) and osseous surgery, which may offer probing depth reduction and gain of clinical attachment. New attachment achieved by these procedures is usually a result of the formation of long junctional epithelium with slight new connective tissue attachment and negligible new cementum formation (**Caton et al 1980**).

Bone grafts and bone graft substitutes were used to enhance bone formation and periodontal regeneration in periodontal osseous defects (**Congé et al 1978**). Among the grafting materials used is Cerasorb<sup>®</sup> which is a resorbable, fully synthetic, pure-phase  $\beta$ -tricalcium phosphate matrix with interconnecting porosity.

Guided tissue regeneration (GTR), the procedure in which a barrier is utilized to exclude epithelium from the root surface, has been shown to partially regenerate lost periodontal tissue with new bone, periodontal ligament and cementum (**Garrett et al 1995**). Several materials, both non absorbable and bioabsorbable barriers, have been applied for guided tissue regeneration.

Among the materials used to affect periodontal regeneration, is the use of Epi-Guide membrane. Epi-Guide is a uniquely structured, resorbable membrane. It is particularly suited for guided tissue

regeneration (GTR) in periodontology and is also successfully implemented in guided bone regeneration (GBR).

Epi-Guide<sup>®</sup> consists of a bioresorbable polymer fabricated from D, D-L, L-polylactic acid which safely degrades to carbon dioxide and water.

Epi-Guide<sup>®</sup> is hydrophilic and possesses a patented multi-layer architecture of hollow spaces which vary in porosity.

To get the best results combined periodontal therapy (CPT) of both bioabsorbable barrier and allograft are used together.

## **REVIEW OF LITERATURE**

Chronic periodontitis is defined as "An inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both."

Chronic periodontitis is the most common form of periodontitis. It is most prevalent in adults but can be observed also in children.

Chronic periodontitis is associated with accumulation of plaque biofilm and generally has a slow to moderate rate of disease progression, but periods of more rapid destruction may be observed. Increases in the rate of disease progression may be caused by the impact of local, systemic, or environmental factors that may influence the host biofilm bacteria interaction.

*Local factors* may influence plaque accumulation, *systemic diseases* such as diabetes mellitus and HIV may influence the host defenses, and *environmental factors* such as cigarette smoking and stress may influence the response of the host to plaque accumulation. Chronic periodontitis may occur as a localized disease in which less than 30 % of evaluated sites demonstrate attachment and bone loss, or it may occur as a more generalized disease in which greater than 30% of sites are affected.

The disease also may be described by the severity as slight, moderate, or severe based on the amount of clinical attachment loss.

A patient may simultaneously have areas of health and chronic periodontitis with slight, moderate and advanced destruction. (**Consensus report 1999**)

Clinical features may include combinations of the following signs and symptoms; edema, erythema, gingival bleeding upon probing and/ or suppuration. Chronic periodontitis with slight to moderate destruction is characterized by a loss of up to one third of the supporting periodontal tissues. In molars if the furcation is involved, loss of clinical attachment should not exceed class I (incipient). Slight to moderate destruction is generally characterized by periodontal probing depths up to 6 mm with clinical attachment loss up to 4 mm (**AAP 2000a**). Chronic periodontitis with advanced loss of periodontal support is characterized by a loss of greater than one third of the supporting periodontal tissues. Advanced destruction is characterized by periodontal probing depths greater than 6 mm with attachment loss greater than 4 mm, loss of clinical attachment in furcation if present will exceed class I (incipient). Radiographic evidence of bone loss is apparent. Increased tooth mobility may be present. (**AAP 2000b**)

The prevalence of severe periodontitis has been reported to vary from 5% to 20% of the different populations investigated according to the criteria employed to measure extent and amount of periodontal destruction (**Papapanou 1994, Papapanou 1996**).

Longitudinal studies conducted to describe the progression of untreated periodontitis have shown that the majority of sites losing attachment belong to a small subset of the population (**Goodson et al. 1982, Loe et al. 1986, Lindhe et al. 1989**).