Textbook of
Periodontology
for Undergraduate Students

According to AAP 2018 Classification

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to

our loving parents

Dr PS Kalsi and Mrs JP Kalsi
and
Er JR Wadhwa and Mrs Kamal Wadhwa

and also to our wonderful children
Meher and Arjan

Foreword

"There is no such thing as a 'periodontal patient,' as all patients with natural teeth have a periodontium." — Dr Glickman

We at times need to remind ourself of the above statement to know the true importance of periodontology in our daily practice. Probably no other specialty of dentistry has an all-encompassing effect on all fields of dentistry as periodontology. Dental surgeons, for several reasons, sometimes however, tend to overlook this fact and confine themselves to the 'task at hand' without managing a patient's periodontal needs adequately.

Periodontology is a seemingly simple but in reality, a vast and complicated subject. Its numerous different, yet intricately interlinked topics and concepts can be difficult to understand and memorize. Often many textbooks on periodontology, though well written, fail to attract a student's attention for being too voluminous. Teachers are in agreement that such 'great but bulky texts' are difficult to understand, memorize and often overwhelm the student into 'inaction'. Students justifiably feel lost and abandoned without access to good educational matter written in simple and student-friendly language.

Apparently, the authors have written this textbook keeping all this in mind. This textbook 'talks directly to the student' and explains what is important and why. It presents knowledge and concepts in a related and simple way. The text is amazingly easy to understand and concise but complete in all aspects. Topics have been so arranged that they act like building blocks that help conceptual learning and build connections between them. Diagrams are thoughtfully drawn and labelled to make the learning easier. Photographs speak volumes for the concept at hand. Keeping with recent developments, the entire text has been written according to the latest and in vogue, 2018 classification of periodontal and peri-implant diseases and conditions. This textbook is also unique due to its emphasis on the importance of a patient-centered approach to learning. What more could one ask for?

Although this textbook would be useful for all undergraduate students of dentistry, but it can also serve as an ideal handbook for postgraduate students and clinicians as well. I congratulate the authors for writing this masterpiece and I am sure, this textbook will find wide acceptance and will benefit both students and practitioners of dentistry.

Chandigarh

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Preface

With the introduction of the 2018 classification of periodontal and peri-implant diseases and conditions, the subject of periodontology and its concepts have undergone a drastic change. Renewed focus is now laid on preserving the health of the periodontium by early diagnosis and preventing initiation and worsening of periodontal diseases. All students of dentistry aiming to be successful practitioners should therefore not only learn intricacies of periodontal treatment but should also learn and master the art and technique of oral examination with uncompromising attention to detail. These notions were kept in mind while writing this textbook.

Due diligence and effort have been made to present the essential and critical topics of periodontology in a simple, flowcharted and 'to the point' manner. Thus, the information presented in this book is an all-inclusive review of the most essential and clinically relevant recent knowledge on basic concepts (*Chapters 1–5*), etiology and pathology (*Chapters 6–12*), planning, diagnosis and nonsurgical therapy (*Chapters 13–16*) and surgical therapy, inter-disciplinary relationships, supportive periodontal therapy and dental implants (*Chapters 17–22*). Commonly asked theory questions have been given at the end of each chapter.

To keep the reader engaged and related to the text at all times, a new idea of a continuous column with commonly asked viva questions running all along the text has been developed and used. These questions are placed in a way so as to precisely point to the section of text where the information related to the question is present. The aim is to help the student understand the underlying scientific concepts better. To help the reader attain and retain more information related to the running text, additional and background information has been presented in separate boxes titled 'Clinical Utility and Short Answer'. Clinically useful facts that are critical to the topic and patient management have been presented in boxes titled 'Alert'. Series of these two boxes contains information that when read additionally or as related information, adds to the understanding of the topic. Presented this way, the information that is indirectly related to the topic at hand does not 'clutter' the reader's mind but contributes immensely in building concepts and learning of the subject.

The authors are sure that dental students will be much better prepared in the subject of periodontology due to this effort by them.

Devinder Singh Kalsi
Parul Kalsi

Acknowledgments

Condensing the sea of information available on each topic of periodontology to fit the requirements of an undergraduate student was a tough task. To accomplish all this and put our ideas and concepts into words, lines and paragraphs and then to organize these in a flowcharted collection of topics in different chapters was not easy. We feel specially blessed to have completed this mammoth task in a short while.

We wish to express our gratitude to all our teachers from whom we have learnt the wonderful subject of periodontology and other dental subjects. Our gratitude also goes to our numerous colleagues for their constant support that has helped us grow as professionals. Special mention must be made here of Drs Ashish Jain, Rajan Gupta, Nymphea Pandit, Gautami, Rajinder Sharma, Shikha Tiwari and Vishakha. We remain especially indebted to Drs Ranjan Malhotra, Viniti, Shalini Gugnani, Kanteshwari Kummathali, Manik Sharma and Vandana for providing us that extra impetus time and again which proved crucial in shaping this book. This textbook has also been molded by the several suggestions and challenging questions posed by our students over time. The need for writing this textbook in its unique style particularly arose to satisfy the quest of knowledge of many such students. We remain indebted to all of them.

We are thankful to Mr SK Jain (CMD) and Mr Varun Jain (Director) CBS Publishers & Distributors Pvt Ltd, New Delhi, for their support in publishing this textbook. Appreciation is also due to Mr YN Arjuna (Senior Vice President—Publishing, Editorial and Publicity) and the able team of professionals under him for designing the impressive layout of this textbook.

Our family has stood by us during ups and downs and has cooperated by sacrificing time and attention that was rightfully theirs, during the writing of this textbook. We appreciate the constant guidance of Dr PS Kalsi a renowned author of several internationally acclaimed books on organic chemistry during the writing of this textbook. Without his help and support, this task would have been impossible. We are thankful to Dr Maninder Sokhey, who has always provided us unending professional support that has gone a long way in making us successful clinicians. Our children, Dr Meher Kalsi, MS, SR Neurosurgery, RML Hospital, New Delhi and Arjan Singh Kalsi, a computer enthusiast bring unmeasurable love and fulfillment into our lives. These have helped us pursue our academic interests, including this textbook, with zeal and vigor.

Devinder Singh Kalsi
Parul Kalsi

Contents

Chapter

1

Periodontology and Periodontics: An Introduction

Periodontology is the study of supporting tissues of teeth and implants in health and of diseases and conditions that affect them. The word periodontology is derived from greek words: 'peri' meaning around and 'odontos' meaning tooth. There are four supporting tissues of teeth: gingiva, alveolar bone, periodontal ligament and cementum; they are collectively called periodontium. ***Clinical practice*** of periodontology is called ***Periodontics***. A dental surgeon who practices periodontology is called a periodontist.

◀ *What is the difference between periodontology and periodontics?*

◀ *What is periodontium?*

Teeth and implants work together as connected units in an individual's dentition. Therefore a good knowledge of their supporting (*periodontal*) tissues is important for maintaining an adequately functioning dentition. Periodontology is one of the many specialties of dentistry. As all dental specialties are interlinked, thorough knowledge of each one of these is necessary to be a successful clinician. This is important even if a clinician prefers to restrict his/her practice to a specific dental specialty. From this point of view, periodontal health is important for success of any and all dental treatments. Afterall, of what use will a well-made bridge or restorative crown, perfect RCT, ideal orthodontic treatment, etc. be, if all these are done on teeth (*dentition*) that are mobile and not likely to adequately function or last for a reasonable time to justify cost and effort involved in treatment.

Periodontal treatment not only improves health of the dentition but it has also been proven to have a positive impact on an individual's systemic health. Periodontal health (*treatment*) also has a huge positive role in management of several systemic diseases and conditions. As both oral and systemic health and disease have an impact on each other, all dental specialties (*especially periodontology*) and medical specialties are therefore interrelated and interdependent.

Periodontal diseases have been known to occur in humans since ancient times. Ancient texts of Ayurveda, Ebers Papyrus, etc. have sections on oral health and treatment of gingival disease. Albucasis, an arabic physician who lived in middle ages not only documented the importance of calculus in etiology of periodontal disease but also designed a set of scalers for removal of calculus.

Modern periodontology has been developing rapidly since the 17th century. Several clinicians including Pierre Fauchard, John Hunter, Antony van Leeuwenhoek and many others are credited for development of a systematic approach to diagnose periodontal disease and its treatment. Pierre Fauchard (*considered to be the father of modern dentistry*), designed and developed periodontal instruments and the technique of scaling. In the 19th century, John W. Riggs recognized the importance of plaque and calculus in etiology of periodontitis and promoted maintenance of good oral hygiene to prevent it. Riggs was the first well-known clinician to restrict his practice to periodontics (*periodontitis was called 'Riggs' disease' at that time*). Major developments in understanding of periodontal health and treatment of periodontal disease occurred in the 20th century due to untiring

◀ *Name some clinicians who have contributed in development of periodontology in the past.*

◀ *What is Riggs disease?*

efforts of Drs Gottlieb, Orban, Widman, Neumann, Glickman, Waerhaug and their contemporaries.

Today, periodontology and periodontics continue to advance at a rapid pace due to clinical and research work of clinicians across the world. However, several details related to periodontal disease and its treatment are yet not known; a lot of work is presently being done to develop better understanding of periodontal disease and its treatment.

Chapter

2

Gingiva

A healthy periodontium is important for health and long-life of teeth. Understanding of normal features of periodontal tissues forms the basis of understanding of periodontal disease. Therefore, good knowledge of normal features or periodontal tissues is important for diagnosis and periodontal treatment.

PERIODONTAL TISSUES

Periodontal tissues are supporting structures of teeth. They are collectively referred to by the term ***periodontium*** (*perio–around; dontium–tooth*). There are ***four*** periodontal tissues:

◀ *How many periodontal tissues are there?*

1. Gingiva (*soft tissue*)
2. Periodontal ligament (*soft tissue*)
3. Alveolar bone (*hard tissue*)
4. Cementum (*hard tissue*)

Gingiva is discussed in this chapter whereas periodontal ligament, alveolar bone and cementum are discussed in chapter 3. Gingiva is masticatory mucosa—a type of oral mucosa.

ALERT!

Types of oral mucosa

◀ *What are the types of oral mucosa and its keratinization?*

There are ***three*** types of oral mucosa:

1. Masticatory mucosa
   a. Gingiva (*covered by keratinizing epithelium*)
   b. Hard palate mucosa (*covered by keratinizing epithelium*)
2. Specialized mucosa
   Dorsum of the tongue (*specialized for perceiving taste; covered by keratinizing but specialized epithelium containing taste buds*).
3. Lining mucosa (*covered by* ***non-keratinizing*** *epithelium*).

CLINICAL UTILITY AND SHORT ANSWER

Does masticatory mucosa (gingiva and hard palate mucosa) help in mastication?

No, only teeth masticate food. Masticatory mucosa only bears the impact of frictional forces of masticated food as it rubs past the gingiva and the hard palate during mastication. Masticatory mucosa is keratinized and is present on gingiva and hard palate.

GINGIVA

Gingiva surrounds and is attached to necks of teeth. A standard line diagram (Fig. 2.1) is used to depict a part of the periodontium in cross section.

Parts of Gingiva

Gingiva has ***three*** parts (Fig. 2.2):

◀ *What are the other names of free gingiva?*

1. Free gingiva (*also called—marginal* ***or*** *crestal gingiva*)

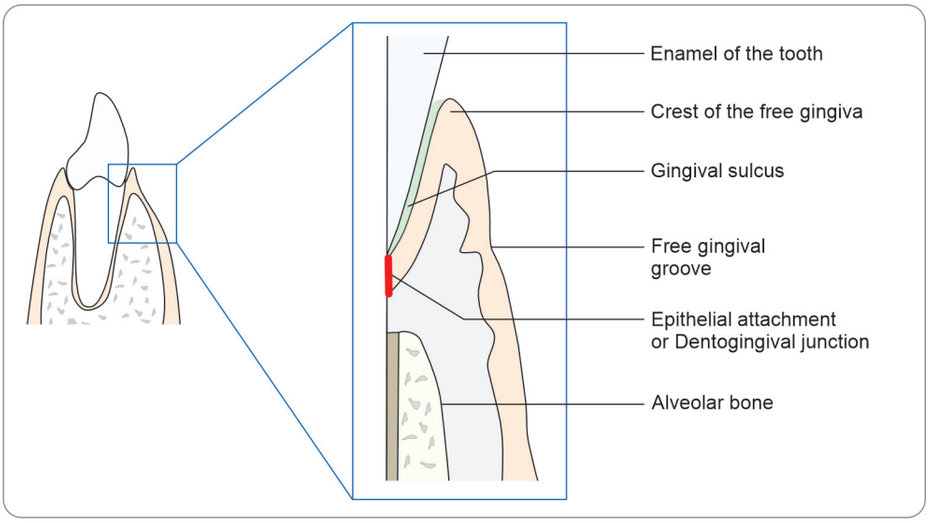


Fig. 2.1: Standard line diagram commonly used to depict a part of the periodontium in cross section

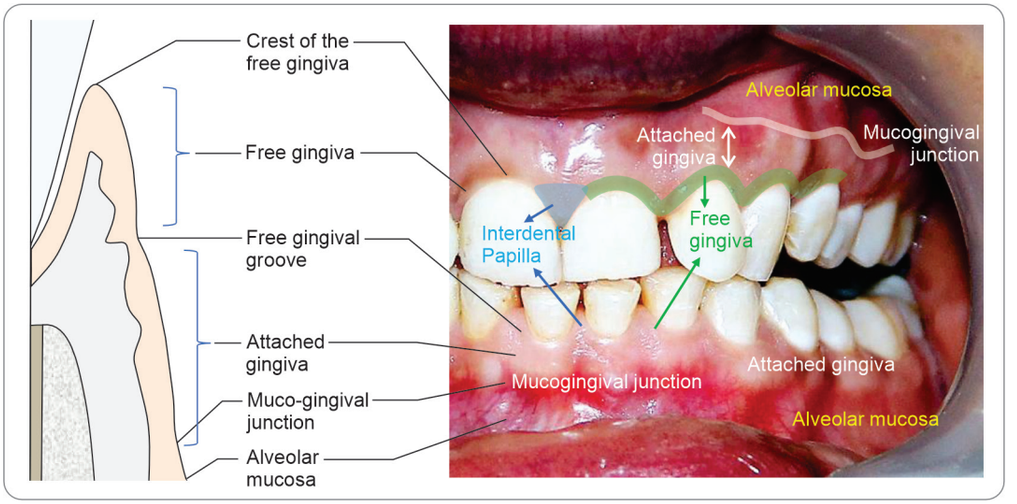


Fig. 2.2: Parts and anatomical landmarks of gingiva (*partially marked*) and corelation between clinical and histological landmarks and boundaries.

2. Attached gingiva
3. Interdental papilla (*also called interdental gingiva*).

Free Gingiva (*also called marginal* ***or*** *crestal gingiva*)

1. It is the coronal-most part of gingiva (Fig. 2.2).
2. It is not attached to the underlying tooth surface.

How does the free gingiva form the gingival sulcus? ▶

3. It surrounds the tooth like a collar and attaches to the tooth surface ***after*** folding inwards to create a pouch-like cavity called ***gingival sulcus***.
4. Free gingiva forms the ***soft tissue wall*** of the sulcus.

Is free gingival groove present in all individuals? ▶

5. Free gingiva is continuous with attached gingiva. A shallow 1 mm wide ***free gingival groove*** forms the clinical boundary between free and attached gingiva (Figs 2.2 and 2.3). Free gingival groove is present at the same level as the base of sulcus but can only be seen in ***50%*** of the population.

What type of epithelium lines the free gingiva? ▶

6. Free gingiva is lined by stratified squamous ***keratinizing*** epithelium on the ***oral/buccal and lingual/palatal surfaces*** and by stratified squamous ***non-keratinizing*** epithelium (*sulcular epithelium*) on the ***sulcular*** side (Fig. 2.12).

Fig. 2.3: Free gingival groove. It forms boundary between free and attached gingiva. As it is present at the same level as base of sulcus, the free gingival groove is a good indicator of level of base of sulcus and sulcus depth.

Gingival Sulcus

Gingival sulcus is a 'V' shaped pouch-like cavity (*shallow crevice/fissure*) formed by inward folding of free gingiva while attaching to tooth surface. The gingival sulcus extends from crest of gingiva to junctional epithelium (Fig. 2.4).

◀ *The gingival sulcus extends between which two anatomical landmarks?*

Depth of Gingival Sulcus

1. Depth of the normal gingival sulcus is ***variable***. It normally varies between ***0.5–3.0 mm*** **(*average depth is 1.8–2.0 mm*)**.
2. In a previously treated case of periodontitis that is now clinically healthy, a sulcus depth of ***4 mm*** is considered to be normal if there is no bleeding on probing at that site (*2018 classification*).
3. The ***ideal depth*** of the gingival sulcus is ***0 mm***.

◀ *What is the normal depth of gingival sulcus?*

◀ *What is the ideal depth of the gingival sulcus?*

Width of Gingival Sulcus

1. The gingival sulcus is ***very narrow*** because healthy free gingiva is very ***firmly adapted*** around the necks of teeth.
2. Due to this reason, sometimes, a periodontal probe cannot be inserted into it. If a periodontal probe is forced into the sulcus, it can cause tearing of free gingiva and pain (Fig. 2.5). However, microorganisms can easily enter and survive in the gingival sulcus.

◀ *Why is it difficult to insert a periodontal probe into a healthy sulcus?*

CLINICAL UTILITY AND SHORT ANSWER

Why does a sulcus that is more than 2 mm deep have greater chance of developing periodontitis?

- Periodontitis is caused by plaque microorganisms.
- The plaque microorganisms are kept in check by natural physical cleansing mechanisms of the oral cavity and body's immune system. These are supported by instituted physical cleansing mechanisms, e.g. tooth brushing, flossing, etc.
- However, toothbrushing and flossing are practically ineffective in maintaining health of the sulcus after a depth of 2–3 mm.
- Therefore, maintenance of a healthy sulcus is difficult if the depth of the sulcus is more than 3 mm. In some cases, sulci up to 4 mm can be normal but such cases are however not common.
- A diseased sulcus is called a pocket. Any factor that leads to increased plaque deposition will favour deepening of the gingival sulcus and its conversion to periodontal pocket.

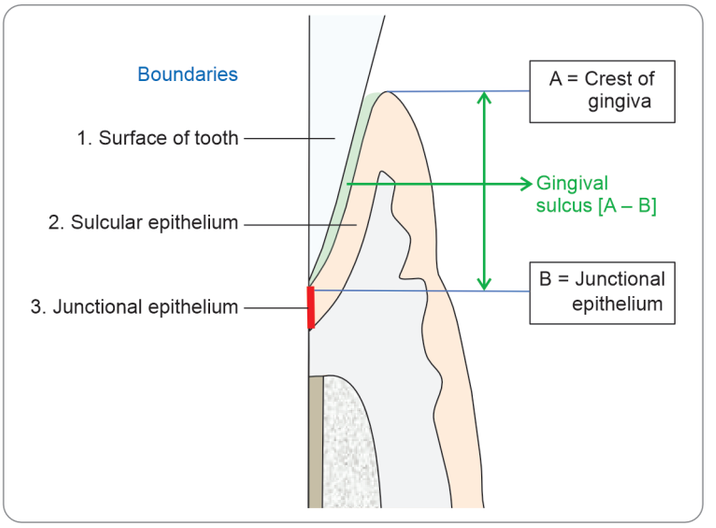


Fig. 2.4: Boundaries of the gingival sulcus (*green-shaded area*)

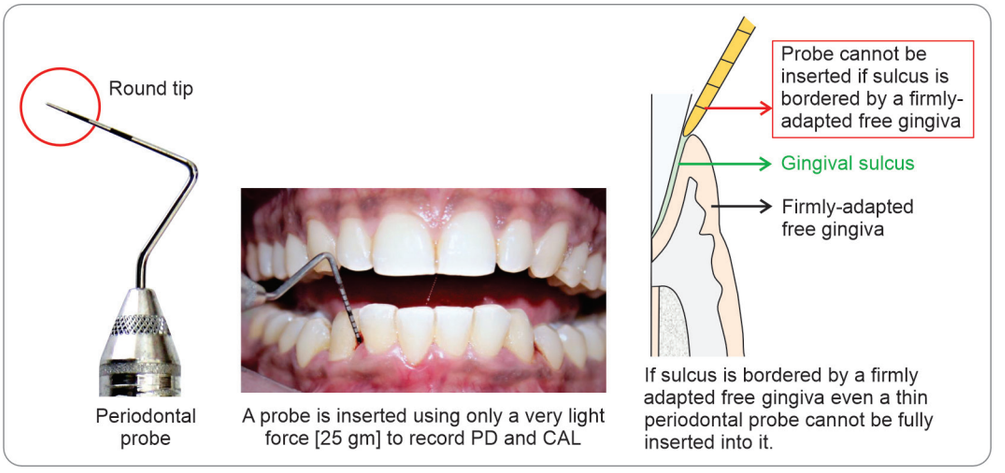


Fig. 2.5: Probing to record clinical probing depth. Probing is done with a periodontal probe that has a round and blunt tip (*end*). Sometimes firm and tightly adapted free gingiva does not allow easy insertion of a periodontal probe without tearing the gingiva.

Boundaries of Gingival Sulcus

The sulcus has ***three*** boundaries (Figs 2.4 and 2.12)

1. **Inner side**—surface of tooth.
2. **Outer side**—sulcular epithelium of free gingiva.

Which cells make the base of the sulcus? ▶

3. **Base**—coronal most cells of junctional epithelium.

How to measure depth of gingival sulcus or pocket

There are ***two*** ways of measuring depth of a sulcus and pockets (*in periodontitis sulcus is called a pocket*):

What is PD? ▶

How much pressure should be used for probing? ▶

I. **Clinical sulcus depth:** Sulcus or pocket depth measured with a periodontal probe is called ***probing sulcus depth or pocket depth*** (*PD*) (Fig. 2.5). For this, a probe is inserted into the sulcus under ***light*** pressure (*approximately 0.25 Newton force, i.e. 25 gm force*) till ***light*** resistance is felt and measurement on the probe is noted. PD tells us the ***current*** status of disease or health at a particular site. William's probe, WHO probe (*it has a 0.5 mm ball tip*), etc. are commonly used periodontal probes. Naber's probe is curved probe used for probing furcations of multirooted teeth (*Fig. 13.8*). Clinical PD is ***more*** than histological depth (Fig. 2.6).

Does PD tell us the current or past status of disease? ▶

II. **Histological:** In a ***histological*** section, distance from crest of free gingiva to base of sulcus (*coronal most cells of the junctional epithelium*) is called histological sulcus depth. Histological depth is ***less*** than clinical PD (Fig. 2.6).

◀ *Which is larger—clinical or histological sulcus depth?*

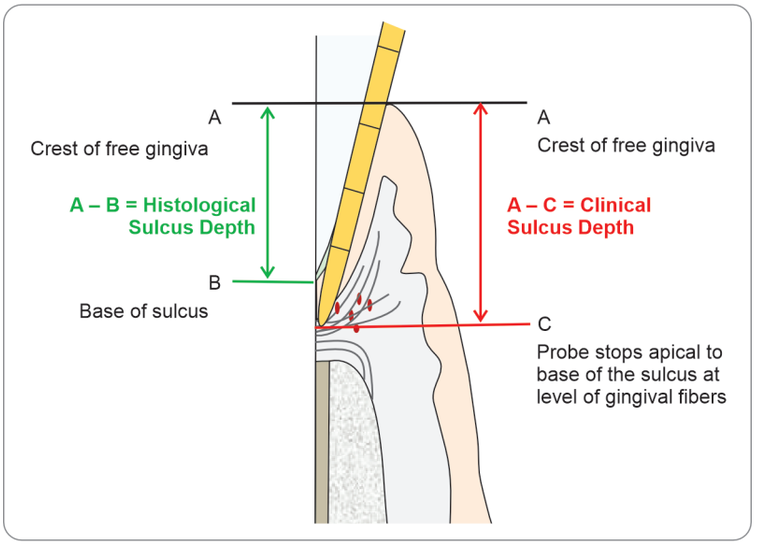


Fig. 2.6: Difference between histological and clinical sulcus depth

CLINICAL UTILITY AND SHORT ANSWER

Why clinical probing depth (PD) is always more than the histological sulcus depth?

For recording PD, a periodontal probe is inserted into the sulcus under slight pressure till a light resistance is felt (*approximately 0.25 Newton force, i.e. 25 gm force*). Because junctional epithelium at base of the sulcus is nonkeratinized, it does not provide any feelable resistance to the probe tip. Therefore, in spite of such light probing pressure, the probe tip penetrates through the base of the sulcus and stops only when resistance is provided to it by the fibers of the dento-gingival fiber group (Fig. 2.6). Thus, recorded clinical probing depth is more than the histological depth.

CLINICAL UTILITY AND SHORT ANSWER

How can you know the correct probing pressure?

25 grams force is approximately the force needed to cause a ***slight*** depression and blanching in the bulb of the thumb of finger when pressed with a probe tip.

CLINICAL UTILITY AND SHORT ANSWER

Why sulci or pockets' depths cannot be measured with an explorer instead of a probe?

An explorer has a sharp tip (*end*) while a periodontal probe has a rounded tip (Figs 2.5 and 13.7). In contrast to the probe, the tip of an explorer being sharp will penetrate easily and more into the gingival tissues (*perhaps stop at crest of the alveolar bone*) and record a greater probing pocket depth (*PD*). An explorer is used to detect hard tissue defects only.

◀ *Why explorer cannot be used for recording PD?*

CLINICAL UTILITY AND SHORT ANSWER

What is clinical attachment level (*CAL*) or clinical attachment loss (*CAL*)?

- The distance from cemento-enamel junction (*CEJ*) to tip of the probe that has been inserted into the sulcus/pocket for taking a probing depth is called Clinical attachment level (Fig. 2.7).
- As CAL quantifies periodontal attachment loss, it is also called clinical attachment loss.
- CAL is zero if base of the sulcus is coronal to the CE junction, i.e. there is no periodontal loss or recession (Fig. 2.21, diag. 1).
- CAL is a measure of total (*past + present ongoing*) periodontal destruction that has occurred due to periodontitis whereas PD tells us the present-day status of periodontitis.

◀ *What is CAL?*

◀ *What does CAL tell us: The current or past status of disease?*

What is the difference between CAL and PD? ▶

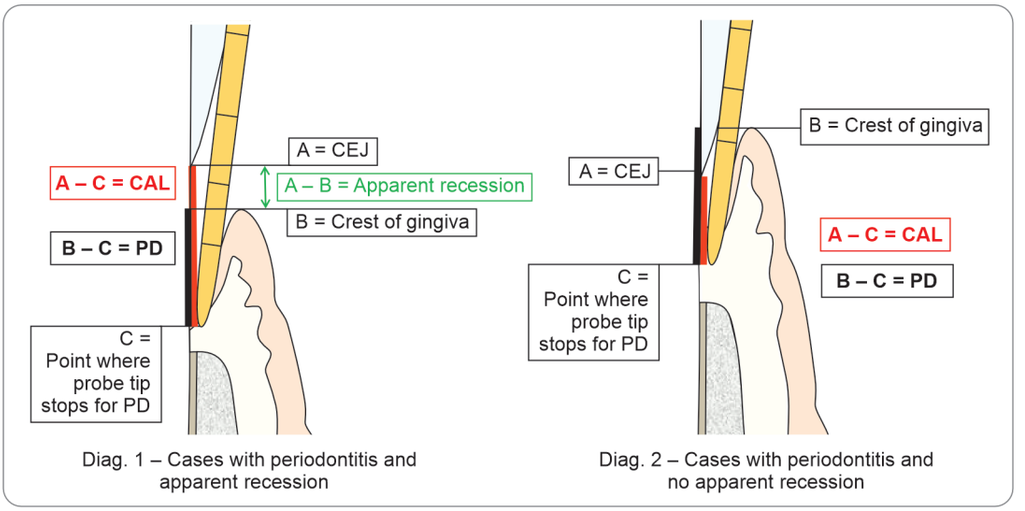


Fig. 2.7: Clinical attachment level (*CAL*) *vs* probing depth

CLINICAL UTILITY AND SHORT ANSWER

When and why can recorded CAL and PD be inaccurate? ▶

Why are CAL measurements not accurate in some cases?

In some subjects, the healthy free gingiva is so firmly adapted to the tooth that insertion of a probe to the base of the sulcus is not possible. This leads to recording of lesser than actual PD and CAL measurements (Fig. 2.5).

In other cases, if the gingiva is inflamed or excess probing pressure is used the probe gets pushed apically to the crest of the bone. In such cases, recorded CAL and PD are more.

CLINICAL UTILITY AND SHORT ANSWER

Which local and systemic factors cause periodontitis and lead to deepening of sulcus?

As the sulcus deepens, it becomes diseased (*periodontitis*); it is then called a pocket. Factors that cause deepening and conversion of the sulcus to a pocket are:

- Increased plaque deposits
- Inadequate natural cleansing, e.g. soft diet, improper tooth contours, etc. and plaque control mechanisms, e.g. faulty tooth brushing, etc.
- Systemic factors leading to xerostomia, (*decreased salivary flow*), decreased immunity, e.g. in diabetes, etc.
- Malocclusion (*makes plaque control difficult*)
- Iatrogenic factors, e.g. overhanging defective restorations, open proximal contacts, etc.

Contents of Gingival Sulcus

What is present in a sulcus and pocket? ▶

a. Food debris
b. Saliva and its constituents like salivary mucin, etc.
c. Gingival crevicular fluid and its constituents
d. Plaque microorganisms and their products
e. Desquamated epithelial cell
f. Leukocytes (*primarily neutrophils*)
g. Pus
h. As the sulcus converts to a pocket, calculus may also be seen.

Gingival Crevicular Fluid (*GCF*)

What is GCF? ▶

A sticky, colourless fluid called gingival crevicular fluid (*GCF*) is present in the gingival sulci and periodontal pockets. It is an ***inflammatory exudate*** and is produced in response to plaque microorganisms and other factors producing inflammation, e.g. physical injury, etc.

As rate of production and flow of GCF increases with increasing of intensity of inflammation, GCF is therefore increased in gingivitis and periodontitis (*pocket formation*).

◀ *Is GCF production more in pockets? Why?*

Constituents of GCF

1. Cells: Epithelial, WBCs and microorganisms.
2. Organic compounds: Carbohydrates, proteins (*immunoglobulins, etc.*), lipids, etc.
3. Metabolic and bacterial products: Prostaglandins, endotoxins, antibacterial factors, etc.
4. Enzymes and enzyme inhibitors: Alkaline phosphatase, proteolytic enzymes, lysozymes, etc.

◀ *What are the contents and functions of GCF?*

Functions of GCF

1. Controlling microorganisms in the sulcus/pocket by:
   a. Flushing-out microorganisms and debris.
   b. Providing a medium in which anti-microbial immune compounds and cells can exist in the sulcus/pocket.
2. Adhesion of free gingiva to the tooth due to its stickiness.

Measuring Rate of Flow of GCF

GCF is produced in very ***small*** quantity. It can therefore only be collected by ***special*** equipment. Methods of collection of GCF are:

◀ *How is rate of flow of GCF measured?*

1. **Absorbing filter paper strips:**
   i. **Intra-sulcular:** Absorbing strip is inserted into the sulcus either only till the entrance or all the way to the bottom of the sulcus. This is the most commonly used method.
   ii. **Extra-sulcular:** Strips are overlaid and adapted on surface of the gingiva and the tooth thereby bridging over the gingival sulcus.

   After the strips are wet with GCF, the rate of flow of GCF can be known by analyzing the strips in three ways:
   i. It is stained with ninhydrin and the area of the stained portion of the paper strip is measured.
   ii. Paper strips are weighed before and after collection of GCF. The increase in weight is co-related to volume of GCF produced.
   iii. An electronic device, e.g. periotron can be used. The strip is inserted into the *periotron* which gauges the wetness of the paper strip and gives a digital reading.
2. **Collecting the GCF in micropipettes (*microcapillaries*).**
3. **Sulcular washing methods:** A known volume of specially prepared solution is flowed into the sulcus and then re-aspirated from the sulcus itself using a collection needle. The entire collected fluid (*prepared solution + GCF*) is then measured and analyzed.
4. **Pre-weighed absorbent-twisted threads:** Pre-weighed absorbent threads can be tied at the necks of teeth at the free gingival margin or inserted into the sulcus/pocket and allowed to absorb GCF. The threads are then weighed to know increase in weight after a set interval of time. The increase in weight can be co-related to volume of GCF produced.

CLINICAL UTILITY AND SHORT ANSWER

What is the clinical significance of GCF?

Increase in rate of flow of GCF is directly proportional to severity of gingival inflammation (*gingivitis and periodontitis*).

ALERT!

Tetracycline is an antibiotic of choice for treating periodontitis because:

- It is a broad-spectrum antibiotic.
- Concentration of tetracycline in GCF is ***8–10*** times more than that in the blood. This high concentration of tetracycline makes it very effective in killing plaque microorganisms in the pocket.
- Tetracycline is used only in ***addition*** to scaling and root planing and other surgical techniques to treat some cases of periodontitis. It must ***not*** be routinely used for treating periodontitis or gingivitis.

◀ *What is the concentration of tetracycline in GCF?*

◀ *Why is tetracycline a drug of choice in treating periodontitis?*

CLINICAL UTILITY AND SHORT ANSWER

What is coral pink color?

It is a brown–orange shade of pink. Corals are sea animals of brownish-orange colour. As base colour of gingiva closely resembles the colour of such corals, the gingiva's colour is described as coral pink color.

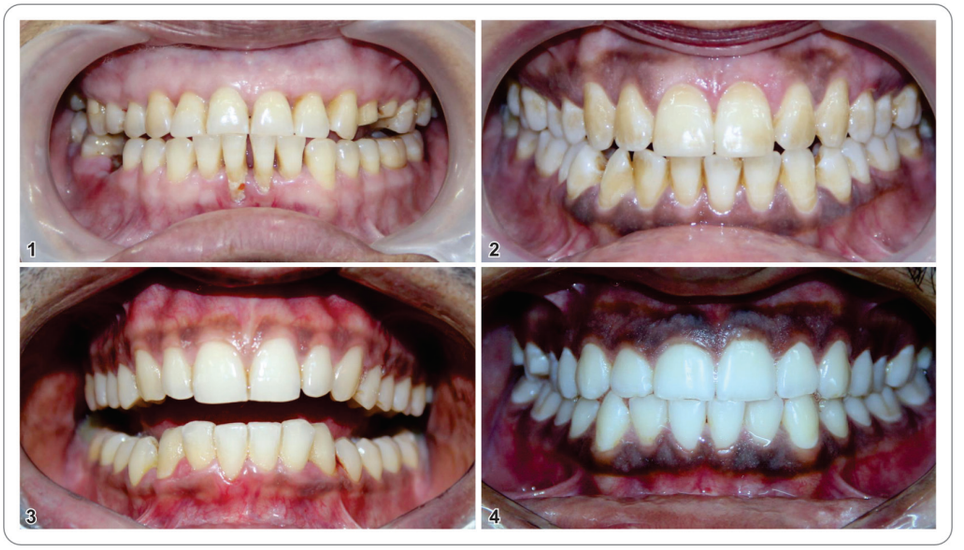


Fig. 2.15: Base colour of gingiva is coral pink and shows several variations due to 'add-on' factors like melanin pigmentation, etc. in different individuals. The alveolar mucosa has a dull dark red color.

Factors on Which Colour of Gingiva Depends

- **Degree of vascularity:** Greater the vascularity (*blood supply*), redder the color. Higher number of vessels supplying the area and/or increased blood supply due to vascular dilatation (*e.g. acute inflammation, i.e. acute gingivitis, etc.*) increases supply of oxygenated blood to the gingiva. This imparts a red color to the gingiva.
  Stasis of blood (*in chronic inflammation, i.e. chronic gingivitis and periodontitis*) causes reduction of oxygen in blood (*cyanosis*). This imparts a ***bluish*** colour to gingiva (*venous blood is blue while arterial blood is red*). If stasis occurs, the color of the gingiva changes to magenta, maroon, burgundy, etc. colours.

◀ *How does inflammation in gingiva affect its colour?*

- **Thickness of epithelium:** Greater the thickness of the epithelium, lighter will be the colour and vice-versa.
- **Degree of keratinization:** Greater the keratinization, lighter will be the color. If there is extreme hyperkeratinization, the gingiva will appear white.

◀ *How do thickness of epithelium and degree of keratinization affect colour of gingiva?*

- **Degree of pigmentation (*melanin*):** Melanin pigment imparts a brown color to the gingiva. Higher melanin pigmentation results in darker coloured gingiva. Melanin pigmentation can be in patches or diffused.

II. Contour of Gingiva

◀ *What is the contour of normal gingiva?*

- The clinically healthy gingiva has a ***'knife edge' deflecting*** and ***scalloped*** or ***arcuate contour*** (*series of semi-circles or arcs*) (*Fig. 2.16, deflecting contour-blue arrow, scalloping shown as yellow line*).

◀ *Is root prominence related to gingival contour?*

- If tooth surface is prominent (*convexity of tooth surface is increased*), the arcuate pattern or scalloping of overlying gingiva increases and the gingival crest is placed at a more apical level, e.g. on the labial surface of teeth in labio-version (Fig. 2.17, blue arrows) and vice-versa (Fig. 2.17, red arrows).

◀ *Is degree of scalloping of gingiva related to thickness of marginal gingiva?*

- As scalloping of gingiva increases, marginal gingiva gets thinner (Fig. 2.17, blue arrows); if on the other hand, scalloping decreases, i.e. becomes flatter, marginal gingiva becomes blunt, rounded and bulbous (Fig. 2.17, red arrows).

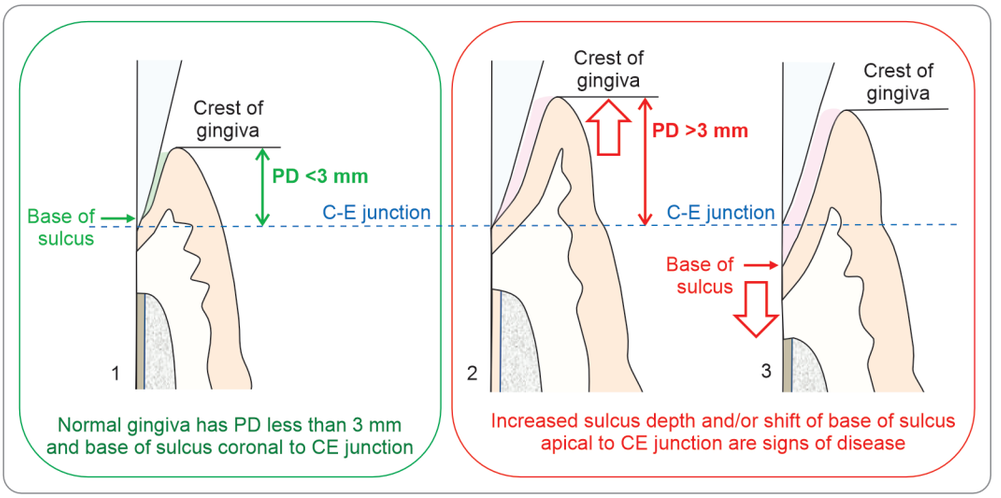


Fig. 2.19: When crest of gingiva is located on enamel, both probing sulcus/pocket depth (*PD*) and location of base of sulcus are taken into account for judging gingival normalcy.
In normal gingiva, both gingival crest and base of sulcus must be located at/coronal to the CE junction (*diag. 1, green arrow*) and sulcus depth (*PD*) must be less than 3 mm (*diag. 1 green double arrow*). If sulcus depth (PD) is more than 3 mm (*diag. 2 red double arrow*) and/or base of sulcus is apical to CE junction (*diag. 3, red arrow*) these are abnormal.

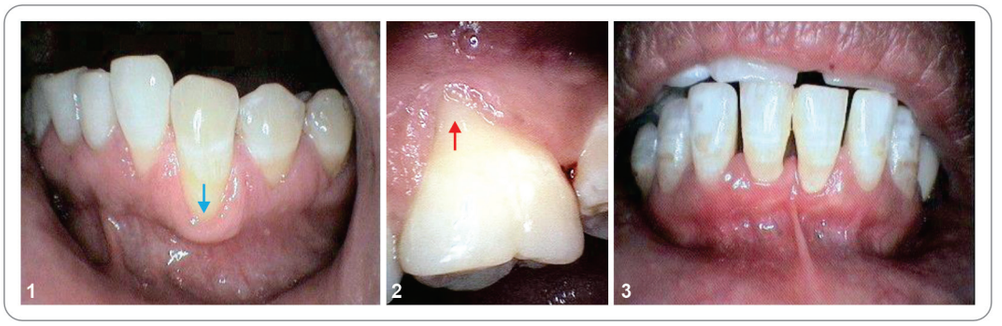


Fig. 2.20: Reduced periodontium due to prominent position of the tooth (*diag.1, blue arrow*), prominent root of multirooted tooth (*diag. 2 red arrow*) and in a successfully treated case of periodontitis (*diag. 3*).

VI. Size of Normal Gingiva

The normal size of the gingiva ***cannot*** be described in physical terms. While deciding on size, i.e. whether it is normal or abnormal, the following have to be kept in mind:

1. Normal-sized gingiva will exhibit normal clinical features.
2. Although gingiva can increase in size (*due to increase in its bulk*), reduction in size below the 'normal' size is ***not*** possible.

◀ *Describe the size of normal gingiva?*

The following can be safely assumed from the above facts:

1. Gingiva is considered to be of normal size if there is no overt increase in size.
2. Gingiva which is not normal will usually be enlarged.
3. Increase in size also alters other normal clinical characteristics of gingiva.

Thickness of Gingiva

Clinically two types of normal gingival thicknesses have been described:

1. Thick gingival biotype (Fig. 2.22, diag. A).
2. Thin gingival biotype (Fig. 2.22, diag. B).
   - Thin gingival biotype is associated with narrow zone of keratinized gingiva and with small teeth.

◀ *Is thickness of gingiva related to width of keratinized gingiva?*

Fig. 6.10: Increased plaque is seen on relatively inaccessible areas of teeth. In this picture plaque is seen in inaccessible furcation area of a mandibular molar whereas other surfaces of teeth are relatively clean.

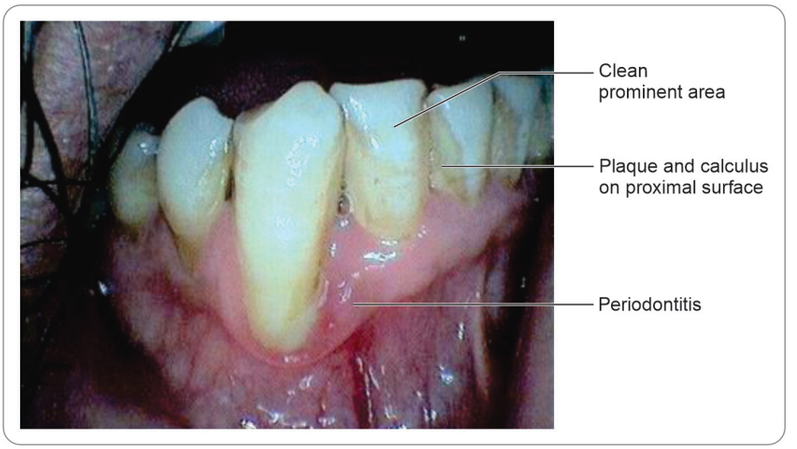


Fig. 6.11: Prominent areas of teeth are cleaned better by natural and instituted mechanisms and vice-versa. In this picture, note that prominent areas of teeth are cleaner while less prominent, receded and less accessible areas, e.g. proximal surfaces of teeth have more plaque, stains and calculus.

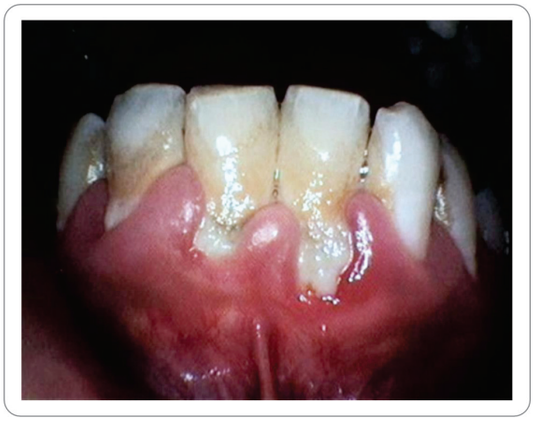


Fig. 6.12: More plaque is seen if there is preexisting gingivitis and periodontitis. This is so because, in such cases, rate of formation of plaque increases (*due to increased nutrition from GCF*), plaque now has more physical protection (*from enlarged gingiva*) and patient with ongoing gingivitis or periodontitis starts to restrict oral hygiene measures due to fear of gingival bleeding after tooth brushing, flossing, etc.

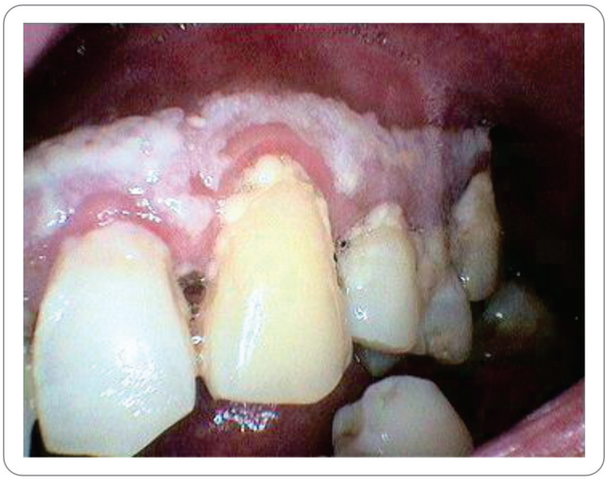


Fig. 6.13: Plaque deposits on gingiva in a 65-year-old male patient who does not brush his teeth due to untreated trigeminal neuralgia (*brushing triggers unbearable severe flashes of pain in this patient*).

Role of Saliva in Formation of Plaque

Saliva plays a role in both formation and reduction of plaque. On the whole, saliva's role in plaque formation and maturation is more of protective nature. Plaque forms in greater quantity and higher speed in patients with relatively less saliva (*xerostomia*) (Fig. 6.14).

◀ *How does saliva increase plaque formation?*

◀ *How does saliva help plaque formation at other sites?*

Saliva *helps* in formation of plaque by:

1. Causing formation of acquired pellicle, which is the first step in formation of plaque.
2. Acting like as a reservoir of microorganisms. Solitary (*planktonic microbes*) are suspended in saliva which scatters them to other oral sites and thus helps plaque formation at new sites.
3. Salivary constituents act as food and nutrients for plaque bacteria.
4. Bacteria clump together using salivary proteins, this helps bacteria survive their dispersion to other sites.
5. Serving as a circulatory medium. Saliva flows in and out of plaque mass via channels within plaque. This crude circulatory system of plaque helps in plaque growth by enabling nutritional and metabolic exchanges, intra and inter-species communication, etc.

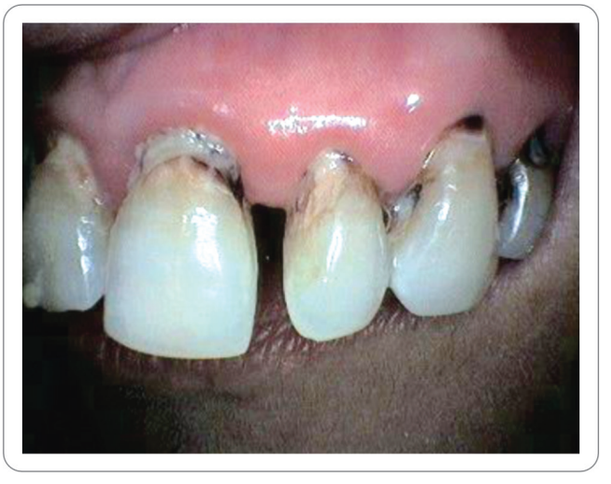


Fig. 6.14: Increased caries and periodontitis (*plaque induced diseases*), in a patient with dry mouth (*xerostomia—less saliva*)

Clinical Features of TFO

Can features of TFO increase or decrease in severity?

Clinical features of TFO can vary in different patients and in different teeth in the same patient. If occlusal forces are reduced, TFO will decrease and continue in a ***chronic*** (***milder***) form or may subside completely. The clinical features of TFO are:

What are the clinical features of TFO?

1. ***Pain*** and ***tenderness*** in:
   a. Affected teeth (*due to inflammation in periodontal ligament*).
   b. Muscles of mastication (*especially in temple region and on sides of mandible*). This is commonly seen in cases where TFO is due to bruxism.
   c. Temporo-mandibular joints (*TM joints*).
2. ***Increased mobility*** in involved teeth.
3. ***Attrition*** (*excessive*) of incisal and occlusal surfaces of teeth (Fig. 9.5, diags B and C) and abfraction cavities (Figs 9.5. diag. D).

Why do teeth with TFO become hypersensitive and non-vital?

4. ***Hypersensitivity*** to cold and hot foods in the affected teeth. This happens when inflammation extends from PDL into pulp (*via apical foramen or accessory canals*).
5. Teeth can become ***non-vital***. This happens if vessels supplying the pulp of the tooth get damaged due to very high occlusal forces.

What are the radiographic features of TFO?

6. Radiographic features:
   a. ***Widening of periodontal ligament and lamina dura*** (Fig. 9.2, diags B and C).
   b. ***Funneling of tooth socket*** (Figs 9.4 and 9.8).
   c. Increased thickness and radiopacity of alveolar bone trabeculae.
   d. Change in direction and orientation of bone trabeculae.

What causes change in position of a tooth?

7. ***Drifting*** or ***pathologic migration:*** If traumatic occlusal forces remain for a long period of time, it can lead to change in position of the tooth (*tooth shifts to a new position where occlusal forces are lesser*) (Fig. 9.16) (*pathologic migration is described in detail later in this Chapter*).
8. ***Acute*** TFO can cause:
   a. Cemental tears
   b. Necrosis in periodontal ligament.
   c. Abscess formation in periodontal ligament.

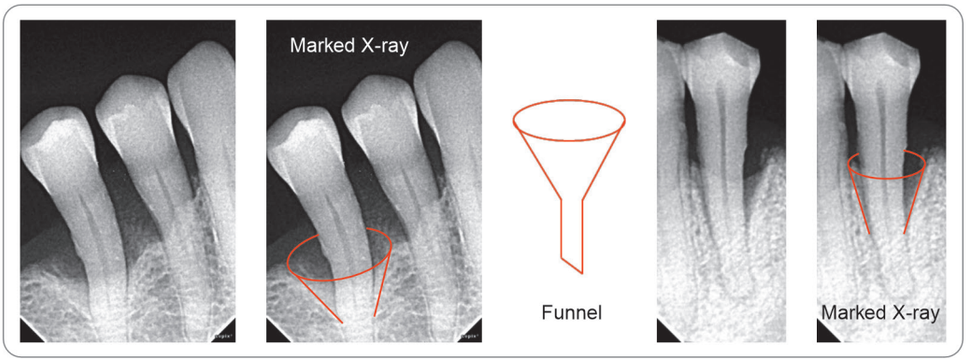


Fig. 9.4: Funneling of socket in TFO (*red marking*) is classical sign of chronic TFO

Stages of TFO

What are the stages of TFO?

If high (*traumatic*) occlusal forces are put on teeth, the periodontium responds to them in ***three*** distinct stages:

Stage I: Injury; Stage II: Repair; Stage III: Adaptive remodeling.

What is the first stage of TFO?

Stage I: Injury

Which periodontal tissue does not get affected by traumatic occlusal force?

- Injury occurs because high occlusal forces exceed load bearing capacity of periodontium. Injury is seen in alveolar bone, PDL and cementum. ***Gingiva*** *is* ***not*** affected by traumatic occlusal force; it therefore shows no features of TFO.
- High occlusal force causes areas of increased ***pressure*** and ***tension*** within the periodontium.

- **Increased pressure** causes:
  - ***Resorption*** of alveolar bone. In extreme cases tooth resorption may also be seen.
  - Compression of blood vessels which results in reduced blood supply to periodontal tissues; this is the chief cause of injury due to TFO.
  - Elongation and compression of periodontal ligament fibers.
- **Excessive tension** (*stretching/pulling*) occurs as a result of high occlusal force. It causes stretching of PDL fibers. While normal tension results in formation, excessive tension results in:
  - ***Resorption*** of alveolar bone.
  - Pulling and tearing of walls of blood vessels leading to hemorrhage in PDL.
  - Tearing and breaking of PDL fibers.

What does increased pressure on the periodontal tissues result in?

What effect does normal tension on periodontal fibers have on alveolar bone?

What does increased tension on the periodontal fibers result in?

CLINICAL UTILITY AND SHORT ANSWER

Which area of the periodontium/teeth is the most susceptible to injury by traumatic occlusal force?
Periodontium of furcation area of multi-rooted teeth is the most susceptible to injury by traumatic occlusal forces (*TFO*).

Stage II: Repair

- Repair activity in periodontium is significantly increased after injury (*TFO*).
- For this, resorptive cells of the periodontium, i.e. osteoclasts, cementoclasts and fibroblasts first remove damaged tissue.
- The formative cells, i.e. osteoblasts, cementoblasts, fibroblasts then lay down new periodontal tissue to increase load bearing capacity of alveolar bone, PDL and cementum. As ***gingiva*** *is* ***not*** affected by traumatic occlusal force; it shows no features of TFO.

What is the second stage of TFO?

Which periodontal tissue does not show increased repair after injury by traumatic occlusal force?

Stage III: Adaptive Remodeling

If TFO (*injury in the periodontium*) continues and the existing periodontal tissues are unable to bear higher occlusal forces, the periodontium then adapts itself by increasing its load bearing capacity to prevent further injury. This occurs by:

i. ***Widening*** of periodontal ligament (Fig. 9.2, diag. B and C white arrows)
ii. ***Thickening*** of lamina dura
iii. Thickening of cementum (*microscopic*).
iv. Formation of reinforcing new bone (*buttressing of alveolar bone*) to reinforce existing alveolar bone. If the reinforcing bone forms within the jaws, it is called ***central buttressing***. If reinforcing bone forms on facial and lingual/palatal alveolar cortical bone plates it is called ***peripheral buttressing***.
Peripheral buttressing may produce a 'shelf' like enlargement of the alveolar bone margin called '***lipping***' (*more commonly seen on facial side*) (Fig. 9.5, diag. B).

What is the third stage of TFO?

What is adaptive remodeling?

What are the features of adaptive remodeling?

What is buttressing bone formation?

What is central buttressing bone formation?

What is peripheral buttressing bone formation?

What is lipping?

After repair and adaptive remodeling, periodontium becomes stronger and can now bear increased occlusal forces better. Due to this, high occlusal forces now do not cause TFO (*injury in the periodontium*).

Can periodontium bear increased occlusal forces better after repair and adaptive remodeling?

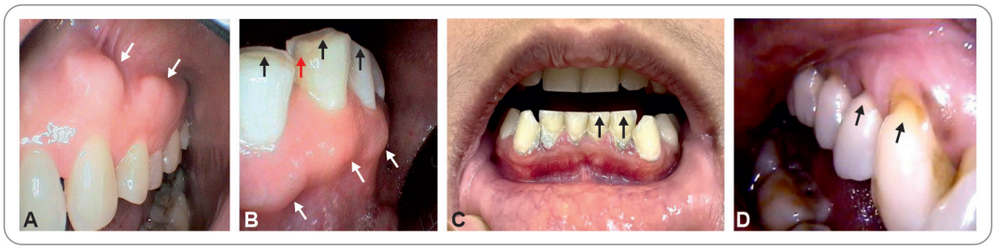


Fig. 9.5: Bone exostosis (*diag. A, white arrows*) and lipping (*peripheral buttressing*) of alveolar bone crest (*diag. B, white arrows*) in a patient of chronic TFO (*bruxism*). Presence of prominent wear facets, incisal and occlusal attrition (*diags B and C, black arrows*), tooth fracture (*diag. B, red arrow*), abfraction cavities (*diag. D, black arrows*) are other signs of TFO.

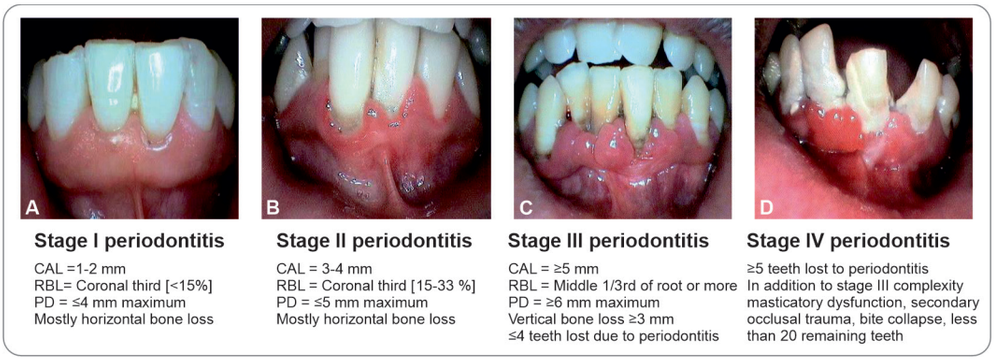


Fig. 10.15: Stage I to IV periodontitis

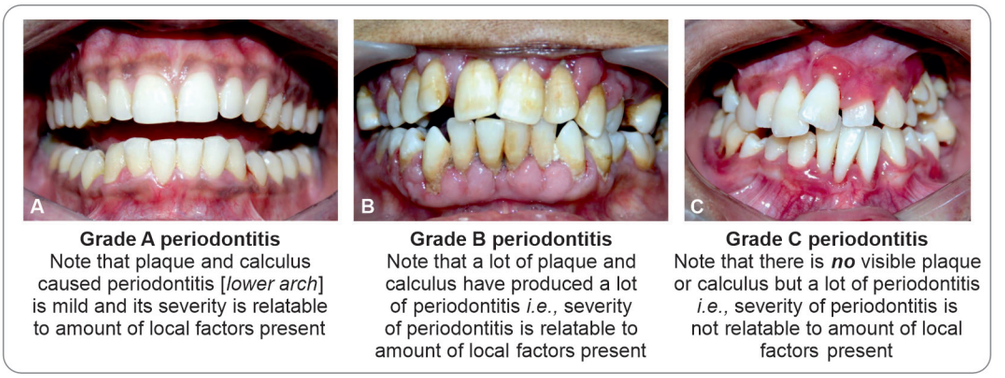


Fig. 10.16: Grade A, B and C periodontitis. In grade A and B periodontitis (*diags A and B*), periodontal destruction and severity of periodontitis is usually co-relatable to the number and amount of local factors (*plaque calculus, etc.*). In grade C periodontitis (*diag. C*), this co-relation is generally missing and there are too little local factors to explain the great amount of disease. In grade C periodontitis with molar-incisor pattern (*periodontitis restricting to all 1st molars and all incisors—localized aggressive periodontitis; 1999 classification*), this correlation maybe totally absent.

clinical features, it deserves special mention in this text. Grade C periodontitis fits the description of aggressive periodontitis (*1999 classification, AAP*) in some aspects. Earlier to 1999 classification, aggressive periodontitis has also been called rapidly progressing periodontitis, juvenile periodontitis, paradontosis, etc.

When compared to grade A and B periodontitis, grade C periodontitis:

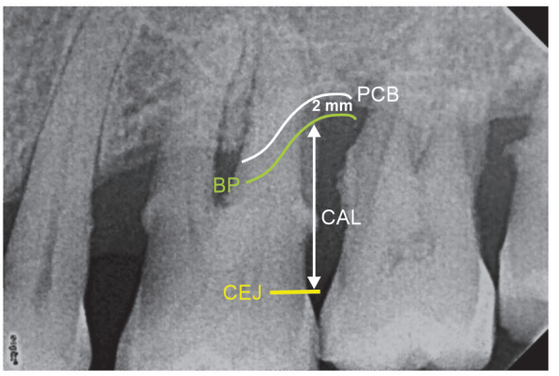
- Progresses at a very fast rate.
- Does not respond to standard periodontal treatment.
- Is usually present in patients with certain systemic diseases and conditions, e.g. diabetes, immune defects, etc.
- Grade C periodontitis especially with molar-incisor pattern, is considered to be an ***extreme variant*** type of periodontitis that is caused by a combination of immune defects and specific microorganisms.

Peculiar and Differentiating Clinical Features of Grade C periodontitis (and comparison of features of grade A and grade B periodontitis with those of grade C periodontitis)

Clinical features in fast progressing grade C periodontitis are ***more evident*** in later stages of the disease, i.e. stage II, III and IV.

How fast do grade A, B and C periodontitis progress?

1. Grade C periodontitis has a very fast rate of progression. It is usually 3–4 times faster than grade A and B periodontitis. Grade A and B periodontitis show ***no*** or up to ***2 mm bone loss*** in ***5 years***. In untreated cases of Grade C periodontitis ***more than 2 mm*** bone loss can be seen in a period of ***5 years*** (*stages III and IV (stages of disease showing severe periodontal loss) are reached very fast*).

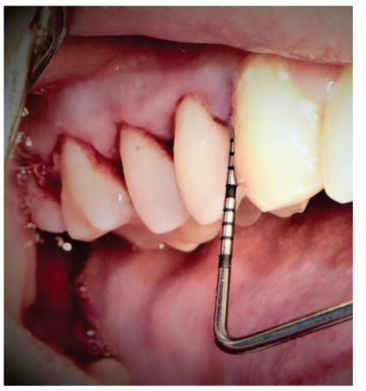


PCB = Present crest of resorbed bone
BP = Estimated level of base of pocket [*2 mm coronal to PCB*]
PCB – BP = Biologic width [*2 mm coronal to crest of bone*]
CEJ = Cementoenamel junction
CEJ – BP = CAL [*CEJ to estimated base of pocket*]

Staging periodontitis using estimated CAL in a IOPA radiograph

Formula

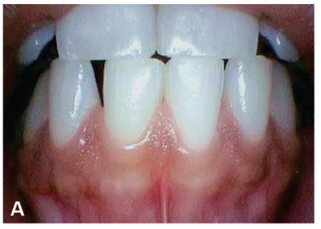
Stage I periodontitis:	CAL 1 - 2 mm
Stage II periodontitis:	CAL 3 - 4 mm
Stage III periodontitis:	CAL more than 5 mm
Stage IV periodontitis:	CAL more than 5 mm



Staging periodontitis using CAL measurements clinically with a probe

Fig. 10A.10: Measuring CAL clinically and radiographically to stage periodontitis.

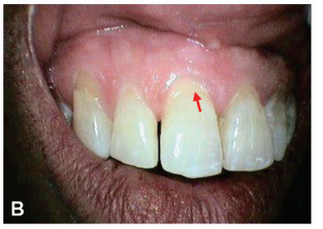
No CAL



Clinical gingival health over intact periodontium
[*no history of periodontitis ever; no CAL*]:

i. PD is less than 3 mm with BOP present at less than 10% sites.
ii. No gingival redness, gingival swelling etc.
iii. No CAL is present; bone crest located not more than 2–3 mm apical to CE junction.

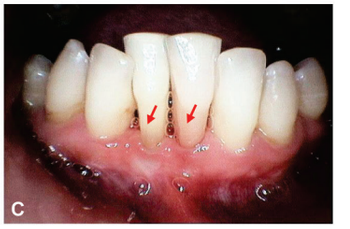
CAL due to protrusion of teeth [*not due to Periodontitis*]



Clinical gingival health over reduced periodontium in a non-periodontitis patient
[*no history of periodontitis ever; CAL present but not due to periodontitis but due to tooth's prominent root*]

i. PD is less than 3 mm with BOP present at less than 10% sites.
ii. No gingival redness, gingival swelling, etc.
iii. CAL is present; bone crest located more than 3 mm apical to CE junction.

CAL due to Periodontitis



Clinical gingival health over reduced periodontium in a stable [*previously treated*] periodontitis patient
[*history of successful treatment of periodontitis; CAL due to earlier periodontitis; now gingiva is clinically healthy*]

i. PD less than 3 mm and BOP present at less than 10% sites. If there is no BOP, sites with up to 4 mm PD are normal.
ii. No or mild BOP maybe present at less than 10% sites
iii. No gingival redness, gingival swelling, etc.
iv. CAL is present; bone crest located more than 3 mm apical to CE junction.

Fig. 10A.11: Diagnostic significance of CAL. Presence of CAL does not necessarily and automatically mean periodontitis. CAL is absent in intact periodontium (*diag. A*). CAL may be present in healthy periodontium if periodontium is reduced (*gingival crest and bone crest are placed apical to CE junction*) due to causes other than periodontitis, e.g. prominent position of tooth root, crown lengthening done earlier, etc. (*diag. B, red arrow*). CAL will remain present even in a previously successfully treated and presently healthy case of periodontitis (*diag. C, red arrow*).

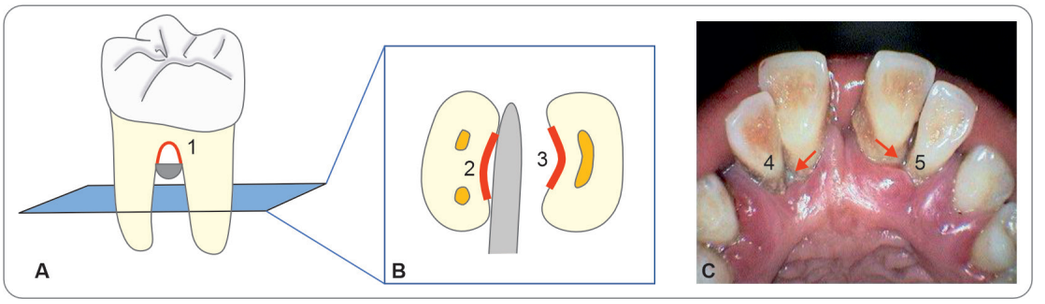


Fig. 13.21: Furcation regions that are narrower than the blade of the scaling instrument (*diag. A, grey semicircle and red lined area 1*), root concavities (*diag. B, red lined areas 2 and 3*) and surfaces of closely placed roots of teeth (*diag. C, areas 4 and 5*) may not be reachable by scalers, curettes or ultrasonic scaler tips. As a result, SRP and oral hygiene maintenance by the patient in such areas is difficult or even impossible. This predisposes such areas to continued periodontitis and worsens prognosis of such teeth.

4. *Teeth with Caries, Root Resorption and Nonvital Teeth*

- Teeth mutilated by extensive caries and/or pulpal involvement and those with extensive resorption of their roots (*due to any cause*, e.g. *high orthodontic forces*, *pathologic processes*, *idiopathic external resorption*, etc.) have a questionable prognosis that is dependent on severity of damage and the probability adequate treatment (Fig. 13.22).
- A ***nonvital tooth has similar prognosis as that of a vital tooth*** if it has been satisfactorily endodontically treated.
- Periodontal healing is similar in both vital and successfully endodontically treated teeth.

◀ *Is the periodontal prognosis of nonvital tooth similar to that of a vital tooth?*

◀ *Is the periodontal healing similar in non-vital and vital teeth?*

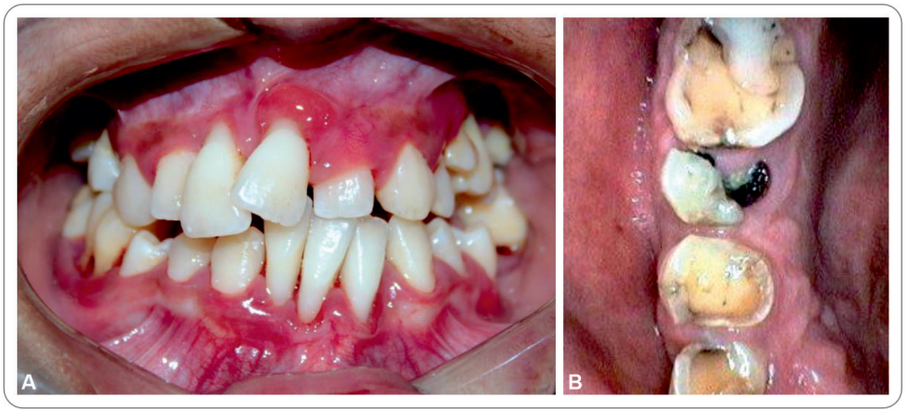


Fig. 13.22: Mucogingival problems like inadequate attached gingiva, high frenal attachments, severe periodontitis, systemic contributing factors that cannot be treated adequately (*diag. A*), teeth mutilated by caries (*diag. B*), etc. contribute to increased severity of periodontitis and worsen prognosis of affected teeth.

5. *Mucogingival Problems in Relation to a Tooth*

- Inadequate width of attached gingiva, location of base of pocket apical to muco-gingival junction, etc. may jeopardise periodontal health of teeth and make treatment complicated and difficult.
- Prognosis of such teeth is dependent on severity of mucogingival problems and degree to which these are corrected.

6. *Location and Restorative Importance of a Tooth*

- As premolars, 1st and 2nd molars and other teeth that are to serve as abutments (*teeth adjacent to edentulous areas in the arch*) have to bear additional (*greater*) occlusal forces, ***more rigid*** standards are employed for determining prognosis of such teeth.

◀ *Why are more rigid standards employed for determination of prognosis of abutment teeth?*

2. **Extra-oral:** This is established by keeping fingers of the working hand on side of the patient's face. Extra oral rest can be:
   a. **Palm down**: Usually established on the left side of the patient's face (*by right-handed operator*) (Fig. 14.22, diag. D, red arrow).
   b. **Palm up:** Usually established on the right side of the patient's face (*by right-handed operator*) (Fig. 14.22, diag. C, green arrow).
3. **Reinforced:** Tip of the first finger or thumb of the non-operating hand is used to provide extra stability to the shank or handle of the instrument being used.

◀ *What are the usual places where palm up and palm down rests are established?*

◀ *What is reinforced finger rest?*

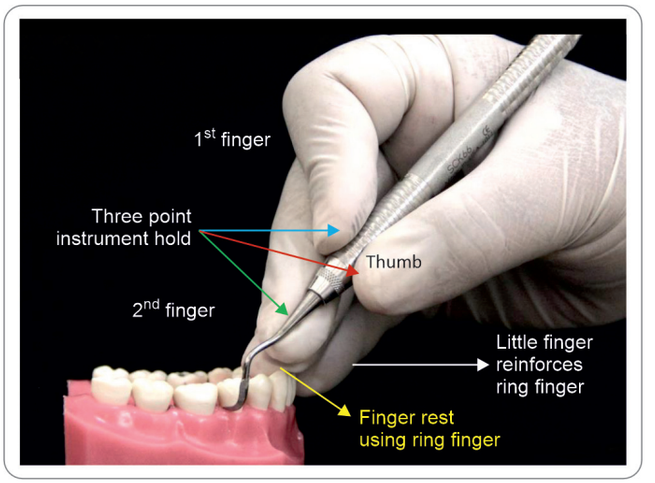


Fig. 14.21: Establishment of fulcrum/finger rest. The modified pen grasp allows good instrument grasp and stability during use. Finger rests allow establishment of fulcrum over which instrument movement can be done.

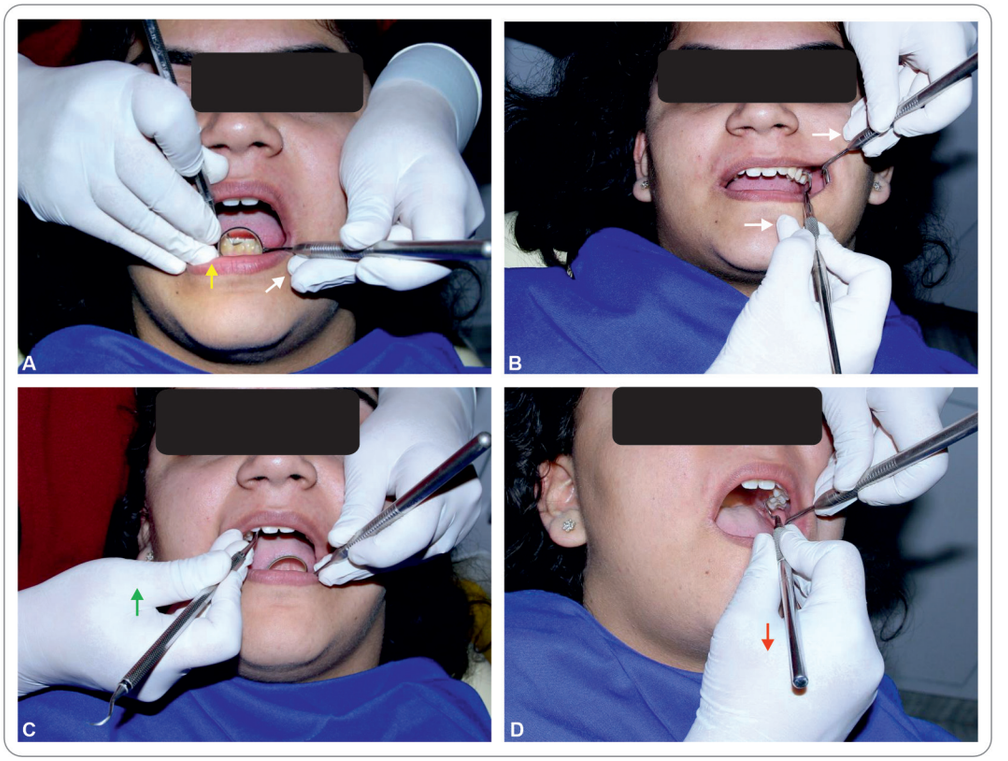


Fig. 14.22: Periodontal instrumentation using intra-oral finger rest (*diag. A, yellow arrow*), extra-oral finger rest (*diag. A and B, white arrows*), palm-up position (*diag. C, green arrow*) and palm-down position (*diag. D, red arrow*). Also note that clinician is holding instrument in modified pen grasp.

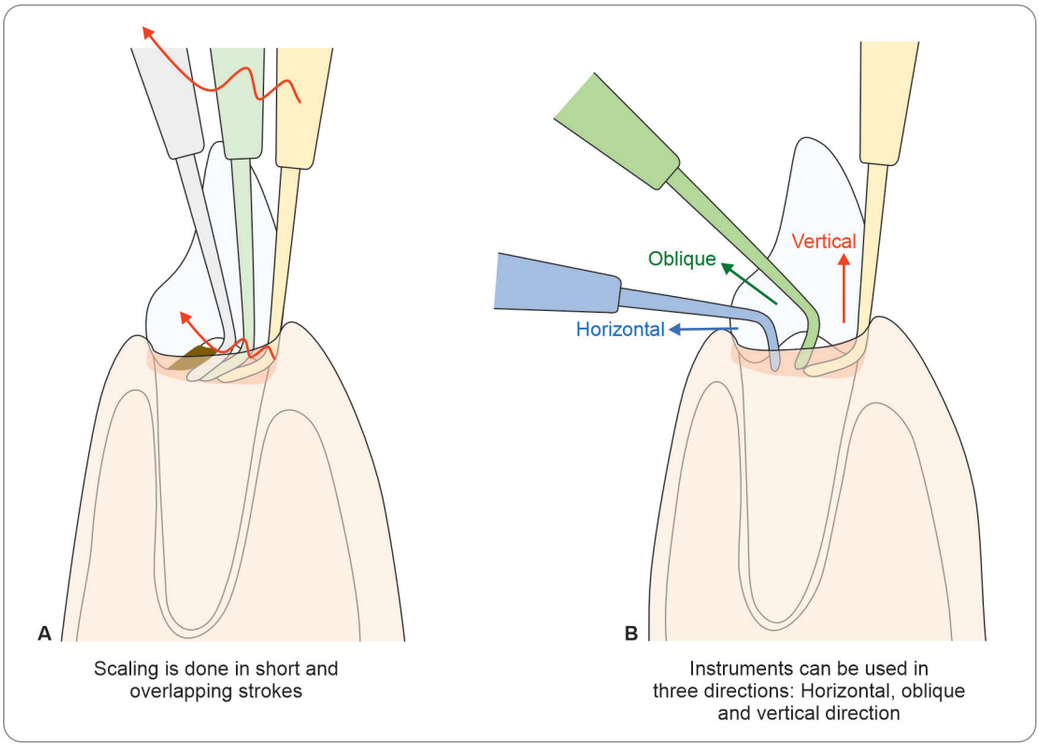


Fig. 14.25: Scaling and root planing must be done in a series of short and overlapping strokes with instruments used in horizontal, oblique or vertical direction during periodontal instrumentation.

patient's right side (Fig. 14.16). Clinician can make minor modifications in work positions according to their convenience and comfort.

1. *Buccal Aspect of Right Maxillary Posterior Region (First Sextant)*

a. **Patient's position:** Back of dental unit reclined at 30°. Patient's head facing straight or slightly to the left.
b. **Clinician's position:** 8 O' clock–9 O' clock position.
c. **Illumination:** Direct for all buccal and mesial surfaces of teeth. Indirect for distal surfaces of teeth (*easier if using a double surface mirror*).
d. **Visibility:** Direct for mesial and buccal surfaces. Indirect for distal surfaces of teeth (*easier if using a double surface mirror*).
e. **Retraction of cheek:** With mirror ***or*** first finger of non-working hand for pre-molars and mesial surface of first molar (*under direct illumination and visibility*).
f. **Hand position and Finger rest:** Palm down hand position, intra oral finger rest on right mandibular incisors-canine area.
g. **Instrument reinforcement:** If required handle and shank of instrument can be reinforced with thumb of non-working hand.

2. *Palatal Aspect of Right Maxillary Posterior Region (First Sextant)*

a. **Patient's position:** Back of dental unit reclined at 30°. Patient's head turned to the right and in chin-up position.
b. **Clinician's position:** 8 O' clock–9 O' clock position.
c. **Illumination:** Direct for all buccal and mesial surfaces of teeth. Indirect for distal surfaces of teeth.
d. **Visibility:** Direct for mesial and buccal surfaces. Indirect for distal surfaces of teeth.
e. **Retraction of tongue:** With mirror.

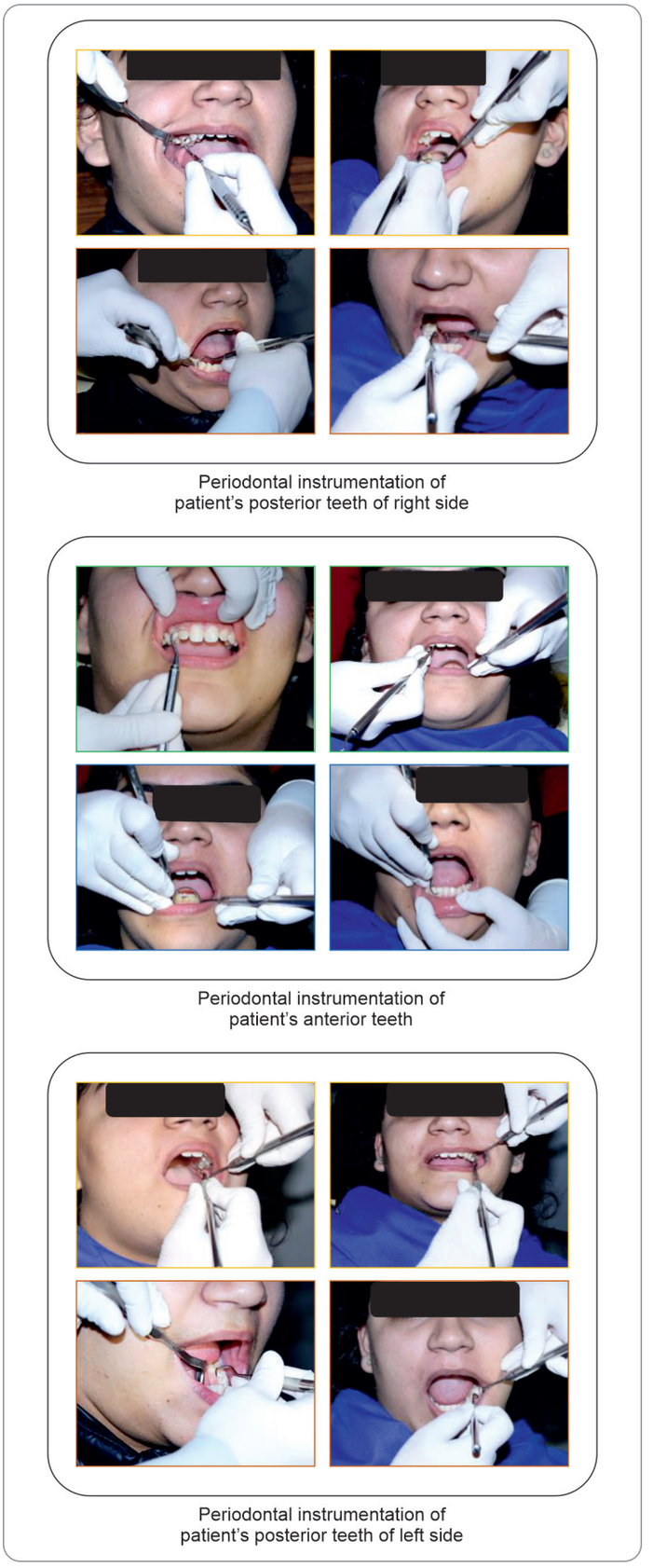


Fig. 14.26: Operator hand and instrument positions for periodontal instrumentation of different areas of the mouth

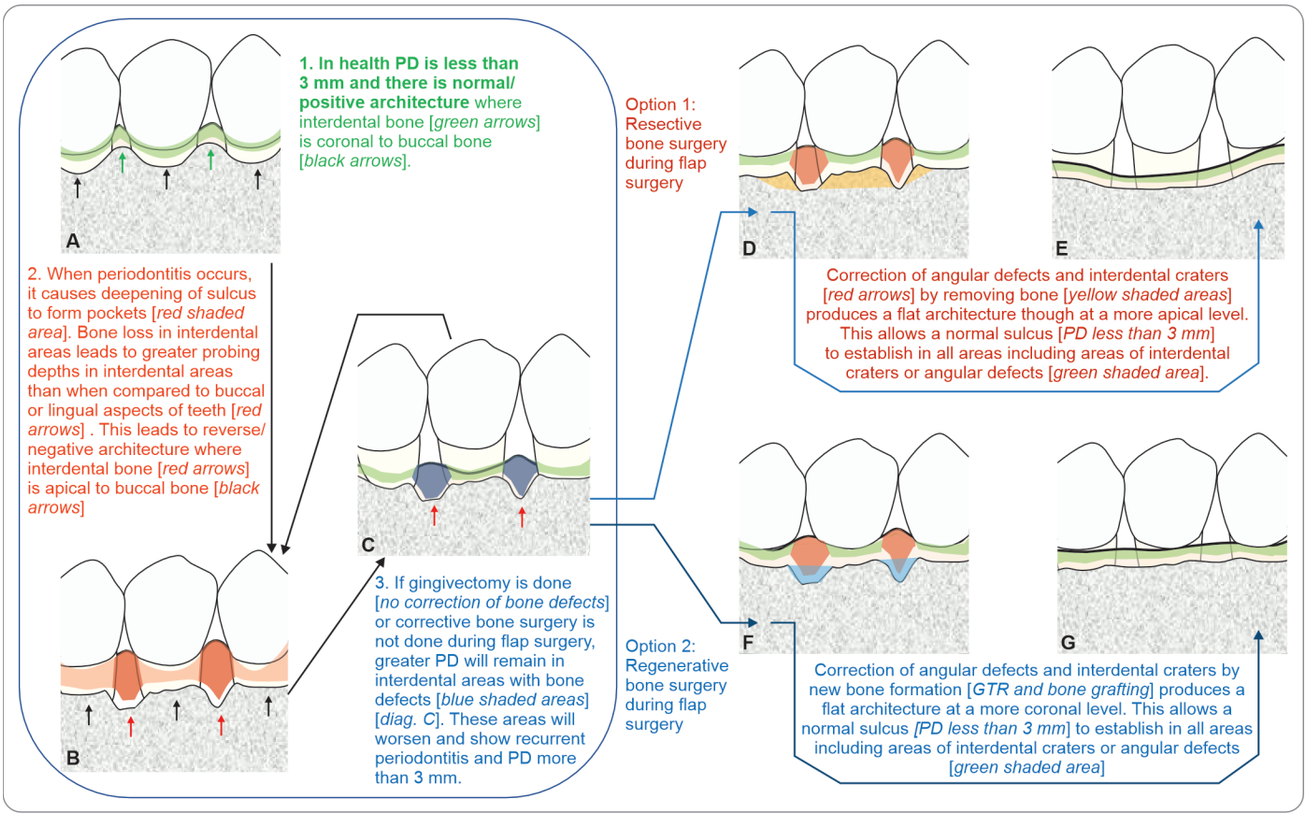


Fig. 18.4: Why and how interdental bone defects must be corrected

ALERT!

Why more bone loss is seen in interdental areas?

More bone loss is seen in interdental bone as compared to buccal and lingual or palatal bone because:

1. Interdental areas are non-self-cleansing areas, i.e. are not cleaned properly by natural physical cleaning mechanisms like scrubbing action of tongue and food during mastication, etc. This leads to initiation and worsening of periodontitis in the interdental areas. On the other hand, buccal and lingual or palatal areas are cleaned very well by natural cleaning mechanisms.

Why does interdental bone resorb faster? ▶

2. Interdental bone septum is composed primarily of cancellous bone overlaid by a thin coat of cortical bone (*crestal lamina*). The crestal lamina (*superficial cortical bone layer*) being more calcified resorbs slowly in periodontitis, but after it gets totally resorbed, the underlying cancellous layer being less calcified (*hence softer*), resorbs at a much faster rate. On the other hand, the buccal and lingual/palatal bone plates are made mostly of cortical bone (*which is harder than the cancellous bone*). They therefore resorb at a slower rate than the interdental septum.

ALERT!

- Periodontitis with ***less than 5 mm*** PD (*probing/pocket depth*) (*critical probing depth; see Chapter 17, 'Surgical Treatment of Gingivitis and Periodontitis', pages 292 and 293 and Fig. 17.2*) and minor bone loss is usually treated with ***SRP*** (*scaling and root planing*) alone. However, if PD does not reduce to less than 3 mm after SRP, pocket reduction must be carried out surgically (*flap surgery or gingivectomy or gingivoplasty*).
- Periodontitis presenting with ***more than 5 mm*** PD and moderate/severe horizontal and/or vertical bone loss or other bone defects before initial treatment (*phase I therapy—SRP, etc.*) has to be treated by flap surgery (*includes resective or regenerative bone surgery*) or by gingivectomy (*if no bone surgery is required*) to bring PD to less than 3 mm.

  If more than 3 mm PD remains few weeks after flap surgery, further pocket reduction must be done to bring PD to less than 3 mm (*by gingivectomy or gingivoplasty*).
- In general, one and two walled intrabony defects and horizontal bone loss ***do not*** show increase in bone height after bone grafting (*regenerative bone surgery*).

CLINICAL UTILITY AND SHORT ANSWER

Which type of bone defect is most and least likely to regenerate bone after bone grafting?

- Bone grafting regenerates bone only if the bone defect (*bone loss*) is ***three-walled***, i.e. it is pit like and is surrounded by bone walls on all sides (Fig. 18.9, diag. A).
- One-walled bone loss defects usually ***do not*** show bone regeneration (Fig. 18.9, diag. C).
- Horizontal height of bone (*around teeth*) usually ***cannot*** be increased.

RESECTIVE OSSEOUS SURGERY (*OSTEOPLASTY OR OSTECTOMY*)

What is the difference between osteoplasty and ostectomy?

Resective bone surgery (*removing bone to re-shape it and correct its contours*) can be done by osteoplasty or ostectomy. If re-shaping of alveolar bone is done by removing bone that does not support the tooth directly, it is called ***osteoplasty***. On the other hand, if ***tooth supporting*** bone is removed, it is called ***ostectomy***.

Indications of Osteoplasty or Ostectomy (*Resective Osseous Surgery*)

When must resective osseous surgery be done?

1. For correcting negative architecture of alveolar bone and converting it to flat or positive architecture.
2. For correction of intra-bony defect around a tooth with sufficient periodontal support.
3. To thin very thick alveolar bone margins, ledges, bone exostoses and correct inter-dental craters, widow's peaks, etc.
4. To widen furcation entrance for providing easy access to instruments and oral cleaning devices.
5. To perform clinical crown lengthening for restorative or prosthetic reasons, etc.

Contraindications of Osteoplasty or Ostectomy (*Resective Osseous Surgery*)

When must resective osseous surgery not be done?

1. If anatomic structures, e.g. mental foramen, maxillary sinus, etc. are present within 5 mm of the bone defect.
2. In aesthetic regions if it will lead to root exposure and opening of interdental space.

ALERT!

Can resective osseous surgery be done in relation to single tooth?

Why is it important to keep bone cool when bone cutting is being done?

1. Resective osseous surgery is ***rarely*** indicated to be done only in relation to a single tooth rather it must be performed on a couple of adjoining teeth to produce physiologic contours of the bone.
2. To prevent overheating of bone, bone cutting if done with surgical burs must be done at ***slow speed*** and under ***constant irrigation with normal saline*** (*if bone temperature rises above 42°C during cutting, it will lead to bone cell death and local bone necrosis*).

Instruments commonly used in osseous surgery

Small tipped bone rongeurs, steel and coarse grit diamond bone cutting burs, Schluger and Sugarman bone files, ochsenbein bone chisels, etc.

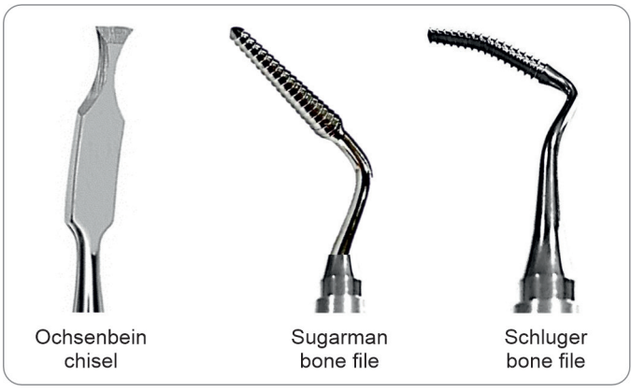


Fig. 18.4A: Commonly used hand instruments for bone recontouring

- Each of these tooth halves is then covered with a restorative crown of the shape of a premolar. This is called bicuspidization.

Root Resection

- Root resection is done on ***maxillary*** molars ***only*** if one of the two ***mesiobuccal*** or ***distobuccal*** root has severe bone loss due to periodontitis while the other two roots have good bone support (*palatal root cannot be resected as such a tooth will not be left with enough periodontal support*).
- Root resection is done only ***after*** successful endodontic treatment (*RCT*).
- The root with severe bone loss is cut (*amputated*) and extracted.
- The rest of the tooth with two remaining roots (*palatal and either mesiobuccal or distobuccal*) is retained as a maxillary molar after covering it with a restorative crown with a smaller occlusal table.

◀ *In which teeth is root resection possible?*

◀ *Which treatment must be done before root resection?*

◀ *Which modification is done in restorative crown of a tooth with resected root?*

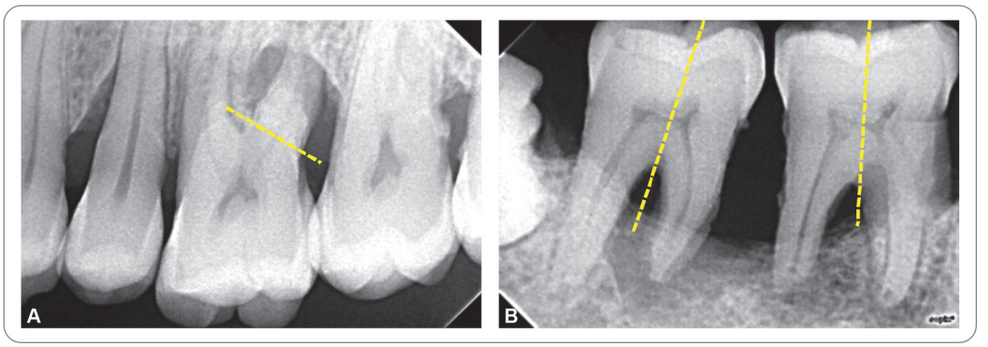


Fig. 20.6: Planned root resection (*diag. A*) and hemisection (*diag. B*). Root resection and hemisection are to be done after endodontic treatment (*RCT*).

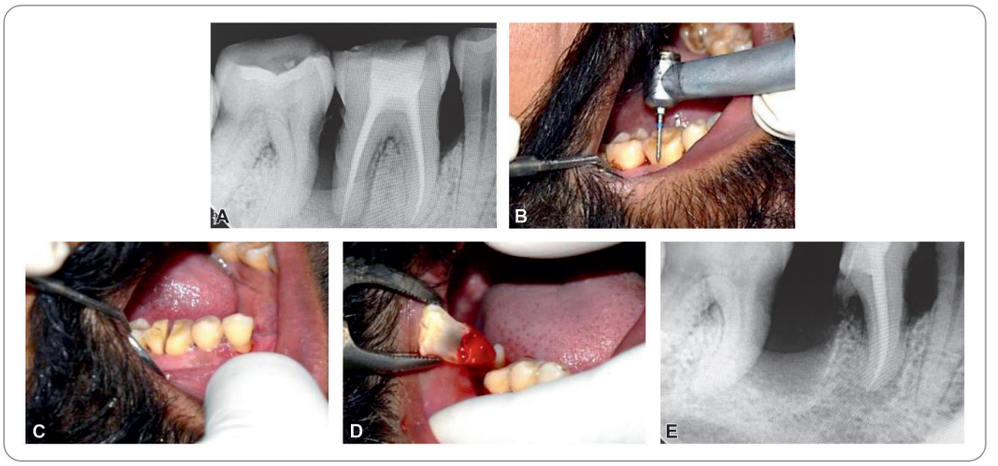


Fig. 20.7: Clinical steps for performing hemisection. Diag. A: Preoperative radiograph after endodontic treatment; Diags B and C: Tooth is cut into two mesial and distal pieces; Diag. D: Distal fragment is extracted and Diag. E: Postoperative radiograph.

CLINICAL UTILITY AND SHORT ANSWER

How does a diseased pulp contribute to furcation involvement?

Infection from the pulp chamber can reach the furcation and result in bone loss in the following ways:

- Pulpal infection can extend into the furcation area through accessory canals (*from the pulp*) opening into the furcation area (*endo–perio complication*).
- Perforation of floor of pulp chamber during endodontic treatment or insertion of retentive posts, etc. can result in furcation involvement.
- Tooth fractures lines passing through the furcations of multirooted teeth can lead to persistent furcation involvement.

◀ *How can infection from pulp chamber and root canals reach furcation area and cause bone loss?*

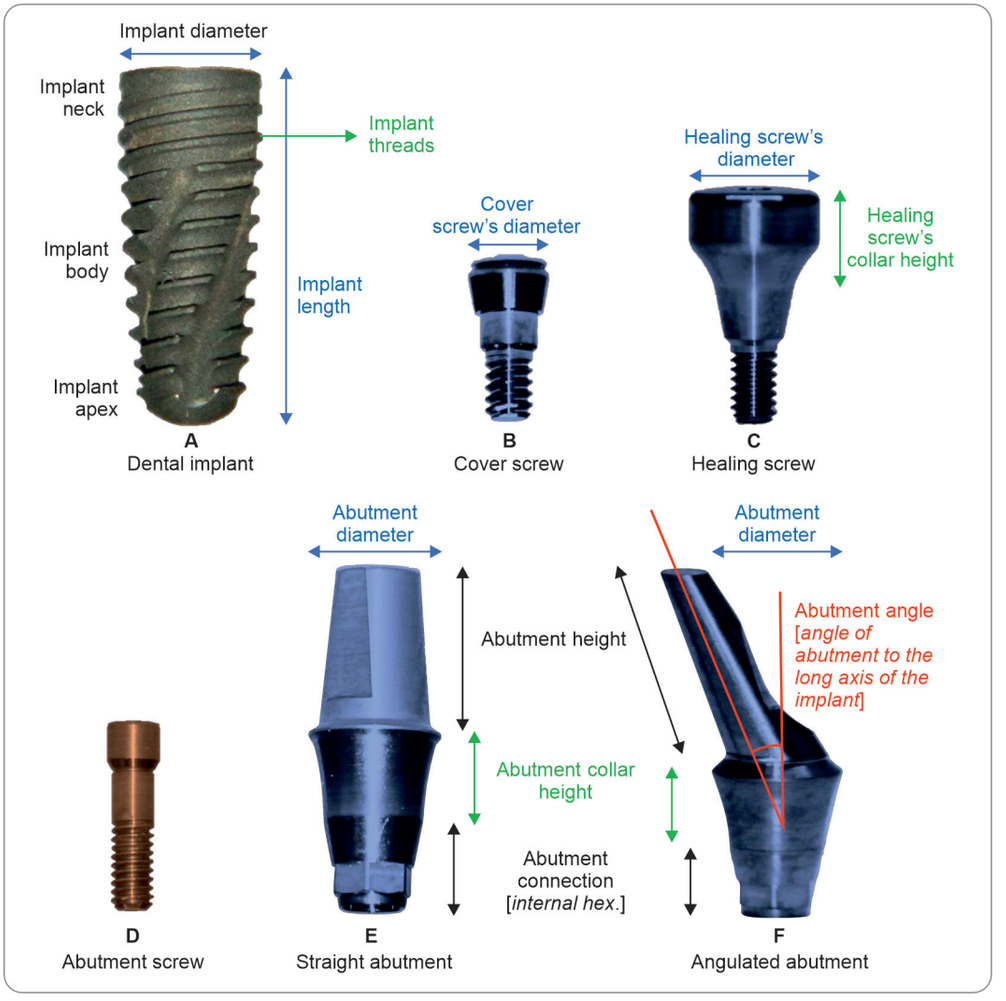


Fig. 22.1: Dental implants and other commonly used implant components

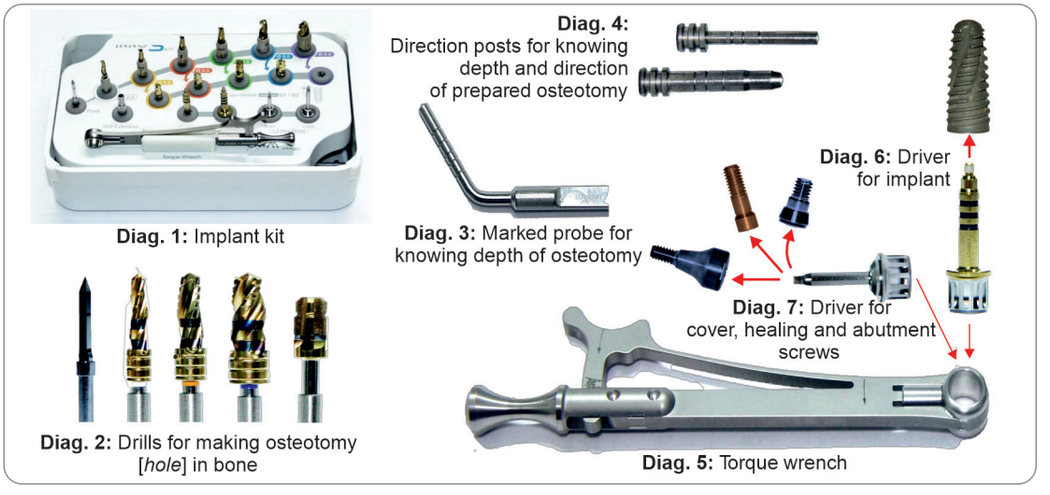


Fig. 22.2: Implant kit and its components. Implant kit (*diag. 1*); Osteotomy drills (*diag. 2*). Marked probe for ascertaining depth of prepared osteotomy (*diag. 3*). Direction posts for knowing direction, parallelism and distance between two or more osteotomies (*diag. 4*). Torque wrench for providing torque for turning implants into and out of the osteotomy (*clockwise and anticlockwise*) and tightening or loosening screws (*diag. 5*). Driver for turning implants into and out of the osteotomy (*diag. 6*). Driver for tightening or loosening screws (*diag. 7*).

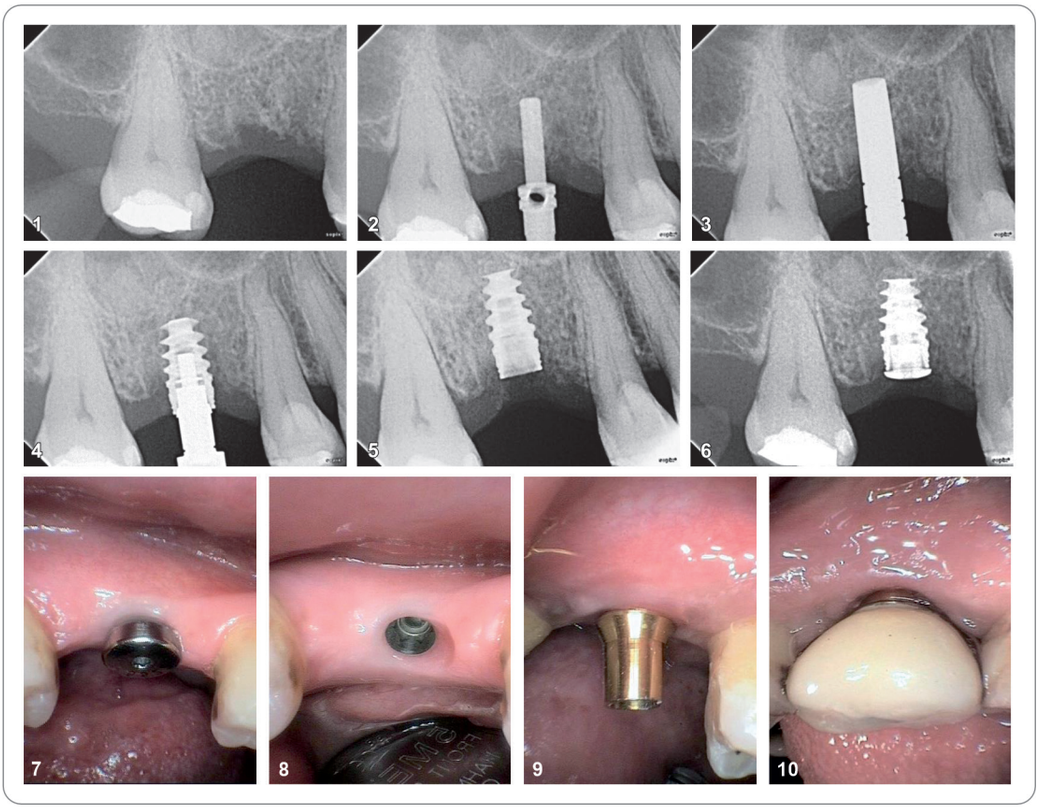


Fig. 22.8: Clinical steps in placing an implant and indirect sinus lift in maxillary posterior area. Diag. 1: Pre-operative radiograph. Diag. 2: After initiating the osteotomy (*1.8–2 mm drill*) and reaching a depth of approximately 5 mm, direction indicating post is inserted into the osteotomy to know the direction of the osteotomy. Osteotomy is prepared to the desired length and diameter using drills of larger diameter in sequence (*base of the maxillary sinus floor in this case*). Diag. 3: Floor of the maxillary sinus at the end of the osteotomy is then fractured and lifted up 2–3 mm by using osteotomes (*indirect sinus lift instruments*). Diag. 4: Implant is then mounted on an implant driver and put into the osteotomy. It is turned clockwise to insert it into the osteotomy with an implant mount (*using physiodispenser and torque wrench*). Diag. 5: Implant is inserted into the osteotomy till the crest of the alveolar bone or just 0.5 mm apical to it. Diag. 6: Cover screw is fixed over the implant to prevent food, plaque and soft tissue growing into it. Diag. 7: Cover screw is replaced with a healing screw. Diag. 8: After allowing gingival healing for about 15 days, gingival collar forms around the healing screw (*abutment*). Diag. 9: Selected abutment is fixed on the implant. Diag. 10: Crown is fabricated and placed on the implant.

Step 4: Re-surfacing the Implant after Healing Period

What does the healing screw (abutment) do? ▶

At the end of the healing phase (*step 4 above*) or any time later, implant is re-surfaced (*exposed*) by making a window in the overlying gingiva. Cover screw placed earlier is replaced with a healing screw (*abutment*) (Fig. 22.8, diag. 7) for 10–15 days to allow shaping and healing of the gingiva around the neck of the implant (Fig. 22.8, diag. 8).

Step 5: Impression Making, Preparation of Plaster Cast and Crown Fabrication

1. **Indirect impression technique**
   - i. After 10–15 days, the healing screw (*abutment*) is removed and an impression coping is screwed into the implant body for making an impression with rubber base impression materials.
   - ii. After making the impression, the impression coping is removed and healing screw (*abutment*) is put back on the implant body.
   - iii. Implant analog (*substitutes for implant body in the plaster cast*) is attached to the impression coping.