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Clinical Diagnosis – The Promising Traditional Platform.

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Abstract: *Periodontal diagnosis is an important label that clinicians place on patients periodontal condition or disease. A well structured and detailed history with a comprehensive and complete examination helps establish an accurate diagnosis. The prognosis and treatment plan for the patient relies almost entirely on proper clinical diagnosis. Despite extensive research to develop novel techniques for improved diagnostic quality the traditional methods clinical diagnosis still remains the mainstay of diagnosis. A good understanding of patient history and findings help provide a customized treatment plan, attending to all specific needs of the individual patient. This article provides details ; on the history of the patient with respect to medical , dental, personal, family aspect as well as thorough clinical examination assessing different components of the periodontium*
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I. Introduction

An integral aspect of patient evaluation and management is the development of a clinical diagnosis. Proper diagnosis is essential to intelligent treatment.[1] A good clinician starts evaluating the patient the moment the patient steps into the clinic An accurate diagnosis can be inferred only by processing the data that has been systematically collected from the patient. Prior to conducting a hand on examination clinician should evaluate in detail the medical and dental histories of the patient.. A valuable aspect of history taking is that helps develop a doctor patient relationship. Clinical examination of the patient should involve evaluation of the general, extra oral and oral components. Periodontal examination is the last component of the oral examination. Following examination analysis of the additional parameters through laboratory and radiographic investigations can be of added value in the final diagnosis.[2] Treatment plan of the patient depends entirely on the diagnosis .Hence the central role of clinical diagnosis is further reinforced.

A well structured and detailed history with a comprehensive and complete examination helps establish an accurate diagnosis. Periodontal diagnosis should first determine whether disease is present or not; If present identify the disease's type, extent, distribution, and severity, and it should finally provide an understanding of the underlying pathologic processes and their causes. The periodontal diagnosis is determined after the careful analysis of the case history and the evaluation of the clinical signs and symptoms as well as the results of various tests. The interest should be in the patient who has the disease and not simply in the disease itself. Diagnosis must therefore include a general evaluation of the patient and consideration of the oral cavity.[1]This article aims to review the detailed periodontal case taking of the patient seeking periodontal care to help in clinical decision making. The following is a sequence for the detailed evaluation of the patient

II. History

Name - for record and communication. Addressing a patient by his/ her name helps build a rapport with the patient and alleviate apprehension**.**

Age-Recorded in years .Certain diseases occur with greater frequency in different age groups.

Elderly	Systemic diseases, Periodontal disease, Root caries, Xerostomia, Candidiasis
Children,adolescents,young adults	Puberty gingivitis, ANUG, Herpetic gingivostomatitis, Aggressive periodontitis

Sex:

Hormonal disturbances during puberty, menstrual period & pregnancy modifies tissue response to local irritation in females.

Females - Autoimmune disorders, Pyogenic Granuloma
Males- Smokers keratosis,

Occupation :[3] Both the present and past occupations should be noted.

Diseases due to physical agents	Miners nystagmus,heat exhaustion ,occupational deafness
Chemical agents	Lead poisoning,silicosis,asbestosis
Biologic agents	Actinomycosis, HIV ,hepatitis
Ergonomic hazards	Musculoskeletal disorders

Residence It gives information about the possible endemic conditions affecting patients. It also gives information about the convenience of patient for recall visits and for future contact with the patient .

III. Medical History

Aids in: diagnosis of oral manifestations of systemic disease, detection of systemic conditions that may be affecting the periodontal tissue, history of previous hospitalisations and operations if any .

Patients on :

Anticoagulants	Monitor INR <3- Infiltration anesthesia, scaling, and root planing <2-2.5- Block anesthesia, minor periodontal surgery, and simple extractions <1.5-2 - Complex surgery or multiple extractions
Hormone supplements	Monitor hormone levels: Thyroid :ensure euthyroid status. Diabetes: normal range of blood sugar levels
Corticosteroids	Non surgical dental procedures no supplemental steroid required. Minor periodontal,oral surgery-5-6 mg prednisone 1-2 hrs before procedure Major surgery- 100mg hydrocortisone i.m 1 hr before surgery. 25mg hydrocortisone for 24 hrs then return to normaldose
Bisphosphonates	invasive treatment, such as extractions, periodontal surgery, implant surgery, and bone augmentation procedures, should be avoided.

Medical conditions and precautions[4]

Liver diseases	Avoid Analgesics:Aspirin,NSAIDs, Opioids,paracetamol Antimicrobials: metronidazole, doxycycline Sedatives diazepam,midazolam	Alternative Codeine COX2 inhibitors Penicillin,tetracycline,cephalosporin Lorazepam
Kidney diseases	Avoids NSAIDs ,Aspirin Avoid tetracyclines	
Hyperthyroid Hypothyroid	Epinephrine and other vasopressor amines should be given with Caution .Antithyroid drugs-agranulocytosis,leading to oral and pharyngeal ulcerations Careful administration of sedatives and narcotics because of the potential for excessive sedation.	

Drug interactions

Doxycyline	Antacids
Minocycline	Pencillins,warfarin
Pencillin	Probenecid,tetracycline
Metronidazole	Alcohol,warfarin,phenytoin
NSAIDS ,Aspirin	Warfarin,ACE inhibitors

Most drug to drug interactions occurwhen drugs are administered concurrently.to avoid drug interactions theyare spaced so that concurrent administration does not occur.if in doubt the patients physician should be contacted.

Oral manifestations of drugs:[3]

Candidiasis	Inhalational steroids
Gingival enlargement	Phenytoin,nifedepine
Gingival hemorrhage	Coumarin,clopidogrel
Drug induced vesicullobullous lesions	Lichenoid : ACEinhibitors,NSAID's Pemphigoid like: amoxicillin,clonidine

IV. Dental History

History of previous dental visits, type of treatment undertaken , duration of treatment, complications during treatment if any. Any orthodontic treatment including duration and approximate date of termination. Previous periodontal problems: Nature & condition of the problem. If treated, type of treatment and approximate period of termination of treatment .In the patient's opinion is the present problem is a recurrence of previous disease

V. Chief complaint (current illness) :

This is to record what made the patient to seek the dentist. It is recorded in patients own words as much as possible, and no documentary or technical language should be used. Recorded in chronological order of their appearance and in order of severity. It aids in diagnosis and treatment planning and should be given the first priority as patient expectations are met only when the chief complaint is attended. Record the nature,site and duration of the complaint as explained by the patient.

VI. History Of Presenting Illness: [5]

Patients response to questions elicited by the examiner for additional information related to chief complaint. The history commences from the beginning of the first symptoms and extends to the time of examination.

In general it can be elaborated under: Mode of onset, Cause of onset , Duration, Progress and referred pain, Relapse and remission, Treatment and Negative history.

Pain : Site, onset ,character, radiation, association, time course, excacerbating /relieving factors , severity
Bleeding gums : spontaneous, or on provocation, with periodicity, associated with menstrual periods, factors that reduce bleeding.
Mobility :location, origin ,onset, duration increased/ progressive, aggrevating factors, relieving factors, associated signs
Migration: Onset (when was it first noted), Duration , Progression, associated with pain / interference with occlusion /function

VII. Family History

Asked to assess the presence of any inherited disease pattern or trait. Common diseases that run in families include diabetes, hypertension, asthma arthritis, aggressive periodontitis.

VIII. Personal History

8.1 Diet : To assess the the presence of a balanced diet,.balanced diet pays a crucial role in maintenanceof goodhealth. Malnutrition consistently impairs innate and adaptive defences of the host. .Highly cariogenic diet should be avoided. Diet rich in natural antioxidants Vitamin C,E and flavanoids favour periodontal health.[6,7]

8.2 Oral habits : Mouth breathing, Thumb sucking , Bruxism , Nail biting, Tongue thrusting

8.3 Tobacco abuse : record the smoking status of a patient(current smoker, former smoker, non smoker). If a smoker record the type, frequency, duration. Current smokers were almost three times more likely to have severe periodontitis than non-smokers.Former smoker were 1.7 times more likely to have periodontal disease than those who never smoked.[1]

8.4 Alcohol abuse: record the amount, frequency and duration.

8.5 Oral Hygiene Practices: Toothbrushing frequency., method of brushing, type of toothbrush type of dentifrice , interval at which brushes are replaced , other aids

8.6 Menstrual and obstetric history: History should obtain a detail on Premenstrual tension, Presence or absence of pain with periods. Whether the patient taking oral contraceptives?.

If a patient is pregnant care should be taken not to expose the patient to any ionising radiations. Surgical treatments should be avoided during pregnancy .[1]

IX. General examinations :

The general examination begins as soon as the patient enters the dental office. The patient's general appearance may give information that relates to his or her medical condition. The clinician will observe the patient's gait, mobility, facial asymmetries, lesions or scars.

9.1 Vital Signs : [8]

Pulse, Respiratory rate, Temperature. , Blood Pressure

Respiratory rate	14-16 breaths/min	Tachypnoea >20 breaths/min
Normal temperature	Orally Woman (33.2-38.1^{0}C), Men (35.7-37.7 ^{0}C)	
Pulse rate	60-100 beats/min(normal), <60 beats/min(bradycardia) >100 beats/min(tachycardia)	
Pulse rythmn	regular(normal) , regularly irregular,irregularly irregular	
Blood pressure	low (90/60),	

	Normal (120/80), Pre hypertension(120-139/80-89) , Stage 1 Hypertension (140-159/90-99) , Stage 2 Hypertension (≥160/ ≥100) .

9.2 Peripheral signs:[8]

Clubbing: **Associated with** **Grades**	 Diseases of the lung, heart, GIT, endocrine system and miscellaneous diseases. Grade 1 – Fluctuation of the nail bed Grade 2 – Obliteration of the nail bed angle Grade 3 – Parrot beak appearance or drum stick appearance Grade 4 – Hypertrophic OsteoArthropathy (HOA).
Cyanosis	Bluish discolouration of the skin and mucous membrane due to increased amount of reduced haemoglobin. Central, peripheral and differential types
Icterus	Yellowish tinge to the skin and sclera. Seen in Acute liver failure, Alcoholic hepatitis, Amyloidosis, Autoimmune hepatitis ,infective hepatitis
Skin	One of the best indicators of general health looked for : appearance (any rashes, sores or itching may reveal a positive history Colour – Pallor , yellow sin (jaundice) Textural changes, Pigmentation (defect – vitiligo), oedema

X. Extra Oral Examination

10.1 Facial Symmetry:[5] To assess the fullness on both the halves of the face and look for any gross disorder that may reveal a significant history. Denoted as Symmetrical or Asymmetrical.

Facial Asymmetry	Inflammatory Congenital, Developmental/acquired

10.2 TMJ :[5] Examined for symmetry ,inter incisal opening (normal 35-50 mm) ,any deviation in opening , range of vertical movement's ,range of lateral movement's, clicking or crepitus sound.

10.3 Lip :[5]

Competance,	Natural seal of the upper and lower lips at rest. Lips may be competent, potentially competant or non competent
Common lesions	Herpetic lesions, actinic chelitis
Localised swellings of lip:	Mucocele
Generalised swelling	allergic manifestations, chelitis graulomatosa,chelitis glandularis
Commissure of lips :	Look for angular chelitis

10.4 Lymph Node Palpation:[9] Lymph nodes are palpated for their size,consisitency,mobility and distribution.

Size Lymph nodes <1 cm Lymph nodes greater than 2 cm,>4 weeks	 Not clinically significant Significant
Consistency: Normal lymph nodes Malignant Inflammatory	 Smooth relatively soft or slightly enlarged Enlarged,irregular,rubbery,hard, matted ,fixed Tender
Mobility: Fixed or matted lymph nodes Freely movable Distribution : Localised Regional Generalised	Metastatic carcinoma Infections, collagen vascular disease, lymphoma Enlarged lymph noes in one region Enlarged lymph nodes in 2 or more contiguous regions Enlarged lymph nodes in 2 or more non contiguous regions

XI. Examination Of Soft Tissues[5]

11.1 Labial mucosa: The labial mucosa should appear wet and shiny. Look for lesions apthous ulcers, pigmentations, swelling (mucocele)

11.2 Buccal mucosa : Pale pink in colour, wet ,shiny. Rule out any pathological lesions like leukoplakia, erythroplakia,, lichen planus, vesiculobullous lesions, OSMF.
11.3 Hard palate, soft palate : Check for clefts, perforations, ulcerations,recent burns or hyper keratinization ,tori, fistulae, swellings hyperplasia.
11.4 Tongue : Volume of the tongue, Integrity of papilla, Cracks or fissures,Swellings or ulcers Mobility
11.5 Frenum : Types of frenal attachment :Mucosal, Gingival, papillary, papilla penetrating. Papillary or papillary penetrating frenum with positive tension test , displaces the gingival margin, leading to plaque accumulation and gingival inflammation.. Such a frenum is pathologic and should undergo frenectomy to to prevent gingival inflammation and its sequelae,
11.6 Floor of the mouth: Check for Colour ,Swellings, Patches, Ankyloglossia .

XII. Examination of hard tissues:

12.1 Type of dentition: Primary Or Permanent.
12.2 Teeth : [10]

Number	normal,/anodontia/hypodontia (third molars,second premolars,lateral incisors)/hyperdontia (supernumerary teeth, mesiodens, distomolar, paramolar)
Size	Macrodontia(isolated :incisors,canines, generalized : pituitary gigantism) Microdontia (maxillary lateral incisor,peg shaped laterals)
Shape	Gemination, Fusion ,Accessory cusps
Developmental alterations	Amelogenesis imperfecta, dentinogenesis imperfecta ,dentin dysplasia,regional odontodysplasia
Represented by	FDI, Palmer ,universal notation

12.3 DMF status ;[11] The decay-missing-filled (DMF) index or decayed, missing, and filled teeth (DMFT) index is one of the most common methods in oral epidemiology for assessing dental caries prevalence as well as dental treatment needs among populations. The DMF Index is applied to the permanent dentition. The def index is a variation that is applied to the primary dentition Maximum DMFT index, score 0 – 28 / 32 .

12.4 Inter arch relations :[12,13] Over jet 2-4 mm , Over bite 2-3 mm(open, normal,deep)

Inter arch Deviation	Types	Site	Impact on periodontium
Deep bite	Skeletal/dental	Anteriors	Impingement of the teeth on the gingiva and food impaction, followed by gingival inflammation, gingival enlargement, and pocket formation.
Open bite	Skeletal/dental	Anteriors mostly	reduced mechanical cleansing by the passage of food may lead to the accumulation of plaque, debris, calculus formation, and the extrusion of teeth
Cross bite	Unilateral/ bilateral	Anteriors/posteriors	TFO, Food impaction, spreading of the mandibular teeth, associated gingival and periodontal disturbances

Other findings			
Proximal contacts	Tight/open	Any	Open contacts allow for food impaction. Several investigators have found open contacts to be a modifying factor in periodontal disease

12.5 Functional occlusal relationships :[13] The examination of functional occlusal relationships is an important part of the diagnostic procedure. Dentitions that appear to be normal when the jaws are closed may present marked functional abnormalities . eg: deflective shift or slide of mandible due to supracontacts in retruded path of closure,.

12.6 Wasting Disease of the Teeth[14]
12.6.1 Erosion : sharply defined wedge-shaped depression in the cervical area of the facial tooth surface. The long axis of the eroded area is perpendicular to the vertical axis of the tooth generally affects a group of teeth .

Classification	Score
Eccles classification	Early, small, advanced
Xhonga and Valdmanis	None , minor <2mm, moderate 3 mm, severe >3mm
Lussi, , Khan et al	Erosion index

12.6.2 Abrasion : loss of tooth substance that is induced by mechanical wear other than that of mastication

Michaels classification Based on morphology	Shallow ,Concave, Wedge shaped, Notched, Irregular,

12.6.3 Attrition : occlusal wear that results from functional contacts with opposing teeth. The angle of the facet on the tooth surface is potentially significant to the periodontium Angular facets of attrition are injuriousto the periodontium.

Brocas classification	
0	No attrition, tooth form retained
1	Enamel worn without cusp obliteration or exposure of dentine
2	Cusp worn down and dentine exposed
3	Appreciable amount of crown of tooth is worn away
4	Most of the crown has disappeared and wear has extended to the neck of tooth).

12.6.4 Abfraction: Results from occlusal loading surfaces causing tooth flexure and mechanical microfractures and tooth substance loss in the cervical area .

Grippos classification	hair line crack , striations , saucer shaped , semi lunar shaped, cusp tip invagination

12.7. Dental stains :[15]The colour of tooth is determined by the translucency and thickness of the enamel ,thickness and colour of the underlying dentin and colour of the pulp.alteration in colour may be physiologic/pathologic and endogenous and exogenous in nature. Tooth disclouration may be extrinsic or intrinsic.

Tooth discolouration Causes and Colors(Abbott 1997)	
Extrinsic discolouration	
Cigarettes,pipes,cigars	Yellow brown to black
Coffee ,tea , foods	Dark brown to black
Poor oral hygiene	Yellow or brown stains
Extrinsic and intrinsic discolouration	
Flourosis	White , yellow,brown , grey,black
Aging	
Intrinsic disclouration	
Genetic conditions	Brown, black
1.Amelogenesis imperfecta	Brown blue
2.Dentinigenesis imperfect	
Systemic conditions	Blue green,brown,purple brown
1. jaundice	
2. porphyria	
Medications during tooth development	Brown, grey,black
1. Tetracycline	
2. Flouride	
Body by products	
1. Bilirubin	Blue,green,brown
2. Haemoglobin	Gray black
Pulp changes	
1.Pulp canalobliteration	Yellow
2.Pulp necrosis	
With hemorrhage	Gray,black
Without hemorrhage	Yellow ,gray black
Iatrogenic causes	
1.Trauma during pulp extirpation	Gray,black
2.Tissue remanants in pulp chamber	Brown,gray,black
3.Restorative dental materials	Brown ,Gray,black
4.Endodontic materials	Gray,black

12.8Hypersensitivity :[16] Root surfaces exposed by gingival recession may be hypersensitive to thermal changes or tactile stimulation. Patients often direct the clinician to the sensitive areas. Diagnosis is made by patient history, clinical examination.

Stimuli to elicit dentinal hypersensitivity	mechanical (tactile) stimuli, electrical stimuli, cold air blast, cold water stimuli, thermo electric device, electrical pulp testers, dental pulp stethoscope evaporative stimuli and air jet stimulator

Scales to assess dentin hypersensitivity	
Visual analog scale	0-10 mm scale.
Numeric pain rating scale	0-10 mm scale. Segmented version of VAS
Faces Pain Scale-Revised (FPS-R)	Used in children

12.9 Trauma From Occlusion:[1] Refers to injury produced by occlusal forces

Clinical findings	Wear facets, attrition, abfractions, V shaped ,gingival recession, tooth mobility
Radiographic findings:	Funnel shaped bone loss, widening of PDL space

12.10 Pathologic migration :[17] Refers to alteration in tooth position .They can lead to premature contacts which are deleterious to the periodontium.

Types	Extrusion, diastema formation, facial flaring ,moving into edentulous space
Etiology	Occlusal forces, soft tissue forces, reduced periodontal support, iatrogenic causes, habits ,extrusive forces
correction	Severe : extraction, replacement Early stages : spontaneous correction following periodontal therapy Limited /adjunctive orthodontic therapy Conventional orthodontic therapy

12.11 Sensitivity to percussion :[1] Sensitivity to percussion is a feature of acute inflammation of the periodontal ligament. Gentle percussion of a tooth at different angles to the long axis often helps with the localization of the site of inflammatory involvement.

XIII. Examination Of The Gingiva

13.1 Colour of the gingiva: [13]Change in colour is an important clinical sign of gingival disease.

Normal	Coral pink
Chronic inflammation	Red or bluish red
Acute gingivitis	Bright red
ANUG,herpetic ginigvostomatitis	Dull whitish gray
Metallic pigmentation	Bismuth,mercury,arsenic- black line Lead- bluish red or deep blue Silver – diffuse bluish gray

13.2 Contour:[13]

Normal	Scalloped, knife edged interdental papilla – pyramidal anteriors col shaped posteriors
Gingival enlargement	Ballooning of interdental papilla and / or marginal gingiva
Gingival recession	Exaggerated contour
Still mans cleft	Apostrophe shaped indentation
McCalls festoons	Life preserver shaped enlargement of marginal ginigva
ANUG	Punched out
Periodontitis	Loss of normal scallop, blunt or flat IDP

13.3 Consistency: both acute and chronic inflammation produce changes in the normal consistency of ginigva. Checked by palpation with digital pressure.

Normal	Firm and resilient attached dgingiva
Inflammation acute	Diffuse puffiness,softening,sloughing, vesicle formation
Chronic inflammation	Soggy puffiness or firm leathery consistency Exudative or fibrotic phase

13.4 Surface Texture : [13]Loss of surface stippling is an early sign of gingivitis.

Chronic inflammation	Smooth ,shiny/firm ,nodular
Senile atrophic gingivitis	Smooth surface texture.
Chronic desquamative ginigvits	Peeling of surface
Hyperkeratosis	Leathery texture
Non inflammatory ginigval hyperplasia	Minutely nodular surface

13.5 Size : It is the sum total of both cellular & intercellular elements. Alteration in gingival size is a ommon feature of gingival disease.

Classification of gingival enlargement

Based on etiology	Inflammatory, drug induced, modified by systemic diseases or conditions, benign,malignant, false enlargements
Location and distribution	Localised ,Generalised Marginal,Pappilary,Diffuse,Discrete

13.5.1 Indices used to measure gingival enlargement [18]

Angelopoulos and Goaz (1972) Vertical component	Grade	Hyperplasia		Tooh coverage
	0 1 2 3	No Minimal Moderate Severe		No Cervicalt hird Middle third More than two third
Seymour RA (1985) Both vertical and horizontal component	0, normal gingiva			
	1,slight, < 2mm increase, ginigva covers cervical 1/3rd of crown			
	2,moderate, 2-4 mm increase, ginigva extends to middle third of crown			
	3,severe,>4mm , covers more than 2/3rd of crown			
Miller and Damm (1992) (Modified Angelopoulos Goaz index)	Grade	Hyperplasia	Size	Tooh coverage
	0 1 2 3	No Minimal Moderate Severe	Normal <2 2-4 >4	No Cervicalt hird Middle third More than two third
Bokenkamp A and Bohnhorst B (1994) based on involvement of the crown	0	No signs of gingival enlargement		
	1	Enlargement confined to interdental papillae		
	2	Enlargement IDP and marginal ginigva		
	3	Enlargement involving three quarters or moreof crown		
Miranda and Brunet index (2001) Vertical component	0-papilla thickness< 1 mm;			
	1- papilla thickness 1–2 mm;			
	2- papilla thickness > 2 mm			

13.6 Position of the gingiva : Refers to the level at which the gingival margin is attached to the tooth. Normally 1mm above CEJ .

Etiology of gingival recession[19]

Anatomical	Dehiscence and fenestration of alveolar bone, tooth position,
Physiological	Orthodontic tooth movement
Pathological	Improper Tooth brusing, flossing

13.6.1 Various classifications of gingival recession[20,21,22]

Sullivan Atkins 1968	Shallow narrow, Shallow wide Deep narrow, Deep wide		
Mlinek 1973	Shallow narrow Deep wide		
Liu,Solt 1980	Visual recession, hidden recession		
Millers classification 1985	Grade I- Marginal tissue recession which does not extend to the mucogingival junction (MGJ). no alveolar bone loss or soft tissue loss in the inter-dental area.Complete root coverage obtainable		
	Grade II- Marginal tissue recession which extends to or beyond the MGJ . No alveolar bone loss or soft tissue loss in the interdental area .Complete root coverage obtainable		
	Grade III- Marginal tissue recession which extends tor beyond the MGJ. Bone or soft tissue loss in the interdental area is present.Partial root coverage related to level of papilla height		
	Grade III- Marginal tissue recession which extends to or beyond the MGJ.The bone or soft tissue loss in the interdental area is present with gross flattening .No root coverage		
Smith 1997	Horizontal component	score	exposure of CEJ

		0 1 2 3 4 5	No evidence 10% 10-25% 25-50% 50-75% 75-100%
	Vertical component	Score	Vertical measure of recession
		0	No evidence
		1	Dentin hypersensitivity,>1 mm recession
		2-8	2-8mm of recession
		9	>8 mm recession
Francesco cairo et al 2011	RT1 :buccal and interproximal CAL same RT2:interproximal CAL ≤ Buccal CAL RT3:Interproximal CAL ≥ Buccal CAL		
Indications for root coverage	Esthetic reasons, hypersensitivity ,root abrasions, disharmony of gingival contour.		

13.7. Gingival Biotype : [23]

Biotype Thick biotype	Thin biotype
>1.5mm thickness	<1.5mmthickness
Prone to recession	Pocket formation
Minimum width of keratinised gingiva	Wide zone of keratinised gingiva

13.7.1 Methods to deteremine thickness

Direct visual inspection (Rouck et al)
Probe transparency (Kan et al 2003)
Ultrasonography (Kydd)
CBCT (Fu JH)
Transginigval probing (Greenberg)

13.8 . Width of attached gingiva : [13]

Normal width	Maxilla anterior 3.5-4.5mm Mandibular anterior 3.3-3.9mm
Causes for inadequate width	Deep periodontal pockets Abnormal Frenal and muscle attachments Recession
Measurement	Total gingival width –pocket depth Schillers potassium iodide (stains glycogen)
Test for adequacy	Rolls test : done by pushing the adjacent mucosa coronally with a dull instrument. If gingiva moves , there is an innadequate AG , if gingiva does not move there is adequate with of attached gingiva.

13.9. Gingival inflammation :[24]

Gingival inflammation is charactereised by changes in : gingival color (redness), gingival contour, gingival bleeding,gingival stippling and gingival crevicular fluid flow. Several gingival indices have been proposed in literature, all of which have relied on one or more of the following criteria .These clinical features can be assessed non-invasively, only visually, (e.g., color, contour, spontaneous bleeding) and/or invasively, with the use of an instrument (e.g.,bleeding on provocation). Whereas some of the indices include both visual and invasive components, others are based on either visual features alone or bleeding on provocation alone. Thus, gingivitis can be evaluated by either quantitative clinical indices that are based on a combination of inflammation symptoms or extent of gingival involvement or on bleeding as a single variable .

13.9.1 PMA Index: developed by Schour & Massler (1947) and described by Massler (1967). It counts the number of gingival units affected rather than the severity of inflammation. The facial surface of gingiva around a tooth was divided into three gingival scoring units : mesial dental papilla (P) , the gingival margin (M) and the attached gingiva (A).Presence or absence of inflammation on each gingival unit was recorded as 1 or 0. The PMA scores were calculated separately and added to express PMA index score per person. P score (0-5),.M and A score (0-3). This index served as a basis for many other indices of ginigivitis.

13.9.2 Gingival index: developed solely to assess the severity of gingival inflammation. Invasive as it uses a periodontal pocket probe to assess bleeding potential. Four sites were assessed: distofacial , mesiofacial papilla ,facial margin and lingual gingival margin.

0	Normal
1	Mild inflammation,no bleeding
2	Moderate inflammation,bleeding on probing
3	Severe inflammation, spontaneous bleeding

Ginigval scores :
0.1-1.0 - Mild
1.1-2.0 -Moderate
2.1-3.0-Severe

13.9.3 Modified Ginigval Index: by Lobene and associates created the modified Gi, by eliminating the bleeding criterion making the MGI a non invasive index.

13.10 Bleeding on probing ; Earliest sign of inflammation and sensitive indicator and an objective measure of gingival inflammation.. Method to check gingival inflammation. Clinicians have used bleeding on probing in their diagnosis and treatment of periodontal diseases to record baseline data related to disease, to identify problem sites that require additional treatment, to screen patients before deciding on need for periodontal treatment, and to motivate patients to improve oral hygiene

Index name	Author	Year	Graded response	Time delay
Gingival Bleeding Score (GBS)	Carter barnes	1974	No	30 seconds
Gingival Bleeding Index (GBI)	AInamo and Bay	1975	No	10
Papillary Bleeding Index (PBI)	Saxer and muhlemann	1975	Yes (0-3)	20-30
Papillary Bleeding Score (PBS)	Loesche	1979	Yes (0-5)	Not stated
Periodontal Pocket Bleeding Index (PBI)	Van der velden	1979	No	30
Modified Papillary Bleeding Index (MPBI)	Barnett	1980	Yes (0-3)	0-30
Bleeding Time Index (BTI)	Nowicki	1981	Yes (0-4)	0-30
Eastman Interdental Bleeding Index (EIBI)	Abrams et al	1984	No	15
Modified Sulcular Bleeding Index (mSBI)	Mombelli	1987	Yes (0-3)	Not stated

These indices exclusively determine gingival bleeding. A range of bleeding responses exists, both with respect to the extent of bleeding and the time it takes for bleeding to occur after provocation.[25]

13.10.1 Pappilary bleeding index(Muhlemann 1975)

0	No bleeding
1	Single bleeding point 20 to 30 seconds after probing
2	Fine line of blood or several bleeding points
3	Blood fills interdental triangle soon after probing
4	Immediate profuse bleeding, fills interdental area, flows over tooth and gingiva

13.10.2 mSBI (Mombelli 1987)

0	No bleeding when a periodontal probe is passed along the gingival margin
1	Isolated bleeding spots visible
2	Blood forms a confluent red line on margin
3	Heavy or profuse bleeding

14. Periodontal Pocket :[1]

Examination for periodontal pockets must include their presence and distribution on each tooth surface, the pocket depth, the level of attachment on the root, and the type of pocket.

14.1 Periodontal pockets are classified into

Based on morphology	Gingival and Periodontal pocket
Based on involvement of alveolar bone	Suprabony, Infra bony
Based on surfaces involved	Simple, Compound, Complex
Based on nature of soft tissue trail	Edematous pocket , Fibrotic
Based on disease activity	Active,inactive pocket

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The only accurate method of detecting and measuring periodontal pockets is careful exploration with a periodontal probe. Pockets are not detected by radiographic examination. Probing can done as a part for diagnosis, monitoring course of treatment and monitoring maintenance.A tooth should be probed in atleast six points (Mesiobuccal, Midbuccal, Distobuccal ,Mesio lingual, Mid lingual, Disto lingual). Special attention should be directed to detecting the presence of interdental craters and furcation involvement..Naber's probe is used specially for easier and more accurate exploration of the horizontal component of furcation lesions.

14.2 Periodontal probe generations:[26]

1st generation /conventional probes	UNC 15, Marquis colour coded probe, Gold man Fox probe, WHO, Williams,
2nd generation probe	Pressure sensitive probes, Vine valley, Viva Care TPS
3rd generation Probe	Controlled force, Automated Florida probe ,Inter probe Toronto automated probe ,Foster miller probe Birek prob , Perio probe comp
4th generation Probe	3 D technology
5th generation Probe	Uses ultrasound technology

14.3 Probing depths [27]

Critical probing depth	Significance
<2.9mm	SRP results in attachment loss
2.9-4.2 mm	SRP indicated, flap surgery results in attachment loss
>4.2 mm	Periodontal flap surgery indicated

14.4 Clinical attachment level

1-2 mm	Mild periodontitis
3-4	Moderate periodontitis
>5mm	Severe periodontitis

14.5 Significance

Treatment need	Deep pocket probing depth is indicative of periodontal interevention
Disease activity	The precise assessment and comparison of the clinical attachment level at different intervals of time can determine whether attachment is being lost , which indicates that the lesion is active
Risk predictor[27]	Residual pocket in treated cases: >4mm , ≤4 pockets -low risk >4mm,>8 pockets- high risk
Reduction in pocket probing depth with attachment gain	Success of regenerative therapy

15. Bleeding On Probing And Disease Activity

The insertion of a probe to the bottom of the pocket elicits bleeding if the gingiva is inflamed and the pocket epithelium is atrophic or ulcerated. It is an earlier sign than color changes. Indices for assessing bleeding on probing: mentioned earlier

15.1 Significance of bleeding on probing

Disease activity	Occurrence of BoP in repeated examination is indicative of disease progression
Periodontal stability	Absence of bleeding on probing is an excellent predictor of periodontal stability. (Meta analysis Armitage 1996).
Future attachment loss	Presence of bleeding and probing in a treated and maintained population is an important risk predictor of increased loss of attachment. (Meta analysis Armitage 1996) . 30% probability of future attachment loss in sites that bleed repeatedly after treatment

16. Palpation [1]: Palpating the oral mucosa in the lateral and apical areas of the tooth may help to locate the origin of radiating pain that the patient cannot localize. Infection deep in the periodontal tissues and the early stages of a periodontal abscess may also be detected by palpation.

17. Suppuration[1] : This sign is present in a very low percentage of diseased sites (i.e., 3% to 5%). Therefore, it is not by itself a good indicator.

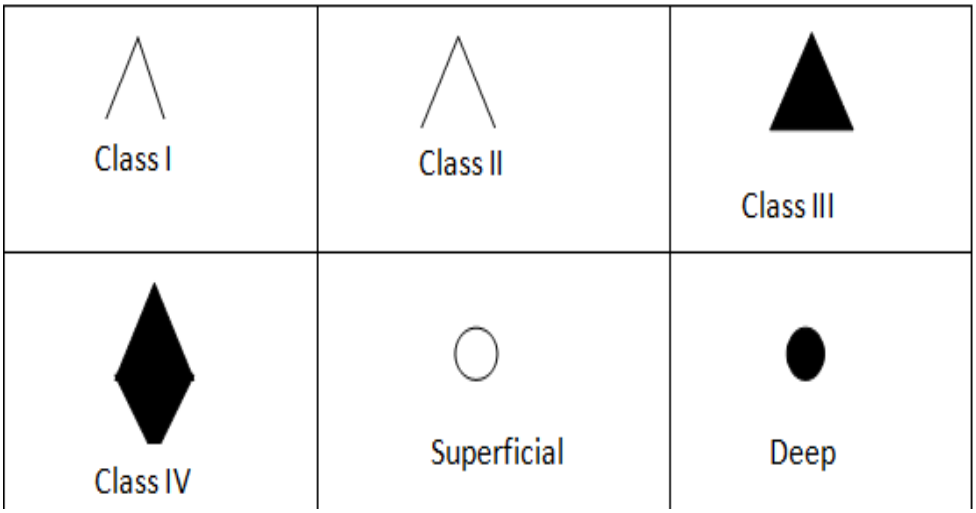
18. Furcation :[27,28,]

In the progression of periodontitis around multi rooted teeth, the destructive process may involve the supporting structures of the furcation area.. Examined with Nabers furcation probe. Not counting the third molars, 24 potential furcation exists. Diagnosis is best made through use of radiography and clinical probing .Probing is with a no. 23 explorer or Naber's no.1 and 2 curved probes. Classification of furcation include:

18.1 classification

Glickman 1953	Grade I:Pocket formation into flute but intact interradicular bone (Incipient) Grade II: Loss of interradicular bone and pocket formation but not extending to opposite side Grade III: Through and through lesion Grade IV: Through and through leion with gingival recession leading to visible furcation
Goldman 1958	Grade 1 : Incipient Grade II: Cul de sac Grade III:Through and through
Hamp et al (1975)	Grade I: Horizontal loss of periodontal support less than 3mm. Grade II: Horizontal loss of support >3mm but not encompassing totoal width. Grade III: Horizontal through and through destruction of periodontal tissue in the furcation.
Ramfjord &Ash (1979)	Class I : Beginning involvement. Tissue destruction <2mm into the furcation. Class II: Cul de sac >2mm, but not through and through. Class III: through and through involvement.
Tarnow & Fletcher (1984)	Subclassification based on the degree of vertical involvement: Subclass A: 0-3mm Subclass B: 4-6mm Subcass C:≥7mm
Eskow & Kapin	Same subclass as Tarnow 1984, but thirds instead of 3mm units are used.
Fedi 1985	Combined Glickman and Hamp; same Glickman gradesI through IV, but Grade II furcation are subdivided into degree I(<3mm) or degree III (>3mm)
Therapy	Grade I : Scaling and root planing, Furcationplasty. Grade II: Furcation plasty., Tunnel preparation , Root resection, Guided tissue regeneration at mandibular molars , maxillary buccal furcations. Grade III:
Prognosis	Regenerative therapy : Grade II furcation. Good clinical outcomes Grade III furcation:outcome unpredictable

18.2 Charting Symbols for Furcation :



19. Tooth Mobility :[1,29] All teeth have a slight degree of physiologic mobility, which varies for different teeth and at different times of the day. Mobility beyond the physiologic range is termed abnormal or pathologic. It is pathologic in that it exceeds the limits of normal mobility values.

Mobility normal	All teeth have a slight degree of physiologic mobility, which varies for different teeth and at different times of the day
Causes	Loss of tooth support (bone loss) Trauma from occlusion / hypofunction Extension of inflammation from the gingiva or from the periapex into the periodontal ligament Increased during pregnancy ,menstrual cycle, use of oral contraceptives. Pathologic processes of the jaws that destroy the alveolar bone
Methods to elicit	Mechanical, Electronic, Optical devices Laser Doppler vibrometry, Periotest.
Classification	Based on Cause of tooth mobility: pathologic , adaptive mobility Manner of tooth movement into : passive , dynamic mobility Direction of tooth movement : transverse , longitudinal. Progression : Increased/static mobility, progressive mobility/Dynamic mobility
Indices	Millers score : • Degree 0 The first distinguishable sign of movement greater than normal (physiologic). • Degree 1: Movement of the tooth which allows the crown to move 1 mm from its normal position in horizontal direction. • Degree 2: visually increased mobility >1mm in horizontal direction • Degree 3: Severe mobility of crown both in horizontal and vertical direction Glickman's score: • Normal mobility' • Grade 1: Slightly more than normal • Grade II: Moderately more than normal • Grade III : Severe mobility facio lingually and/or mesio distally combined with vertical displacement Prichard's index: • 1.Slight mobility • 2.moderate mobility • 3.Extensive movement in a lateral or mesiodistal direction combined with vertical displacement in the alvelolus. Wasserman's index • 1.Normal • 2.Slight Mobility: less than 3/4th mm of BL dimension • 3.Moderate : upto 2mm of BL movement. • 4.Severe mobility: more than 2 mm of BL movement.
Therapy	Primary occlusal cause : fixed ,removable splinting. Restorative therapy (Selective grinding) Primary periodontal cause: Periodontal therapy

XIV. Assessment of etiological factors:

Etiological factors include Plaque and calculus .

Indices to assess plaque and calculus[11]

Plaque biofilm forms the main etiologic agent in the initiation of periodontal disease. Calculus provides arough surface for the formation of plaque thus acting as a scaffold for the pathogenic biofilm in the pathogenesis of periodontal disease. Various indices have been used to estimate the extent of surface area of the tooth covered by plaque.

Plaque index

By Loe and Sillness(1964)

Unique in that it assess only the the thickness of plaque at the gingival third of tooth, most widely used, good validity and reliability.

Index teeth: 16,14,26,34,36,44

Surfaces : Mesiofacial,Facial,Distofacial,Lingual

Scoring:

0	No plaque
1	Film of plaque adhering to FGM,seen only with disclosing agent
2	Moderate accumulation of plaque gingival third,seen with naked eye
3	Abundance of plaque within gingival pocket,tooth, gingival margin

Turesky-Gilmore-Glickman Modification Of Quigley –Hein Plaque Index

This modification of the index was done to strengthen the objectivity of the Quigley Hein Index criteria by redefining the scores of the gingival third area .Provides a comprehensive method for evaluating anti plaque procedures and chemical anti plaque agents.

0- no plaque 1- separate flecks of plaque at cervical margin of tooth 2- thin continuous band 1mm at cervical margin of tooth 3- band of plaque >1mm covering <1/3rd of crown of teeth 4- plaque covering at least 1/3rd but < than 2/3rd of crown 5- plaque covering 2/3rds or more of crown

Other indices

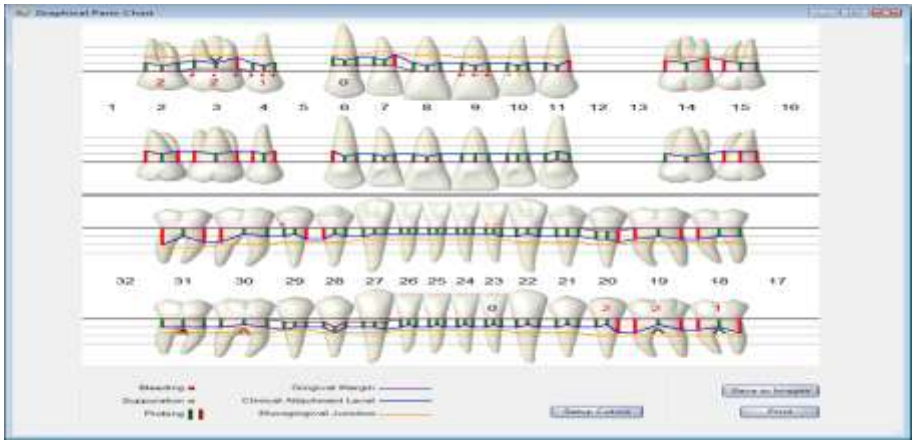
Shick & Ash modification of Plaque Criteria	The original criteria of the plaque component of Ramfjords Periodontal Disease Index was modified . Consists of examinig six selected tooth excluding interproximal areas ,restricting scoring of plaque to gingival half of facial and lingual surfaces of index toot
Glass index	This index assess the presence and extent of debris accumulation for evaluating the tooth brushing efficacy.
Navy plaque index	
Modified navy plaque index	The use of this new index enables the examiner to evaluate and record both the gumline (or marginal areas) and interproximal areas of the tooth, thus giving these anatomical areas an increased importance

Calculus Indices :

Calculus surface index	CSI assesses the presence or absence of supra and/or subgingival calculus by visual or tactile examination, regardless the quantity of calculus. Criteria: 0 – Absence 1 – Present.. 4 or 6 mandibular anterior teeth are examined. CSI =Total number of scores 0 -16 or 0 – 24
Marginal line calculus index	Another index used in short term clinical trials of anti calculus agents. Asses the accumulation of supra gingival calculus along the margin of gingiva. Used for assessing patient progress, patient motivation
Volpe manhold index	To assess the presence and severity of calculus formation specifically new deposits of supragingival calculus , following an oral prophylaxis.

Softwares For Periodontal Charting: Praktika ,Open dental software manual , Periodontal dental hygiene diagnosis ,Shire dental system ,OCS dental software ,Clear Dent electronic charting.

Periodontal charting :



Investigations Include : [30,31,32,33,34]

Blood investigations	Values	Increase	
Haemoglobin TLC: DLC: Neutrophil	13.5-18 mg% 12-16mg% 4500-1100cells/mm^3 50-70	Polycythemia Infection,drugs,abscess	Anaemia Viral disease, drugs, agranulocytosis
Lymphocytes	20-40	Leukaemia, allergy, tuberculosis	HIV,AIDS
Monocytes	0-7	Infection, SBE	
Eosinophils	0-5	Parasitic disease,allergy	Aplastic anaemia, cortisone therapy
Basophils	0-1	Leukaemia	Anaphylactic reaction
Bleeding time:	1-6min	Bleeding disorders	
Clotting time:	30-40 min	Coagulation disorders	
ESR:		Chronic infections :TB	
Urine analysis	Apperance and colour: Normal- yellow or amber Yellow brown/green brown-obstructive jaundice Yellow to orange urine – dehydration ,fever Black urine- malignant melanomas Specific gravity : Normal - 1.001-1.035 High specific gravity - dehydration, diabetes mellitus Low specific gravity –diabetes insipidus, Ph: normal -6 Range 4.6-8 Acidic pH: diabetic comaprolonged fever Alkaline pH: systemic alkalosis,renal insufficiency Protein : Normal- no protein in urine High protein Values : golemulardisease, congestive heart failure Glusoce- normally- negative strip test Positive strip test : Diabetes mellitus,hyperthyroidism Ketones: Not found ususally Abnormal values : severe diabetes mellitus, starvation, dieting Bilirubun : Normal : 0-0.02mg/100ml High values : obstructive jaundice,hepatitis Urobilinogen : Normal- 0.5-2.5 mg in 24 hr High values : liver disease, haemolytic disease Urinary sediment : Normal- one red cell in every two or three high power field Higher- bleeding due to acute infections,TB, WBC's- increased- urinary tract diseases,urethritis Casts –Few- Normal Higher numbers – urinary tract disorders Crystals – normal, note shape and colour.		
Serology	In case of desquamative gingival lesions , to confirm the type of lesion		
Radiographic in	IOPA : 17 periapical films (gold standard) 4 bite-wing films(vertical bite wings for assessment of alveolar crest level relative to CEJ.) OPG: overall radiographic picture of the distribution and severity of bone destruction with periodontal disease,		
Biopsy	Incisional biopsy- extensive lesions,central lesions Excisional biopsy- small lesions Aspiration Biopsy – intra osseous and soft tissue masses FNAC-isolatedneoplastic lesions Bone Marrow Aspiration-Anaemia,leukaemia,Myeloma Exfoliative vyoloty- screening of oral cancers, premalignant lesions Stab and roll- gingival biopsy, prevents peeling off epithelium from connective tissue Velscope- determine appropriate margin for biopsy		

Diagnosis :[1]

Diagnosis may be : Provisional Differential Final	
AAP classification	Gingival Diseases • Plaque-induced gingival diseases • Non–plaque-induced gingival lesions Chronic Periodontitis • Localized • Generalized Aggressive Periodontitis • Localized • Generalized Periodontitis as a Manifestation of Systemic Disease • Necrotizing Periodontal Diseases • Necrotizing ulcerative gingivitis • Necrotizing ulcerative periodontitis Abscesses of the Periodontium • Gingival abscess • Periodontal abscess Pericoronal abscess • Periodontitis Associated With Endodontic Lesions • Endodontic–periodontal lesion • Periodontal–endodontic lesion • Combined lesion Developmental or Acquired Deformities and Conditions • Localized tooth-related factors that predispose an individual to plaque-induced gingival diseases or periodontitis • Mucogingival deformities and conditions around the teeth • Mucogingival deformities and conditions on edentulous ridges • Occlusal trauma

Prognosis :[1] Defined as prediction of the probable course, duration and outcome of a disease based on a general knowledge of the pathogenesis of the disease and presence of risk factors for the disease .
Prognosis may be classified into
1. Overall prognosis of the whole dentition
2. Individual tooth prognosis

Factors to be considered while determining prognosis:

Overall Clinical Factors	Patient age Disease severity Plaque control Patient compliance
Systemic and Environmental Factors	Smoking Systemic disease or condition Genetic factors Stress
Anatomic Factors	Short, tapered roots Cervical enamel projections Enamel pearls Bifurcation ridges Root concavities Developmental grooves Root proximity Furcation involvement Tooth mobility
Local Factors	Plaque and calculus Subgingival restorations
Prosthetic and Restorative Factors	Abutment selection Caries Nonvital teeth Root resorption

25.2 Individual tooth prognosis depends on:
Mobility
Periodontal pockets
Mucogingival problems

Furcation involvement
Tooth morphology
Teeth adjacent to edentulous areas
Location of remaining bone in relation to the individual tooth surfaces
Relation to adjacent teeth
Caries, nonvital teeth and resorption.

25.3 Classification

Mc Guire and Nunn 1996 (based on tooth mortality)	Good, Fair ,Poor ,Questionable ,Hopeless
Kwok and Caton 2007 (stability post treatment)	Favourable, Questionable, Unfavourable, Hopeless

In many of the cases, it may be advisable to establish a provisional prognosis until phase I therapy is completed and evaluated.

Treatment Plan:[1,27]

Treatment of periodontal disease is a complex and multidisciplinary procedure, requiring periodontal, surgical, restorative, and orthodontic treatment modalities. According to Lindhe, treatment phases consist of : Systemic phase of therapy ,Initial (or hygiene) phase of periodontal therapy, Corrective phase of therapy, Maintenance phase (care).According to Carranza the different treatment phases include : Preliminary phase, Phase I, Phase II,Phase III, Phase IV. The commonly used classification of treatment phase by Caranzza is described below:

Preliminary	Treatment of emergencies: Dental or periapical Periodontal Other
Phase I	Plaque control and patient education: Diet control (in patients with rampant caries) Removal of calculus and root planing Correction of restorative and prosthetic irritational factors Excavation of caries and restoration (temporary or final, depending on whether a definitive prognosis for the tooth has been determined and the location of caries) Antimicrobial therapy (local or systemic) Occlusal therapy Minor orthodontic movement Provisional splinting and prosthesis
Phase II	Periodontal therapy, including placement of implants Endodontic therapy
Phase III	Final restorations Fixed and removable prosthodontic appliances Evaluation of response to restorative procedures Periodontal examination
Phase IV	Periodic rechecking: • Plaque and calculus • Gingival condition (pockets, inflammation) Occlusion, tooth mobility Other pathologic changes

XV. Conclusion

Periodontal diagnosis is an important label that clinicians place on patients periodontal condition or disease.[35] Proper diagnosis demands a thorough knowledge of the patients physical, medical and dental status. A good understanding of the normal periodontal healthy periodontium and changes associated with disease is imperative for early and correct diagnosis of the periodontal condition. Clinical diagnosis has been the main stay of disease diagnosis since ages. Despite intensive research efforts to develop new technologies to improve diagnostic ability, traditional diagnostic procedures based upon clinical signs of inflammation, probing depths and clinical attachment loss still form the basis upon which periodontal diagnosis is made. This reinforces the need for mastering this skill of clinical diagnosis

The methods described above for the examination of patients with respect to periodontal disease provide a thorough analysis of the presence , extent and severity of the disease in the dentition.[1] The classification of the patient and the correct diagnosis for each individual tooth should form the basis for a pre-therapeutic prognosis and the treatment planning of the individual patient. As the diagnosis of the case determines, the prognosis and treatment plan of the disease, Every effort should be made to utilise all the relevant clinical findings and form a customised treatment plan to benefit the individual patient.

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