

**Fibrous Architecture of Cemento-Dentinal Junction
in Health and Disease – A Scanning Electron and
Light Microscopic Study**

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CERTIFICATE

Certified that the dissertation on **“FIBROUS ARCHITECTURE OF CEMENTO-DENTINAL JUNCTION IN HEALTH AND DISEASE – A SCANNING ELECTRON AND LIGHT MICROSCOPIC STUDY** *done by* **Dr. PRATEBHA. B,** *Part II: Post Graduate Student (MDS), Branch II : Periodontics, Saveetha Dental College and Hospitals, Chennai submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment for the M.D.S. degree examination in February 2005, is a bonafide Dissertation work done.*

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INTRODUCTION

Periodontium is simply defined as the tissues supporting and investing the tooth. It consists of cementum, the periodontal ligament, the alveolar bone derived from dental follicle and that part of gingiva facing the tooth which is an adaptation of oral mucosa.^{41,51}

Like the specialized fibrous joint that roots the tooth to the socket; like the interfacial tissue, cementum, that forms a biological and structural link between dentin and periodontal ligament, the CEMENTO-DENTINAL junction, cements the cementum to dentin and forms a functional union between the tissues.⁸

After the root dentin is laid, the Hertwigs root sheath breaks-up and its cells secrete enamel like proteins. This is a 10 μm layer formed from an admixture of ectomesenchymal and epithelial products and is known by names like the hyaline layer of Hopewell and Smith and intermediate cementum.⁵¹ This layer gets mineralized later and forms the cemento-dentinal junction. The primary cementum is laid onto and adjacent to this layer.⁵⁰

Controversies regarding the nature of this layer and whether it is a form of dentin, cementum (or) a tissue in its own right were prevalent.⁵⁰

At present it is best regarded as a distinct tissue with the role of cementing cementum to dentin. It is distinct because it is atubular and has a unique organic matrix and is more mineralized than either the cementum (or) dentin.⁵⁰

It is reported from recent studies that the cemento-dentinal junction is a 2– 4 μ wide fibril poor groove and the attachment is created mainly by adhesion of proteoglycans and strengthened by mineralization.^{48,49}

At this juncture the nature and structure of the CDJ is somewhat clear but the feasibility of regeneration of this crucial structure that holds the key to regeneration of the periodontium remains elusive.⁴⁴

But, before addressing questions on regeneration it is imperative to develop a detailed understanding of the pathological alterations that may occur at the CDJ and its clinical implications if any. Studies pertaining to pathological aspect of CDJ are few and do not give conclusive reports.

The objective of this study therefore is to observe the collagenous architecture of cemento - dentinal junction in healthy and periodontitis affected teeth under scanning electron microscope; and also to study the pathological alterations in cementum and CDJ under a light microscope in healthy and periodontitis samples.

REVIEW OF LITERATURE

DEVELOPMENT OF CEMENTO-DENTINAL JUNCTION

Root development in human and non-human primates results from dentinogenesis and cementogenesis which are embryologically dependant on the apical proliferation of the cells of the Hertwig's epithelial root sheath (HERS).^{35,41} These cells subsequently degenerate or migrate away from the developing root surface into dental follicle. The sheath is bilaminar extension of the cervical loop of the enamel organ and is composed of outer

and inner enamel epithelium HERS proliferates in an apical direction, separating the dental papilla (the future pulp) and dental follicle or sac³⁵ (the future periodontal ligament & cementum).

During the root development, the cells of inner enamel epithelium of HERS do not become ameloblasts and initiate amelogenesis, unlike their counterparts in enamel organ of crown. These cells according to the traditional theory, only include adjacent ectomesenchymal cells of the dental papilla to become odontoblasts by cell-cell interaction. After deposition and mineralization of dentin matrix, the HERS degenerates & migrates into adjacent follicle (epithelial cell rests of malassez).^{8,41} The exposure of newly formed dentinal matrix to the cells of the dental follicle results in cell-matrix interactions and differentiation of adjacent cells of follicle into cementoblasts, which then deposit cementum on the newly formed dentinal surface. Thus both dentinogenesis and cementogenesis is sequentially dependant on the presence and subsequent absence of HERS at the developing root surface.⁴¹

Of particular interest is the absence of any mention of intermediate cementum at the cemento-dentinal junction in the traditional/classical theory of embryologic development. **Slavkin** suggested that the classical/traditional theory of root formation is inconsistent with the available evidence regarding the existence of this layer.⁴⁴ Many other investigators^{31,32} have supported the contention that the classical theory of embryonic tooth development does not recognize the formation and existence of IC.

The observation of a microscopically unidentifiable layer at the cemento-dentinal interface was first recorded more than a century ago⁸. This observation has been confirmed by light microscopy, scanning electron

microscopy, histochemical analysis, micro radiography and other investigative methods⁹⁻²². The development (origin) and function of intermediate cementum (IC) are controversial but heading recently towards clarity.

The structure and composition of this layer have been investigated by variety of methods that provide strong evidence of its existence. There are different theories regarding its origin.¹⁸ These theories differ with the chronology of the investigation, the sophistication of the investigative methods and the animal models used.

Intermediate cementum was first described by **Bodecker**⁷ in **1878** as the interzonal layer between dentin and cementum.

Hopewell-Simth 1920²¹ described a thin homogenous layer devoid of any identifiable histologic elements between the granular layer of Tomes and internal acellular layer of cementum. He speculated that this layer acts as a barrier to the external passage of medicaments placed in the root canal in the treatment of pulpless teeth.

The term intermediate cementum was first used by **Bencze**³ in **1927** (he initially called it the intermediary layer of cementum). In his article he described an ill defined area between cementum and dentin that had microscopic characteristics unlike either tissue (ie) the dentin (or) cementum. It was declared a tissue in its own right.

Stones 1934,⁴⁵ **Gottlieb**,¹⁵ **Blackwood (1957)**,⁶ **Held AJ**¹⁹ (**1951**) studied the biology of intermediate cementum. They reported similar non-descriptive, morphologic function of a narrow \pm 10 micron meter layer of calcified tissue outlining the dentin of the root (ie) the cemento-dentinal junction and commonly referred to as the “hyaline layer of Hopewell and

Smith” **Blackwood 1957**⁶ observed protoplasmic bodies in the intermediate cementum layer of human teeth and interpreted these as odontoblastic processes.

Osborn,³² **Owens**³¹ in **1972** reported similar observations indicating that IC was an unusual form of dentin and was therefore a product of dentinogenesis and not cementogenesis.

Cellular inclusions in this layer in the developing teeth of rats was observed by **Lester**²² in **1969**. With enhanced resolution of the electron microscope he determined that those inclusions were likely from cells of epithelial origin. He postulated that cells of HERS became entrapped in the rapidly calcifying matrix of cementum and subsequently underwent degeneration as a result of lack of nutrients.

The cementodentinal junction and cementogenesis was studied by many investigators using animal models using a variety of investigative methods during the 1960s and 70s . The results of these investigations^{38,39,40} contributed a large amount of information often contradictory, and a myriad of opinions and speculations regarding the development and function of the interface. The only areas of general agreement about the IC layer were that it is a noncollagenous matrix and is hypercalcified.

Hirchfeld et al 1973²⁰ reported the light microscopic observation of the presence of an enamel layer on the root analogs of unerupted teeth.

Slavkin et al 1974⁴³ confirmed the presence of epithelial secretory products (enamel matrix proteins) on the root analogs of rat and rabbit incisors. With use of light, scanning and transmission microscopy and histochemical and autoradiographic techniques they reported that the root-analog surfaces were covered with a thin layer of insoluble, non-

collagenous, epithelial, secretory products that were structurally and chemically identifiable as enamel matrix proteins containing glycosylated tryptophan, proline and histidine. They postulated that deposition of this matrix on these surfaces reflected reciprocal, interdependent- mesenchymal interactions.

Schonfeld, Slavkin et al (1974, 1976)^{36,44} immunised young adult rabbits with enamel matrix proteins & these proteins elicited an alloimmune response as demonstrated by the specific binding of antiserum to enamel matrix visualized by indirect immunofluorescence microscopy. They suggested that enamel proteins could possibly be auto antigens and is a constituent of the acellular cementum extracellular matrix which can provoke an “autoimmune reaction” on the part of the hosts immune response. They concluded that if validated this concept would explain why some periodontal pockets remain unchanged for years but suddenly progresses rapidly to cause severe destructions of supporting hard and soft tissues when exposed by periodontal disease.

Schonfeld, Slavkin 1977⁴² examined the continuously erupting incisors of rabbits by scanning & transmission electron microscopy biochemical fractionation and immunohisto chemistry. They reported qualitative similarities between the enamel matrixes in crown and root analogs. They said that root analog also demonstrated the characteristic honey comb- like appearance of mineralizing enamel matrix resulting from Tomes process pits. However they reported a quantitative difference in the thickness and geometry of enamel matrix. The thickness in the root analog surfaces demonstrated less than half the thickness observed on crown analogs. Transmission electron reports revealed the absence of collagen

fibres in IC and therefore the authors said that it was possibly a thin layer of mineralised enamel matrix proteins. They also concluded that the enamel matrix proteins were a product of inner enamel epithelial cells of HERS and noted a high level of cytodifferentiation within these cells.

Lindskog, Hammarstrom 1982²³ (a) investigated the presence of a distinct intermediate cementum with enamel proteins in higher animals. The study supported the observation of the existence of an aprismatic enamel and intermediate cementum that occupies the external interface of junctions between dentin and enamel in crown and junction between dentin and cementum in the root. Acknowledging the diversity of opinions regarding origin of IC, they used tetracycline labeling and concluded that the epithelial root sheath actively participates in the formation of IC but not in that of cementum proper (i.e.) acellular and cellular cementum.

Andreasen JO 1981¹ reported on the barrier function of intermediate cementum. Even as early as 1920, Hopewell-Smith first suggested that the “homogeneous layer of dentine, immediately external to the granular layer of Tomes and entirely devoid of any histological elements whatsoever” acts as an efficient barrier to the outward passage of drugs or medicaments placed in the root canal in the treatment of pulpless teeth. He concluded that “fortunately for mankind, one may use indiscriminately, and without fear of injuring the cementum or periodontal membrane, any or every drug of an escharotic or other nature. Andreasen in his study proposed that this layer acts as a barrier to the stimulation of inflammatory root resorption by the necrotic tissues (or) microbial contents of the root canal after trauma. His findings suggest that surface resorption (i.e) resorption of external cemental surface always occurs after trauma to the dentition and supporting structures

takes place. However when surface resorption penetrates IC layer, more severe resorption results. According to his hypothesis once intermediate cementum is violated by surface resorption it allows noxious stimuli of root canal to pass through, highly permeable dentin and this stimulates more aggressive resorption and destruction.

Lindskog 1982²⁴ (B) determined that IC layer was actually an enameloid layer. Their study provided evidence that before fragmentation occurred, the cells of HERS produced an enamel-like material on the surface of newly formed root dentin. This layer was reported to be 10-20 micron thick formed prior to cementum proper deposition and rapidly calcified. Also morphologic changes like distal microvilli and proximal bulb-type functions were demonstrated in here as in presecretory ameloblasts under SEM.

Lindskog, Hammarstrom 1982²³ (c) in order to provide additional evidence of the origin of IC, used labeled Tryptophan and proline injecting these amino acids into the periodontal tissues of monkey with incomplete root development. Results of this study showed that Tryptophan uptake was significant in the cells of HERS but not evident in odontoblast “predentin”. Tryptophan is an amino acid that is incorporated into enamel matrix but not into the collagen matrix of dentin (or) cementum proper and therefore was incorporated only in the HERS.

Moon-II Cho, Philius R Garant 1988³⁰ conducted a study to determine cells responsible for root formation in rats by light and electron microscopy. In this study special attention was focused on initial cementoblast differentiation into pro cementoblasts. In contrast to the results of many studies that reported that break-up of inner epithelial cells on

the surface of dentin was due to degeneration of Hertwigs epithelial root sheath, in this study the authors noted no evidence of cellular degeneration in the epithelial cells. Their observations suggest that detachment of the inner epithelial cells from the dentin surface and fragmentation of the underlying basal lamina may result from the active migration of pre cementoblasts toward dentin surface and their penetration between the dentin surface and inner epithelial cells. It is suggested that the cells of the dental follicle proper are cementoblast precursors which respond to chemoattractant substances newly released from deposited dentin matrix/basal lamina associated material of root sheath origin.

Craig K R, Harrison J.W 1993⁹ postulated that the presence of the smear layer should be considered in the root end resection procedure in periradicular surgical procedures and that removal of smear layer may enhance reparative cementogenesis on resected root surfaces. By Snook's reticular staining, the collagenous matrix of both dentin and reparative cementum, forms a nonstaining zone about 10µm thick which exists between reparative cementum and demineralised resected root ends. The nonstaining reaction zone represents the absence of collagenous matrix in both dentin and cementum. Under the light microscope this zone was identical to a zone observed along the lateral root surfaces at the CDJ in the area that corresponds to the expected location of IC layer.

Little disagreement exists that there is a permeability barrier external to root dentine. What is not known is the exact tissue or tissues that form this barrier. It is unlikely that cellular cementum with its vast system of interconnecting canaliculi could function as an effective barrier. This

probability indicates that acellular cementum or the IC layer or both function in this role.

Harrison, Roda 1995¹⁸ studied extensively the intermediate cementum and in their review article on IC, supported the view that HERS product is involved in formation of IC and they also stated the formation of IC clearly as (1) a permeability barrier between cementum and dentin (i.e) between external root surface and internal (pulpal) root surface (2) a precursor for cementogenesis in root development (3) a precursor for cementogenesis in wound healing.

L. Hammarstrom, I Atali, C.D. Fong 1996¹⁶ investigated the diverging opinions about the role of Hertwigs epithelial root sheath in the formation of cementum. In their study they lend support to the opinion that HERS is actively involved in the formation of both pre cementum, acellular and cellular cementum. The development of acellular cementum is consequent to the secretion of enamel related proteins by cells of Hertwig's epithelial root sheath.

Goldberg et al 2000¹⁴ has suggested in his study that the inductive agent for cementum matrix formation is not necessarily amelogenins. He has demonstrated the possibility of pulp-derived signals that might have an impact on cell differentiation in ameloblasts as well as HERS cells (or) cementoblasts. They also suggested that candidate molecules include basement membrane components like laminin and fibronectin, integrin cell surface receptors and peptide growth factors that interact with extra cellular matrix components.

T.G.H. Diekwisch 2001⁴⁷ (a) studied the mesenchymal origins of cementum and investigated the role of amelogenins in cementogenesis.

They have strongly emphasized that presence of amelogenin (or) enamel like proteins in acellular and intermediate cementum is questionable in contradiction to reports and studies by Schonfeld and Slavkin. In his study where he used in situ hybridization reaction using an amelogenin probe he demonstrated the absence of detectable amounts of ‘amelogenin RNA’s. He also reasoned out that the possibility of demonstrating amelogenins in other studies may be due to the fact that a number of polyclonal amelogenin antibodies cross react with keratins found in many epithelial cells including HERS.

Thomas G.H., Diekwisch 2001⁴⁷ (b) studied the developmental Biology of cementum. He studied the origin of the initial cementum matrix secreted/deposited against which cementoblasts differentiated (in rat molar). The electron micrographs of developing mouse molar root surfaces revealed that initial cementum matrix deposition exclusively occurred in areas in which mesenchymal cells had access to root surface and in which adjacent epithelial cells were separated from root surface by a basal lamina did not deposit any cementum matrix. This finding suggests that the first cementoblasts that appear during root development are mesenchymal cells of dental follicle and confirms previous studies on mesenchymal origin of cementum forming cells.

CEMENTO-DENTINAL JUNCTION IN HEALTH AND DISEASE

Prolonged exposure of cementum to a microbial environment occurs during periodontal disease. Numerous chemical and structural changes have been associated with cementum with this exposure. Among them one of the more pronounced changes is the presence of “pathologic granules”.⁴ Based on electron microscopic observations, it has been proposed that granules

might be sites where unmineralized fibrils may have been destroyed. Because of the association of cemental granules with exposure of root surfaces to the oral environment it has been suggested that they result from the action of microbial products on cementum. In general the heaviest concentration of these granules was near cemento-dentinal junction.¹² In the beginning the hypothesis that granules were artifacts were entertained. It was considered that they might be (i) fixation artifacts (ii) decalcification artifacts (iii) freezing artifacts.¹¹ But consequently as results were obtained from both fixed and unfixed tissues and granules were present in both it was confirmed that they were not fixation artifacts. Also the granules did not appear to differ in tissue which was decalcified with either acid or chelating agent. The hypothesis that when tissue was frozen prior to sectioning, formation of ice crystals might allow ingress of air into specimen, was also negated when granules were seen in decalcified ground sections which were never frozen.¹¹

Arimtage C.G, Christie T.M 1973(a)¹² studied structural changes in exposed human cementum, under light microscope. 56 teeth that were periodontally involved and exposed to oral cavity were sectioned, demineralised in formic acid. After demineralisation frozen sections were made with a cryostat and every fifth section was viewed under microscope. The granules like area were irregular and present along the surface of cementum and then number of granules increased towards the cemento-dentinal junction. It is suggested that the granules represent decomposed collagen fibers. The granules were not present in unexposed areas of the root cementum and CDJ.

Arimtage C.G. , Christie T.M et al 1973(b)¹³ studied structural changes in exposed human cementum under electron microscope. The periodontitis affected samples were demineralised, decalcified and frozen sections made. The frozen sections were viewed under light microscope and the areas with pathologic granules were demarcated. About 1mm of tissue from frozen sections were scraped and processed to be viewed under transmission electron microscope to confirm the presence of granules(or) Vacuoles. The vacuoles frequently followed morphologic patterns of collagen fibrils in the cementum and underlying dentin. Both granules and vacuoles have a similar distribution and are in the same size range and the same basic shape. Samples not exposed to oral environment did show any presence of granules.

Claude Bigarre, Michael Yardin et al 1977⁴ demonstrated lipids in pathologic granules in cementum and dentin in periodontal disease. In this study frozen sections of decalcified, periodontally diseased teeth were stained with Sudan Black and Schultz- Hershberger methods which indicated contained cholesterol or steroids. The lipid content of cementum suggests that the outer layer of cementum has been penetrated by exogenous substances of bacterial or salivary origin.

K.A Selvig, Hals E 1977³⁷ studied periodontally diseased cementum by correlated microradiography, electron probe analysis and electron microscopy microradiography studies [in essence illustrates the distribution of calcium] have indicated that the zone of cementum alteration may be limited to a superficial layer. It was concluded that in the absence of detectable caries, exposed cementum may develop a highly calcified surface layer with light F content and as well as a sub surface condensation of

organic material of exogenous origin (i.e.) bacterial endotoxin and gingival exudate.

C.G.Daly, G.J.Seymour, J.B. Kieser et al 1982¹⁰ assessed periodontally involved cementum for presence of lipids histologically and compared it with uninvolved cementum. Frozen sections were prepared from decalcified roots of 36 periodontally diseased teeth. Serial sections were stained for either lipid(Oil-red-O) or polysaccharide(Alcian Blue-PAS) and also with H&E. Specimens were then examined under light microscope for assessment of differences. Involved cementum exhibited strongly PAS +ve stained process penetrating 3-7 μ m into surface of cementum. Such processes were not present / observed in uninvolved cementum. H&E specimen revealed presence of microbial deposits in surface defects and defects within cementum. This suggests bacterial contamination upto CDJ.

Gray C. Armitage, Mark I. Ryder et al 1983¹¹ studied cemental changes in teeth with heavily infected root canals. Forty teeth that were periodontitis affected were decalcified and frozen sections made. Each section was observed under light microscope for presence of pathologic granules. The results of the study were that numerous bacteria were observed in the root canals of infected group and no bacteria were observed in either the root canals or adjacent dentinal tubules of any of non-infected group. Pathologic granules in cementum and CDJ were observed in infected group and not in the uninfected group. The results indicate that bacteria may play an important role in the formation of a structural change in cementum and underlying dentin known as pathologic granules.

Bjorn Eide, Tryggene, A. Selvig 1984⁵ studied surface coatings of dental cementum and concluded that the coating is probably identical to the

dental cuticle and stems from adsorption of components of gingival inflammatory exudate to the root surface. They also said that the endotoxins may adhere to the coating only rather than penetrating the cementum surface deeper. Mineralisation of the coating as demonstrated in the study would then act to strengthen the retention of toxic substances. They have also added that if this hypothesis, presented in the study and in a previous study by Eide et al 1983 is correct, then there would be a whole new outlook to treating periodontitis- involved root surfaces. It would mean that sacrifice of excessive amounts of hard tissue is unnecessary and that only mineralised (or) unmineralised surface coating only needs to be removed instead of whole of cementum rendering regeneration difficult.

Patrick A. Adriaens, Jan. A. De. Boeuer et al 1987³³ studied the viability and the distribution of bacteria within radicular dentin and pulp of periodontally diseased caries-free teeth. Healthy teeth served as controls. Samples obtained from the pulp tissue and from the radicular dentin(from interdental surfaces in sub gingival area of approximately 1mm thick layers of dentin). Samples were processed and cultured using anaerobic culture. Bacterial growth was detected in 87% of periodontally diseased teeth.

Tsuneyuki Yamamoto , Minoru Wakita 1990⁵² studied initial attachment of principal fibres to the root dentin surface in rat molars. The initial attachment of principal fibres to dentin surface was investigated in developing rat molars by light and electron microscopy. Ruthenium red was used to intensely stain the first deposited material onto dentin surface. Observations suggested that when principal fibrils became embedded in the ruthenium red material, there was no complicated interweaving between principal fibers and dentin- matrix. Therefore it is likely that initial principal

fiber attachment to the root dentin matrix is mediated by ruthenium red positive material.

T.Yamamoto, T.Domon et al 1999⁴⁸ studied the structure and function of cemento- dentinal junction in human teeth under light and electron microscopy. The cemento-dentinal junction was an approximately 1-3 μ m thick layer full of proteoglycans with muco polysaccharides but containing fewer collagen fibrils. By enzymatic treatment of decalcified specimen with hyaluronidase and trypsin, the CDJ decreased or lost staining affinity to toluidine. Prolonged treatment caused separation of cementum from dentin. These data suggest that proteoglycan is more important than the intermingling of dentinal and cemental fibrils for the C-D attachment.

T. Yamamoto, T. Domon, S.Takahashi et al 2000⁴⁹ studied the fibrous structure of cemento- dentinal junction in human molars under scanning electron microscope. This was done to observe the fibrous architecture in detail throughout the root sections of CDJ. They used NaOH maceration technique and observed that in macerated specimens, CDJ was a fibril poor groove. Some cemental fibrils or fiber bundles penetrated in the groove and appeared to intermingle with dentinal fibrils and intermingling was point- like at few places only . It is established that NaOH maceration removes only interfibrillar substance without damaging the collagen architecture. The study showed that interfibrillar adhesive substances are more important than fiber intermingling for C-D attachment.

T. Yamamoto, T. Domon, S.Takahashi et al 2001⁴⁶ have studied the fibrillar structure of the cemento- dentinal junction under scanning electron microscope in different kinds of human teeth. They have

demineralised teeth collected for 2-3 months in EDTA, sectioned, macerated them in NaOH to remove inter fibrillar structure and to observe the details of collagen architecture. Observations suggest that intermingling occurs in only few places in both cellular and acellular cementum. They have also observed that the protoglycan is a main factor for the cemento- dentinal attachment and that fibril intermingling is only an accessory factor in all kinds of human teeth.

Thus the junctional tissue has been extensively investigated and different theories regarding its origin and structure differed with the chronology of investigation and the sophistication of investigative methods used. The pathological aspects of this junctional tissue and its clinical implications is still not clear. This study intends to make an attempt to understand the cemento – dentinal junction in health and disease.

SUMMARY AND CONCLUSION

Cementum dentinal junction forms the biological and structural link between cementum and dentin and the evidence for existence for distinct layer between external root dentin internal acellular cementum is strong and undeniable from numerous meticulous investigations.

The aim of the present study was to observe the collagenous architecture of cemento-dentinal junction in periodontally affected and healthy teeth and to compare and report the differences as observed under scanning electron microscope.

Healthy and periodontitis samples were also observed under light microscope to study the pathological alterations in cementum and CDJ.

A total of 15 healthy and 30 periodontitis teeth were collected and sectioned into two halves and this amounts to 30 healthy samples and 60 periodontitis samples.

One half of the samples were processed for scanning electron microscopy and the other half for light microscopy. The SEM observations showed :

1. Increased width of interface at cemento-dentinal junction in periodontitis samples (7.1 μ m) compared to that of healthy samples(3.9 μ m)
2. Fewer and less dense areas of fiber intermingling in periodontitis as compared to more number and dense areas of fiber intermingling in healthy samples.
3. Detachment of cementum from dentin was seen in (43.33%) of periodontitis samples whereas only in 10% of samples detachment was observed in healthy samples.
4. Under light microscope , pathologic granules were observed in cementum and maximum concentration of granules were observed along CDJ compared to healthy samples.

From the present study we can conclude that the cemento–dentinal attachment is affected during periodontal pathology. This probably reflects as an increased width at CDJ, more number of periodontitis samples with cementum detachment , and fewer areas of fiber intermingling. In frozen sections pathologic granules denote decomposed collagen fibers and their concentration at CDJ may denote that the collagen present along CDJ may be more prone to destruction. This is because of more amount of unmineralised collagen at CDJ.

The present study deals with only the structure of cemento-dentinal junction. Further studies on composition of the junctional tissue at CDJ in periodontally affected teeth would reveal the differences in composition of proteins and proteoglycans that are actually involved in maintaining the integrity of the junction.

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