THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED SIXTY ONE

The Cementum: Its Role In Periodontal Health and Disease*

by DONALD A. KERR, D.D.S., M.S.,** *Ann Arbor, Michigan*

THE cementum is a specialized calcified tissue of mesenchymal origin which provides for the attachment of the periodontal fibers to the surface of the root. It consists of 45 to 50 per cent inorganic material and 50 to 55 per cent organic material with the inorganic material in a hydroxyl apatite structure.

The primary cementum is formed initially by appositional growth from the dental sac and later from the periodontal membrane under the influence of cementoblasts. It is formed in laminated layers with the incorporation of Sharpey's fibers into a fibrillar matrix which undergoes calcification. Cementum deposition is a continuous process throughout life with new cementum being deposited over the old cemental surface. Cementum is formed by the organization of collagen fibrils which are cemented together by a matrix produced by the polymerization of mucopolysaccharides. This material is designated as cementoid and becomes mature cementum upon calcification. The significance of the continuous deposition of cementum has received various interpretations.

1. Continuous deposition of cementum is necessary for the reattachment of periodontal fibers which have been destroyed or which require reorientation due to change in position of teeth. It is logical that there should be a continuous deposition of cementum because it is doubtful that the initial fibers are retained throughout the life of the tooth, and therefore new fibers must be continually formed and attached by new cementum. There is without question some wear and tear damage due to traumatism which would necessitate replacement. However, some question the need of new cementum for reattachment of fibers associated with functional reorientation. If we accept the premise that the fibers of the periodontal membrane are not continuous from bone to cementum but that cemental fibers and alveolar fibers are united by an intermediate layer, then functional reorientation can occur in the intermediate layer and reattachment by new deposition of cementum is unnecessary. The report of Gustafson and

*Read before the Annual Meeting of the American Academy of Periodontology, October 12-15, 1960, in Santa Monica, California.

**Professor and Head, Department of Oral Pathology and Periodontia, The University of Michigan School of Dentistry, Ann Arbor, Michigan.

Fig. 1. Resorption of cementum, dentin, and bone in an area of trauma. The resorption is inactive and the widened periodontal space is filled with fibrous tissue. Note active osteogenesis on outer surface of alveolar process.

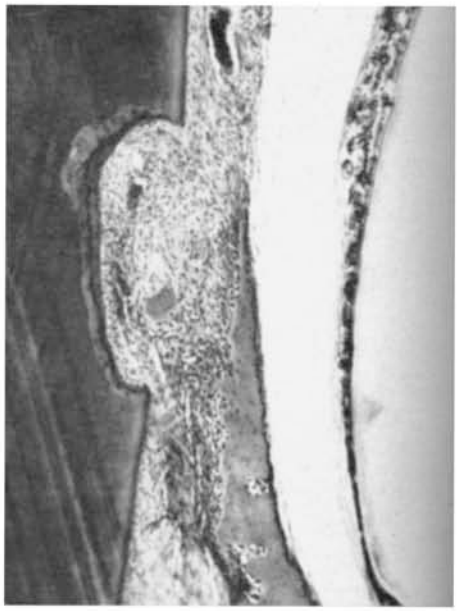

Fig. 2. Area of resorption of cementum and dentin repaired by cellular and acellular cementum. This is the root of a deciduous tooth. The enamel organ of the succedaneous tooth is evident at the right.

Persson[4] demonstrated that change in direction of Sharpey's fibers in cementum and bone has a relationship to the deposition of both cementum and bone. This suggests that new layers of cementum become necessary when change in position produces bending of Sharpey's fibers at the point of insertion into cementum.

2. Various authors have suggested that after deposition, cementum underwent aging and became nonvital. When the surface cementum became nonvital, the attachment to the tooth had to be re-established by apposition of a new layer of vital cementum.

3. It also has been indicated that cementum apposition is dependent upon tooth movement to provide the necessary space for apposition of new layers. This is in part true, but cementum deposition can be stimulated by hyperfunction associated with the widening of the periodontal space, by resorption of alveolar bone in response to occlusal traumatism, and cementum deposition may also be induced during the reparative phase of periodontal injury or tooth fracture.

4. The continuous deposition of cementum may be considered as an example of a normal biologic characteristic of all forms of calcified tissue. When tissue is fully calcified, it is susceptible to resorption, while uncalcified matrix is resistant to resorption. In intrachondrial bone formation the portion preformed by cartilage does not undergo resorption or replacement until calcification has occurred. After the cartilage has calcified, it is then resorbed by osteoclastic action. Bone, cementum, and dentin are all protected at the point of contact with connective tissue by a layer of uncalcified matrix. With destruction of the formative tissue uncalcified matrix is not present and the calcified tissue is resorbed by osteoclastic action in the zone of repair. Resorption of all calcified tissue can be produced by osteoclastic action. It is logical to postulate that the continuous deposition of

Fig. 3. Epithelial attachment in area of resorption at the bottom of a periodontal pocket. The space between epithelium and cementum is due to shrinkage in preparation.

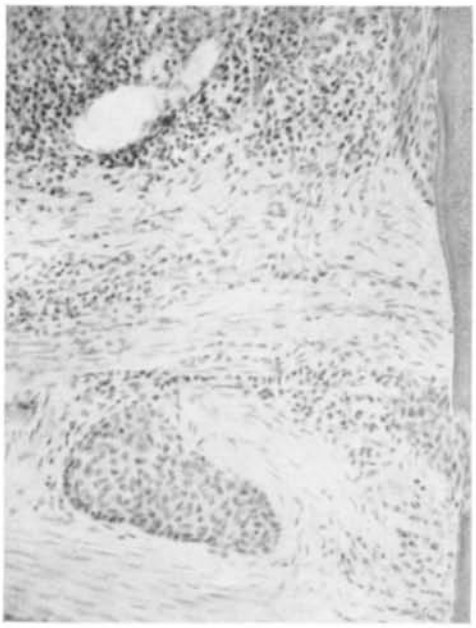

Fig. 4. Small area of cemental resorption filled with proliferating epithelial rest. Larger nests of epithelium in the periodontal membrane. The bottom of the epithelial attachment at the base of the pocket is evident in the upper right corner.

cementum is a protective biological process without which the supporting structure could not be maintained. This is supported by Zander and Hürzeler's[10] study in which they observed a straight line relationship between the thickness of cementum and age. This would suggest that continuous deposition of cementum was a biologic characteristic of calcified tissue which of necessity had to occur at a constant rate to provide the necessary protection of the mature calcified tissue. The cementum is more resistant to resorptive action than bone because of the nature of its vascular supply. When pressure is increased in the periodontal space, there is an increased intravascular pressure within the periodontal tissue which is transmitted by the vessels contained within the periodontium to the marrow and haversian spaces of the adjacent bone. The altered vascular supply effects the endosteum and stimulates bone resorption. Bone changes cannot be interpolated into cemental changes because all bone is vital and influenced by circulation, while only the surface of the cementum can be affected by changes in the circulation of the periodontal membrane. The only positive statement which we can make relative to the resorption of the two substances is that cementum is more resistant to resorption than bone even though it is produced in both tissues by osteoclastic action.

Cementum which is deposited after the initial layer covers the root may be of two varieties: it may be acellular like the initial cementum, or it may be cellular in character. The cellular cementum resembles bone in that formative cells are incorporated in lacunae spaces from which there are radiating canaliculi. Because of morphological similarity to bone, it has been designated as osteocementum. The lamina of osteocementum are thick and bulky in comparison to the lamina of primary cementum. The lamina contain a variable number of lacunae oriented parallel to the surface and with anastomosis of canaliculi. This provides a limited circulation to maintain the vitality of a thicker lamina. Cellular cementum appears to be deposited in broad

Fig. 5. Area of resorption at the bottom of the pocket just below the level of the epithelial attachment. No evidence of repair and very limited inflammation.

lamina when there is a rapid demand for a large amount of cementum to compensate for extensive movement or repair. Held[5] states that cellular cementum is produced by the incorporation of fibroblasts in the matrix at the time of formation and produces the cementocytes, while cementoblasts are not incorporated into the matrix of primary cementum. Thus the formation of the two types of cementum involve different degrees of histodifferentiation. This is analogous to the formation of fibrillar and mature bone. Cellular cementum can be covered by acellular cementum.

Investigations by Stones[9] indicate that primary (acellular) cementum is impervious while cellular cementum is porous. When cellular cementum is in contact with dentin, the cementocytes at the cementodentinal junction remain vital, while those slightly removed from the junction become devital with subsequent apposition of new lamina. This observation may carry some significance when we consider the cementum in disease.

Cemental hyperplasia occurs in response to heavy functional demand. When individual teeth or small groups of teeth are placed in heavy function, the surface of the cementum becomes irregular with spurs extending into the areas of principal fiber attachment. This undulating pattern increases the area of periodontal attachment and strengthens the supporting mechanism (Fig. 7). In heavy function the cementum is considerably thickened in the apical portion of the root. These changes indicate that cementum deposition is influenced by function as well as by movement of the teeth.

Increased deposition of cementum also occurs in association with periodontal inflammation, and changes which stimulate reparative proliferation may be manifest as hypercementosis.

Certain alterations in cementum have been suggested or postulated to be of importance in periodontal disease, and certain observations have been made which perhaps are worthy of note although their full significance cannot be explained.

Some authors, Gottlieb[1,2] in particular, have suggested that periodontal disease was a disease process of cementum, so-called cementopathia. It is difficult to see how this could be true when cementum is basically a nonvital calcified material which has to be continually "vitalized" by apposition

Fig. 6. Areas of resorption of cementum exposed in a periodontal pocket. The exposed cemental surface is covered by calculus.

of new cementum. Cementum is a product of mesenchymal tissue, and if there is a dysplastic process present, it must be one of the mesenchymal formative tissue. Such processes are rarely demonstrated, but a few examples have been observed in children in which there was spontaneous shedding of deciduous teeth, especially incisors, without attendant root resorption. Microscopic sections of such teeth demonstrate a complete absence or a very limited quantity of cementum on the entire root surface. This appears to be a dysplastic process of mesenchymal origin. All of the cases I have studied involved the deciduous dentition except one which involved the permanent teeth of a boy aged ten.

Hürzeler and Zander[6] studied cementum apposition in periodontally involved teeth and concluded that cementum apposition was less in periodontally involved teeth than in normal teeth. It appeared that cemental apposition decreased with advancing age in periodontally involved teeth. According to the authors this would support the contention that periodontal disease was a disease of the attachment apparatus, and as a result of the process cementum apposition was reduced. However, it does not indicate that the process is a dysplasia of the cementum.

Fig. 8. Enamel pearl in the bifurcation which is covered by a nodule of hyperplastic cementum.

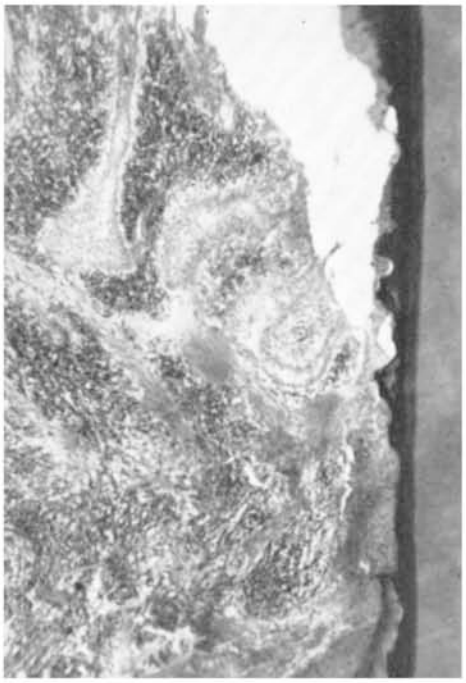

Fig. 7. This shows an area of cemental hyperplasia which has now been exposed by the development of a periodontal pocket. This provides a rough cemental surface for the accumulation of debris and the firm attachment of calculus similar to the pattern produced by resorption.

One would anticipate that periodontally involved teeth would have less cementum because of the detachment of periodontal membrane associated with pocket formation. Also, because these teeth were mobile, attrition with associated continuous eruption and apical thickening of cementum did not occur. This does not indicate that periodontal disease is associated with decreased cementum deposition.

Several writers have suggested that the cementum and dentin of the cervical area exposed in a periodontal pocket was softer than unexposed cementum and that it also was more porous than unexposed cementum. The cementum at the cervical area is acellular which Stones[8] has demonstrated

to be nonporous. However, exposed cementum may become decalcified after exposure and would then be soft and porous and an unsatisfactory environment for reattachment of periodontal fibers.

Rautiola[7] studied the hardness of cementum in both normal and periodontally involved teeth. He found that the cementum exposed by periodontal disease was of the same hardness as unexposed cementum of the same tooth or of normal teeth. This would indicate that the clinical impression expressed by Riffle[8] and others was incorrect.

In studying the root surfaces of periodontally involved teeth, areas of cemental resorption are frequently observed and their character and location suggest a varied etiology. One type of cemental resorption is seen in association with traumatism of a severity to produce compression of the periodontal membrane to the point of necrosis. As a part of the reparative process, cemental resorption is produced (Fig. 1). Such areas of resorption may be partially or totally repaired by new cementum deposition (Fig. 2). If apical migration of the epithelial attachment reaches such a level of cemental resorption, it appears to establish the same attachment as is provided on primary cementum (Fig. 3). In some areas of resorption due to traumatism there is proliferation of epithelial debris as a part of the reparative process and the epithelium attaches to the area of resorption (Fig. 4). These changes are identical to those described by Gottlieb[3] as typical of periodontosis.

Another form of cemental resorption observed in periodontitis is seen at the level of active inflammatory response in the bottom of the pocket, just apical to the epithelial attachment. In this zone principal fibers are partially destroyed or detached, there is active inflammation and fibroblastic proliferation, and cemental resorption. The resorption is usually shallow and limited in area. There is no evidence of repair in such areas, and they may be surfaced either by mesenchymal or epithelial tissue (Fig. 5).

A third type of resorption is seen in the cementum of the root exposed in a periodontal pocket. It is shallow and often appears to have been multicentric in origin. It does not have the same pattern as described for the other two types. The impression obtained from studying these areas is that of resorption of devital cementum by mesenchymal elements of the granulation tissue replacing the epithelial lining of the pocket. However, it is impossible to determine whether this might not have been initiated prior to exposure of the cementum into the pocket. Such areas are conducive to the accumulation of debris and the deposition of calculus (Fig. 6). Deposition of calculus into the irregular areas of resorption provides a firm attachment and makes complete removal extremely difficult and may explain some failures of reattachment and/or pocket elimination (Fig. 7).

Localized areas devoid of cementum, not to be confused with resorption, are present due to the persistence of Hertwig's epithelial sheath in selected areas. When the epithelium persists on the surface of the root, the dental sac or periodontal membrane is not influenced to produce cementum. Such areas of agenesis may ultimately be involved in pocket formation and become exposed into the pocket to be confused with areas of resorption.

From these observations it is suggested that cemental resorption may be a manifestation of periodontal disease before, during, and after pocket formation.

Anomalies of cementum deposition may also be associated with enamel pearls either in the form of aplasia of cementum or hyperplasia with irregular nodular masses over the pearl. Because of the frequency with which they occur in the bifurcation areas, they may influence the development or progress of the pocket into the bifurcation (Fig. 8).

BIBLIOGRAPHY

1. Gottlieb, Bernhard. The biology of cementum. J. Periodont., 13:13-17, Jan. 1942.

2. Gottlieb, Bernhard. The new concept of periodontoclasia. J. Periodont., 17:7-23, Jan. 1946.

3. Gottlieb, Bernhard. Etiology and therapy of alveolar pyorrhea. Zschr. f. Stomatol., 18:59, 1920.

4. Gustafson, A. G., and Persson, P. A. The relationship between direction of Sharpey's fibers and the deposition of cementum. Odontologisk Tidskrift, 65:458, 1957.

5. Held, A. J. Cementogenesis and the normal and pathological structure of cementum. Oral Surg., Oral Med., and Oral Path., 4:53-67, 1951.

6. Hürzeler, B., and Zander, H. A. Cementum deposition in periodontally diseased teeth. Helv. Odonto. Acta, 3:1-3, 1959.

7. Rautiola, C. A. The microhardness of cementum and underlying dentin of normal teeth and teeth exposed to periodontal disease. In press, J. Periodont.

8. Riffle, A. B. The cementum during curettage. J. Periodont., 23:170-7, July 1952.

9. Stones, H. H. The permeability of cementum. Brit. Dent. Jr., 56:273, 1934.

10. Zander, H. A., and Hürzeler, B. Continuous cementum apposition. J. Dent. Res., 37:1034-44, 1958.

NEW DIPLOMATES OF THE AMERICAN BOARD OF PERIODONTOLOGY

1961

The American Board of Periodontology announces that the following dentists were certified as diplomates at their last meeting:

Henry S. Brenman, B.S., M.S., D.D.S. . .	1913 Walnut St., Philadelphia, Pa.
Alvin D. Cederbaum, D.M.D.	140 Lockwood Avenue, New Rochelle, N.Y.
Sanford Frumker, B.S., D.D.S.	746 Rose Bldg., Cleveland 15, Ohio
Daniel A. Grant, D.D.S.	501 Maple St., San Diego 3, Calif.
Arthur B. Hattler, B.S., D.D.S.	843 Lancaster Ave., Bryn Mawr, Pa.
Sam W. Hoskins, Jr., D.D.S.	USAF Hospital Lackland, Lackland AFB, Texas
Walter N. Johnson, D.M.D.	U.S. Naval Hospital, San Diego, Calif.
Jules Klingsberg, B.A., M.S., D.D.S. . . .	100 Brooklyn Ave., Massapequa, L.I., New York
Edward R. Loftus, D.M.D.	68 Russell Park, Quincy 69, Mass.
Robert H. Loving, B.S., D.D.S.	U.S. Naval Training Center, Great Lakes, Ill.
Emanuel H. Malamed, D.D.S.	1147 Magee Ave., Philadelphia, Pa.
Bernard S. Moskow, D.D.S.	240 East Palisade Ave., Englewood, New Jersey
Billy M. Pennel, B.S., M.S., D.D.S. . . .	847 Monroe, Memphis, Tenn.
Sigurd P. Ramfjord, L.D.S., M.S., Ph.D. .	Univ. of Michigan, Sch. of Dent., Ann Arbor, Mich.
Bruce H. Rice, D.D.S., M.Sc., Ph.D. . .	3909 Van Buren Blvd., Arlington, Calif.
Sheldon J. Ross, D.D.S.	136 East 36th St., New York, New York
Albert Salkind, D.D.S.	650 Central Ave., Cedarhurst, New York
Daniel M. Staub, A.B., D.D.S.	465 Beach 133rd St., Belle Harbor 94, N.Y.
Jack L. Thaller, B.S., D.D.S.	1502 West 5th St., Brooklyn, New York
George J. Whinston, D.D.S.	132 East 72nd St., New York 21, New York
Jack K. Whitman, D.M.D.	307 Medical Arts Bldg., Jacksonville, Fla.
Charles H. M. Williams, B.ScD., D.D.S. .	University of Toronto, Faculty of Dentistry, Toronto, Canada