STUDIES IN THE PATHOGENESIS OF PERIODONTAL DISEASE*

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It can be said with certainty that the underlying causes of periodontal disease are not peculiar to modern times. There is ample evidence to suggest that the factors responsible for the destruction of tooth-supporting tissues have had their counterparts for as long as man has inhabited the earth. Early fossils have shown clear evidence of pre-mortem alveolar bone loss corresponding in appearance to the apparently atrophic alveolar ridge so often seen in patients with a history of periodontal disease preceding the edentulous state. Early writings, too, point to the prevalence of this disease in the times of their authors.

There are many variations of nomenclature in connection with periodontal disease, but fortunately the fundamental nature of the disease is incontrovertible. It is characterised by progressive disintegration of the supporting tissues and normally terminates in exfoliation of the affected teeth. This essential concept of the disease should not be obscured by the multiplicity of terms coined to describe intermediate stages in the pathological process.

Figure 1, a section of a newly erupted monkey molar, provides an illustration of the supporting tissues of a normal tooth. These constitute the bony walls of the socket, the fibres of the periodontal ligament which attach the tooth to the bone and the epithelium which surrounds part of the crown and insulates the underlying fibres and bone from the oral environment; to these three constituents could be added a fourth, the cementum covering the root, since this continuously deposited tissue affords attachment for the periodontal fibres.

Theories have been propounded at different times to suggest that the initial lesion of periodontal disease may arise in any of these components of the periodontium. The possibility of *primary damage to the cementum*

* Paper, read before the Nederlandse Vereniging van Tandartsen, Utrecht, 21st April 1961.

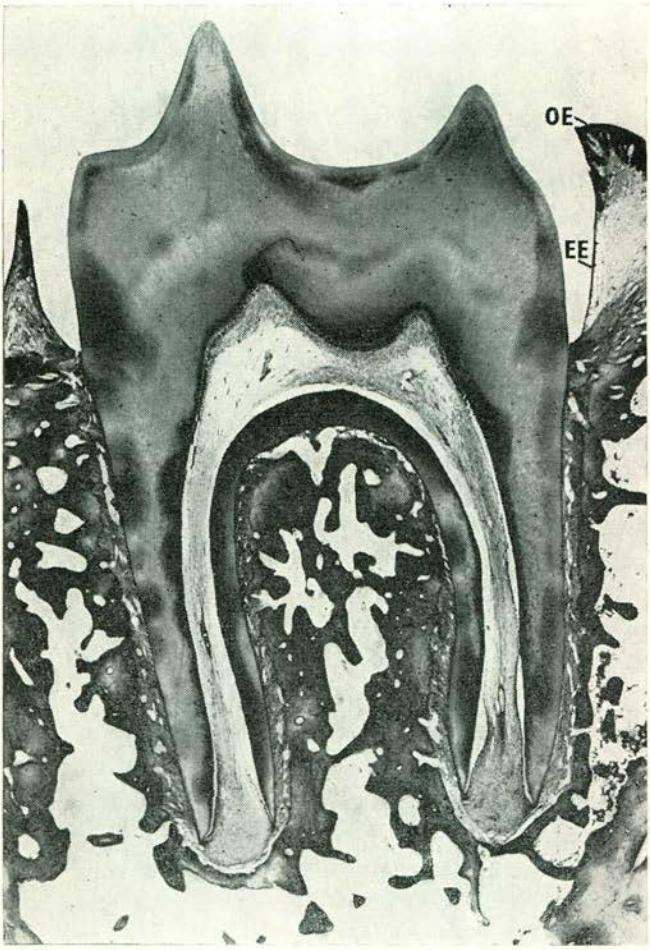

Fig. 1. Mesiodistal section of a molar tooth illustrating the essential components of the periodontium, i.e. bone, fibres, cementum and epithelium. The enamel has been removed in the course of preparation. OE = oral epithelium; EE = enamel epithelium. Haematoxylin and eosin. x 9.

was put forward by GOTTLIEB (1942) when he pointed out that the attachment of new fibres replacing old could not be effected without new deposits of cementum; he therefore regarded the viability of cementum as essential to the maintenance of periodontal health. However, no evidence has been adduced to demonstrate a failure of cementogenesis preceding periodontal disease, and the role of cementum has become obscured by assertions that the detachment of fibres from cementum can be effected by proliferation of the epithelial attachment. It is a common observation

that epithelium proliferates apically to cover denuded cementum, but the suggestion that the attachment of fibres can be destroyed by pressure or proteolytic action of epithelium postulates properties normally associated only with neoplastic conditions. So far as *origin in bone* is concerned it is certainly possible that a metabolic disorder affecting the skeleton as a whole could result in loosening and even loss of teeth. Generalised osteolytic conditions of such intensity are however extremely rare, and it is unlikely that any patient could long survive generalised skeletal resorption to the extent seen locally in periodontal disease. Moreover, the bone changes brought about in profound metabolic disorders, such as hyperparathyroidism for example, are reversible, so that restoration of bone contours, in a manner unknown in periodontal disease, can be effected by eliminating the cause of excessive resorption. It would be ludicrous to infer that systemic disorders cannot profoundly influence the course and severity of periodontal disease; but it is suggested that these should be regarded as modifying rather than exciting factors in its pathogenesis.

Leaving aside the rarities of metabolic osteodystrophies, the only other suggested means by which periodontal disease could be initiated in bone is that put forward by adherents to the theories of traumatic occlusion. Experimental and clinical evidence has shown that oblique occlusal force leads to resorption on the pressure side of the tooth socket and deposition on the tension side. Many different attempts have been made to improvise or imitate so-called occlusal trauma in order to produce periodontal disease. In no instance however, has this been done. No occlusal force has ever been contrived which can produce the selective and irreversible resorption of the alveolar crest so distinctive of periodontal disease. Indeed the daily experience of orthodontists indicates that teeth can readily be moved by imposed forces without the sacrifice of their ultimate rigidity. It is undoubtedly true that once the attachment of a tooth has been disrupted by periodontal disease, mobility and migration will be aggravated by undue occlusal forces; but it seems equally certain that traumatic occlusion can in no way account for the inception of the syndrome.

The possibility that periodontal disease could *commence as a lesion of the periodontal fibres* is embodied in the concept of periodontosis put forward by ORBAN and WEINMANN (1942). According to their belief this particular variety of periodontal disease is first manifested by migration of the affected teeth, which occurs as the consequence of a non-inflammatory degeneration of the periodontal fibres and subsequent proliferation of epithelium along the root surface. The criteria for the recognition of the

early stage, either clinically or histologically, have never been clarified; ORBAN's subsequent article (1942) on the aetiology of periodontosis incorporated so wide a spectrum of systemic causes that it did nothing to delineate the concept of a specific disease. Today periodontosis has come to be defined as a generic term for diseases of the periodontium in which degeneration is dominant; as such it is difficult to distinguish as a specific entity, and provides no inkling of an origin exclusively within the periodontal ligament.

There now remains to be discussed the possibility of an *initial lesion in the epithelium*. FISH (1944) traced the course of periodontal destruction from the outer surface, i.e. the epithelium, towards the deepest part of the periodontium, i.e. the alveolar bone, and subsequent studies have confirmed the general accuracy of his description. Histologically, a breach in the epithelium can often be detected before damage to either fibres or bone is evident at a microscopic level; occasionally fibre destruction is seen without signs of bone change, but never without evidence of epithelial injury being revealed by examination of serial sections; likewise, when bone loss is seen together with the disintegration of fibres an associated epithelial defect is invariably to be found.

Apart from the constancy with which evidence of damaged epithelium accompanies periodontal disease, there are other reasons for believing that the initial lesion is epithelial. Studies of periodontal lesions in laboratory animals (COHEN, 1959a and 1960) have demonstrated that progressive periodontal destruction can be initiated as a consequence of trauma to the epithelial surface. In every case the site of injury was related to a specific area of the periodontal epithelium – that is, to the enamel epithelium.

The Two Constituents of the Periodontal Epithelium

Before eruption, the crown of the tooth is covered by epithelium derived from remains of the enamel organ. It is not possible for this tissue to survive exposure on a masticatory surface, but in areas of the tooth not so exposed, such as the interproximal areas, enamel epithelium may persist long after eruption and well into adult life. In the retromolar area of figure 1, for example, it can clearly be seen that the periodontium is covered by two different epithelia; one of these, multilayered and robust, is the stratified squamous epithelium of the oral mucosa, while the other, frail and slender, is composed of enamel epithelium. The two epithelia merge at a site corresponding to the bottom of the gingival crevice.

This concept of a dual origin of the periodontal epithelium has occa-

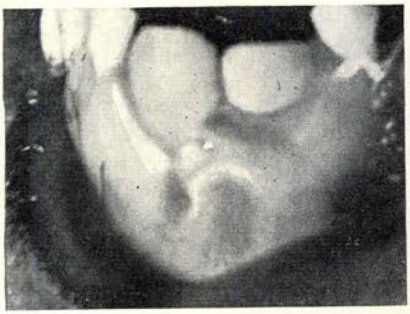

Fig. 2. Photograph of erupting lower central incisors in a monkey. 1) had been exposed prior to eruption and stripped of enamel epithelium, after which the flap of oral epithelium was sutured back into position. Note the difference in outline of the gingiva surrounding this tooth from that of the control.

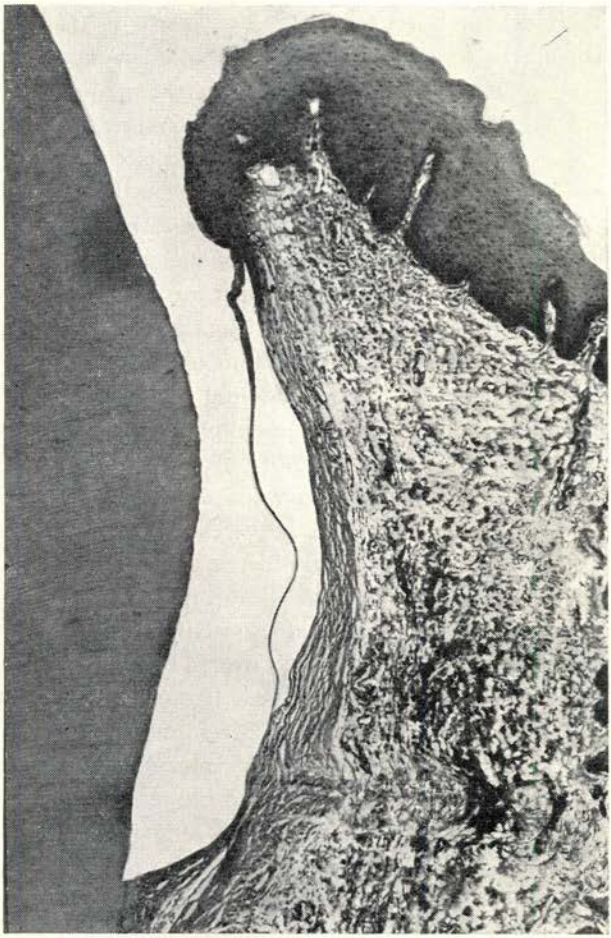

Fig. 3. Longitudinal section of the gingival crevice of a tooth from which the enamel epithelium had been removed prior to eruption. Note that a thin tapering extension of oral epithelium lies between the periodontium and the space previously occupied by enamel. Haematoxylin and van Gieson. x 65.

sionally been contradicted by the suggestion that the deeper layer is merely a tapering extension of the stratified squamous epithelium of the surface. In order to provide evidence of the essential participation of enamel epithelium, experiments were carried out (COHEN 1961) in which enamel epithelium was extirpated from the crowns of unerupted permanent teeth. These teeth later erupted uneventfully but showed two major differences from the normal:

(a) clinically, the gingival festoon followed the outline of the cement-enamel junction giving the appearance normally expected in a much older tooth (see figure 2);

(b) microscopically, the characteristic arrangement of two confluent epithelia was entirely absent and the periodontium was covered only by stratified squamous epithelium tapering abruptly from the gingival crest to the cement-enamel junction (see figure 3).

These findings provide evidence suggesting that the normal recession of the gingival margin with age is indicative of gradual involution of the enamel epithelium; it also suggests, incidentally, that a real attachment of enamel epithelium to enamel must exist in order for the gingival level to be sustained at an appreciable distance from the cement-enamel junction.

From theoretical considerations it would be expected that of the two tissues comprising the periodontal epithelium the stratified squamous element would be by far the better suited to withstanding injury. This multilayered epithelium is not only capable of rapid proliferation when injured but reacts to the stimulus of wear by producing an outer coat of resistant keratin. By contrast the enamel epithelium is a vestigial tissue, no more than a few cells in thickness, and ill-fitted to act as a protective outer surface. It is not theoretical consideration alone however, that provides a basis for the hypothesis that the most vulnerable area of the periodontium is that which is covered by enamel epithelium, since observations on experimental animals (COHEN 1959a, 1960) have implicated this tissue in the production of periodontal lesions.

A study of the distribution of enamel epithelium on the periodontal surface is an obvious corollary to the recognition of its vulnerability. It has previously been shown (COHEN 1959b) that although enamel epithelium is a constituent of the periodontal surface of recently erupted teeth, it is always buttressed by adjacent stratified squamous epithelium on the buccal/labial and lingual/palatal aspects (see figure 7). Only in the interdental area can the situation arise where enamel epithelium alone comprises the surface covering of the periodontium, and then only when teeth have erupted in contact. To find the reasons underlying this circumstance

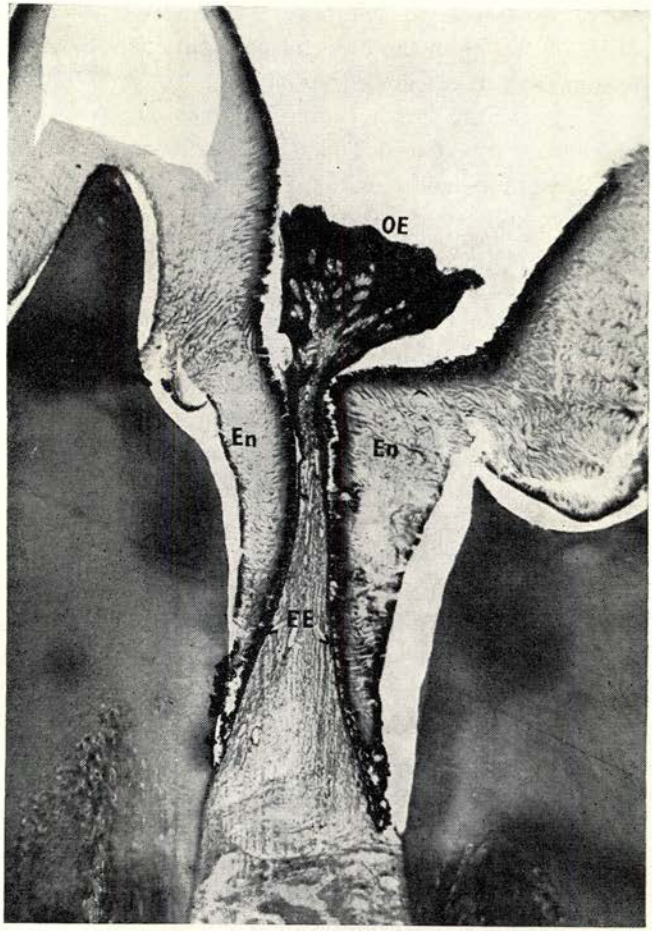

Fig. 4. The interdental septum between an erupted molar and one in the course of eruption. The enamel structure has been preserved. OE = oral epithelium; EE = enamel epithelium; En = enamel. Haematoxylin and eosin. x 20.

it is necessary to go back to the time of tooth eruption, even though consequences may only become manifest many years later.

It frequently happens, when a tooth erupts, that it is guided into its position in the arch by the adjacent previously-erupted tooth. As contact is established between the two teeth the oral epithelium surrounding the erupted member must inevitably be undermined by the emerging crown and subsequently divided into two processes of oral mucosa which, separated by the contact area, come to constitute the gingival papillae oc-

cupying the buccal and lingual embrasures. It is not easy to demonstrate this severance of oral epithelium by conventional methods, but the preservation of enamel in decalcified sections (BRAIN and EASTOE, 1961) enables exact phases in the process to be identified microscopically. Such a section is that shown in figure 4. This shows the interdental area between the first and second molars of a monkey, the latter in the course of eruption. Above the contact area a strand of stratified squamous epithelium can be seen in transverse section; its persistence in this situation is clearly impossible and it is destined to disintegrate even before the new tooth has come into full occlusion. This means, as reference to figure 4 will again confirm, that the periodontium will be covered by no more than the interproximal enamel epithelia of the contacting teeth, and so it must remain until the oral epithelium has contrived to reunite by growing through from embrasure to embrasure beneath the contact area.

The extent of the area covered only by enamel epithelium depends upon the morphology of the adjoining teeth, and it can be demonstrated histologically only by cutting sections in the buccolingual plane. When this was first done it was found that the interdental septum between teeth in contact had the outline, not of a papilla or a pyramid as has so often been said, but of an irregular depression between two prominences, these being constituted by the buccal and lingual papillae; the structural outline of the septum in this plane conforming to that of a col between two mountain peaks. The col-shaped structure of the interdental septum can be identified microscopically if contacting teeth of a young subject are extracted with sufficient care to preserve the soft tissues in an undisturbed state. Figure 5 shows the molar and premolar area of a monkey's maxilla in which this has been done, and it can be seen that the papillae which occupy the buccal and lingual embrasures constitute peaks in relation to the col of the interdental septum. If a section be cut through one of these interdental septa soon after tooth eruption it is possible to demonstrate that stratified squamous epithelium is not continuous from papilla to papilla. Such a section is shown in figure 6. This not only illustrates the conspicuous difference between the two epithelia but demonstrates the considerable extent of the more vulnerable area of the periodontium, that is the area covered only by enamel epithelium. This vulnerability is of course offset by the fact that the interdental area is normally protected from direct injury by the teeth in contact above it.

These morphological features provide a reasonable explanation for the common clinical experience that the intrabony pockets of destructive periodontal disease are most often to be found interdentally. Damage to

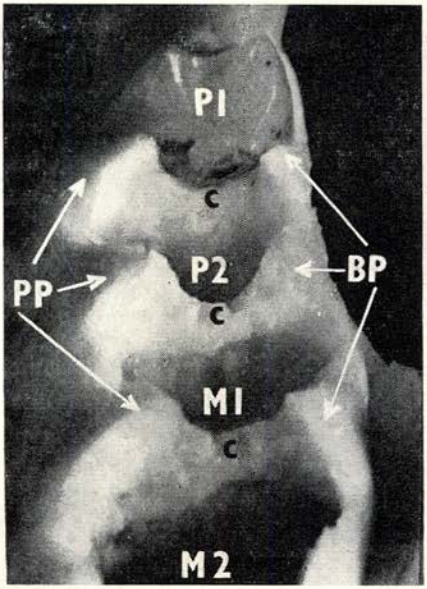

Fig. 5. Portion of the left maxilla of a monkey from which teeth have been extracted to reveal the gingival contours. BP = buccal papilla; PP = palatal papilla; PI = first premolar; P2 = second premolar; MI = first molar; M2 = second molar; C = col.

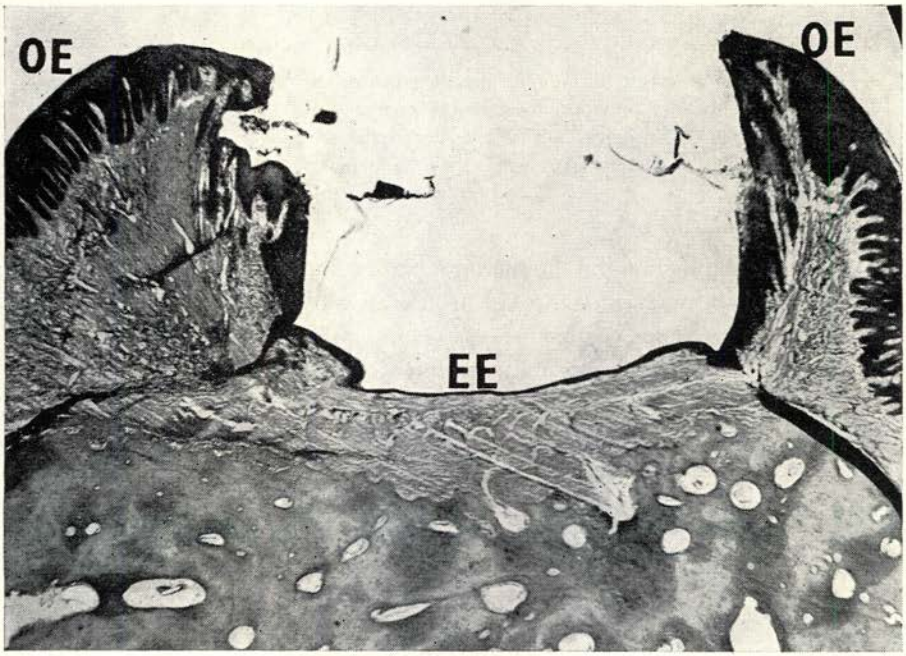

Fig. 6. Buccolingual section cut through the septum between two molar teeth. OE = oral epithelium covering papillae; EE = enamel epithelium covering the surface of the col. Haematoxylin and eosin. x 28.

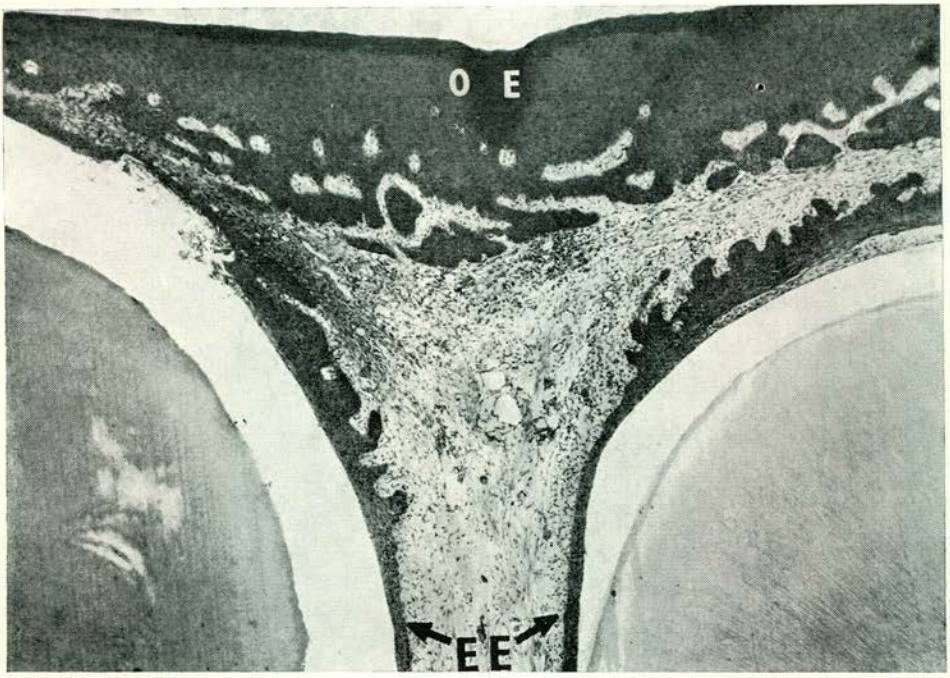

Fig. 7. Horizontal section through the lingual embrasure between two molar teeth. The spaces denote the area previously occupied by enamel. OE = oral epithelium in close relationship to enamel epithelium on the lingual surface. EE = enamel epithelium. Note that no oral epithelium is present as the interdental area is reached. Haematoxylin and eosin. x 55.

the enamel epithelium on the buccal/labial or lingual/palatal aspects can presumably be made good through proliferation of the stratified squamous epithelium by which the enamel epithelium is buttressed in these situations; no such protection is afforded interdentally (see figure 7). Support is lent to this theory by the results of a survey of the gingival condition of some 400 adolescents (COHEN and TAYLOR, 1960), in which it was found that bleeding could be elicited with a blunt explorer far more frequently interdentally than anywhere else around the tooth.

Calculus and Periodontal Disease

The fact that labial and lingual crevices are always well protected by stratified squamous epithelium affords an explanation for the fact that deep pockets are rarely seen in these situations, except when they extend from an interdental lesion. For this reason calculus, which is so commonly

deposited around the necks of teeth on lingual surfaces, is of little consequence as an exciting factor in the development of deep periodontal pockets.

It is, indeed, extraordinary that so much attention is still paid to the significance of calculus in the pathogenesis of periodontal disease. In the tenth century Abulcasis described instruments designed to scrape the teeth free of scale which would otherwise communicate corruption to the gums and thus denude the teeth; we are still scraping at the scale, and descendants of Abulcasis continue to design scalers, but the incidence of periodontal disease does not appear to have been greatly altered as a consequence. In spite of this it might seem to some that the acquittal of calculus is akin to heresy. It might therefore be as well to emphasise that the incidence of periodontal disease is certainly as high in the upper anterior teeth as it is in the lower anterior segment; most clinicians would probably suggest that it is, in fact, considerably higher. Yet the lower anterior teeth constitute the commonest site of calculus deposition in the mouth, the upper anteriors the rarest. If there were any sort of positive correlation between the presence of calculus and the development of periodontal disease, the lower incisors should be the first teeth to succumb to this disease; in fact, they are much more frequently the last survivors!

It is not suggested that the removal of calculus is unnecessary or undesirable. The presence of deposits around the necks of teeth is often responsible for congestion and inflammation of the marginal gingiva, and their removal is invariably attended by improved gingival health. However, scaling should be recognized for what it is, namely a local debridement desirable in the interests of local oral hygiene; it is a delusion to attribute more far-reaching benefits to this procedure and to suggest that it can serve to prevent the development of periodontal disease in interdental regions already the site of early periodontal breakdown. All the evidence suggests that this early lesion in the pathogenesis of intrabony pockets has no causal connection with calculus. It is as well to remember, perhaps, that the heaviest deposits of calculus occur along lingual surfaces and that both lingually and buccally the gingival crevice is amply protected by stratified squamous epithelium, as has been illustrated microscopically in figure 7. The robust epithelium of the embrasure and lingual surfaces shown in figure 7 is well equipped to protect the underlying tissues from such minor irritants as calcareous deposits; the flimsy covering of the interdental area is unquestionably the more likely site for damage affecting the entire periodontium.

It is therefore suggested, on the basis of these studies, that the first

responsibility of the dentist in preventing periodontal disease is to ensure that the periodontium is adequately epithelialised. It is just as important to examine patients carefully for a breach in the integument of the periodontium as it is to examine their teeth minutely for a breach in the enamel. An important difference is that once the enamel is breached, it cannot heal; the gingival epithelium, on the other hand, has an immense capacity for repair, and the prevention of periodontal disease should therefore be within the reach of dental science.

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