THE PREVENTION OF PARODONTAL DISEASE *)

BY SIR E. WILFRED FISH

1. Introduction

When discussing new developments in dentistry it is usual, and I believe prudent, to examine both the history and the outcome of corresponding activities in the sphere of general medicine. If we apply this to our present problem we find that in recent years very considerable interest has been focussed on Preventive Medicine as a department of medical science and training. The ideal of positive health, rather than mere freedom from demonstrable and active disease, has led to intensive study of prevention based on more exact and early diagnosis and more accurate knowledge of aetiology. The significance of heredity, race, of constitutional predisposition and even of underlying emotional and mental factors became more evident as increasing interest led to more carefully recorded observation.

During the last War in Great Britain an Inter-Departmental Committee was set up to inquire "Into the organisation of Medical Schools, particularly in regard to facilities for clinical teaching and research". This Committee, usually called the 'Goodenough Committee' after their Chairman Sir William Goodenough, laid special stress on the importance of inculcating the principles of preventive medicine in medical students. Indeed in enumerating some of the problems which constitute a large part of the general practitioner's work they said, "Specially important in this connection are the prevention of disease and an appreciation of the part played by social environment in health and sickness".

They further expressed the view that medical training was deficient in this respect and added that "There is growing recognition that it is on the teachers of the pre-clinical subjects that the responsibility rests for laying the foundations of the student's realization of the importance of health and the prevention of disease".

This is indeed sound advice and is particularly appropriate in any discussion of prevention in relation to disorders of the parodontal tissues, so much so in fact that when the General Medical Council were revising the "Recommendations concerning the Dental Curriculum" in 1952 they said, "Instructions should be given in Preventive Dentistry, including the theories of causation, incidence and prevention of oral disease"; adding a note: "During courses of instruction in the periods of preclinical and clinical studies, the attention of the student should be

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continuously directed by his teachers towards the prevention of dental disease."

The importance of this recommendation becomes apparent when one reflects that the healthy development of both the calcified and the soft connective tissue as well as the normal growth of the epithelium and the enamel are of the greatest importance in the prevention of both parodontal breakdown and caries.

Unfortunately so far as actual practice is concerned, in contrast to precept, interest has been chiefly focussed on the prevention of caries rather than pyorrhoea. This can easily be understood since "prevention" and "Children's dentistry" have usually been bracketed together in the organisation of our schools and institutes. This arrangement is no doubt inevitable not only for economic reasons but because, in dentistry, prevention, if it is to be practised at all, must be practised on children, but that does not mean that our attention should be limited to the prevention of caries. It is true that caries is predominently the disease of childhood and early adult life and that parodontal breakdown is more commonly observed later on, but the seeds of parodontal disease are often sown in childhood, even if only by inadequate instruction in methods of maintaining oral hygiene or by the extraction of a carious first lower molar, which allows the bite to close. Moreover once the parodontal tissues have been destroyed they can never be regenerated and it would be a serious criticism of our preventive service if we were to delay or even prevent the onset of caries only to find later on that the attachment of the teeth had become hopelessly undermined by pyorrhoea.

It is, however, obvious that the practice of prevention requires a close and careful study not only of the pathology but also of the aetiology and social and racial incidence of the disease: and this cannot be made in the children's department alone without access to clinical material and laboratory facilities in other departments of the hospital. It seems unlikely therefore that the prevention of *parodontal* disease will make much headway unless there can be a closer liaison established between these various departments. It would be unfortunate if the parodontal departments of our great teaching establishments were to limit their interest to adults who are suffering from the disease and be concerned wholly with their treatment and with the pathology of the various manifestations of parodontal breakdown, while at the same time those responsible for the teaching in the preventive dentistry departments were concerned only with children, in whom the disease is seldom conspicuous, and were excluded from a close study of the pathology and from daily contact with the clinical reactions of parodontal disease. Para nevertheless in the course of investigations into the pathology of parodontal breakdown, and in clinical experience of treating and preventing the recurrence of the disease in adults, it is possible to draw some tentative conclusions as to aetiology and prevention ab initio.

2. *Chronic Marginal Ulceration*

There can be little doubt that in the vast majority of cases where the

attachment of the teeth is undermined the first step in the gradual deterioration of the parodontal tissues is the ulceration of the gingival trough; but there are of course predisposing causes even to this ulceration. Not only are there predisposing conditions which lead to the ulceration itself but there are conditions which aggravate the marginal inflammation and cause it to spread more or less rapidly to the deeper structures.

The predisposing causes of marginal ulceration are chiefly mechanical such as inconsistencies in the diet, and irregularities in the arrangement of the teeth. The absence of attrition is also important, for owing to the soft nature of civilised diet the teeth are left with long clinical crowns, and the gums get very little friction. The conditions which aggravate the ulceration and lead to a more rapid extension of the inflammation causing destruction of the deeper tissues, are associated with occlusal trauma. But deeply underlying both the phenomena which lead to marginal ulceration and those which predispose to the more rapid spread of the inflammation to the deeper structures is a constitutional and often an hereditary predisposition which so far has eluded our efforts to track it down. Many of these predisposing causes may be found to operate or have their origin in childhood, and some may be preventable.

The proximate predisposing cause of marginal ulceration is lack of mechanical toughness in the gum margins to resist abrasion and this in civilised communities is often due to lack of friction. Friction is the one method at our disposal for rendering tough the surface layers of stratified squamous epithelium. The familiar phenomenon of the horny hands of a labouring man or the callous soles of the feet in those who walk barefoot will serve to illustrate the value of friction in this context. It is therefore just as important for dental health to restore the abrasive qualities of the food we eat, or supplement them, as it is for general health to restore the vitamins which are often similarly lost in the course of preparing food for the table. It is however extremely unlikely that we shall ever persuade people to revert to primitive diet. Indeed it would be impossible to feed the vastly increased population of the earth on fresh food. We have therefore inevitably, to take into our calculations the occasional excessive onslaught of the extra tough or unusually sharp food particles impinging on a gum margin, which is soft from lack of constant friction; and it is that which causes the damage. If the food was uniformly fibrous and hard the gum margins would have a better chance to become tough, and their epithelium to remain firmly keratinised. But even so the long clinical crowns of civilized teeth prevent the gum margins from getting regular uniform friction whether our food be soft and pulpy or fibrous and tough.

The problem therefore is to devise and teach some method of applying artificial friction to the gum margin regularly which will be as simple and as little irksome as the swallowing of vitamin pills which in their attractive red, blue and green jackets have a wonderful fascination for some people, indeed perhaps for those very people who need them least.

By "friction" one does not mean "massage". The purpose of the exer-

cise is to harden the keratinised surface layer of the gum margins so that they shall be able to resist mechanical damage from rough and hard food particles and the consequential attack of bacteria through small abrasions. This friction must be applied to the right spot, the edges of the gums and the actual point of attachment of the epithelium to the tooth at the bottom of the sulcus. In children this is accessible enough on the buccal or labial and lingual aspects of the teeth, but is almost entirely inaccessible between the teeth. Nevertheless effective use of the tooth brush in a direction from the tooth to the gum, and not from the gum to the tooth as is generally taught, will allow the bristles to enter the parodontal sulcus and increase the keratinisation of the surface layers of epithelium. Brushing in this direction will not strip the gum off the tooth as lay people seem to imagine but will render it harder and less likely to recede. Surface epithelium thrives on stimulation. The soles of the feet do not wear out if one walks barefooted.

Massage of the gum margin with the fingers would, however, seem to be useless. It is designed, we are told, to increase the blood supply to the gums; but the epithelium is completely avascular and the connective tissue when healthy is not a very vascular tissue either. It is only when it is inflamed that it becomes highly vascular. We are further told that massage is "to relieve stasis", but there is no stasis in normal tissue nor in chronic inflammatory conditions of the gum or anywhere else; it is only in acute inflammation or cardiac insufficiency that stasis occurs, and massage of an acutely inflamed part would spread the infection and do immense damage. If however, lingual and labial and buccal friction, in contradistinction to massage, were regularly maintained with a soft toothbrush for young children one might hope that the interdental papillae would not become inflamed. They might not ulcerate and split into a buccal and a lingual section with the ulcer in between as so often happens, but would remain intact and gradually shrink, until at length some kind of interdental friction, perhaps with wood-points or even darning wool, could be instituted.

Local friction directly applied to the point of onset of parodontal disease, to wit the gum margin, is therefore the obvious, first and essential exercise in prevention, yet many who never make any attempt to apply friction escape the early onset of the disease and others no less careless and often more assiduous in the care of their teeth and gums develop the disease in the most severe and progressive form.

That there are general factors concerned is reasonably certain and will be considered later, but the other local factors referred to above also play an important part. Just as the long clinical crowns of the teeth of civilized man prevent the gums from getting even such modified friction as our soft cooked food would provide, so irregularities in the arrangement of the teeth give rise to stagnation areas where no friction is ever experienced. Tartar accumulates, caries destroys the interproximal surface of the teeth themselves to which the interdental papillae of the gum should be attached; and even when these carious cavities are filled cervical edges of the fillings may very easily constitute a

serious irritant to the gum margins unless they are carefully polished.

Even that does not discharge the whole responsibility of the dentist who does the interproximal filling for if the interdental papilla has split into two parts leaving swollen buccal and lingual papillae these must be excised and the patient taught to use a woodpoint between the teeth to maintain the keratinised cuticle of the gum and clean the cervical margin of the filling. The dentist must carry out regular scaling and see that the patient keeps up his woodpoint drill.

Similarly the stagnation areas related to irregularities in the arrangement of the teeth place a very heavy responsibility on the orthodontist. Perhaps the close bite is of all factors the most prolific in setting up marginal ulceration, and any procedure likely to predispose to close-bite should be avoided at all costs. Unfortunately elaborate orthodontic treatment may itself predispose to stagnation and marginal gingivitis and in planning orthodontic work one must not lose sight of this danger. Damage may nevertheless be unavoidable and it may even be necessary to carry out a modified "gingivectomy" on a child, after orthodontic treatment, for example on the palatal aspect of upper incisors after retracting them.

That aspect of prevention, therefore, which falls under the heading of preventing the initial marginal inflammation, resolves itself into avoiding anything which might irritate the gum margins and encouraging anything which may promote their better keratinisation.

Things to avoid are inconsistencies in the diet, unpolished cervical margins of fillings, tartar, irregularities of the teeth, ill-designed orthodontic appliances, and unfilled interstitial cavities. Procedures which make for better keratinisation are, first and foremost, accurate systematic brushing of the gum margins with a soft brush, soft enough to be pleasant to use and comfortable at all times. Every part of the gum margin all round every tooth must be brushed or subjected to some kind of friction every day.

This operation is easy enough so far as the buccal and lingual gum margins are concerned. It is only necessary to divide the mouth up into six areas (the back teeth, left and right, and the front teeth, in both the upper and lower jaws) and teach the child to brush both lingually and buccally or labially (say thirty strokes in each area) to ensure adequate friction, provided that the child can be persuaded to do it. As the child grows up however, the interdental papilla becomes detached and a space develops below the contact point. Generally also an ulcer is formed just below the contact point and, by deepening, splits the interdental papilla into two — a buccal and lingual portion *). If interstitial caries intervenes this ulceration and splitting of the interdental papilla is, as we have seen, inevitable and no more salutory contribution to the prevention of parodontal disease could be imagined than that after or even before doing an interstitial filling for a young person the detached buccal and

*) Fish E. W. Parodontal Disease. Second Ed. Page 92. Fig. 18. Eyre & Spottiswood. London.

lingual papillae should be excised and wood point drill instituted. It would draw both the dentist's and the patient's attention to the importance of positive parodontal health, and keep this constantly in mind. It may be that if proper brushing of the interdental papillae were taught from the earliest age, the ulceration of the tips of the papillae at the contact point might be avoided except where interstitial caries supervened. The papillae might shrink down so that interdental friction with woodpoints might become possible without papillectomy. In this event the splitting of the papilla by an ulcer into a buccal and a lingual part might never occur, at least in many young people. These are matters which are open to experiment by those who are in charge of our preventive dentistry departments.

The other approach to establishing better keratinisation of the gum margins is by securing a more uniformly fibrous diet of a firm consistency, devoid of sharp fragments which might tear the gum margins. Apart from the disinclination of people to change their habits this is not a very hopeful approach, for the long clinical crowns of civilised man keep the gums out of the way of most of the food, and it is unlikely that we would agree to mix sand with our children's food just to produce attrition, which aesthetically amounts to actual mutilation, however desirable it may be from the point of view of securing adequate friction to the gum margins. One is however inclined to believe that there are constitutional factors not to mention orthodontic problems which would still defeat one in many cases.

Before discussing these constitutional factors however, some references must be made to masticatory trauma, which also causes marginal ulceration, and to occlusal trauma which occasions the spread of inflammation to the deeper structures. It is reasonably certain that the severity of the reactions which these forms of trauma produce are also influenced by the same or perhaps other constitutional factors.

3. Occlusal and masticatory trauma

These terms seem to be used indifferently to denote the damage caused to the parodontal tissues by masticatory stress. Such damage is of two kinds. In one case the teeth of one jaw actually impinge upon, or force food against the gum margins of the teeth in the other jaw. In the second case the impact of a tooth in one jaw against its opposite number in the other, transmits an undue proportion of the weight of the bite to that tooth, or transmits it in a direction which the second tooth is unable to tolerate without damage to its attachment.

It would be convenient to call the first masticatory trauma and the second occlusal trauma, but of course we never shall, except perhaps it may avoid confusion to do so here.

Masticatory trauma then, is seen in its most aggravated form in cases of close bite where the lower incisors impinge on the palatal gum margins of the upper ones, or even the upper inclined incisors on the labial gum margins of the lowers. The damage is obvious but the results are not uniform. In one patient the gum margin becomes scarred and notched

by the teeth and is hyper-keratinised, but remains firmly attached and is hardened to such a degree that it must be quite an efficient masticatory organ. In other patients the gum margin is red, swollen and chronically inflamed, while examination with a blunt probe shows it to be detached from the tooth, there is a deep pocket and the tooth is often drifting out of place. It is easy to see how each type of reaction occurs. In the former the degree of trauma falls within the physiological limit of tolerance and the more buffeting the tissue gets the stronger it grows. In the latter case the trauma oversteps this limit, the tissues are torn, bacteria are thrust into the deeper tissues and destroy the cells and fibres of the parodontal membrane, an abscess forms and, opening into the parodontal sulcus, produces a pocket, the bone is contaminated by the soluble toxic products of the abscess and resorbed. Thus having lost much of its attachment to the bone, the tooth moves out of place in the direction of the force applied to it by the opposing tooth.

What is not easy to see, is why the same degree of traumatic irritation should fall within the bracket of physiological tolerance in one patient, and outside it in another. We may return to this point later.

The other kind of trauma inflicted by the bite for which the term "Occlusal trauma" is here reserved, is the result of the strain imposed by one tooth on another when the muscles of mastication bring the jaws into contact. An obvious example is a case where all the upper back teeth have been removed and the lower arch remains perfect. An arch is an architectural device designed to resist force applied in a centripetal direction, but it is very ill-designed to support force applied in a centrifugal direction. The result therefore is that when the teeth close in the case cited, the lower teeth are pressed together and no harm is done to their attachment, but the upper ones are pushed outward, separated and loosened. Not only does this loosening occur as a result of mechanical pressure but periodically the strain on an individual tooth tears the attachment and, as with masticatory trauma, causes bacteria to be carried into the parodontal membrane on the palatal side of the tooth root and a parodontal abscess supervenes. This of course is followed by a deep pocket and further loosening of the tooth.

Similar but slighter degrees of occlusal trauma are said to emerge as a result of irregular arrangements of the teeth which do not fall markedly outside the normal range. These are dealt with by careful "bite analysis" and selective grinding. In a state of nature where a considerable amount of grit is often mixed with very tough food, there is no need for selective grinding, but it is certainly true that theoretically a case can be made out for it amongst civilised people. The difficulty is of course to find the high spots, and too much zeal is liable to be rewarded by a very disgruntled patient.

The particular difficulty lies in the fact that those cases of traumatic occlusion which need relief most are those with a deep overbite of the front teeth, and obviously grinding front teeth is a very unpopular procedure, but the only alternative is a parodontal splint. In practice however, it is found once more that there are the widest variations in susceptibility

between individuals and while some patients with occlusal trauma suffer severe parodontal breakdown, others, who have a most exaggerated overbite, and whose occlusion is completely locked, show no sign of parodontal damage and their teeth remain firm until they become quite old.

How far then, can we usefully go along this path in our search for a means of prevention of parodontal breakdown? Certainly orthodontic treatment is the obvious portal through which we must enter, and the rule of the road will be to preserve or restore the vertical height of the bite. In a more general sense one may say it is necessary to maintain or restore freedom of forward and lateral movement.

Fortunately for me I can disclaim any pretence to be an orthodontist, but I may say I have dabbled sufficiently to know how difficult it is to put this precept into practice. It may well be that when the orthodontist has done his best a careful bite analysis will show undue lateral trauma on individual teeth, and a little spot grinding may help, but the risk of recurrence is by no means negligable and if the bite is locked it may be better to watch the parodontal tissues carefully and if signs of pocketing or loosening of the teeth appear, to consider a parodontal splint as offering the best solution.

4. General factors and individual susceptibility

This brings me to my last point, that of individual susceptibility, whether to early marginal ulceration or to the spread of infection to the deeper tissues. Here again there may be many factors which predispose an individual to parodontal breakdown. For example endocrine disturbance is observed in pregnancy, and toxic states are exemplified by the hypertrophy observed in epanutin therapy. The leukaemias produce typical gingival lesions, and Vincent's stomatitis is a common symptom of glandular fever. There are nutritional upsets such as vitamin C deficiency which affect the gum margin.

Apart altogether from these specific causes of parodontal damage however, there is a very wide degree of constitutional variation observed in the susceptibility of the parodontal tissues to septic attack, and this may one day prove to be hereditary. On the other hand it may be nutritional. The intercellular fibres of soft connective tissue (which include the parodontal fibres) and the matrix of bone are both made of collagen which is laid down during the growth period, and is not replaced unless damaged, or contaminated. Even so if the collagen fibres, and even bone matrix are resorbed once growth has stopped, they are not usually replaced — certainly not if resorbed because of their proximity to a septic lesion such as a parodontal pocket.

On the other hand some people have chronic ulceration of the parodontal sulcus which persists for years and only very slowly do these sulci deepen. The individual variation may of course be due to an endocrine disorder or a Vitamin deficiency, but there are no concomitant symptoms which suggest this. These fortunate people may have some specific resistance to the common septic organisms and their products, but it

seems more likely that there is a structural difference; that is to say that the parodontal fibres are made of better stuff in one person than another. They are stronger, tougher and more resistant to strain. Moreover these fibres have guardian cells. The fibroblasts lie between the fibres and the cementoblasts watch over their attachment to the tooth. The more competent these cells are the less likely will it be that the fibres will be contiminated by toxic seepage from the pockets. They will be less liable to be resorbed so that the sulcus will not deepen. Similarly the bone matrix is protected by osteoblasts and osteocytes. The more efficient they are in building the matrix, the better the material they have to hand, and the better they protect it from contamination the less risk of bone resorption.

Whether the efficiency of one's connective tissue cells as collagen producers depends primarily on the Genes one has inherited or on the materials that are presented to these cells by the bloodstream while they are building fibrous tissue or bone, has not been determined.

If it were found that the presentation of some particular amino-acid in the food is all that is necessary to ensure a perfectly constructed collagenous framework for the body, it would be a simple matter to see that all adolescents had an adequate supply, and we should indeed be building better and tougher babies.

Once more, as I have said of the effect of regular friction with a soft tooth brush on the gums of children, it is a matter for experiment. Here there is room for a much more elaborate and fundamental experiment involving a close study of the mechanism by which collagen is formed in the body, and of the factors which modify its molecular structure. Such research is proceeding at last in many places, and it would be pleasing to think that dental research men were taking an appropriate part in it. At least we may hope that it is not a fixed hereditary principle which determines the pattern of these structures, but that by timely interference we may build a tougher breed.