Long in the tooth

'Long in the tooth' is a phrase often used in colloquial speech, but its origin is obscure. The *Dictionary of Idioms and their Origin* (1992) states that 'long teeth' meant that the wearer of the smile was past the first flush of youth. In further clarification it records that in 1666 the French playwright Molière was well aware of the problem since in *Le Médicin Malgré Lui* he wrote, 'the teeth have time to grow long while we wait for the death of someone'. In the same vein there is a Scottish saying 'you need not look in his mouth to know how old he is'.

'Long in the tooth,' while implying ageing, also has a clinical meaning. Kronfeld (1936) wrote 'the dental operator should always be aware of the fact that the level of the gingival margin, as he finds it in the patient's mouth, is only temporary'. He believed that as time progressed, a gradual physiological recession of the gingival border (margin) was inevitable, resulting in an exposure of additional tooth surface. At the time of Kronfeld's article terminology was less precise than at present.

Gingival recession now has a specific definition. It is the location of the gingival margin apical to the cemento-enamel-junction (*Glossary of Periodontal Terms*, 2001). The normal gingival margin position is found to be 0.5 - 2.0 mm coronal to the cemento-enamel junction (Fig. 1(a)).

Present-day terminology may also cause confusion because 'long in the tooth' implies gingival recession to our patients, but the periodontal literature uses the term 'attachment loss' instead of recession. These clinical notes are presented as a handy reference for busy clinicians to clarify matters.

The **attached gingiva** is that portion of the gingiva which is firm, dense, stippled, and tightly bound down to the underlying periostium, tooth and bone *(Glossary of Periodontal Terms*, 2001).

A Volchansky, BDS, PhD

Honorary Professor, MRC/University of the Witwatersrand Dental Research Institute and Senior Specialist Department of Orthodontics, University of the Witwatersrand
Address for correspondence: Prof A Volchansky, Private Bag 3, Wits 2050. Tel: (011) 442 6243, fax: (011) 717 2121,
e-mail: ***078avol@cosmos.wits.ac.za***

The **attachment apparatus** is formed from the cementum on the tooth root, periodontal ligament and alveolar bone (*Glossary of Periodontal Terms*, 2001) (Fig. 1(a)).

Clinical attachment level is when the epithelial attachment is below the cemento-enamel junction (CEJ) and represents the vertical distance in millimeters from the CEJ to the point of clinical periodontal attachment. This could be described as either attachment loss or the presence of probing depth. According to Carranza (1996) pocket depth or probing depth is the distance between the base of the pocket and the gingival margin. The level of attachment is the distance between the base of the pocket and a fixed point on the crown such as the CEJ (Fig. 1(b)). The *Glossary of Periodontal Terms* (2001) describes the clinical attachment level as 'the distance from the CEJ to the tip of the periodontal probe during periodontal diagnostic probing'.

Attachment loss is an indicator of periodontal disease and tells us that a destructive process has occurred. Naturally we need to consider how and why this has happened.

It is now generally accepted that periodontal disease is a slow and continually progressive condition (Burt, 1994) resulting in attachment loss. There are however bursts of activity, quiescent and repair periods. Some evidence for this is as follows.

The natural history of periodontal diseases was studied as long ago as 1978 by Loe *et al.* and in 1979 by Anerud *et al.* on Norwegian and Sri Lankan populations. These studies suggested that without interference the periodontal lesion progresses at a slow, relatively even, continuous pace. According to the authors the Norwegian subjects were well-cared-for students. Among these 50% of the 17-year-olds had no loss of periodontal support, in the other 50% there was slight localised loss of attachment primarily on the buccal surfaces of the first molars and first premolars of both jaws. At the age of 21 all the students showed one or more of these lesions as well as loss on the interproximal surfaces. This continued through to the 40-year-olds among whom mean individual loss of attachment was slightly above 1.5 mm or 10% of the total periodontal support. In contrast, the Sri Lankan population showed advanced periodontal disease when there is major loss of bone support, usually accompanied by tooth mobility and furcation involvement (American Academy of Periodontology, 1996).

Progression of periodontal disease was also studied more recently by Papapanou *et al.* (1998) using intra-oral radiographs of individuals aged 25 - 75 years. They observed that the prevalence of angular bony defects generally increased with age.

A further look at **age-related periodontal disease** was undertaken by Albander *et al.* (1999) who studied the severity of periodontal disease in adults over the age of 30 years in the USA. The percentage of persons with periodontitis (mild, moderate, or advanced forms combined) increased with rising age from 30 to 70 years, and then levelled out between 70 and 90 years. The prevalence and severity of periodontitis were higher in men than women, as well as in blacks and Mexican Americans than in whites. A conclusion that one may draw from this investigation is that the prevalence of moderate and advanced periodontitis increased with age, so this population grew 'longer in the tooth'.

Continuous attachment loss is one of the major problems confronting periodontists and their patients. This may take the form of **refractory periodontitis.** This condition is defined in *Parameters of Care of the American Academy of Periodontology* (2000) as destructive periodontal disease(s) in patients who, when longitudinally monitored, demonstrate additional attachment loss at one or more sites, despite well-executed therapeutic and patient efforts to stop the progression of the disease. These diseases may occur in situations where conventional therapy has failed to eliminate microbial reservoirs of infection, or has resulted in the emergence or super-infection of opportunistic pathogens. They may also occur as a result of complexity of unknown factors which may compromise the host's response to conventional periodontal therapy. The primary clinical feature is additional attachment loss. These obser-

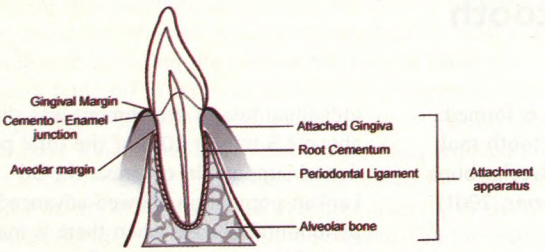


Fig. 1(a). A diagrammatic representation of the attachment apparatus showing the cementum, periodontal ligament and alveolar bone.

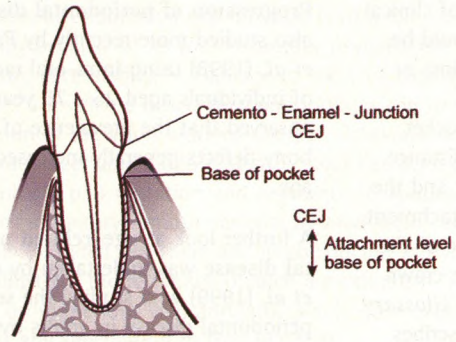


Fig. 1(b). Probing depth or pocket depth is the distance between the base of the pocket and the gingival margin. Level of attachment is the distance between the base of the

vations were also made by Colombo *et al.* (1998, 1999). In their 1999 paper they maintained that the microbial profile can contribute to distinguishing refractory subjects. In this investigation, a profile that exhibited high levels of *Streptococcus constellatus, Porphyromonas micros* and *Capnocytophaga* species accompanied by low levels of *Actinomyces naeslundii* genospecies 2 was found to be associated with the refractory state.

Refractory periodontitis probably includes multiple 'syndromes' involving distinct aetiological agents, such as the profiles mentioned above, different serum antibody responses to subgingival species, and undoubtedly differences in genetic background and smoking status. The desired outcome should include arresting or controlling the disease. However control may not be possible in all instances. In such cases, a reasonable treatment objective is to slow the progression of the disease (*Journal of Periodontology*, 2000). Should periodontal disease be left unabated, this will result in attachment loss, so these individuals may grow 'long in the tooth'.

The ageing of our population could have an impact on dental care and it is therefore essential that dental care practitioners have an understanding of the ageing process with particular reference to the psychological and physiological changes that may occur with age.

Ageing and the periodontium

The entire volume 16 of *Periodontology 2000* (1998) is devoted to periodontal therapy of the older adult. These papers are of importance to all dental practitioners as we are seeing many more elderly patients.

A comprehensive review (72 references) of epidemiology of periodontal disease among older adults was carried out by Locker *et al.* (1998). The most important clinical conclusion to be drawn from these studies concerning the effects of ageing is that increased age poses some increased risk for periodontal destruction, but the degree of destruction due to age alone is probably consistent with 'successful ageing' rather than an accelerated pathological process. Another conclusion from this paper is that although periodontal disease is time-associated, and ageing itself appears to be responsible for some attachment loss, it is of a magnitude that is unlikely to be of clinical significance.

An interesting comment by these authors was: 'Indeed, it was probably appropriate to view the ageing-periodontitis association as a rationale for redefining appropriate endpoints of periodontal therapy, such that the objective of treatment is to maintain a functioning dentition rather than a perfect level of periodontal attachment'. This could be interpreted that slightly longer in the tooth is acceptable.

In the same volume, Wennstrom (1998) summarised his paper on 'The treatment of periodontal disease in the older adult', by saying that the literature does not indicate that older adults differ from younger individuals in their response to periodontal treatment. However, in older adults, it is more common to find compromised general conditions that may negatively affect the patient's ability to maintain adequate plaque control. It has been shown that in the older adult periodontal disease development can be prevented or reduced by control of dental plaque. This is part of 'supportive periodontal therapy', an attempt to prevent the progression (attachment loss) of periodontal disease. 'Longer for as long as possible'.

How long in the tooth can one get?

Taking the observation of ageing and the periodontium a stage further, radiographic periodontal attachment loss was used by Soikkonen *et al.* (2000) as an indicator of death risk in the elderly. In the first instance three categories were cited: infrabony pockets, horizontal bone loss and furcation lesions, and then they also included apical periodontal lesions. These were termed 'potentially infectious foci'. In their results they state: 'Incidence of death did not differ in edentulous and dentate groups. Age was not significantly associated with death, neither was gender', but they go on to say 'mortality tended to be increased in subjects having 5 - 14 periodontal infrabony pockets, moderate to advanced horizontal bone loss, apical periodontal lesions, and in association with the pooled sum of all potentially infectious findings'. Before these strong statements can be generally accepted more evidence from around the world is needed.

The American Academy of Periodontology uses case types to describe degrees of severity of periodontal disease. It is a useful yardstick, not an absolute definition of each disease entity, but in the context of this paper they can be used as an indicator of disease progression.

Case type I – Gingival disease

Inflammation of the gingiva characterised clinically by changes in colour, gingival form, position, surface appearance, and presence of bleeding and/or exudate.

Case type II – Early periodontitis

Progression of the gingival inflammation into deeper periodon-

tal structures and alveolar bone crest, with slight bone loss. There is usually a slight loss of connective tissue attachment and alveolar bone.

Case type III – Moderate periodontitis

A more advanced stage of the above condition, with increased destruction of the periodontal structures and noticeable loss of bone support, possibly accompanied by an increase in tooth mobility. There may be furcation involvement in multi-rooted teeth.

Case type IV - Advanced periodontitis

Further progression of periodontitis with major loss of alveolar bone support usually accompanied by increased tooth mobility. Furcation involvement in multi-rooted teeth is likely.

Case type V - Refractory periodontitis

Includes those patients with multiple disease sites which continue to demonstrate attachment loss after appropriate therapy. These sites presumably continue to be infected by periodontal pathogens no matter how thorough or frequent the treatment provided.

Local factors

Carranza (1996) in his discussion of the aetiology of recession said that gingival recession increases with age; the incidence varies from 8% in children and 100% after the age of 50 years. This could suggest that recession may be a physiological process related to ageing. Many people believe this and that is why so many patients express the view that ageing is synonymous with their teeth looking longer, even though there is no absolute proof for it. Carranza went on to say that the gradual apical shift is most probably the result of cumulative effect of minor pathological involvement and/or direct trauma to the gingiva. He lists the following factors as being implicated in the aetiology of gingival recession: faulty tooth brushing technique, tooth malposition, friction from soft tissues, gingival inflammation and high frenal attachment. Also mentioned are labial tooth movement and the possibility of occlusal trauma. Prichard (1965) who is often quoted, placed emphasis on bone morphology, stating that a defect is related to the original anatomy of the bone and influenced by tooth anatomy and position.

Carranza (1996) dealt with bone morphology in the following way: 'bone morphology is related to tooth position, tooth shape and tooth relationships. With even moderate malalignment the bone margin may be apically placed so that the gingiva is not supported by bone'. On the same subject Manson (1975) wrote 'where a tooth or individual root is displaced out of the arch the facial or lingual plate may become very thin or even perforated so that fenestrations or dehiscences are found'.

The *Glossary of Periodontal Terms* (2001) defined dehiscence as 'a condition in which the buccal and less often the lingual aspect of the root of a tooth is without all or a portion of its bony covering'. The definition continues: 'such defects are most often associated with the roots of teeth in prominent positions in the arch'. A fenestration (fenestra) is defined as a window-like aperture or opening, often found over the roots of teeth covered with thin bone.

Dehiscences and fenestrations are therefore considered to be a possible underlying cause of attachment loss. Volchansky and Cleaton-Jones (1978) showed in a study on dried human skulls that the prevalences of dehiscences and fenestrations in the mandible occurred most frequently on the buccal aspect of the canines and then the first premolars (Fig. 2(a)). Fig. 2(b) shows dehiscences on the labial aspect of mandibular incisors.

Fig. 2(a). A dried human mandible showing the presence of fenestrations (window-like openings) on the buccal side of the canine and first premolar, and to a lesser degree on the second premolar and first molar.

Fig. 2(b). A dried human mandible showing dehiscences on the labial aspect of the incisor teeth.

Fig. 3. An adult periodontal patient who presented with advanced recession/attachment loss on the labial aspect of the mandibular incisors. Part of the aetiology could be the absence of bony support on the labial side of these teeth.

In Fig. 3 a clinical implication is seen of an adult patient seeking periodontal attention. The complaint was marked 'recession'. This condition was confined to the lower incisors. The remainder of the mouth was healthy. The reasons for this recession could be the effect of local irritation combined with an underlying bony defect and malalignment of the incisors.

While the lower incisors were not the most common sites for dehiscences these are areas of concern for the orthodontist. Fig. 4 illustrates a case of a 14-year-old undergoing orthodontic treatment with lower incisor attachment loss that may have had its origins in the underlying bony anatomy.

Malocclusion and/or oral habits could result in loss of attachment on the labial aspect of the lower incisors. These were concerns expressed by Ronnerman (1977), Larsson and Ronnerman (1981) and Ronnerman and Larsson (1982), who suggested that the position of the gingival margin as measured by clinical crown height may be a useful diagnostic measure in determining deviations from the 'normal' due to the suspected effects of malocclusion or oral habits.

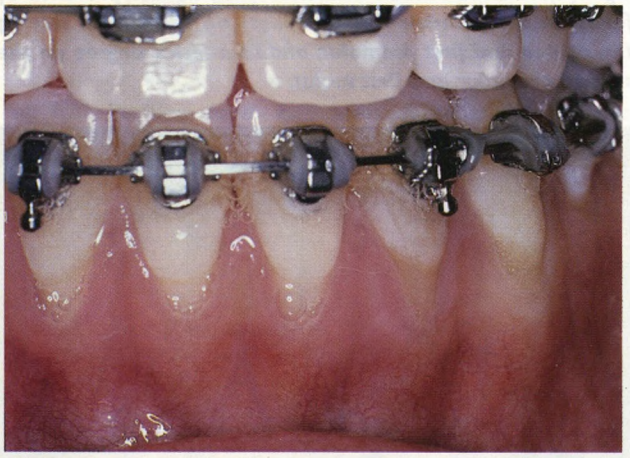


Fig. 4. Clinical photograph of a 14-year-old undergoing orthodontic treatment. The recession on the labial side of the lower incisors could present a problem in the treatment of this young patient.

Fig. 5(a). Clinical photograph of an adult thumb-sucking patient with periodontal involvement and attachment loss on the labial side of the lower incisors.

A counter view was expressed by Sabine *et al.* (1998), who found that orthodontic proclination of lower incisors in children and adolescents seemed not to result in gingival recession.

Should an oral habit such as thumb-sucking persist as seen in the adult patient in Figs 5(a) and 5(b) combined with bacterial activity a dramatic picture could result, with attachment loss and an anterior open bite.

The **position of the tooth** in the arch has also been indicted as a cause of mucogingival problems with resultant tissue loss in both the mandible and the maxilla. Fig. 6 shows a case of attachment loss/recession on the buccal side of a mandibular canine. This observation could follow on from an understanding of fenestrations and dehiscences of the alveolar bone (as seen in Fig. 2).

In the maxilla, Volchansky and Viera (1981) found that the most common sites of fenestrations and dehiscences were the buccal aspects of the first molars and then the canines. Fig. 7 illustrates these defects in the maxilla.

The underlying bony anatomy could predispose to attachment loss when trauma such as vigorous or incorrect tooth brushing

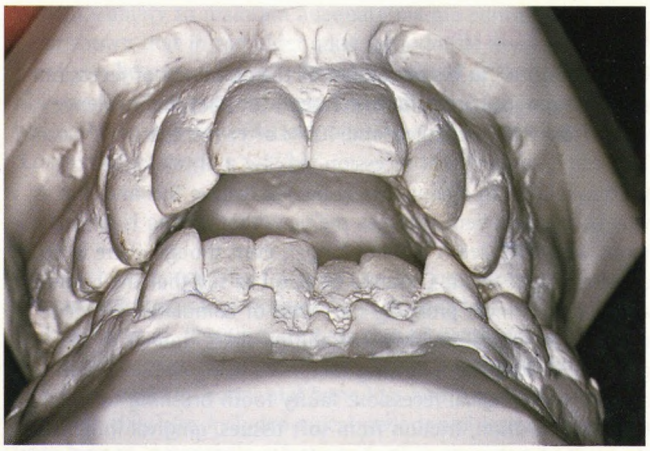


Fig. 5(b). The study models of the patient with a thumb-sucking habit showing the resultant anterior open bite.

Fig. 6. Attachment loss/recession on the buccal side of a mandibular canine which could be due to the position of the tooth in the arch and the absence of bony support.

Fig. 7. Dehiscences on the buccal aspect of the canine and maxillary first and second molars on a dried human skull.

is applied to these areas. Fig. 8 is a clinical picture of an adult periodontal patient showing attachment loss on the mesiobuccal root of the first molar and the canine.

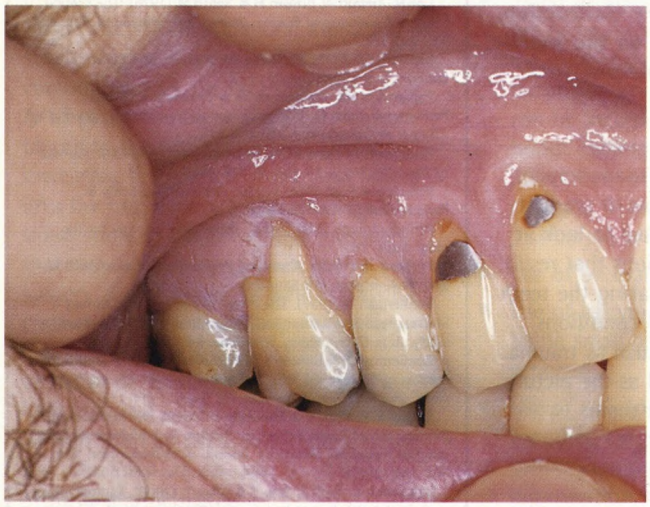


Fig. 8. Clinical photograph of a periodontal patient with recession of the mesiobuccal root of the first molar. The presence of a dehiscence could be part of the aetiology.

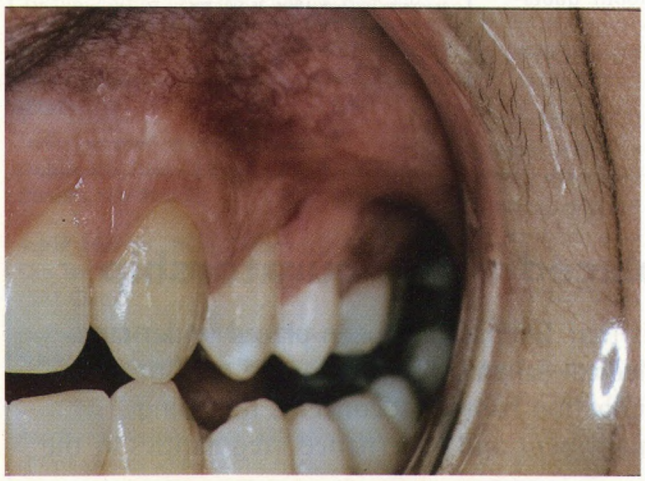


Fig. 9. Gingival clefts on the labial side of the lateral incisor and first premolar. They have been characterised as simple clefts as the cleavage occurs in one direction.

Gingival clefts may also result from incorrect tooth brushing and/or occlusal trauma, although the latter cause has not been substantiated (Carranza, 1996). He goes on to say that clefts are divided into simple clefts, as seen in Fig. 9 on the lateral incisor and the first premolar, in which cleavage occurs in a single direction (the most common type), and compound clefts, in which cleavage occurs in more than one direction.

Loss of teeth and non-replacement, particularly premolars and molars allows opposing teeth to over-erupt and in this way give the impression of recession, as the gingival margin is remote from the cemento-enamel junction. This is shown in Fig. 10, where the first molar has erupted into the mandibular molar space. The photograph also shows marked recession and 'toothbrush abrasion' on the buccal side of the canine and first premolar.

Denture trauma, in the context of persistent local irritation, may be the cause of recession (attachment loss) on the lingual aspect of the lower incisors and canines as seen in a long-standing mandibular partial denture wearer (Fig. 11).

Fig. 10. Loss and non-replacement of a maxillary molar with over-eruption of the maxillary first molar and concomitant gingival recession. The clinical photograph also shows marked recession and 'toothbrush abrasion'.

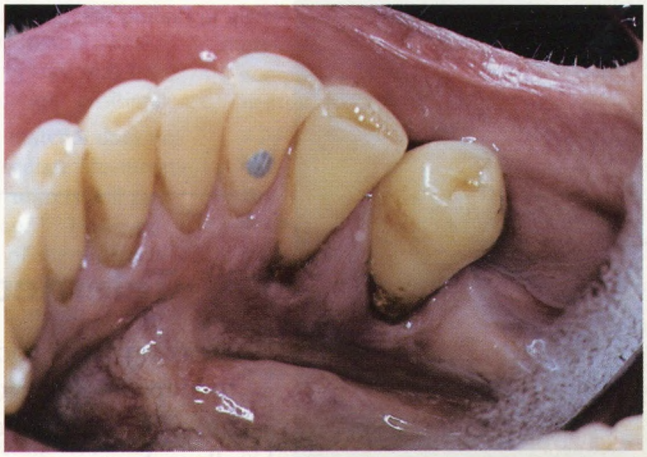
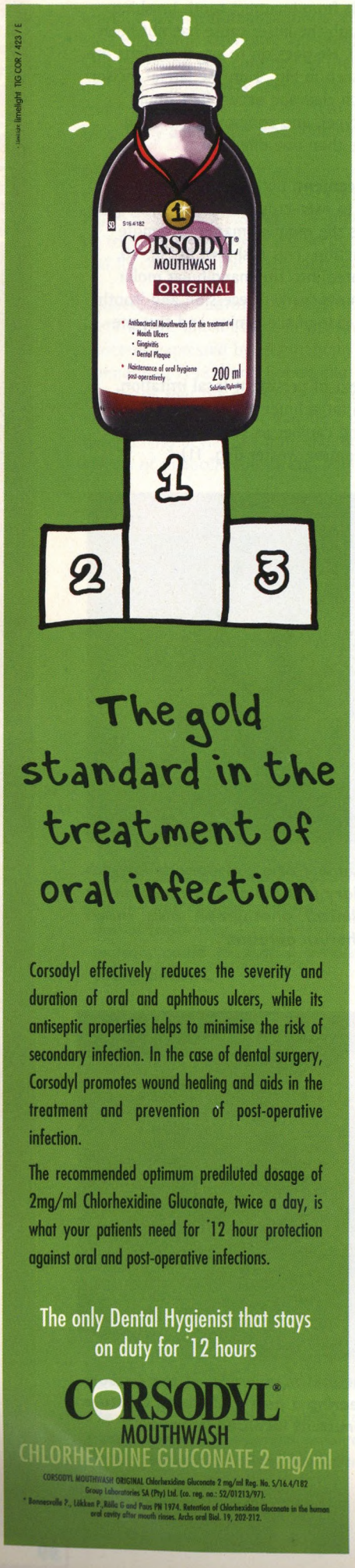


Fig. 11. Attachment loss on the lingual side of the incisors and canine in a patient wearing a partial denture. Denture trauma could be due to persistent localised irritation.



Acute and aggressive gingival and periodontal conditions such as acute necrotising ulcerative gingivitis (ANUG) or periodontitis result in the rapid loss of tissue. Starting in the papillae, the more advanced conditions involve the periodontium as well. The clinical photograph (Fig.12) shows typical papillary destruction and a more advanced lesion on the maxillary left central and lateral incisor.

Fig. 12. Clinical photograph of a post-ANUG patient, with typical papillary destruction, and a more advanced lesion on the maxillary left central and lateral incisors.

'Long in the tooth' in everyday usage implies getting older. This is often a slow or gradual process. This article has attempted to look at some of the periodontal conditions that result in attachment loss/recession, making the teeth look longer. There are also local conditions that produce attachment loss, such as trauma. These have also been illustrated as the picture would not be complete without them.

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Telemed placed under curatorship

Breaking news today is that Telemed has been placed under curatorship. Registrar of Medical Schemes Patick Masobe confirms he brought the curatorship action based on 'concerns regarding alleged serious mismanagement and governance irregularities at the scheme'.