Letters to the Editor

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LETTERS

WHAT LIES BENEATH

Sir, a 29-year-old male went to see his usual dentist one morning after discovering a large left sided facial swelling on awakening. He had a history of toothache of a throbbing nature from the lower left quadrant of his jaw which had been affecting him for the past three days.

His past dental history demonstrated him to be a regular attendee at his dental practice undergoing routine check-ups every six months. His oral hygiene was found to be remarkable on previous visits and no signs of caries or periodontal disease had previously been noted. His dental records indicated previous radiographs were last taken over four years ago.

Extra-oral examination revealed a firm, tender swelling in the pre-auricular region of the left side of his face and mouth opening was found to be limited to approximately 13 mm. Tooth 37 was found to be significantly tender on percussion. No obvious caries was noted clinically and no soft tissue swelling could be palpated intra-orally. His airway was not compromised at this time.

An orthopantomogram radiograph was taken showing a large, well defined unilocular radiolucency within the left ascending ramus of the mandible. Tooth 37 was also found to have mesial caries present with evidence of periapical pathology. This tooth further appeared to be displaced superiorly above the occlusal plane (Fig. 1).

The patient was sent to the nearest maxillofacial surgery unit where he was admitted and placed on intravenous antibiotics and analgesia.

A CT Dentscan was taken showing the location and extent of bone loss (Figs 2a-c).

Fig. 1 Orthopantomogram radiograph showing a large, well defined unilocular radiolucency within the left ascending ramus of the mandible

Figs 2a-c CT Dentscan showing the location and extent of bone loss

Extraction of tooth 37 was later carried out under general anaesthetic, as well as marsupialisation and a soft tissue biopsy of the lesion. Histopathology results revealed that of a radicular cyst. The patient has since undergone continuous decompression of the cystic cavity. He has made good post-operative progress.

In this case, more recent radiographs may have picked up signs of early caries or dental pathology. General dental practitioners should ensure appropriate radiographic recall in all patients, including those that are asymptomatic and who may clinically appear dentally fit. Such radiographs might be in the form of bitewings which can reveal early dental caries allowing for intervention to be carried out, or similarly may indicate the need for further radiographic investigations such as long cone periapicals, a full mouth orthopantomogram or CT scan.

A. Puri

Middlesbrough

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LACKING FOUNDATION

Sir, having specialised in periodontics for the last 28 years, during that time, like most periodontists, I have either been trying to create recession as the pockets heal or, rather less frequently, correct its outcome! We may be the one discipline in dentistry which welcomes recession in difficult financial times!

The paper regarding the origins and management of gingival recession, in a recent issue (*BDJ* 2011; 211: 251-254) by Patel *et al.*, provides a welcome reminder of this much misunderstood topic, but in my opinion fails to stress one fundamental point.

I have always found it useful to discuss with patients (and referring practitioners) one simple point which appears to have been overlooked in this paper to date: that recession cannot occur when the bone contours are normal. Physiologically, no amount of vigorous tooth brushing will remove the bone through the gingival tissues, yet a lack of bone is essential for recession to occur.

Many teeth simply have an altered crestal bony contour associated with either anatomical variations of tooth position, tooth width and alveolar disproportion, which, combined with the thin gingival biotype, will predispose to recession in the presence of the traumatic and inflammatory factors described. Not all teeth erupt into a 'perfect' envelope of alveolar bone; indeed orthodontic treatment to bring them into that envelope can also be associated with spontaneous improvement of localised recession.

This lack of basic foundation underwrites and determines the extent of all gingival recession and of course influences the stability of attempts to repair this surgically, although these have been strengthened in recent years by the introduction of regenerative techniques.

I have always tried to encourage students and colleagues, when considering their periodontal patients and problems, to 'look at the outside, but think of the inside'. By trying to conceive the underlying bony anatomy, the effects of the secondary factors contributing to recession become easier to understand and for the patient to appreciate.

With that in mind, one might ask your readers to consider whether recession is a sign of health or disease?

A. Woodman
Portsmouth

The authors M. Patel, P. J. N. Nixon and M. F. W-Y. Chan respond: We would like to thank the reader for his interest in our paper. Dr Woodman has highlighted a key underlying point with regards to gingival recession. It is clearly true that the aetiological factors mentioned in the article can only result in recession in the absence of normal bone structure underneath the gingival tissues. Full bony coverage of the root can be altered

or lost at healthy sites as a result of development (ie tooth size discrepancy and tooth position in the bony envelope), orthodontic movement or traumatic displacement of teeth outside the bony envelope and possibly due to other factors which are still poorly understood. Normal bony contours can also be lost in sites of disease through an inflammatory disease process resulting ultimately in bone loss.

In the absence of clinical attachment loss, the status of the bony contour at healthy sites cannot simply be observed or easily assessed clinically without surgical intervention. Therefore it is difficult to predict which patients are at risk of developing gingival recession based on the underlying bony contours. Any operator who reflects mucoperiosteal flaps will have observed sites where there are significant bony dehiscences around roots and yet the gingival margin has remained at or just above the CEJ. It should therefore be emphasised that dehiscences of the alveolus, resulting in root(s) not being covered by bone do not necessarily always lead to recession.

It is, however, known that in the presence of altered bony contours the mechanical factors mentioned in the article can result in gingival recession. If these factors can be identified early on, then preventative advice should be offered to patients.

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GLUCOSE AND SUGAR

Sir, I refer to the paper by Dr Lucy Wray, *The diabetic patient and dental treatment: an update* (BDJ 2011: 211: 209-215).

Whilst a paper on what is an increasingly prevalent disorder with oral implications is to be welcomed, it is unfortunate that the author has used the words 'sugar' and 'glucose' interchangeably throughout, implying that they refer to the same compound. Some of your readers must have found this very confusing.

Glucose is a monosaccharide and is indeed 'a sugar'. Poor hormonal control of its concentration in blood is the underlying cause of diabetes mellitus. However, the compound generally referred to as sugar is in fact a disaccharide consisting of a glucose molecule

linked to a fructose molecule. Although a dietary constituent, it does not occur in blood, being degraded to glucose and fructose in the alimentary canal before absorption. Dietary sucrose, however, is especially cariogenic because it can be metabolised by oral bacteria not only to acidic end products, but the glucose and fructose moieties can also be polymerised to form glucans and fructans which contribute to the matrix of dental plaque with the associated adverse effects on the gingivae and dentition.

It is unfortunate that the lay press still all too frequently refers to blood glucose as 'blood sugar'. This terminology probably came into use decades ago when the disorder was poorly understood and clinicians tasted the urine of diabetic patients to differentiate between diabetes mellitus and diabetes insipidus! That from the diabetes mellitus patient tasted sweet or honey like, thereby giving it its name.

The paper also implies that ketones are acidic and are the products which cause ketoacidosis. What actually happens is that when cells are deprived of glucose because of lack of insulin (some tissues require insulin for glucose uptake), fat is degraded to form a group of compounds known as 'ketone bodies'. Two of these, acetoacetate and β -hydroxybutyrate, are indeed acidic, but only the first is a ketone. The third one, acetone, is the ketone which gives the breath its characteristic smell, but it is not acidic.

Lastly, haemoglobin is not 'attached' to the erythrocytes (red blood cells), but is contained within them. Erythrocyte membranes are glucose permeable which is why the % haemoglobin A1c reflects the long term blood glucose concentration.

I hope these comments will be helpful and lead to a better understanding of this disorder.

J. A. Beeley
Glasgow

Dr Wray responds: Josie Beeley is of course perfectly correct in saying that 'blood sugar' and 'blood glucose' are different entities.

However, the words 'blood glucose' and 'blood sugar' are routinely used