We, as part of our micro-educational opportunities,[2] engage with the community by sending our Year 5 dental students and hygiene/therapy students to a variety of community settings together with qualified dental nurses. These include a homeless shelter for young adults, a drug and alcohol detoxification unit, drug and alcohol recovery services, and the Probation Service (community drop in centres). The students talk with the residents and service users disseminating oral health advice, providing oral health screening and free toothbrushes and toothpaste. Patients requiring treatment are then offered an appointment at the Dental Academy where all treatment is provided by students free of charge through our NHS primary care contract.

Although our provision of community service is much less comprehensive than that reported, as it is just one element of what we do at the Dental Academy, it does embed in the students a new dimension to their professional career. Despite reducing budgets, we have continued to provide the services due to their significant positive impact on the patients, as well as broadening the experience of our students. Last, we will point students to this excellent paper to give them greater insight into dental care for the homeless and hard to reach, to enhance their understanding in this important area of dental care provision.

D. R. Radford, G. Potts, S. Dampier, L. Davda, Portsmouth

1. Caton S, Greenhalgh F, Goodacre L. Evaluation of a community dental service for homeless and 'hard to reach' people. *Br Dent J* 2016; **220:** 67–70.
2. Radford D R, Weld J A. Micro-educational opportunities in outreach clinical dental education. *Br Dent J* 2013; **215:** 389–391.

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PERI-IMPLANT DISEASE

Not the grim reaper?

Sir, I read with interest the article on combating peri-implant disease (*BDJ* 2016; **220:** 48–49). It certainly makes for grim reading and as someone who is actively involved in implant therapy as well as assessing implant cases caught in the net of dento-legal litigation, I am only too aware of the problem.

However, I would caution your readers about the premise upon which much of the periodontal community has drawn its data, since it does not differentiate between aetiologies of peri-implant disease. The need for a classification of aetiology is long overdue since many initiating factors that can lead to peri-implantitis can be avoided and response to treatment can vary widely according to the initiating or causative factor. Recently my colleagues and I published a classification which aims to dispel much of the myth that surrounds the prevalence data, recognising as it does that true peri-implant disease, defined as being caused by the presence of biofilm, is only one such category.[1]

Few would argue that peri-implantitis caused by the presence of excess cement is the same disease process as a biofilm-induced peri-implantitis in a patient who has a genetic pre-disposition to periodontal disease, especially if they are also a smoker. However, the literature and data upon which the European Workshop on Periodontal Disease relies fails to recognise these differences. The same is true of physiologically or surgically induced peri-implantitis. This occurs when the buccal or labial bone is naturally thin (>0.5 mm) or is rendered too thin by virtue of the osteotomy preparation to be sustainable. Resorption ensues and the surface of the implant becomes exposed to the soft tissue environment. The effects of biofilm then come into play. Had there been adequate bone thickness no such vulnerability to the biofilm would be exposed.

I was also concerned by the suggestion that the work of Addison was either new or in some way indicated in the pathophysiology of peri-implantitis. It has been known for very many years that titanium corrodes in a physiological environment. A quick search on Medline will attest to numerous studies going back as far as the 1960s demonstrating this very effect[2] and many recent studies have demonstrated that titanium particles, which can be found in distant tissues including lymph nodes, lungs etc appear to be very well tolerated with little or no side effects.[3] It is a leap to suggest that such nanoparticles or corrosive by-products of titanium are a causative agent in peri-implantitis, although I await the findings of Professor Addison's five-year NIHR study with interest.

Additionally, numerous studies have identified the threshold of clinical parameters used to measure and define peri-implantitis as being very variable. Depending on the level set for these thresholds the prevalence can vary tremendously, and there remains some considerable debate as to where these thresholds lie. In short peri-implantitis is certainly a problem, but with good planning and execution, and a better understanding of the data included and thresholds set in our evaluation of this disease, we may find that it is not quite the grim reaper we all fear.

M. Norton, by email

1. Sarmiento H, Norton M R, Fiorellini J. Development of a classification system for peri-implant diseases and conditions. *J Dent Res* 2015; **94:** 3283.
2. Condon R E, Beder O E. A study of titanium implants in a corrosion-producing environment. *Surgery* 1964; **55:** 714–717.
3. Weingart D, Steinemann S, Schilli W *et al.* Titanium deposition in regional lymph nodes after insertion of titanium screw implants in maxillofacial region. *Int J Oral Maxillofac Surg* 1994; **23:** 450–452.

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ORAL CANCER

Link with early coitus

Sir, a 22-year-old mother presented to us, complaining of a sore area on the left side of her tongue, present for about two weeks. There was no other relevant medical history nor adverse oral habits. She had initially reported to a physician who suspected a local traumatic ulcer arising from an unerupted lower third molar. She was prescribed a topical steroid and chlorhexidine mouthwash. Reviewing the patient one week later with no signs of healing and progressive trismus and dysphagia, she was referred to us for further management.

Extra oral examination revealed a tender, hard, enlarged right jugulodigastric and submandibular lymph nodes. Intraoral examination demonstrated a coated tongue, a tender, indurated erosive endophytic ulcer 2 × 3 cm

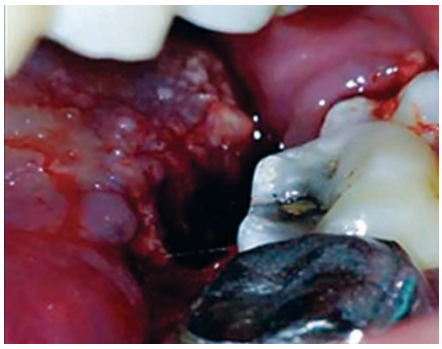

Fig. 1 A single non-healing erythematous endophytic ulcer involving the left lateral border of the tongue and floor of the mouth

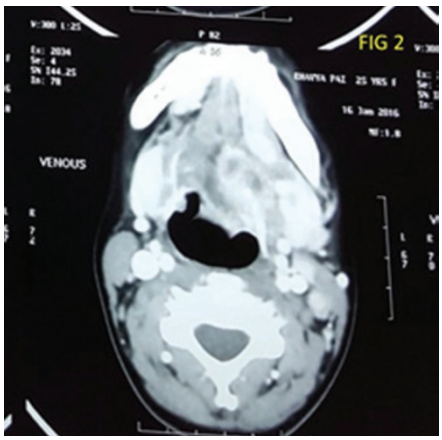

Fig. 2 Axial CECT shows a large left-side tongue base carcinoma. Note the extension approximating the midline and the ipsilateral enlarged jugulodigastric node

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