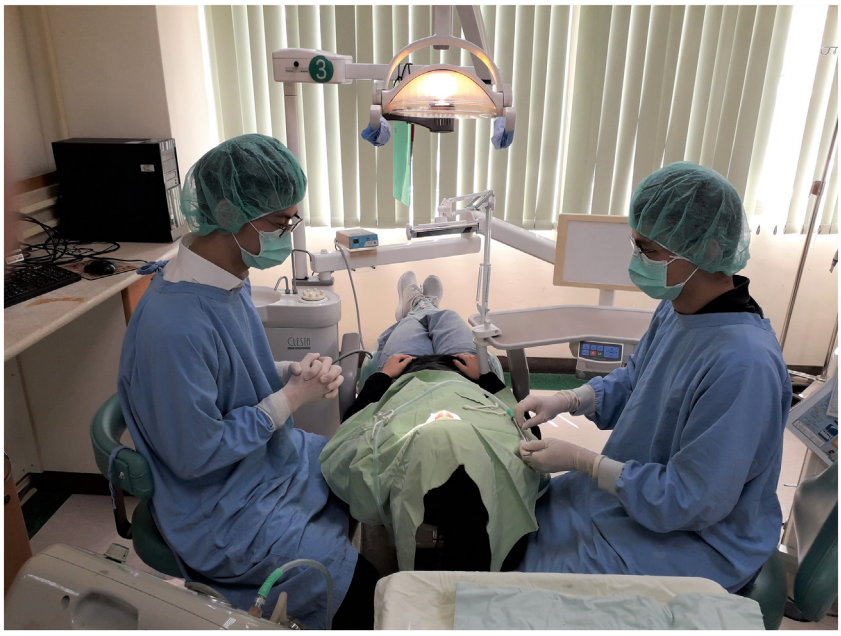

Fig. 2. The front-facing camera allows the surgeon to clearly see the area being filmed and does not interfere with the surgical process.

Oral mucosal desquamation induced by sodium lauryl sulphate

Sir,

We describe a 49-year-old white woman who presented to the oral diagnosis clinic with a 10-year history of asymptomatic desquamation of the oral mucosa. Her medical history showed Hashimoto's thyroiditis that had been treated with 50 mg of levothyroxine sodium daily for 20 years. No history of allergy or local trauma was reported. Intraoral examination showed greyish-white strips coming off the buccal mucosa. These layers came off spontaneously, or could be peeled off easily, leaving beneath a clinically normal mucosa with no erosion (Fig. 1).

Exfoliative cytology showed a group of epithelial cells and bacterial colonies, but no hyphae or yeast-like structures.

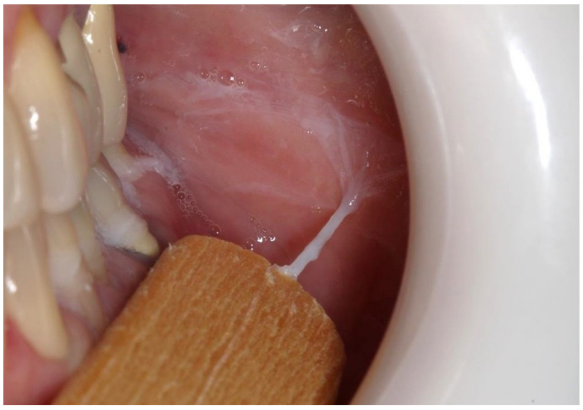

Fig. 1. Grey-white strips sloughing from the left buccal mucosa.

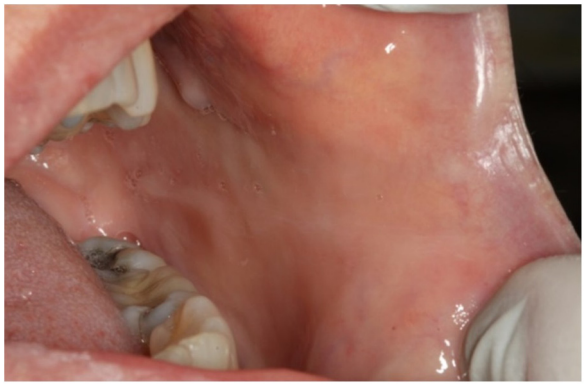

Fig. 2. Complete resolution of the peeling after oral hygiene products containing sodium lauryl sulphate was stopped.

In the absence of any other sign, we made the diagnosis of desquamation of the oral mucosa. We instructed the patient to use toothpaste and mouthwash that did not contain sodium lauryl sulphate, and after two days without it, the peeling stopped (Fig. 2). To confirm that the desquamation had been caused by the products, we asked her to use them again, and the symptoms recurred. The final diagnosis was therefore oral desquamation caused by toothpaste and mouthwash containing sodium lauryl sulphate. There was complete resolution of the peeling 2 days after discontinuation.

This condition is characterised by partial loss of the integrity of the oral epithelium, and may be caused by certain oral hygiene products, although in some cases it seems to be idiopathic.[1] Different terms have been used for it, including oral peeling, oral epitheliolysis, and shedding of the oral mucosa, but the term oral mucosal desquamation induced by oral care products seems to be the most commonly used

in recent publications.[1,2] Reactions caused by sodium lauryl sulphate have been described previously, and are related to the duration and frequency of its contact with epithelial cells in the oral mucosa.[3]

The area most commonly compromised by epitheliolysis is non-keratinised oral mucosa,[4] as was seen in our patient. The pathophysiology of the condition is not fully understood, but it is thought to be caused by irritation of the oral mucosa, which then leads to the denaturisation of the proteins in the saliva, permeability of the oral mucosa, and, eventually, damage to the integrity of the epithelial surface.[2] By the lack of any clinical signs of inflammation in the tissue (after removal of the desquamated epithelial layer), we can also rule out causes such as erosive lichen planus, autoimmune disorders with oral manifestation, and hypersensitivity reaction.[5] If the peeling is associated with mucosal erosion, a biopsy sample should be examined.[5]

Although reactions to sodium lauryl sulphate are considered to be common, its true incidence is uncertain, and rarely discussed. We have shown that, when in contact with the oral mucosa in some patients, sodium lauryl sulphate can cause scaling of the epithelium. Dentists therefore need to be aware of this possible reaction and to inform patients with such symptoms to discontinue its use.

Conflict of interest

We have no conflicts of interest.

Ethics statement/confirmation of patient's permission

Ethics approval not applicable. The authors have the consent of the patient to publish information and clinical images.

References

1. Hassona Y, Scully C. Oral mucosal peeling. *Br Dent J* 2013;**214**:374.
2. Green A, Crichard S, Ling-Mountford N, et al. A randomised clinical study comparing the effect of Steareth 30 and SLS containing toothpastes on oral epithelial integrity (desquamation). *J Dent* 2019;**80**(Suppl 1):S33–9.
3. Plonait DR, Reichart PA. E Epitheliolysis of the oral mucosa (mucosal peeling) as a side effect of toothpaste. *Mund- Kiefer- und Gesichtschirurgie* 1999;**3**:78–81 (paper in German).
4. Vannet BV, De Wever B, Adriaens E, et al. The evaluation of sodium lauryl sulphate in toothpaste on toxicity on human gingiva and mucosa: a 3D in vitro model. *Dentistry* 2015;**5**:325.
5. MacDonald JB, Tobin CA, Hurley MY, et al. Oral leukoedema with mucosal desquamation caused by toothpaste containing sodium lauryl sulfate. *Cutis* 2016;**97**:e4–5.

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Cleft and craniofacial surgery training: is it time for a new solution for UK trainees?

Sir,

I read with interest the editorial article entitled 'Cleft and craniofacial surgery – how do we inspire and recruit the next generation of OMFS trainees?'[1] I wanted to add my own thoughts to the debate.

As a young consultant the impressions garnered from multiple trainers are still fresh. Similarly, career-path decisions I faced are also recent. It is not straightforward to select a subspecialty interest from the wide variety on offer in OMFS, yet in considering cleft and craniofacial surgery a number of disincentives come to mind readily. This is despite it actually being a very appealing area of our speciality; one that I gave serious consideration to during my training.

The centralised nature of some surgical services has left many trainees exposed to a bare minimum number of cases in cleft and craniofacial, essentially sufficient for the award of CCT but perhaps not enough to foster a career-long vocation. Moreover, there remains a sizeable pool of experienced OMFS consultants with a wealth of expertise who were thwarted from practicing as specialist cleft and craniofacial surgeons due to service reconfiguration in the UK. It is hardly surprising that savvy OMFS trainees would be disinclined to be led up a garden path, left without a substantive job in which to practice their subset of skills.

The inception of the Training Interface Group (TIG) programme for all the right reasons, has fallen victim to the law of unintended consequences. In the move to match

TIG cleft fellows to substantive jobs in NHS trusts concerns about a bottle-neck effect have been exacerbated. A lack of success for OMFS trainees in TIG application processes is open knowledge via social media and other forums, with a suspicion that a less than meritocratic approach has been taken by some involved in the recruitment process. Disenchantment with the TIG process is also a significant problem in other subspecialty areas of OMFS, most notably head and neck oncology, where a number of leading units have actively withdrawn participation.

In my experience OMFS trainees are amongst the highest achievers overall and most dedicated in their training journey through necessity. It seems unlikely that they will be significantly assisted therefore by intensified interview preparation sessions, in contrast perhaps to trainees in sister specialties.

Ultimately, given that it is unlikely to prove feasible to reverse historical trends of centralisation of services, the best recourse would appear therefore to facilitate an increase of flow in to cleft and craniofacial training opportunities from OMFS. This could be achieved through reform of the current TIG process to make competitive selection more meritocratic and provide equal opportunities for candidates regardless of specialty of affiliation, as was originally intended. Alternatively, the creation of designated RCS approved fellowships in cleft and craniofacial around the UK would also serve to increase OMFS participation in this area, and consideration should be given to this idea at association level.

Conflict of interest

Not applicable.

Ethics statement/confirmation of patient's permission

Not applicable.

Reference

1. Fullarton M, Sweet C, Robertson B, Begley A, Magennis P. Cleft and craniofacial surgery — how do we inspire and recruit the next generation of oral and maxillofacial surgeons? *Br J Oral Maxillofac Surg* 2019;**57**:395–6.

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Functional compensation of a hypertrophied sublingual gland and the absence of the ipsilateral submandibular gland

Sir,

The absence of the submandibular gland and the enlargement of the ipsilateral sublingual gland has been reported previously, and may occur unilaterally or bilaterally. The finding may be incidental, as a symptom associated with reduced saliva, or an enlarged gland, or it can manifest as a mass in the neck.[1–5] Although aplasia or atrophy of the submandibular gland is thought to cause hypertrophy of other glands, including the sublingual gland, compensation of function by the glands has not been shown so far. To the best of our knowledge, 4 cases of the bilateral absence of the submandibular gland and hypertrophy of the sublingual gland have been reported to date[1–4]; one had relatively poor oral hygiene and several cavities, and the others had no symptoms of salivary gland dysfunction. A 41-year-old woman was also diagnosed with aplasia of the submandibular gland and ipsilateral hypertrophy of the sublingual gland (with symptoms such as xerostomia and dysphagia), but showed no uptake on ^{99m}Tc-pertechnetate scintigraphy in the submandibular-sublingual area.[5] We think, therefore, that functional compensation may be independent of hypertrophy of the sublingual gland.

We report a patient who presented with enlargement of the sublingual gland that appeared to compensate for the atrophied ipsilateral submandibular gland. To the best of our knowledge, no reports on this condition have been published before.

A 66-year-old man visited the otorhinolaryngology department with a 7-day history of voice change. He was had no dryness of the mouth and his oral hygiene was good. Past diagnoses included Behçet's enteritis and uveitis, and ischaemic heart disease, but there was no history of cervicofacial surgery, radiotherapy, or salivary gland-related disease.

Physical examination showed no ulceration or scarring indicative of Behçet's disease in the oral cavity. Bimanual palpation, however, indicated an absence of the right submandibular gland, and no saliva was being expelled through the papilla. Laryngitis was diagnosed on endoscopy, then after informed consent was given, computed tomography (CT), magnetic resonance imaging (MRI), and sialoscintigraphy were also done. CT showed no abnormality except the absence of the right submandibular gland, and a $3.3 \times 1.3 \times 2.9$ cm enlargement of the ipsilateral sublingual gland. T2-weighted MRI showed complete replacement of the right submandibular gland with fatty tissue, with isointense and enlarged right sublingual and left submandibular glands (Fig. 1). The maximal uptake on ^{99m}Tc-pertechnetate scintigraphy occurred 30 min after the injection in the right submandiular-sublingual region, and was reduced in comparison with the left side (Fig. 2).