COMMENT

Letters to the editor

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Priority will be given to letters less than 500 words long. Authors must sign the letter, which may be edited for reasons of space.

Anaesthesia

(DGA) deaths since *'A conscious decision'*

Sir, mortality from general anaesthesia declined greatly in the second half of the twentieth century, therefore any death from dental general anaesthesia (DGA) received even greater condemnation. This led to the publication of what was known as '*The Poswillo report*' in 1990 which recommended DGA should be avoided wherever possible and other practices utilised. DGA use declined and sedation increased; however, deaths still occurred. Therefore, in 2000 the Department of Health published '*A conscious decision*'[2] which recommended 'General anaesthesia for dental treatment should only take place in a hospital setting'. It also recommended 'better data must be obtained on fatal and non-fatal complications'.

Before '*A conscious decision*', DGA activity was recorded by the Dental Practice Board and from this data we know there were approximately 300,000 patients who underwent DGAs each year in the 1990s, with two patients dying each year. The data available to us now is taken from hospital episode statistics which don't record whether general anaesthesia was used, so estimates are made from the procedures carried out. The number of surgical extractions, simple extractions and 'excision of dental lesion of jaw' procedures done in the four nations was 221,574 in 2017;[3] however, the number of these that required GA is not known.

What effect has this change in practice had? It was the author's aim to find the number of DGA-related deaths since 2000.

Freedom of Information (FOI) requests were sent via email to each health board or trust in the UK requesting 'the number of deaths per year since 2000 from general anaesthesia for dental procedures'.

Table 1 Responses from each health board/trust in each of the four nations from the FOI requests sent

	England	Scotland	Wales	Northern Ireland	Total
Full reply of records since 2000	159	11	7	3	174
Incomplete records	19	1	0	2	21
Failed to reply	12	1	0	0	13
Unable to reply due to cost or lack of records	16	2	0	0	18
Total FOI sent	206	15	7	5	233
Number of deaths	15	0	0	0	15

There was a total of 15 deaths (see Table 1). However, even these could not be directly attributed to the DGA as the death was linked with the care episode, not a specific cause. Also the interpretation of the FOI may be subjective.

If this data is to be accepted then there has been a decrease in the number of deaths per year from DGA since the 1990s (from 2 per year to 0.88). However, given the variability in responses the study highlights the lack of knowledge about:

a) The number of DGAs carried out and
b) The morbidity and mortality rate as a result of DGA.

A universal monitoring system has been advocated previously.[4]

I. Murphy, J. Szuster and D. Richards, by email

1. Department of Health. *A conscious decision: a review of the use of general anaesthesia and conscious sedation in primary dental care*. London: Department of Health, 2000.
2. Wildsmith J A. Death in the dental chair – an avoidable catastrophe? *Br J Anaesthes* 1998; **80:** 877–877.
3. NHS. Hospital Admitted Patient Care Activity 2016-17. Available online at https://digital.nhs.uk/catalogue/PUB30098 (accessed 11 May 2018).
4. Robertson S, Chaollaí A N, Dyer T A. What do we really know about UK paediatric dental general anaesthesia services? *Br Dent J* 2012; **212:** 165–167.
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Assessment of anxiety

Sir, good measurement is the keystone to the scientific method. It is heartening to see therefore the paper by Shokouhi and Kerr in this Journal reviewing the Indicator of Sedation Need (IOSN).[1] We note under the section of 'patient anxiety' that the Corah's dental anxiety scale (CDAS) might be considered as more accurate than the current measure: modified dental anxiety scale (MDAS). The basis of Shokouhi and Kerr's argument is a review of 15 dental anxiety measures by Newton and Buck published 18 years ago.[2] We would caution substituting MDAS with CDAS based upon a review that is almost two decades old. The MDAS has now replaced the CDAS in study reports dated this century (88 vs 13 PubMed hits MDAS vs CDAS dental anxiety) and has extensive age/gender UK normative values.[3] The recommendation should therefore hold to retain the MDAS as the recognised

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assessment of dental anxiety in the IOSN. We value the attention to the often neglected subject area of psychometrics in clinical assessment and decision making. It is hoped that this note will have made a useful addition to the continuing development of the IOSN.

R. Freeman, G. Humphris, by email

1. Shokouhi B, Kerr B. A review of the indicator of sedation need (IOSN): what is it and how can it be improved? *Br Dent J* 2018; **224:** 183–186.
2. Newton J T, Buck D J. Anxiety and pain measures in dentistry: a guide to their quality and application. *J Am Dent Assoc* 2000; **131:** 1449–1457.
3. Humphris G, Crawford J, Hill K, Gilbert A, Freeman R. UK population norms for the modified dental anxiety scale with percentile calculator: adult health survey 2009 results. *BMC Oral Health* 2013; **13:** 29.

DOI: 10.1038/sj.bdj.2018.450

Oral medicine

Chronic hyperplastic candidosis

Sir, in their review of the diagnosis and management of oral candidosis,[1] Lewis and Williams raise the issue as to whether anti-fungal therapy prior to biopsy for suspected chronic hyperplastic candidosis (CHC) will enable the histopathologist to state whether any epithelial atypia which is identified microscopically is genuine dysplasia or merely a reactive change to the infection.

Unfortunately, pre-biopsy antifungals might complicate rather than simplify matters, since the histological changes in the oral mucosa caused by candidal infection may persist even after antifungal medication but without any demonstrable fungal hyphae, even with special stains. The problem in this situation is that the microscopic features of candidal infection are not specific and might prompt the pathologist to consider other diagnostic possibilities such as non-specific chronic hyperkeratosis and inflammation, migratory stomatitis/glossitis (ie geographic tongue) or rare entities such as irritant contact stomatitis, plasma cell stomatitis, Reiter's syndrome and psoriasis, a potentially misleading differential diagnosis that could confuse subsequent clinical management.

On the other hand there are occasions when, as the reporting oral pathologist, I add 're-biopsy after antifungal therapy might be helpful' at the end of the histopathology report if, in the presence of histologically-proven candidal infection, I have been unable to decide whether there is genuine epithelial dysplasia or not. Some cases of CHC resolve after anti-fungal medication and thus re-biopsy is not justifiable on clinical (or ethical) grounds. It is assumed that in such cases the epithelial atypia was indeed reactive, but the possibility also exists that anti-fungal therapy cures genuine dysplasia in some patients with CHC.

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1. Lewis M A O, Williams D W. Diagnosis and management of oral candidosis. *Br Dent J* 2017; **223:** 675–681.

The authors M. A. O. Lewis and D. W. Williams respond: We thank Dr Barrett for his comments regarding the use of pre-biopsy antifungal therapy in relation to chronic hyperplastic candidosis (CHC) and his interest in our recent paper on the management of oral candidosis.[1] CHC has a characteristic clinical presentation consisting of bilateral white patches in the commissure regions of the mouth in a patient who invariably has a smoking habit. In the majority of suspected cases, a dramatic clinical improvement occurs following the provision of fluconazole 50 mg daily for seven days, to the extent that a diagnostic biopsy is subsequently not required. The significant impact of fluconazole, both clinically and histopathologically, has previously been reported.[2] However, it is essential that the patient stops smoking, otherwise recurrence is likely.

The response to pre-biopsy antifungal therapy in this situation also removes the need for a post-antifungal therapy re-biopsy of CHC in case where there has been obvious clinical improvement. We present here two illustrations (Figs 1 and 2) of a recent patient of ours, which demonstrate the significant improvement from what clinically appeared to be a highly suspicious mucosal abnormality at initial presentation, to a far less worrying mucosal changes following fluconazole therapy. In addition, the use of pre-biopsy antifungal therapy in this patient made selection of biopsy site and the biopsy procedure itself far more straightforward. As is often the case with diagnosis of mucosal disease, a decision has to be made on an interpretation of the information available, specifically a combination of the clinical signs and symptoms supported where necessary by the findings reported by an oral pathologist. The clinical features can differ widely between patients but ultimately the clinician has the responsibility to decide on the management of an individual case and the requirement of a biopsy.

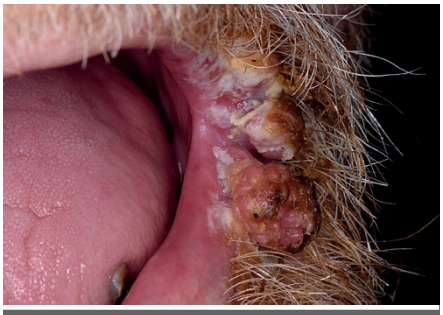

Fig. 1 Crusting at the left angle of the mouth at initial presentation

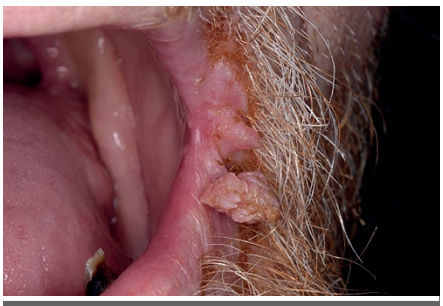

Fig. 2 Appearance following systemic antifungal therapy

In our practice, the use of pre-biopsy antifungal does have a role in investigation of the majority of patients presenting with suspected CHC. Biopsy is certainly indicated whenever there is doubt concerning the clinical diagnosis and likewise re-biopsy of a mucosa abnormality is necessary when the histopathological findings, in particular the presence of epithelial dysplasia, do not reflect the signs seen clinically. We suspect that the true relationship between the presence of candida in the tissues and dysplasia will remain unresolved.

1. Lewis M A O, Williams D W. Diagnosis and management of oral candidosis. *Br Dent J* 2017; **223:** 675–681.
2. Lamey P-J, Lewis M A O, McDonald D G. Treatment of candidal leukoplakia with fluconazole. *Br Dent J* 1989; **166:** 296–298.

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It's occlusion, stupid

Sir, after almost 40 years I continue to be confounded by my profession's inability to reconcile the connection between occlusal disease (OD)/temporomandibular disorder (TMD) and occlusion. Having had to actually *treat* patients with TMD symptoms and/or failing dentitions, in my experience I can unequivocally say that the occlusion is the primary aetiologic factor in the vast majority of cases.

So why such a discrepancy between what some of us do every day, and what 'the literature' says? I believe part of the problem is understanding the aetiology. OD/