I thank Mr Caen for comparing myself and my anaesthetic colleagues with Jenson Button. Next time I have surgeons bemoaning the fact that I am taking too long with an anaesthetic I will refer them to his remarks!

Useful resources

1. Standing Dental Advisory Committee. *Conscious sedation in the provision of dental care. Report of an Expert Group on Sedation for Dentistry*, 2003. Available at: http://www.advisorybodies.doh.gov.uk/sdac/conscious_sedationdec03.pdf
2. Standing Committee on Sedation for Dentistry. *Standards for conscious sedation in dentistry: alternative techniques*. London: Royal College of Surgeons of England, 2007. (Guideline Ref ID 15950) Available at: http://www.rcseng.ac.uk/fds/publications-clinical-guidelines/docs/SCSDAT%202007.pdf
3. National Institute for Health and Clinical Excellence (NICE). *Sedation in children and young people* (CG112). December 2010. Available at: http://guidance.nice.org.uk/CG112
4. British Dental Association. *BDA advice. Conscious sedation*. November 2011. Available at: http://www.bda.org/Images/conscious_sedation_-_nov_11.pdf

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GLAD TO BE RETIRING

Sir, I read Dr Dobson's letter (*BDJ* 2012; **212:** 206) with interest and with resignation. I think he is probably saying nothing new. The medical and dental non-consultant anaesthetists providing dental general anaesthesia (GA) in 1998 would agree, I am sure, that this was less stressful than sedation. I personally found intubation anaesthesia was the least stressful. I would agree that the GDC obsession with GA being dangerous and therefore banning it was and still is irrational and I am certain it was a politically motivated decision.

The GDC of course, able to dispense with the tedium of evidence and ignore the patient and their need for properly trained professionals who think pain and anxiety/physiology control matter during dental treatment, stopped GA in the dental surgery in 1998. Their concern probably being the then increasing numbers of deaths in clinics run by a particular doctor, who has subsequently died. They apparently ignored the evidence that trained anaesthetists had very low death rates (calculated to be approx 1:750,000 for the dentally qualified by Brett and Jack[1]). In all the time I had access to coroners' reports relating to dental GA deaths, all except one were probably preventable where advice and recommendations (usually regarding monitors) were ignored. The one death I would have said was unavoidable back then was an odd allergic response but even so a prominent anaesthetist of the day said an aggressive approach could have worked. I know anaesthetic colleagues were horrified that a BDS was enough of a qualification to perform GA and that no postgraduate training existed after the Wylie and Seward Hospital training SHO posts stopped.

Once again the anaesthetic community observe dentists now using sedative drugs, in most cases expertly I'm certain, where there is no mandatory training, registrable qualification or postgraduate specialty and audit, despite years of discussion and astonishingly deaf ears, and are expressing concern. I don't know where to place the blame for dentistry's shameful lack of interest in this aspect of patient care. Is it not time for the GDC to be replaced or enhanced with a competent body that places patients' needs above all, sticks to evidence-based decisions and organises/enables postgraduate training in anaesthesia and all aspects of it including local and sedation the way doctors and vets do?

Incidentally, I can understand the GDC attitude of the day. I rang the Royal College of Anaesthetists in 1998 to ask what its view was regarding the competence of dental 'non-consultant' anaesthetists. I was told by a very senior member, very aggressively amongst other derogatory comments, that '*we don't need dentists anymore*'. What a pity. It would seem that the UK BDS is heading toward a technical rather than clinical qualification. I hope I am wrong but I am glad to be retiring.

W. A. D. Jack
By email

1. Brett I I, Jack W A. Deaths associated with dentistry. *Anaesthesia* 1993; **48:** 1102-1103.

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IN COMPLETE AGREEMENT

Sir, in my career as a Consultant in Special Care Dentistry I was privileged to work in a hospital environment with highly skilled anaesthetists. They gave me the ideal operating conditions of a paralysed patient with a cuffed nasal endotracheal tube and I gave them

liberal local analgesia so that they could reduce the dose of parenteral analgesics to a minimum. The patients were mostly at the upper end of the learning/physical/medical/psychological spectrum of disability. Many were ASA III and a memorable few ASA IV. The steady advance in pharmacology, techniques and monitoring paved the way for total day case lists. Patients had recovered sufficiently at the end of the day to be safely discharged to their own home environment even after an operation involving oral surgery and full mouth restorations lasting two hours. This was highly cost effective. The invention of the laryngeal mask airway reduced the battle between operator and anaesthetist in paediatric exodontia. So, I find myself in complete agreement with Dobson[1] that general anaesthesia is safe and sedation fraught with problems.

Studies of the electroencephalogram (EEG) in hypnosis and nitrous oxide sedation showed the same easily recognisable pattern of a dominant alpha rhythm and suppression of the lower (<4 Hz) frequencies. I came to the conclusion that nitrous oxide sedation should be classified as chemically assisted hypnosis. We tested new anaesthetic agents as they became available and observed a similar phase of sedation before onset of general anaesthesia which was characterised by loss of the alpha and emergence of dominant delta rhythms.[2] However, the dose-response curve of some agents, notably sevoflurane and propofol, is so steep that it is difficult to envisage titrating the patient to a stable sedation state. This has been tragically illustrated in the case of Michael Jackson, and the very high incidence of adverse airway events observed in the USA. Should the patient lose consciousness, however fleetingly, a different set of rules apply. Hypoxia is the enemy; a lot can happen in a short time and the PaO_2 can run away precipitously down the oxyhaemoglobin dissociation curve. As Haldane remarked, hypoxia not only stops the clock but can wreck the clockwork.

We did not find easily identifiable changes to the EEG in sedation with benzodiazepines. There is no simple way to monitor 'depth' of sedation using these drugs. How frequently should response to verbal commands be tested? Continuous monitoring of auditory evoked potentials is a possibility but few monitors are commercially available or affordable. Individuals vary considerably in their response to drugs so a single bolus dose of midazolam, however skilfully administered, may easily overshoot or undershoot the desired level of sedation, and in any case the time available for dental work is severely constrained.

The situation is not helped by the definitions of sedation. The GDC states that it is 'depression of the central nervous system'. Does that mean all of the CNS? Cardiovascular and respiratory centres? Sensory and motor cortices? The American Society of Anesthesiologists calls it 'drug-induced depression of consciousness' and goes on to say, worryingly, that in moderate sedation spontaneous ventilation is 'adequate' and cardiovascular function is 'usually' maintained.

I do not deny that there is a role for sedation in the spectrum of need, but with defective definitions, lack of specific monitoring, absence of audit and risk of hypoxia I would rather have a GA than intravenous sedation.

M. Griffiths
By email

1. Dobson A P. A contrary view. *Br Dent J* 2012; **212:** 206.
2. Griffiths M J, Preece A W, Green J L. Monitoring sedation levels by EEG spectral analysis. *Anesth Prog* 1991; **38:** 227-231.

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CONCENTRATE ON RESEARCH

Sir, I enjoyed reading Dr Islam's witty and insightful 'opinion paper' (*BDJ* 2012; **212:** 163-164) on the failings of modern undergraduate dental education. There is much truth in his contention that there has been a worrying decline in the clinical experience of our undergraduate dental students.

I have been an undergraduate teacher for 30 years and I am increasingly concerned by the lack of clinical exposure of more recent dental graduates. In my view, this is at least partly due to the effect the RAE (and its successor the REF) has had upon shaping the goals and efforts of university dental academic staff.[1,2] Certainly the easiest way to obtain promotion, as rapidly as possible, is to ignore teaching and clinical care as much as you can get away with, and concentrate on research.

It is time that the decline in the clinical exposure of undergraduate dental students was reversed. Certainly we should not follow the medical model, where it appears that medical students are able to graduate, yet be incapable of carrying out many basic clinical procedures expected of a doctor.

J. R. Drummond
Dundee

1. Drummond J R. Presidential address. *Br Dent J* 2009; **206:** 557-559.
2. Royal College of Surgeons of England. Balancing teaching and research in dental clinical academic life. *Faculty Dent J* 2010; **1:** 71-72.

DOI: 10.1038/sj.bdj.2012.398

SEIZURE STIMULI

Sir, the recent article by Lisowska and Daly on vagus nerve stimulation therapy (VNST) (*BDJ* 2012; **212:** 69-72) mentions pre-seizure auras being recognised in visual, auditory or olfactory systems.

This set me thinking because I have recently been asked to give orthodontic advice for a child who uses VNST, and who seems to experience his epileptic auras in the upper incisors.

Is the type of aura linked to the type of seizure inducing stimulus which an individual is sensitive to? For example are flashing lights more likely to cause fits in epileptic patients who have visual auras than other types of aura?

Might there be a risk that by applying a stimulus such as orthodontic forces to my patient's upper teeth, a seizure could be provoked?

M. Anderssohn
By email

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