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 Dobson¹ 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 PaO₂ 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

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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.

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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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