LETTERS TO THE EDITOR

Send your letters to the Editor, British Dental Journal, 64 Wimpole Street, London, W1G 8YS
Email bdj@bda.org. Priority will be given to letters less than 500 words long. Authors must sign the letter, which may be edited for reasons of space. Readers may now comment on letters via the *BDJ* website (www.bdj.co.uk). A 'Readers' Comments' section appears at the end of the full text of each letter online.

MEDICAL EMERGENCIES

Auto-injector confusion

Sir, over a number of years, whilst training dental professionals in the management of medical emergencies, we have become aware of concern and some confusion around the use of auto-injectors to administer epinephrine as an intra-muscular injection to patients suffering from acute anaphylaxis. This has mainly centred on the fact that an auto-injector which delivered the epinephrine dose recommended by the Resuscitation Council[1] was difficult to obtain.

We felt that your readers may be interested in the product Emerade which is new to the market and satisfies resuscitation guidance not only on epinephrine dose but also the needle length of auto-injectors used in the management of anaphylaxis.

C. Bryant, G. Umar
London

1. Resuscitation Council (UK). *Emergency treatment of anaphylactic reactions. Guidelines for health-care providers.* Working group of the Resuscitation Council (UK). January 2008, annotated with links to NICE guidance July 2012. Reviewed 2013.

DOI: 10.1038/sj.bdj.2014.600

ORAL HEALTH

Bamboo salt

Sir, bamboo salt has been used in Korean medicine for centuries as a folk medicine for the prevention and treatment of various diseases. Studies have shown bamboo salt to have anti-cancer, anti-oxidant, anti-inflammatory and anti-microbial effects.[1]

Bamboo salt (also called as Jookyeom) is specially processed according to a traditional recipe using normal salt, bamboo, pine tree wood, pine resin, and yellow soil, combined at very high temperature.[1] Sea salt is put into cases made from bamboo trunks with three years of growth and the ends are plugged with natural yellow clay. The assembly is roasted one or more times to make bamboo salt. The main ingredient of bamboo salt is sodium chloride salt. It is believed that the trace elements in the mud and bamboo make this form of salt healthier.[2]

Fig. 1 Bamboo salt toothpaste

Bamboo salt has been added to toothpaste and according to the manufacturer this toothpaste can prevent cavities, reduce plaque and gingivitis, soothe sensitive teeth, fight bad breath, whiten teeth, strengthen tooth enamel, prevent receding gum line and decrease mineral loss. Numerous bamboo salt toothpastes are available in Japan, Hong Kong, Korea and worldwide through the Internet (Fig. 1).

Choi *et al.* conducted a study to evaluate the laboratory remineralisation effects of a dentifrice with bamboo salt and NaF on artificial caries-like enamel lesions, at both the surface and deep areas. The authors concluded that there was a significant increase in the level of the surface hardness and decreased mineral loss of the artificial caries-like enamel lesions. The test dentifrice also significantly decreased the lesion depth.[3] Sohn *et al.* showed that when bamboo salt is used in dentifrices, it had an anti-plaque and anti-inflammatory effect.[4]

Over the years, dentistry has evolved from a practice based on folk cures to a structured medical discipline that relies on science and technology. Dentistry today is based on scientific evidence. More research and scientific data are needed before dentists can advise the use of dentifrices like the bamboo salt dentifrice for oral use. Dentists and the general public should be cautious before using products which have not been proven scientifically.

Preena Sidhu, Sathya Kannan
Senthilkumar Muthusamy, Kavitha Muthu
SEGi University, Malaysia

1. Moon J H, Shin H A, Rha Y A, Om A S. The intrinsic antimicrobial activity of bamboo salt against *Salmonella enteritidis*. *Mol Cell Toxicol* 2009; **5:** 323–327.
2. Hyun Jun Ju, Hong Jin Bae, Young Tai Shin. Severe hypernatremia by excessive bamboo salt ingestion in healthy young woman. *Electrolyte Blood Press* 2013; **11:** 53–55.
3. Choi C H, Ha M O, Youn H J *et al.* Effect of bamboo salt-NaF dentifrice on enamel remineralization. *Am J Dent* 2012; **25:** 9–12.
4. Kim C Y, Chung S C, Sohn W S. Comparison of the antiplaque and anti-inflammatory effect of the dentifrices containing NaCl and bamboo salt. *J Korean Acad Dent Health* 1991; **15:** 269–280.

DOI: 10.1038/sj.bdj.2014.601

PHARMACOLOGY

A new bleeding issue

Sir, drugs producing a bleeding tendency include vitamin K antagonists (VKAs such as warfarin), newer oral anticoagulants (NOACs such as dabigatran), heparins and anti-platelet agents such as aspirin or clopidogrel. All may cause post-operative bleeding although, for minor dental surgery, drug dose modifications are rarely needed. Current oral anti-platelet therapies, used for the management and prevention of ischaemic events (acute coronary events, stroke, peripheral vascular disease) are based mainly on the inhibition of two pathways of platelet activation namely thromboxane A2 (blocked by aspirin) or adenosine diphosphate (ADP)-P2Y12 receptors (blocked by clopidogrel, prasugrel, and ticagrelor). Despite these therapies, patients who have experienced atherothrombosis (eg myocardial infarction) remain at risk for recurrent ischaemic episodes.[1] Therefore, a new class of platelet antagonists targeting thrombin-mediated platelet activation via protease-activated receptors (PAR) have been developed to address these issues.[2] The first drug in this group, vorapaxar, a PAR1 antagonist, has just been approved by the FDA[3] for the reduction of thrombotic cardiovascular events in patients with a history of myocardial infarct and peripheral arterial disease but not in those with a history of stroke or transient ischaemic attack (due to the increased risk of intracranial haemorrhage observed in clinical trials in these two groups).[4–6]

Vorapaxar is rapidly absorbed after oral administration with onset of activity at two hours. It has a long-half-life of

UNRELIABLE DATA

Sir, the study by Elmer *et al* (*BDJ* 2014; **216:** E10) claims promise for a new way to assess the effectiveness of fluoridation. However, it relies on data of questionable validity from the Hospital Episode Statistics system (HES).

Other authors found HES data unreliable, missing as many as 80% of actual general anaesthesia hospitalisations (GAs).[1-2] Many practitioners did not even report to the HES system. Since Elmer compared just two health regions, and the number of practitioners doing hospital extractions is small, there is a high risk of bias from regional differences in reporting rates.

Referral practices may also vary by region and can be strongly influenced by health policies.[3] Modest referral differences can produce large differences in GA rates since GAs are a small fraction of all extractions.

Regional differences in dental care are another potentially confounding factor. The dental care index (ft/dmft) was 35% higher in Elmer's fluoridated West Midlands region than his comparison unfluoridated region North West, suggesting a better level of care in the fluoridated region.[4] This contrast was amongst the most extreme in England, and was significant (p = 0.002, based on sub-region variance, 2-tailed t-test).

These problems also apply to a recent PHE fluoridation monitoring report that relied on HES data.[5]

Elmer says evidence on fluoridation from randomised controlled trials (RCTs) remains 'understandably absent' because RCTs are 'very challenging'. While a community-level fluoridation RCT would be difficult, an individual-level RCT using coded bottled water would not. Pharmaceutical companies do thousands every year. A few RCTs could settle the question of fluoridation's effectiveness. As the authors of the York Review said, existing evidence is 'poor quality' and '... only high-quality studies can fill in the gaps ... Recourse to other evidence of a similar or lower level than that included in the York review..., no matter how copious, cannot do this.'[6]

C. Neurath, S. Peckham, H. Limeback, H. S. Micklem, W. Osmunson, by email

1. Ní Chaollaí A, Robertson S, Dyer T A, Balmer R C, Fayle S A. An evaluation of paediatric dental general anaesthesia in Yorkshire and the Humber. *Br Dent J* 2010; **209:** E20.
2. Robertson S, Ní Chaollaí A, Dyer T A. What do we really know about UK paediatric dental general anaesthesia services? [editorial]. *Br Dent J* 2012; 212: 165-167.
3. UK Department of Health. *A conscious decision: a review of the use of general anaesthesia and conscious sedation in primary dental care.* London: UK Department of Health, 2000.
4. NHS Dental Epidemiology Programme. *NHS Dental Epidemiology Programme for England oral health survey of 5-year-old children 2007/2008.* Public Health England, 2009.
5. Public Health England. *Water fluoridation: health monitoring report for England 2014.* London: Public Health England, 2014.
6. Centre for Reviews and Dissemination, University of York. *What the 'York Review' on the fluoridation of drinking water really found.* York: Centre for Reviews and Dissemination, University of York, 2003.

DOI: 10.1038/sj.bdj.2014.603

up to 311 hours. Although considered a reversible platelet antagonist, because of its slow dissociation from PAR-1, it may not be practically functionally reversible (after a single loading dose of vorapaxar, platelet function is restored [>50% of baseline] within 2-3 weeks). Vorapaxar is extensively metabolised in the liver by cytochrome P450 (CYP) 3A4. Thus administration of inhibitors of CYP 3A4 activity (eg azole antifungals such as miconazole, and macrolide antibiotics such as erythromycin) could potentially affect its antiplatelet effects. The route of drug elimination is mainly faecal and to a minor extent renal (<5%).[7]

Dental professionals should be aware of this drug. Until more clinical data become available, management principles for dental patients on this new platelet antagonist should follow existing guidelines available for other anti-platelet drugs, with consultation with the prescriber, avoiding drug interactions, avoidance of interruption of the anti-platelet drug regime, using local measures to control bleeding and ensuring stability of the underlying cardiovascular and other co-morbidities particularly before proceeding with any form of invasive dental treatment.[1]

A. N. Robinson, Singapore
C. Scully, London

1. Scully C. *Scully's medical problems in dentistry.* 7th ed. Churchill Livingstone, Elsevier, 2014.
2. Brito F D S, Tricoci P. Novel anti-platelet agents: focus on thrombin receptor antagonists. *J Cardiovasc Transl Res* 2013; **6:** 415–424.
3. FDA News release, 8 May 2014. FDA approves Zontivity to reduce the risk of heart attacks and stroke in high-risk patients. Available at: http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm396585.htm (accessed 19 May 2014)
4. Morrow D A, Braunwald E, Bonaca M P *et al.* Vorapaxar in the secondary prevention of

atherothrombotic events. *N Engl J Med* 2012; **366:** 1404–1413.
5. Tricoci P, Huang Z, Held C *et al.* Thrombin-receptor antagonist vorapaxar in acute coronary syndromes. *N Engl J Med* 2012; **366:** 20–33.
6. Morrow D A, Alberts M J, Mohr J P *et al.* Efficacy and safety of vorapaxar in patients with prior ischemic stroke. *Stroke* 2013; **44:** 691–698.
7. Kosoglou T, Reyderman L, Tiessen R G *et al.* Pharmacodynamics and pharmacokinetics of the novel PAR-1 antagonist vorapaxar (formerly SCH 530348) in healthy subjects. *Eur J Clin Pharmacol* 2012; **68:** 249–258.

DOI: 10.1038/sj.bdj.2014.602

VIEW FROM MY WINDOW

Venerable views

Sir, I was interested to see your article about 'view from my window' (*BDJ* 2014; **216:** 549) and a request that people submit their own photographs showing the views from their windows.

I remember a similar series of articles and photographs published (I think in *Dental Update*) in the mid-late 1980s – certainly during the years when my son (born 1981) was young. I remember that the clear winner was a practice that could boast a beautiful view of the beach in Jersey from their practice window.

This created a backlash from practices with awful views.

I remember that I considered submitting a photograph taken from the window of my mobile clinic which was parked in the grounds of a secondary school adjacent to a building shrouded in plastic that was being stripped of asbestos by men in hazard suits. However, I didn't do so and even if I had I would have doubtless still have lost out to the eventual winner who could boast the back entrance to the local VD clinic in an old Victorian building as his winning view!

I look forward to seeing the results of your current 'competition'.

J. Papworth
By email

Send the view from your workplace window to k.quinlan@nature.com.

DOI: 10.1038/sj.bdj.2014.604

FACIAL BURNS

Acid drops

Sir, a patient who sustained a facial burn during a routine bond-up procedure in which 37% phosphoric acid etch was used (Fig. 1) is a timely reminder of the potential hazard of acid burns to the skin during procedures using dental etching. A number of materials can be used for etching, but the most common approach is to use 30-40% phosphoric acid in the process of 'total etching'.

At present there are no formal guidelines on precautions, however, these

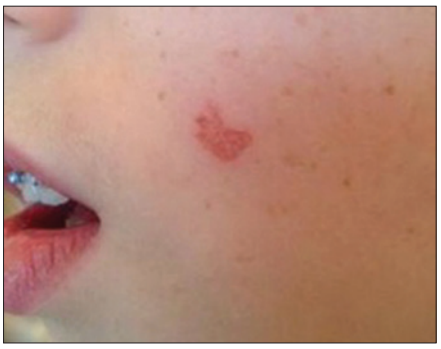

Fig. 1 Patient who sustained a facial burn during a routine bond-up procedure

can include using etch in the form of a coloured gel to allow for identification and prevent unwanted spreading of acid. Furthermore, etch should only be left in contact with the tooth for 30-40 seconds and then be removed using cotton wool followed by high speed aspiration. During the procedure, surrounding teeth can be protected using cotton wool rolls or a rubber dam and the patient should wear protective glasses and a bib. The application brush used to apply etch should not be overloaded and a direct application syringe can be used rather than transferring etch into a Dappens pot and then to the tooth.

Clinical features suggestive of a burn following the use of etch are the development of an area of erythema or an area of intense pain. If a burn is suspected then the most important intervention is to thoroughly irrigate the area using running water, taking care not to spread the acid from the affected area. Irrigation should be continued until such a point that the patient no longer feels pain in the affected area. If the acid contamination involves the eye then the eye should also be immediately irrigated very thoroughly.

Further treatment will depend upon the depth and extent of the burn but all patients sustaining facial burns should attend their local hospital, usually via Accident & Emergency.

J. E. Steele, K. Parker, J. L. Atkins, D. S. Gill
By email

DOI: 10.1038/sj.bdj.2014.605

SYSTEMIC DISEASES

Periodontitis and Alzheimer's

Sir, I read with great interest the recent article *Chronic non-communicable diseases* (*BDJ* 2014; **216:** 487) and the evidence for periodontitis and its relationship with systemic conditions.[1] Preliminary results suggest that periodontitis contributes to cognitive impairment and people with poor oral hygiene and periodontitis may be at a greater risk of developing Alzheimer's disease (AD).[2] The data supporting this association are, however, still limited.[3,4]

In March 2014 US National Institutes of Health founded prospective research in Germany (NCT02109705) which may explain the role of periodontal disease and especially the role of periopathogen *Porphyromonas gingivalis* in the initiation and progression of Alzheimer`s disease by determination of correlations between selected AD indicators (beta-amyloid protein) and periodontal disease determinants (enzyme activity, inflammatory mediators).[5]

Laboratory research conducted post-mortem has demonstrated the presence of products from bacteria *Porphyromonas gingivalis* in brains from patients suffering from dementia[6] while other research suggests that several types of spirochetes, including periodontal pathogens, may be involved in the pathogenesis of AD with a probable causal relationship.[7]

Plausible biological mechanisms linking periodontitis and AD may include three possible routes: a) direct effects of oral pathogens; b) inflammatory response to perio pathogens; and c) the influence on vascular integrity. Immune responses of the brain tissue exposed to certain periodontopathic bacteria and/or their endotoxins may hypothetically lead to nerve cell damage.[8] It is important to determine whether degenerative processes in the brain are initiated by inflammatory reactions as a result of periodontitis.

A. Dziedzic
By email

1. Chapple I L P, Wilson N. Chronic non-communicable diseases. *Br Dent J* 2014; **216:** 487.
2. Kamer A R, Craig R G, Dasanayake A P, Brys M, Glodzik-Sobanska L, de Leon M J. Inflammation and Alzheimer's disease: possible role of periodontal diseases. *Alzheimers Dement* 2008; **4:** 242–250.
3. Shaik M M, Ahmad S, Gan S H *et al.* How do priodontal infections affect the progression of type 2 diabetes and Alzheimer's disease? *CNS Neurol Disord Drug Targets* 2013; **13:** 460–466.
4. Sparks Stein P Steffen M J, Smith C *et al.* Serum antibodies to periodontal pathogens are a risk factor for Alzheimer's disease. *Alzheimers Dement* 2012; **8:** 196–203.
5. Porphyromonas Gingivalis and Alzheimer's Disease (PGNEURO). ClinicalTrials.gov. Onine information available at http://clinicaltrials.gov/show/NCT02109705 (accessed July 2014).
6. Poole S, Singhrao S K, Kesavalu L, Curtis M A, Crean S. Determining the presence of periodontopathic virulence factors in short-term postmortem Alzheimer's disease brain tissue. *J Alzheimers Dis* 2013; **36:** 665–677.
7. Miklossy J. Alzheimer's disease – a neurospirochetosis. Analysis of the evidence following Koch's and Hill's criteria. *J Neuroinflamm* 2011; **8:** 90.
8. Watts A, Crimmins E M, Gatz M. Inflammation as a potential mediator for the association between periodontal disease and Alzheimer's disease. *Neuropsychiatr Dis Treat* 2008; **4:** 865–876.

DOI: 10.1038/sj.bdj.2014.606