<u>LETTERS</u>

atherothrombotic events. *N Engl J Med* 2012; **366:** 1404–1413.

- Tricoci P, Huang Z, Held C et al. Thrombin-receptor antagonist vorapaxar in acute coronary syndromes. N Engl J Med 2012; 366: 20–33.
- 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.
- 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 (betaamyloid protein) and periodontal disease determinants (enzyme activity, inflammatory mediators).⁵

Laboratory research conducted postmortem has demonstrated the presence of products from bacteria *Porphyromonas gingivalis* in brains from patients suffering from dementia⁶ 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-communica-
- ble diseases. Br Dent J 2014; 216: 487.
 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.
- 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.
- 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.
- Porphyromonas Gingivalis and Alzheimer's Disease (PGNEURO). ClinicalTrials.gov. Onine information available at http://clinicaltrials.gov/show/ NCT02109705 (accessed July 2014).
- 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.
- Miklossy J. Alzheimer's disease a neurospirochetosis. Analysis of the evidence following Koch's and Hill's criteria. J Neuroinflamm 2011; 8: 90.
- 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