LETTERS TO THE EDITOR

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ORTHODONTICS

Treating avulsed permanent teeth

Sir, I read with interest the research summary in September's edition of the *BDJ* entitled 'Primary care dentists' experience of treating avulsed permanent teeth'[1] and also the research paper itself.[2]

T he study reported that 39% of respondents had replanted an avulsed tooth before, and that this most often took place in a primary care setting. It is interesting and perhaps unsurprising to note that the vast majority of respondents reported following the British Society of Paediatric Dentistry (BSPD) guidelines on *Treatment of avulsed permanent teeth in children*.[3]

The BSPD guidelines recommend that a composite-wire type splint is placed to stabilise replanted avulsed teeth, which is sensible given the availability of these materials. The International Association of Dental Traumatology guidelines[4] also recommend the placement of a splint, but advise that there is currently no evidence base for the best type of splint to use.

Although the materials for composite-wire splints are readily available, they can be somewhat tricky to place, especially in a field contaminated with blood from a traumatic injury, and the wire has to be stabilised whilst all composite elements are polymerised. A more stress-free alternative may be to splint using orthodontic brackets. The benefit here is that each bracket can be placed individually before the wire is secured, and anecdotally they are much easier to remove and to keep clean. Importantly, the wire can be easily removed and replaced to allow accurate appraisal of tooth mobility and vitality at subsequent review. Naturally they require the availability of orthodontic brackets and modules; however, a small number of these could be obtained relatively cheaply and stored as part of a 'trauma pack' kept handy for this very reason. Indeed, working in secondary care, bracket-wire type splints are the first choice for adult and paediatric patients having undergone dental trauma.

I would also direct colleagues to the very intuitive Dental Trauma Guide (www.

CASE REPORT

Improved gum health

Sir, in July 2014, one of us (Rob Stepney) was bitten on the leg by a street dog in Sarajevo. The university hospital prescribed seven days' clarithromycin 500 mg bd. Rob experienced a metallic taste shortly after taking each dose, and, some days into the course, noticed that his gums no longer bled after brushing his teeth. Rob had experienced remission of gum disease on two previous occasions when taking short courses of ciprofloxacin and metronidazole as antibiotic cover for prostate biopsies, but both times bleeding had quickly resumed.

Rob, who has an MSc in Pharmacology, was intrigued at the apparent link with clarithromycin, especially since in this instance the effect on his gum health was prolonged.

At his next three-monthly check-up, Alison Zalinski – his dental hygienist who was unaware of her patient's recent history – noticed a marked improvement in Rob's gum health. This has been maintained over fourteen months. Rob's account of his experience reminded Alison of two recent cases which seemed similar.

Since data are from a retrospective review of everyday clinical records details are incomplete. However, the theme common to the three cases is of improved gum health following a macrolide antibiotic taken for unrelated reasons - see Table 1.

Table 1 Patient characteristics, antibiotics taken, and periodontal scores at the last visit before antibiotic use, first routine visit following antibiotic use and at most recent check-up

Patient (sex)	Agent(s) prescribed	Reason	Date	Periodontal scores at visit		
				Pre-antibiotic	Post antibiotic	Most recent
1 (M) – RS author	Clarithromycin 500 mg bd 7 days	Dog bite	Jul-14	3,3,4	3,3,3	3,0,3
				3,2,3	1,2,1	0,2,3
2 (M)	Ceftriaxone 2 g plus Azithromycin 500 mg 10 days	Pneumonia	Nov-12	X,4,X	X,3,X	X,3,X
				4*,4,4*	3*,2,4*	3*,2,4*
3 (F)	Erythromycin	Chest infection	Oct-13	4,4,4*	4,0,4*	3,0,4*
				4,4,4	4,4,4	0,4,0

In 2008, Burrell and Walters reported that the concentration of clarithromycin in gingiva is several times higher than in serum and higher in inflamed than in healthy gum tissue.[1] Although this study involved induction of experimental gingivitis in healthy subjects (through use of a maxillary stent), the clarithromycin schedule (500 mg bd) was the same as given to Rob, though for only six doses.

Along with this evidence of relevant drug distribution, our case histories of patients with gingivitis (although small in number) suggest that a short course of macrolide taken for reasons unrelated to dental health may result in periodontal improvement that lasts and may even increase with time.

Such an effect does not seem to have been reported (or, at least, is not widely known). Have others had similar experiences? If so, it might be worth conducting a randomised trial of these agents in gingivitis patients, with periodontal health and potential adverse events as endpoints.

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1. Burrell R C, Walters J D. Distribution of systemic clarithromycin to gingiva. *J Periodontol* 2008; **79:** 1712–1718.

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dentaltraumaguide.org) which is useful as an evidence-based guide to managing all types of trauma to primary and secondary teeth.

J. R. Allison and G. Garlington, Newcastle

1. Gilchrist F. Primary care dentists' experience of treating avulsed permanent teeth. *Br Dent J* 2015; **219:** 216–217.
2. Kenny K P, Day P F, Douglas G V A, Chadwick B L. Primary care dentists' experience of treating avulsed permanent teeth. *Br Dent J* 2015; **219:** E4.
3. Day P F, Gregg T A. Treatment of avulsed permanent teeth in children. British Society of Paediatrc Dentistry, 2012. Online information available at http://bspd.co.uk/Portals/0/Public/Files/Guidelines/avulsion_guidelines_v7_final_.pdf (accessed November 2015).
4. Andersson L, Andreasen J O, Day P, Heithersay G, Trope M, Diangelis A J *et al.* International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 2. Avulsion of permanent teeth. *Dent Traumatol* 2012; **28**: 88–96.

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PRESCRIBING

Congratulations

Sir, we congratulate N. Beacher *et al.* for bringing the antibiotic-associated complications of Clostridium difficile-associated disease (CDAD) to the attention of dentists.[1] Although their coverage of the topic was comprehensive, there are recently published data specifically related to the dental use of amoxicillin and clindamycin for the prevention of infective endocarditis and the associated incidence of CDAD.[2] We surveyed yellow card adverse reaction reports between 1963 and 2014 for all prescriptions of a single 3 g oral dose of amoxicillin or a single 600 mg oral dose of clindamycin. The adverse reaction rate for amoxicillin was very low with zero fatal and 22.62 non-fatal adverse reactions reported per million prescriptions – of which 40% were allergy-related and 15% could have been CDAD-related. In contrast, the adverse reaction rate with clindamycin was much higher with 12.6 fatal and 149.1 non-fatal adverse reactions per million prescriptions with all but one of the fatal reactions due to CDAD and the majority of the non-fatal reactions (57%) likely due to CDAD with only 22% allergy-related. While demonstrating that CDAD can occur with amoxicillin, our data suggest that a single 3 g oral dose used for antibiotic prophylaxis is extremely safe. In contrast, a single oral dose of clindamycin appears to cause CDAD with a much higher frequency and severity, including death, than amoxicillin. Indeed, the propensity for a single dose of clindamycin to cause CDAD appeared to be similar to that of more prolonged courses of clindamycin used for treating infections. This was somewhat unanticipated as it had previously been thought that a single dose would be unlikely to predispose to the development of CDAD.

M. Thornhill, Sheffield; M. Dayer, Taunton; B. Prendergast, London; L. Baddour, Rochester, USA; S. Jones, New York, USA; P. Lockhart, Charlotte, USA

1. Beacher N, Sweeney M P, Bagg J. Dentists, antibiotics and Clostridium difficile-associated disease. *Br Dent J* 2015; **219:** 275–279.
2. Thornhill M H, Dayer M J, Prendergast B, Baddour L M, Jones S, Lockhart P B. Incidence and nature of adverse reactions to antibiotics used as endocarditis prophylaxis. *J Antimicrob Chemother* 2015; **70:** 2382–2388.

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Anti-thrombotic agents

Anti-thrombotic agents are used for treatment of thrombosis – to prevent a thrombus enlarging, or to prevent thrombosis in people at risk, such as those who have:

- Atrial fibrillation
- Blood disorders
- Endocarditis
- Mechanical heart valves
- Mitral stenosis
- Hip or knee replacements.

All anti-thrombotic agents produce a bleeding tendency and may cause post-operative bleeding. Dental preventive care is thus especially important in order to minimise the need for surgical intervention. In general, anti-thrombotic agents should be stopped before surgery only where the risk of post-operative bleeding is high (eg major surgery) or where the consequences of even minor bleeding are significant (eg retinal and intracranial surgery) though, for other minor surgery, drug dose reductions are rarely needed and indeed may put the patient at risk from thromboses which can be lethal.[1] This should be discussed with the patient, who also must be warned of the risk of intra- and post-operative bleeding and intra/extra-oral bruising.

The two main classes of anti-thrombotic drugs are anticoagulants and antiplatelet drugs. Oral anticoagulants include:

- Vitamin K antagonists (VKAs -such as warfarin/coumarins)
- Newer oral anticoagulants (NOACs - such as dabigatran). The latter, such as direct thrombin inhibitors (DTI) (gatrans) and anti-Xa (xabans), target respectively the single coagulation enzymes thrombin (dabigatran) or factor Xa (apixaban, rivaroxaban, and edoxaban). In contrast to warfarin, NOACs have less thrombotic events and lower rates of major bleeding events and do not require monitoring in the same way as for warfarin using the prothrombin time or INR.2 and are thus replacing VKAs for many situations. Dabigatran and rivaroxaban are quickly absorbed and have short half-lives compared to warfarin so, in the event of excessive anticoagulant activity, discontinuing the drug is usually sufficient. Dabigatran and rivaroxaban have to date had no antidotes and if reversal is essential, haemodialysis, coagulation factor concentrates, and an antibody fragment which binds dabigatran (aDabi-Fab) were recommended to reverse the effects.[3]

The U.S. Food and Drug Administration (FDA) has now just granted approval of Praxbind (idarucizumab),[4] a humanised antibody fragment, or Fab, which binds specifically to dabigatran molecules only, neutralising their anticoagulant effect without interfering with the blood coagulation cascade, for reversal of the dabigatran anticoagulant effects if needed for emergency surgery/urgent procedures or in life-threatening or uncontrolled bleeding.[5,6]

Biological agents clearly will have increasing utility in healthcare well beyond those already reported.

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

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2. Griffiths M J, Scully C. New anticoagulants. *Br Dent J* 2012; **213:** 96.
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ERRATUM

Letters *BDJ* 2015; **219:** 420-421

Case report: A bridge too far!

The caption to Figure 2 in this letter was incorrect. It should have read as follows:

'Fig. 2 The extracted 9-unit cantilever bridge'.

We apologise to the author and our readers for any inconvenience caused by this error.

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