Other journals in brief

A selection of abstracts of clinically relevant papers from other journals.

The abstracts on this page have been chosen and edited by John R. Radford.

CLONIDINE V ADRENALINE

Is clonidine an adequate alternative to epinephrine as a vasoconstrictor in patients with hypertension?

Patil PM, Patil SP. *J Oral Maxillofac Surg* 2012; **70:** 257–262

It would appear that clonidine has few advantages compared with adrenaline in local anaesthetic solutions.

Local analgesic agents containing adrenaline (epinephrine) should be 'limited or avoided' in ASA Class III and Class IV patients. The aim of this study was to compare the analgesic and therapeutic properties of lidocaine/clonidine to lidocaine/adrenaline. Clonidine is a central antihypertensive drug. In a double blind study, 25 patients received 2% lidocaine with clonidine (15 µg/mL), and 25 patients received 2% lidocaine with epinephrine (12.5 µg/mL) for extraction of upper third molar teeth. All patients were taking antihypertensive medication. Taken in the round, there were few differences between the analgesic efficacy and haemodynamic properties of the two solutions. The investigators carried out a *post hoc* power calculation. They concede that they should have recruited at least 94 patients to show a possible 'difference of 10 mm Hg between the treatment groups.'

DOI: 10.1038/sj.bdj.2012.439

'TOOTH WEAR MAY BE CYCLICAL'

In vivo measurements of tooth wear over 12 months

Rodriguez JM, Austin RS *et al. Caries Res* 2012; **46:** 9–15

It would appear there was <15 µm tooth wear in six months, in the majority of a cohort of patients referred to secondary care.

The topology of tooth surfaces were recorded at baseline and then after 6 months in 63 subjects (and in some of these patients at 12 months, although this data could not be analysed). Silicone impressions of the teeth were recorded. Tooth wear was measured on gypsum replicas, using non-contacting laser profilometer and surface matching software. At the subject level, 77.7% of subjects had <15 µm of tooth wear after six months. However when recruited to the study, all subjects demonstrated at least 20 worn teeth with dentine exposure. Such observations could be reconciled by the hypothesis that tooth wear occurs in bursts. When analysing heartburn symptoms and vomiting only, the coefficient of variation R^2 (how well future outcomes are likely to be predicted by the model) was 0.10 ($p = 0.001$).

DOI: 10.1038/sj.bdj.2012.440

A COMPLEX DISEASE

Genetic susceptibility to periodontitis

Laine ML, Crielaard W *et al. Periodontol 2000* 2012; **58:** 37–68

Epigenetic changes ('changes in gene function that occur without a change in the sequence of nuclear DNA') may be caused by the dental biofilm and smoking.

This narrative review uses 14 summary tables and cites 164 papers. Periodontitis has commonality with, for example, Crohn's disease in that it is a 'complex disease' involving multiple, low penetrance genes, each with a limited role. In addition, genetic risk factors for one population may not be the same as for another population. The authors cite a meta-analysis that found moderate/weak associations between IL1 composite and IL1B-511 genotypes and those with chronic periodontitis. In another meta-analysis, the TLR4 +399 R allele showed a protective effect for aggressive periodontitis. The reviewers concede that many studies in this field 1) lack statistical power, and 2) the classification of periodontal diseases is 'clearly unsatisfactory'.

DOI: 10.1038/sj.bdj.2012.441

PERI-IMPLANTITIS

Residual periodontal pockets are a risk indicator for peri-implantitis in patients treated for periodontitis

Cho-Yan Lee J, Mattheos N *et al. Clin Oral Implants Res* 2012; **23:** 325–333

Does the data support the conclusion that 'the maintenance of periodontal health, rather than the previous history of periodontitis, is the critical determinant of increased risk of peri-implant disease'?

This retrospective case-control study followed 60 patients who received implants, for a minimum of five years. Of these, 30 patients had periodontal disease and underwent treatment before implant placement and the rest were periodontally healthy patients (PHP). Then, only those in the group that had periodontal disease before implant treatment, were categorised at the conclusion of the observational period, as either having at least one periodontal pocket ≥6 mm ('residual periodontitis'), or with 'no residual periodontitis'. When analysed at the implant level, the number with peri-implantitis were significantly higher in those with 'residual periodontitis'. Yet 'patients treated for periodontitis prior to implant therapy were found to have a significantly higher prevalence of peri-implantitis compared with PHP'.

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