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LETTERS TO THE EDITOR

Re: Aspirin and post-surgical haemorrhage

Dear Sir,

Prolonged post-surgical haemorrhage is an unpleasant, relatively uncommon complication of oral surgical procedures. It can occur as an unexpected complication in healthy young patients, and in these situations aspirin is a possible cause. Aspirin has been implicated as a factor contributing to post-surgical haemorrhage at least since 1945, when Singer suggested that aspirin was a cause of post-tonsillectomy haemorrhage. Aspirin-associated post-surgical haemorrhage in oral surgery appears to be relatively uncommon, at least as a serious complication, although prolonged post-operative bleeding necessitating blood transfusion has been reported (Lemkin, et al, 1974). A number of non-steroidal anti-inflammatory agents have a capacity to inhibit the enzyme cyclo-oxygenase, which catalyzes the production of endoperoxide precursors of the prostaglandins from arachidonic acid. This in turn inhibits the production of endoperoxides and thromboxane A2, leading to poor platelet aggregation. Aspirin in particular, irreversibly acetylates this enzyme, leading to an inhibition of cyclo-oxygenase for the lifetime of the platelet (about 10 days). This is manifested as a prolonged bleeding time in normal subjects.

This potential interference with haemostasis is usually not of clinical significance, and indeed aspirin is used commonly as a post-surgical analgesic. Nevertheless, it would be wise as a general rule to avoid aspirin therapy for approximately 2 weeks prior to elective surgical procedures, especially if major. Such advice to patients would be appropriate, for instance, ***in those that are referred for removal of third molars, and especially in those that are referred for orthognathic surgical procedures.*** It is sobering to note that in the case described by Lemkin et al., (1974), the surgical procedure was simply the removal of several teeth.

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[Editor: Orthodontists should also bear this advice in mind in those instances ***where patients are referred during orthodontic treatment for extractions.*** The caution would be most relevant where an archwire change preceded the extractions in patients with sensitive teeth and who commonly take aspirin in this situation.]

re: TMJ 'sprain' or 'strain'

Dear Sir

I read, with great interest and appreciation in your March 1984 issue, Professor Peter Reade's article on the temporomandibular joint pain-dysfunction syndrome. His use of the word 'sprain' in explaining to the patient the nature of his complaint is a very useful concept. I can think of no condition in which it is more important that a patient understand the cause of his symptoms, and the ***use of language fully meaningful*** to him is invaluable. Very useful though the word 'sprain' is however, it may not be entirely accepted by those patients who deny the single traumatic occurrence it conjures up or sufficiently explanatory to a patient concerned about the steady worsening of his complaint.

My purpose in writing this letter is to suggest the use of a similar and supplementary term, 'strain', defined by Blakiston's New Gould Medical Dictionary as 'a condition produced in a part by over-use or wrong use'. It will be apparent that this term covers things like faulty occlusal contacts and parafunctions which can then be placed in a more meaningful and therapeutic context which the patient can easily appreciate. The term can be used to explain why certain people are more likely to acquire this condition than others and why, once the condition has occurred, it is unlikely to undergo spontaneous cure.

In a typical case where there is historical or clinical evidence of both sprain and strain, a sprain constitutes the exciting cause and strain the predisposing cause and perpetuating factor.

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Reply from Professor Reade

Dear Sir,

Thank you for the opportunity to reply to Dr. Somers' interesting letter and thereby to ***expand upon a point which to me is fundamental to an understanding of temporomandibular joint pain-dysfunction syndrome*** (TMJPDS). Dr. Somers has reiterated what I believe are serious misconceptions in stating that '...certain people are more likely to acquire this condition than others...' and '...it is unlikely to undergo spontaneous cure...'. Of course the condition can undergo spontaneous cure and it probably does in most cases. It is the first of these points, however, which is the most serious of the conceptions. Let me explain my position by asking some questions: If there are such things as 'predisposing causes' why then don't we all suffer from TMJPDS because we all have some occlusal disharmonies or psychological stress; why don't all those with parafunctional habits have TMJPDS; why don't all those with deep overbites, crossbites, open-bites, missing teeth, over erupted teeth and so on have TMJPDS? A further question is probably the most telling: why is it that those patients who have been treated for TMJPDS by occlusal splint therapy with no irreversible changes to the occlusion commonly can return to their unchanged occlusion once therapy is complete and apparently have no predisposition to further episodes. This realization has reinforced my strong opinion and supports the proposition that irreversible therapy such as occlusal grinding, orthodontics, occlusal rehabilitation and surgery are not the treatment of choice for TMJPDS as has been quite strongly voiced by Griffiths (1983).

In seeking an answer to the questions posed above, it seems obvious to me that TMJPDS does not have a 'predisposing cause', that it is precipitated by a minor or major traumatic event (a sprain) either overt or covert to the patient, and that it can resolve spontaneously or be maintained (or perpetuated) by those factors mentioned above and commonly considered to be the cause of a TMJPDS. It seems reasonable to me that these maintaining factors can be described as 'strains' to use Dr. Somers' word. In the management of TMJPDS it is the maintaining factors which have to be temporarily negated and the strain on the temporomandibular joints

thereby reduced allowing resolution of the joint disturbance, and return to normal function of the involved muscles. Adequate occlusal splint therapy, with the addition in appropriate cases of psychotherapy, provides a well-tested means of treatment of this painful problem and avoids the obvious disadvantages of irreversible procedures.

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re: Maximising glass ionomer usage

Dear Sir,

In answer to your letter requesting information on the correct handling of glass ionomer cement for luting orthodontic bands, may I submit the following points.

A good starting point for information on the setting reaction and the setting speed of glass ionomer cements is an article in the most recent issue of the Australian Dental Journal by Wong and Bryant (1985), although the title suggests it may only cover 'dispensing and strength'.

There is no question in my mind that the *glass ionomer cements require isolation for some hours following their placement.* This most certainly applies to the restorative cement and it should apply to the luting cement as well because the setting reaction is identical. Wong and Bryant (1985) show that the cement increases in strength for 24 hours or more although it reaches a respectable level at 2 or 3 hours. This coincides with my earlier work using indentation measurements to follow the setting reaction (Mount 1982). There has been quite some confusion in the literature on this point particularly concerning the restorative cements because many operators will maintain that the cement looks satisfactory while it remains wet. This is quite true, but appearances can be quite deceptive. In fact if water or saliva contaminates the cement or it is allowed to dehydrate unduly within that first hour or two the physical properties will be notably reduced.

This of course justifies your use of 'Dry Foil' just as you previously used to cover zinc phosphate cements. This would isolate the glass ionomer for long enough to allow it to achieve acceptable physical properties. We have recently decided that none of the varnishes currently advocated by the manufacturers is sufficiently watertight to seal the cement adequately (Earl, Hume and Mount 1985). We have just completed experimental work which will lead to a second paper along these lines showing that Visio-Bond is in fact the material of choice for a surface sealant. Visio-bond is a light activated bonding agent used with composite resins made by Espe. Its greatest advantage is its wetability because it flows very freely over the surface and 'wets' the glass ionomer very adequately and penetrates into all the nooks and crannies.

I would suggest that a light covering of Visio-Bond over the periphery of the newly cemented band and activated would give you a total seal for the cement which would in turn allow it to mature to its full potential.

I was interested to note your technique for attaching the material to surgically exposed canines which were in danger of becoming grown over by mucosa. You suggest there that 'of course, etching was necessary'.

I would disagree quite strongly with this because the glass ionomer cements require the retention of whatever calcium and phosphorous ions are available. Etching is strictly contraindicated but on the other hand conditioning is necessary. 'Conditioning' to me means the removal of plaque and pellicle and any other contaminants from the surface of the enamel and/or dentine. This can be achieved readily by scrubbing the surface with a pumice and water slurry or alternatively painting with polyacrylic acid for 5-10 seconds. The surface should then be thoroughly irrigated, lightly dried, and the material placed immediately. Once again having allowed the cement to achieve its initial set at about 4 minutes from the commencement of mix it should be covered with a layer of Visio-Bond to seal it against water loss or water uptake.

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