Send your letters to the Editor, British Dental Journal, 64 Wimpole Street, London W1G 8YS. E-mail bdj@bda-dentistry.org.uk
Priority will be given to letters less than 500 words long. Authors must sign the letter, which may be edited for reasons of space.

Dentists practising CAM

Sir, – Mr Darby (*BDJ* 2002; 193: 244) feels it is a matter of great concern that some dentists are practising complementary or alternative medicine (CAM) without adequate training. I would omit the final three words of this statement.

As others have said, there is really no such thing as CAM. Rather, there is medicine which works, and medicine which doesn't. No amount of House of Lords' Select Committees is ever going to change the brute facts of science.

I have met some really nice, pleasant CAM practitioners. I have also met many very nice, pleasant dentists and medical practitioners. I even try to be pleasant myself. Pleasantness and taking an interest in one's patients are important for good human relations. What it does not do is provide specific treatment for anything other than anxiety, but this is why some patients seek CAM.

It is therefore of considerable importance that the BDA and the GDC should not 'recognize' any purported systems of medicine which proclaim their fundamental difference from medicine which has been scientifically established.

By all means test such claims if there are resources to do so, but do it rigorously, and protect patients from adverse effects, which may vary from herbally-induced kidney damage to ignoring scientifically established treatment for serious diseases. And at some point there should come a time when the accumulation of negative evidence is allowed finally to outlaw some alleged therapies which are useless, as happens with treatments found to be useless in orthodox scientific medicine.

T. L. P. Watts
London

Treatment of carious deciduous teeth

Sir, – I would like to compliment Drs Levine, Pitts and Nugent on their enthusiasm for clinical research ('What happens to unrestored carious deciduous teeth?" (*BDJ* 2002; 193:99-103).

We GDPs have access to a huge amount of clinical data. If we all kept records as carefully as these authors, and took the trouble to look back at them, we could probably make a significant contribution to science.

However, there is a difference between retrospective studies and prospective ones. Prospective ones of this nature would require Ethics Committee approval. I doubt if it would be regarded as ethical to withhold treatment from children to see what the consequences would be.

Was it really ethical to withhold it back in 1976? The reason given was, ". . . the prevalence of caries was much higher than today and the pressure on the available resources within the newly established practices required priority to be given to pain relief and treatment of carious permanent teeth." In other words, they were too busy to do anything about the carious deciduous teeth. Or perhaps, to be more charitable, they considered their responsibility to the community to be of greater importance than their responsibility to their individual patients.

The dmft in their area is currently 2. We all know that decay is not evenly distributed and the probability is that one third of that population have a dmft of 6. Call it 5, and remember that in 1976 it was much worse. If 80% of those teeth were lost painlessly, 1 in 5 caused pain. It seems probable that nearly every one of those children whose carious teeth were not restored suffered pain. If I had been one of the parents, and had thought that my child had been looked after by a professional person whom I trusted, I would be pretty peeved to learn that my child's suffering had been caused by professional neglect.

In the review by Dr M. Tickle, who was co-author of four of the five references quoted in support of the apparent need for us to re-evaluate our approach to carious deciduous teeth, he wrote, "In the studies reported by our team there was no difference in the outcomes of restored and unrestored primary teeth." In this study three times as many painful teeth were extracted than were restored.

I find this study, and the review, worrying for a number of reasons. Firstly, if GDPs take upon themselves the role of those responsible for managing health at a community level, and ration health care according to the perceived need, there is a risk that health problems will be underestimated.

In this case, it must have seemed splendid that all those children were under the care of a dentist even though they were not getting treatment. This is an important issue; our prime responsibility should be to our individual patients. The health care of the general population is a matter for the politicians.

Secondly, if those carious teeth that were eventually extracted had been restored earlier, perhaps extraction could have been avoided. We all know how extractions can lead to orthodontic problems and we all ought to understand the disadvantages of demonstrating that teeth are disposable. Also, extraction must seem like more of a violation to a child than restoration would. We need to consider our patients' long-term approach to dentistry.

Thirdly, if restoring carious primary teeth really makes no difference to the outcome, should we not improve our technique rather than give up?

I suppose what worries me most is the apparent assumption that we, the

readership, would understand and agree with the decision to withhold treatment from patients in our care. I really hope this study prompts other letters from your readers so we can get a broader picture of our attitudes to this question.

N. Cole
Devon

The author R. S. Levine responds:

My colleagues and I thank Mr Cole for his interest in our paper (BDJ 2002; 193:99-103) *and note that his criticism is not of the analysis of the data or our interpretation of the results, but of my personal care of children with carious deciduous teeth.*

We agree that prospective trials are ideal and some are underway, but in the interim, retrospective studies can be useful in highlighting the issues and providing evidence on outcome. However, the thrust of his argument is that I 'withheld' care from these children in the interest of clinical research and later in his letter, that practitioners like myself are 'managing health at a community level and rationing health care'. This suggestion is dismissed.

Firstly, as is increasingly recognised, care of carious deciduous teeth does not have to be based on the traditional concept of conventional restoration of all cavities without regard for age, and the dental, emotional and medical status of the child. All of the children in the reported study were given appropriate care with an emphasis on prevention and wherever pain presented, it was treated. Mr Cole suggests that I was 'too busy' to restore the children's carious deciduous teeth. Quite true!

As a single-handed practitioner starting a new practice in an area of deprivation and very high caries levels, priority had to be given to relief of pain and infection and secondly the restoration of carious permanent teeth. Mr Cole is unlikely to have experience of such pressures from his practice in beautiful Devon, where he clearly provides intensive restorative treatment for those fortunate children under his care.

Secondly, Mr Cole's extrapolation of our data is erroneous. Contrary to his suggestion, very few of the children suffered pain from the non-restored teeth. Where pain occurred it was promptly treated. No unnecessary operative treatment was provided and 82% of carious teeth remained symptomless until being lost.

Mr Cole confuses his argument with the distinction between 'individuals' and 'communities' and the suggestion that he cares for individuals while I do not. For Mr Cole to suggest that serving the children in deprived areas of Leeds and Halifax indicates that I consider my 'responsibility to the community to be of greater importance than that to individuals' suggests a detachment from the reality that dentists working in areas of endemic dental disease face every day.

His philosophy is clearly displayed by this assertion that 'all those children were under the care of a dentist even though they were not getting treatment'. As we enter the 21st Century can we not abandon the dogma that the 'individual' is not being treated properly unless the dentist is drilling away at the child's teeth?

Increasingly, the profession is looking critically at the patient benefit from the various forms of treatment we provide - evidence based dentistry. Such an approach will point the way to the best range of strategies for sound and ethical patient care and that is the purpose of our research. On one thing we do agree, a debate is needed.

Amalgam carriers

Sir, – I read with interest Mr Meacher's letter regarding the appearance of beads of mercury within amalgam carriers. As far as I am aware this has not been reported before and adds yet another item to the list of potential sources of mercury uptake in dental personnel.

My first reaction was to look at the amalgam carriers within the dental hospital in which I work. However I could find little evidence of mercury contamination other than the presence of 'smears' of amalgam within the nozzles. Certainly I found no beads of mercury itself. Admittedly the numbers viewed were small and the guns were all from the same manufacturer and therefore this could not be regarded as in-depth study. I also looked at my own 'all-metal' 'gun' purchased when I was student in 1958 and used throughout my student career. This item had never gone through a hot sterilisation process in keeping with the philosophy of 'kitchen cleanliness' in vogue at that time. Boiling water sterilisers, which were used at that time, were regarded as potentially damaging and therefore carriers were cleansed with disinfectant solution. Again I could find no sign of contamination as described.

The question therefore is where does the mercury come from? The obvious answer is that it is expressed from the fresh amalgam mixes as the result of the pressures involved in the delivery to the tooth being restored. However I cannot really believe this. It is known that mercury is released from amalgam at temperatures of 65°C.[1] Many years ago, in a letter to the *BDJ* (*BDJ* 1977: July 19th) I reported the finding of visible droplets of mercury on amalgam-filled teeth which had been sterilised in boiling water prior to use in phantom head teaching. I suspect therefore that the heating of Mr Meacher's amalgam carriers in an autoclave has created the drops of mercury which he describes.

The next question is; is this of any real significance with respect to occupational hazards to staff? Certainly, cosmetically and psychological it is undesirable. However the real question is 'does the vaporisation of the mercury in an autoclave lead to unacceptably high levels of mercury in the working environment?'

I suspect that the answer is 'no' but I have no evidence to justify this opinion. Referring to unpublished data of my own, I can say that placing eight teeth containing amalgam fillings in a boiling water sterilizer in my own office in the 'old' Sutherland Dental School in 1978 generated levels of mercury in that room from zero to an average of 81 mgs Hg/m^3 air in a space of an hour.

However in Mr Meacher's case, the original amount of amalgam in the carriers is very much less compared to the eight fillings in the extracted teeth used in the above study. In any case autoclaves should be used in well-ventilated rooms and ideally exhaust outdoors. In the dental hospital the latter is the case, which makes it difficult to assess if mercury vapour is released into the workspace.

Handling of the contaminated carriers might be construed as constituting a hazard. Although mercury is said to be capable of passing through the intact skin, this is thought to be an insignificant route.[2] The wearing of rubber gloves will reduce the risk and washing hands will also help. The real risk is to the smoker, the heat of whose cigarettes will volatilise the mercury on fingers and cause it to be inhaled together with the other products of this habit.

However the thought of mercury contamination is off-putting and this should make us think of ways of preventing it. One such way is to adopt the use of direct placement-capsules rather like those supplied by Southern Dental Industries that are attached directly to a delivery device similar to those used for composite resin.

There are of course other instruments that can be contaminated by amalgam before sterilisation. These include burs and perhaps more importantly serrated amalgam condensers – one of my

hobbyhorses. These can be readily clogged with amalgam that may be detrimental to subsequent fillings and also be a source of Hg vapour from sterilisers. I have never understood why they are serrated in the first place and feel that they should be ground flat to overcome their problems.

E. R. Smart
Newcastle upon Tyne

1. Mitchell J A *et al* (1955). X ray diffraction studies of mercury diffusion and surface stability of dental amalgam. *J Dent Res* 34: 744.
2. Berlin M. (1986) Handbook on the toxicology of metals . 2nd Edition Vol II Specific Metals Ch 16 Ed Friberg, Nordberg G E, Vouk V B, Kessler E. Elsevier Amsterdam.

Amalgam carriers II

Sir, – I read D. J. Meacher's letter (*BDJ* 2002; 193: 126) as it confirms my own concerns about autoclaving syringe type amalgam carriers.

I am sceptical of the efficacy of dismantling carriers prior to autoclaving as they may be mercury free when they go in the autoclave but where do the droplets of mercury go and will it always be carried out effectively in a busy practice?

Some seven years ago, we discarded all syringe types in favour of Hu Friedy double ended carriers (No AC5202) which do not collect residual 'pressed out' mercury. Initially awkward to use, the dentists soon get used to them, and they last for years.

R. Cole-Morgan
Thornbury

Who sets regulations?

Sir, – There is a very unfortunate (typographical) error in the second paragraph of the second letter in a recent *BDJ* (*BDJ* 2002; 193: 242).

The GDSC does not set regulations to be applied by the Dental Practice Board. The regulations are of course laid before Parliament by the Health Departments, after consultation with the GDSC (or the home country Dental Practice Committees). The HDs do not always follow the advice of the GDSC when finalising regulations.

Anthony S Kravitz
Chairman, GDSC

Root cause of trauma

Sir, – The statement, 'Trauma, especially that caused by improper tooth brushing, is a well-recognised cause of gingival recession', appeared recently in the *BDJ* (*BDJ* 2002; 192: 615-616).

Undoubtedly there are some cases when this is true but 'improper tooth brushing' is often blamed for recession even when evidence of ulceration or trauma is lacking. In the absence of such evidence and in the presence of a normal periodontium (bone level 2-3mm apical to CEJ), I would be interested to know the presumed pathogenesis of the recession.

Even if trauma is implicated, we would have to accept the scenario of the trauma being sufficiently prolonged to cause a chronic inflammation followed by bone loss, or the patient brushing through the soft tissues and the underlying bone! Although possible, as in some cases of gingivitis artefacta, this seems unlikely. Surely in the majority of such cases the patient would experience some discomfort from the trauma, remove the cause and the damage would heal.

The danger is that in the absence of evidence of trauma, patients may be advised to alter their brushing habits in a way that enables plaque to accumulate. That in turn could induce a chronic inflammation, leading to possible bone loss and recession.

A more likely scenario is the possibility of an underlying deficiency of alveolar bone dehiscence that may be developmental in origin (or perhaps following orthodontic treatment?), combined with a very thin overlying gingiva.

The capillary bed of such tissue is so thin that the poor blood supply could lead to atrophy and hence recession. This could happen even in the absence of trauma or inflammation and should be considered whenever there is recession without visible signs of inflammation or ulceration.

In the case presented, the presence of a lip stud may be coincidental or perhaps it has caused prolonged pressure on such gingiva, further restricting its blood supply, but not causing trauma.

When recession due to a combination of bone dehiscence and thin gingiva does occur, it can be rapid but is not necessarily progressive. The reason for this, under this hypothesis, is that further recession will stop at the stage that the remaining gingiva has an adequate blood supply. This is likely to be when it approaches the bone margin. However, the more apical position of the margin may then make plaque control more difficult for the patient leading to plaque-induced chronic inflammation and possible bone loss. This situation will require specific oral hygiene advice from the dentist or hygienist. It is also possible that a gingival graft may be indicated in order to bring the margin into a more cleansable area and to provide a thicker gingiva with an adequate blood supply.

D. G. Hillam
Prenton