Correspondence

When the dentist said: "Be still your beating heart!"

Dear Sirs,

We all often encounter a patient history and apparent presenting complaint that we can not precisely and cleverly explain. Our patient, Mr BW, a fit and active 53-year-old man, attended a routine appointment as an outpatient. He had done this many times previously as he was experiencing difficuties with heart rate control and troubling symptoms secondary to atrial fibrillation (AF). Coincidentally, he had also had amalgam dental fillings drilled some 18 months previously.

Since then, his cardiac problems had escalated. There appeared no clear causality between the fillings and the patient's AF but he was convinced there was a link. Interestingly, on his most recent visit, he reported that he had been well and had remained symptom free. First thoughts were: "What have we done that was so different after all this time? How have we stumbled upon the solution?" The only change since the patient's last visit was that he had insisted in having his dental fillings removed and replaced.

Looking through internet archives, we found many patients being convinced of an apparent link connecting the development of their specific arrhythmia to dental fillings. This led us to carefully assess if there were any plausible links.

At its most basic level, there are two possible theories to explain this – an electrical or a chemical phenomenon.

First, the electrical theory concerns the metals in the amalgam itself, which can apparently act as either a cathode or an anode depending on the metals' composition, leading to the possible formation of an electrical circuit with the body. One study has shown a significant electrical current generated between dental filling alloys and the body.[1] This current has been measured[2] at up to 109 mV and it may be that these currents are enough to tip susceptible individuals into arrhythmias.

The second possible theory regards the exact composition of the fillings. A variety of metals used in dentistry have potential biochemical effects. Mercury poisoning, for example, can have an arrhythmogenic effect.[3] This may occur due to small but constant amounts being released into the blood stream from corrosion of the filling.

Whilst this is not a clearly defined problem, some studies have shown that the removal of dental amalgam has reduced patients' complaints with on a variety of issues.[4]

We therefore suggest that a large retrospective analysis is carried out on those patients attending with arrhythmias to assess if this is a genuine problem or just an unfortunate coincidence.

Ewan J Mckay
Cardiology Imaging Fellow
Aintree Cardiac Centre, University Hospital Aintree Hospitals NHS Trust, Longmoor Lane, Liverpool, L9 7AL
(ewan.mckay@aintree.nhs.uk)

References

1. Nogi N. Electric current around dental metals as a factor producing allergenic metal ions in the oral cavity. *Nippon Hifuka Gakkai Zasshi* 1989;**99**:1243–54.

2. Ciszewski A, Baraniak M, Urbanek-Brychczynska M. Corrosion by galvanic coupling between amalgam and different chromium-based alloys. *Dent Mater* 2007;**23**:1256–61. doi: 10.1016/j.dental.2006.11.006

3. Ravasini JA, Mossop P, McLachlan CS. Homocysteine and heavy metal interactions in atrial fibrillation and ablation treatments. *Europace* 2008;**10**:1458; author reply–7.

4. Melchart D, Wuhr E, Weidenhammer W *et al*. A multicenter survey of amalgam fillings and subjective complaints in non-selected patients in the dental practice. *Eur J Oral Sci* 1998;**106**:770–7. doi: 10.1046/j.0909-8836.1998.eos106303.x

Could coronary artery calcium scores replace exercise stress testing? A DGH analysis

Dear Sirs,

The merits of coronary artery calcium scoring (CAC) were recently discussed by Purvis and Hughes, particularly its higher sensitivity compared to the exercise stress test (EST). The authors highlighted that one of the main disadvantages of CAC was the potential for increased workload. In the absence of ESTs, however, this would be equivalent to less than four CACs and less than one computer tomography (CT) per week in high coronary artery disease (CAD) areas.[1]

To address this issue, the CAC can be technician-led and lasts 15 minutes.[2] It is therefore plausible to argue that it involves less workload than the same number of ESTs organised otherwise. The only true challenge on human resources would be the CT scans, and perhaps one extra scan per week is acceptable. Furthermore, in our unit at University Hospital Wales, Cardiff, we found a much smaller cohort (4%) of low CAD risk patients (NICE risk score 10–29%); thus for other areas with a similar prevalence, the extra workload would be even less than the estimates reported by Purvis and Hughes.

Whilst changing from EST to CAC in low-risk patients *per se* is important, what is of greater significance in these cash-strapped times is the change in inertial culture of over-using ESTs for all groups of patients including those with very high CAD risk. In an audit of our Rapid Access Chest Pain

Clinic (RACPC), we found: exercise tolerance test (ETT) was inappropriately ordered for one in 10 patients with non-cardiac chest pain; 100% of patients with CAD risk <10%; and, 60% of patients with CAD risk >30% which included approximately half of patients with CAD risk >90%.

We recognise that in many hospitals, EST remains much more accessible than CAC. However, attitudinal barriers need to be addressed as a priority in order to ensure appropriate usage when CAC does become more widely available.

To decrease workload further, we must find a way to improve GP referrals. We found that almost a quarter of the patients seen in our RACPC were inappropriately referred, with 17% patients with non-cardiac chest pain and 5% very low CAD risk (<10%). Many of these patients went on to have unnecessary investigations with half having been ordered ETTs. In a previous qualitative survey conducted in our catchment area, it was found that GPs with higher referrals often had the lower perceived clinical confidence, and many felt that they would benefit from access to specialist advice.[3]

Thus, in order to improve referrals to our chest pain service, we plan to implement a simple pro forma encompassing NICE guidelines in order to direct further diagnostic management and, furthermore, to organise a GP training session with a consultant cardiologist to ensure clarity and fluidity for future referrals.

In summary, the 'misuse' of EST is a common problem across all risk groups in RACPC. Resource-smart changes include tailoring appropriate tests for each risk group and liaising with GP colleagues to minimise inappropriate referrals.

Tina Tian
(tina.m.tian@gmail.com)

Andrew Carson-Stevens

Nick Gerning
Cardiff University, Cardiff

References

1. Purvis JA, Hughes SM. Could coronary artery calcium scores replace exercise stress testing? A DGH analysis. *Br J Cardiol* 2011;**18**:120–3

2. Rajani R, Underwood SR. The exercise ECG - here today, gone tomorrow? *Br J Cardiol* 2011;**18**:7–8.

3. Daniels A, Moss M,Wilshere S. Improving cardiology referrals from primary to secondary care project report. South-East Wales Cardiac Network 2010 (unpublished data).

Dear Sirs,

We applaud Purvis and Hughes for a very relevant study to facilitate cardiologists building a business case for running full cardiac CT services in their departments.[1] They have clearly shown that previous studies of calcium score are relevant in a British cohort and the cost-effectiveness model can be applied similarly.

They have evaluated the pre-test likelihood of coronary artery disease (CAD) after analysing their investigative data. We note that after including the pre-test likelihood of CAD, investigative strategies would have altered so that patients in their cohort who went through coronary artery calcium scoring could have only been offered invasive coronary angiography. Only one patient whose zero calcium score suggested a low pre-test likelihood of CAD score was found to have significant CAD (2%). This reiterates the point that investigations organised according to the pre-test likelihood of CAD, as suggested by NICE, sounds reasonable.

However, it seems that the authors have not included patients who had a negative exercise stress test. Some patients in this cohort could have had a significant pre-test likelihood of CAD, and may have had a different diagnostic test according to NICE guidance. We do not have retrospective studies from the UK to show the outcome in such cohort of patients.

Yours faithfully,

Chris Sawh
Cardiology Registrar with special interest in Cardiac Intervention
Doncaster Royal Infirmary, Doncaster

Pankaj Garg
Cardiology Registrar with special interest in Cardiovascular Imaging
Northern General Hospital, Sheffield
(pankajvic@gmail.com)

Reference

1. Purvis JA, Hughes SM. Could coronary artery calcium scores replace exercise stress testing? A DGH analysis. *Br J Cardiol* 2011;**18**:120–3.

The authors reply

We serve as a semi-regional centre with the majority of cases attending our unit following an equivocal exercise stress test result. It is currently difficult for us to collect cases with negative results.

We believe it is important for clinicians to know that the current NICE strategy of stopping the CT after CAC = 0 may miss a small percentage of patients with significantly obstructive non-calcified plaque as well as those in whom myocardial bridging is responsible for symptoms.[1]

Innovations that substantially reduce radiation dose, such as iterative reconstruction[2] may in future permit full coronary CT angiography with doses that are currently only achieved with CAC. We hope that such innovations are widely available by the time of any future NICE guidance on chest pain and will permit ultra low dose CT angiograms for all-comers.

John Purvis
Consultant Cardiologist
(john.purvis@btinternet.com)

Sinead Hughes
Consultant Cardiologist

Altnagelvin Hospital, Londonderry, BT47 6SB

References

1. Leschka S, Koepfli P, Husmann L *et al*. Myocardial bridging: depiction rate and morphology at CT coronary angiography - comparison with conventional coronary angiography. *Radiology* 2008; **246**:754–62.

2. Renker M, Ramachandra A, Schoepf UJ *et al*. Iterative image reconstruction techniques: applications for cardiac CT. *J Cardiovasc Comp Tomog* 2011;**5**:225–30.

Controversial salt paper published – a response

Dear Sirs,

The editorial in *The Lancet* (May 12th 2011) that you quote in the June issue of the journal[1] is incorrect. I can assure you that diligent search of the literature will not reveal a single paper that shows that either salt loading, or high salt intake, causes increased mortality or morbidity in normal people with normal renal function. The recommendation that everybody should avoid salt is based only on the fact that salt deprivation lowers blood pressure (by less than 10 mmHg). The findings of Stolarz-Skrzypek *et al*[2] are entirely consistent with the literature, as summarised briefly below.

"Low salt intake increases mortality; high salt intake does not increase mortality," said Stolarz-Skrzypek *et al*, who followed up 3,681 normal subjects and measured how much salt came out in the urine, as an index of salt consumption (normal kidneys excrete any excess in the body). Although very high salt intake was associated with a very minor increase in blood pressure, this was not associated with any harm. In *Heart Wire*, MacGregor attempts to discount this study on methodological rounds;[3] he is a foremost advocate of advising normal people to restrict salt intake.

Previous publications with the type of study design used by Stolarz-Skrzypek have had the same result. In addition, studies in which normal people have been loaded with large quantities of extra salt in their diet have failed to show a rise in blood pressure, or any evidence of harm. Low salt intake was proposed as an idea since salt is supposedly believed to cause high blood pressure. If that were the case, patients with developing and/or established high blood pressure would have more salt in their bodies; they do not. The erroneous idea of the benefits of low salt intake is based on the fact that in patients with high blood pressure, low salt intake reduces blood pressure slightly; this is not relevant to normal people.

The most worrying aspect of salt restriction in normal people is that it increases mortality. That low salt intake causes harm is well established in the medical literature. Some elderly people and pregnant women have an inability of the kidneys to retain salt. This causes a condition called hyponatraemia, which is an increasingly common problem in the elderly. This condition is associated with swelling of the brain and consequent damage, possibly associated with the development of Alzheimer's disease. Low salt intake combined with high water intake (a 'detox diet') caused 5,259 hospital admissions in England in 2006–7 (one such patient with brain damage after 'detox' was awarded £800,000 compensation). Low salt intake has also been shown to cause increased obesity and alcoholism. Alderman states: "Sodium restriction generates other, sometimes undesirable effects, including increased insulin resistance, activation of the renin-angiotensin system, and increased sympathetic nerve activity".[4] Hollenberg, who does not advocate salt deprivation, concludes that: "prognosis in high blood pressure patients is improved substantially by the array of antihypertensive drugs available today";[5] we probably do not have to deprive even hypertensive patients of salt if we have effective drugs with fewer dangers than salt restriction.

It is time the media and government alerted the public to the dangers of low salt intake, and stopped pro-low salt propaganda.

Yours faithfully,

Mark Noble

Cardiovascular Medicine, University of Aberdeen

References

1. Controversial salt paper published (news). *Br J Cardiol* 2011;**18**:111.

2. Stolarz-Skrzypek K, Kuznetsova T, Thijs L *et al*. Fatal and nonfatal outcomes, incidence of hypertension, and blood pressure changes in relation to urinary sodium excretion. *JAMA* 2011;**305**:1777–85. doi:10.1001/jama.2011.574

3. Alderman MH, Cohen HW. Impact of dietary sodium on cardiovascular disease morbidity and mortality. *J Am Coll Nutr* 2006;**25**:256S–61S.

4. Hollenberg NK. The influence of dietary sodium on blood pressure. *J Am Coll Nutr* 2006;**25**:240S–6S.