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A REAPPRAISAL OF BENJAMIN BELL (1749–1806)

I read with great interest the article on Benjamin Bell which appeared in the last volume of the Journal.[1] Its author cannot have realised that in the *Practitioner* of June 1977 I wrote a similar article entitled 'Benjamin Bell, 1749 to 1806' without however his erudite comments on Bell's surgical practice, writings and reputation. Obviously Mr Macintyre did not know of my article when he compiled his bibliography.

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I am grateful to Dr Wilson for these comments, and was not previously aware of his excellent paper which gives useful insights into Benjamin Bell's character. I should for the sake of accuracy have described Bell as 'the oldest surviving child...' as he did have an elder sister who died in infancy. Dr Wilson and I both stated in our papers that Blacket House was sold in 1775, the date given in my cited reference.[1] A visit to the Register of Sasines confirms that the correct date was indeed 1777. It shows the value of using primary sources wherever possible for historical articles.

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CENTRAL PONTINE MYELINOLYSIS WITHOUT HYPONATRAEMIA

We read with interest the Case of the Quarter by Bose et al.[1] on central pontine myelinolysis (CPM) without hyponatraemia. Of note, with the help of physiotherapy, their patient walked again despite an initial quadriparesis. We have a patient who developed CPM in the usual manner, after an inadvertent rapid correction of serum sodium of 24 mmol/L in 38 hours to 126 mmol/L. She developed flaccid paraparesis and magnetic resonance imaging showed central pontine patchy enhancement with sparing of the lateral pons. She received intensive rehabilitation in the neuro-rehabilitation unit, and was discharged on day 32 able to walk independently. She remains well seven years later. It is clear that CPM is not unique to sodium, and is probably a common phenotype of rapid osmotic shifts in the central nervous system (CNS). More encouragingly, it is possible for patients with CPM to make good functional recovery. The possibility of recovery may be predicated upon early recognition and treatment, so as to limit further CNS insult.

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THE UNDERSTANDING OF ANGINA PECTORIS IN THE PRE-SURGICAL PERIOD

I am puzzled that the contribution of Dr John Fothergill (1712–1780), Edinburgh graduate and Quaker physician, to the understanding of the chest pain known to William Heberden as angina pectoris, receives no acknowledgement in your article on angina.[1] It is true that William Heberden himself did not realise that the chest pain he so clearly described was due to an abnormality of the heart. It is also true that Edward Jenner and Caleb Hillier Parry made significant contributions to the subject in the last decade of the eighteenth century. But it was John Fothergill who first associated angina pectoris with coronary artery disease. In a patient that he treated before his client's sudden death in 1775, and whose autopsy was reported in the *Medical Observations and Inquiries* in 1776, the autopsy, performed by John Hunter, revealed that the coronary arteries 'were become one piece of bone'. These findings, which antedated Jenner by twenty years, were published by me in my article on Dr Fothergill and the angina pectoris in the first edition of *Medical History* 1957; 1:115–122. I sent a reprint of that article to my old chief Sir Ian Hill and received a courteous note of thanks. So it is also puzzling that Sir Ian made no reference to Fothergill in his reminiscences.

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I am grateful to Sir Christopher for his observations on John Fothergill and his role as a pioneer in the understanding of angina. Fothergill deserves a place in the story; moreover he helps weave together a tale of eighteenth century contributors to it, for, as Sir Christopher points out, it was John Hunter who

performed the autopsy that Fothergill reported. The irony of it is that Hunter's own coronaries were found, by Everard Home in 1793, to be 'in the state of bony tubes', very much resembling the 'become one piece of bone' that Hunter described to Fothergill in 1775.

As in so many stories, one item of information raises several new questions. Hunter's own first heart attack was in 1772, three years before the autopsy on Fothergill's patient; his next ones in 1776 and 1777, when Jenner came to see him recuperating at Bath. We may wonder how much of those 1775 autopsy findings Hunter described to Jenner at that visit (as their correspondence shows, they shared a good deal of reflection and Hunter continued to mentor Jenner over the years). To what extent, we may wonder, would Jenner's own awakening perception of the angina/heart disease link have been influenced by his old chief's reminiscences? We may also wonder how much Hunter's memory of those bony tubes would have contributed to his own perceptive comment about his life being 'at the mercy of any rogue who cares to anger me'.

Careful observation and shrewd speculation – these were about all the diagnostic resources that the physicians of the eighteenth century had to employ, but they made remarkably good use of them.

As for Sir Ian Hill's failure to acknowledge Fothergill's contribution, I can only remark that sins of omission are not unique to the Anglican prayer book.

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NON-TUBERCULOUS MYCOBACTERIAL INFECTIONS IN THE SCOTTISH BORDERS

I read with interest the McCallum, Watkin and Faccenda paper on non-tuberculous mycobacterial (NTM) infections in the Scottish Borders.[1] As they aptly state this is a field where treatment is potentially contentious and idiosyncratic. The increasing incidence in the region is noted in the context of England, Wales and Northern Ireland.

Because of closeness of geographic, social, family and genetic ties to Scotland including the Scottish Borders, the epidemiology of NTM in the South West region of the Republic of Ireland should also be noted. This region consists of Counties Cork and Kerry, population 549,500 and like the Scottish Borders has few large urban areas. The annual incidence of NTM was studied from 1987 to 2000. The mean annual incidence during this period was 0.4 per 100,000 but was significantly higher ($p<0.01$) at 0.62 per 100,000 from 1995 onwards – with for example three clinical cases in 2000.[2] The incidence of *M. tuberculosis* infections also decreased, and routine Bacillus Calmette-Guérin (BCG) vaccination was discontinued in most of Cork between 1976 and 2010, so the question of decreases in cross-immunity between *M.tuberculosis* and BCG on the one hand and NTM on the other, arises in this region also. Other reasons for the NTM infections upward trend, like increasing age, and chronic lung disease mentioned by McCallum and colleagues appear to apply in South West Ireland also. With the longer historical view it may be relevant to recall that *Mycobacterium leprae* was once an important cause of human disease in the British Isles but ostracisation and other factors including possibly cross- immunity between *M.tuberculosis* infections and leprosy, protecting TB patients against leprosy, caused the latter disease to disappear,[3] apart from occasional cases in migrants from endemic countries.[4]

This poorly explained disappearance of a disease occurred centuries before the concept of cross-immunity was understood or even enunciated.

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SHOULD WE BE GIVING ENHANCED VITAMIN D INTAKES TO ALL?

Arguing against universal dietary supplementation with vitamin D, Miles Witham states that 'we do not have the understanding of its biological effects'.[1] My view is that no physician should fail to replenish vitamin D stores if deficiency is found. The following facts convince me that it would be unethical not to give vitamin D supplements to residents of Scotland:

1. In a Scotland-wide study of 2,235 healthy adults by Edinburgh University,[2] 84% had serum 25-hydroxy-vitamin D (25[OH]D) levels below 50 nmol/L, a level below which vitamin D deficiency is diagnosed. This threshold is conservative and most vitamin D experts consider that optimal levels are above 75 nmol/L.[3]

2. Also, of healthy postmenopausal women in the Aberdeen area, studied over all seasons, 100% had mean levels below 50 nmol/L.[4] In our Edinburgh practice in 2011, 70% of 350 tested individuals (age group 15–87) had levels below 50 nmol/L.[5]

3. We should note that 400 IU (10 micrograms [mcg]) supplements daily are not sufficient to raise 25(OH) D levels above 50 nmol/L in adults and pregnant women[2,6,7] and that 4,000 IU (100 mcg) daily are safe.[3] Also, two meta-analyses of randomised controlled trials (RCTs) (36,000 and 94,000 participants) showed 7% and 6% reductions in mortality, respectively.[8,9]

While we await updated guidance, including considerations on food fortification, from public bodies such as the Food Standards Agency and Scottish Intercollegiate Guidelines Network in Scotland or the Scientific Advisory Committee on Nutrition and National Institute for Health and Clinical Excellence in England, physicians should not deny patients advice and treatment based on up-to-date science. So we should prescribe supplements in adequate dosage to all residents of Scotland to prevent deficiency. The need to research biological effects and to complete randomised controlled trials should not distract us from the obligation to treat deficient patients. Public health services must now embrace this task with effective information campaigns for health professionals and the general population, thus ensuring prompt prescribing by primary care professionals for those with evidence of deficiency. In addition, with significant occurrences of rickets in Scotland,[10] the most urgent need is prevention of deficiency in babies from birth onwards.

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