# Letters to the Editor

## TORASEMIDE

Sir, Torasemide, a long acting loop diuretic, was a notable absentee from the 'Birmingham Hypertension Square',[1] notwithstanding previous documentation of comparable antihypertensive efficacy between this drug and the thiazides.[2] Absence of trials undertaken to demonstrate the effect of torasemide on cardiovascular risk does not necessarily signify lack of efficacy in this respect, especially in view of recent recognition that, for any given antihypertensive regime, attainment of an optimum target blood pressure is more crucial than specific drug choice in reducing the risk of adverse cardiovascular events.[3] An important consideration is that, in comparison with thiazides, loop diuretics (probably including torasemide) confer less risk of hyponatraemia because, unlike thiazides, they do not render the collecting tubules more permeable to water, nor do they potentiate the action of antidiuretic hormone at this site.[4] In my own series of 54 patients with severe hyponatraemia (defined as plasma sodium <120 mmol/l), compiled over a period of 15 years, 25 of the 28 diuretic-associated cases showing reversibility on withdrawal of the offending drug, proved to be on thiazides (including bendrofluazide 2.5 mg/d in five instances). Plasma sodium ranged from 101 mmol/l (in a patient taking hydrochlorothiazide and triamterene) to 119 mmol/l, with corresponding osmometric documentation of plasma osmolality in 18 instances, yielding values ranging from 208 to 266 mOsm/kg. Diabetes was not an associated feature. Underlying causes in the other 26 patients included drugs such as antidepressants and non-steroidal anti-inflammatory agents, intrathoracic as well as extrathoracic neoplasms, chest infection, secondary hypoadrenalism, heart failure, cirrhosis with ascites, and inappropriate administration of hypotonic intravenous fluids. Plasma electrolytes therefore need to be monitored diligently in patients treated with thiazides. The corollary is that relative freedom from hyponatraemia, and from biochemical derangements such as hypokalaemia, hyperglycaemia, hyperuricaemia, and dyslipidaemia[5,6] may render torasemide a more attractive proposition than thiazides for the busy executive who does not want to be lumbered with too many biochemical checkups.

OMP Jolobe, Geriatrician

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## ROYAL COLLEGE OF PAEDIATRICS AND CHILD HEALTH LECTURE: NEW INSIGHTS INTO THE CAUSES OF CEREBRAL PALSY

Sir, In her enjoyable lecture given on 5 November 1999, Dr Karin Nelson emphasised the epidemiological evidence that cerebral palsy did not seem to originate from hypoxia-ischemia. However, there is evidence that obstetrics diagnoses and successfully treats fetal hypoxia-ischemia.

In a recent review on the assessment of the fetus in labour[1] the simple method of detecting meconium in amniotic fluid is given a major role. However, meconium is a poor indicator of future cerebral palsy.[2,3] These apparently contradicatory conclusions are readily reconciled by biochemical evidence of fetal hypoxia-ischemia derived from quantitative analyses of meconium stained amniotic fluid[4,5] which justifies subsequent rapid delivery by the obstetric team. In some cases the mother accelerates the delivery of a fetus which shows biochemical evidence of hypoxia-ischemia.[6]

After the above evidence became available, meconium staining of amniotic fluid even more rarely was associated with fetal distress in Northwick Park Hospital, Harrow. This is consistent with recent surveys, the widely recognised West Australian epidemiology and Dr Nelson's own work. There have long been pleas for objective evidence of hypoxia-ischemia but the present blood gas-acid base measurements only provide data on the preceding hour, in which postural and anaesthetic treatment with a time delay before delivery restore blood gas-acid base results to normal.[7] Scalp sampling need not provide data on the general circulation. Quality control of blood gas-acid base measurements in obstetric units has been poor.[8] The interpretation of complex patterns like fetal heart rate tracings is also subject to error.[9] Despite these reservations hard data suggests that obstetrics and the mother's own efforts are effective and validate an easily detected, easily interpreted, clinically robust sign - that is, meconium staining of amniotic fluid.

More effective diagnoses, especially of the increasing number of genetic defects, may reveal more satisfactory causes of cerebral palsy. The most powerful diagnostic method available seems to be to use the age at which the disease presents as an initial discriminant to search an expanding literature.[10,11]

(I am grateful to the Royal College of Physicians of Edinburgh for funding further research on Age Related Diagnosis.)

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## REMEMBERING SIR MELVILLE ARNOTT

Sir, Your obituary of Sir Melville Arnott gave prominence to his distinguished war service which lasted for six years, starting in China in 1939 and ending in Germany in 1945. In his first and last postings, he coped with unusual circumstances of contrasting nature.

Dispatched to the International Settlement in Shanghai in 1939, a later move in competition with a fellow officer gave a choice of Hong Kong or Singapore. The toss of the coin was in his favour and he chose the latter. Unlike his colleague, he evaded the Japanese.

During the campaign in North-West Europe in 1945, he was lieutenant-colonel in charge of the medical division of 109 British General Hospital (BGH) at Duffel in Belgium. The hospital was selected as an army teaching unit to train young medical officers, most of whom had spent several years in the field, for future specialist service in what promised to be a long campaign in the Far East. His rich academic background and experience of tropical diseases made him an ideal tutor. The *Lancet* referred to 109 BGH as the 'University of Duffel'.

Arnott's enthusiasm was infectious and quick success in post-war membership examinations was, for a few, due in no small measure to his efforts. I recall Melville Arnott with great admiration and affection.

Hugh Conway, Consultant Physician (retired)

## REMEMBERING PROFESSOR IAN HILL

Sir, The article published in Vol. 30, No. 1 of the *Proceedings* on Professor Ian Hill[1] took me down memory lane to the year 1963 when I was a house man under the late Dr W. Frain-Bell, dermatologist at Dundee Royal Infirmary.

The authors of the article were then consultants under Professor Hill. I was appearing for the Edinburgh MRCP with dermatology as the special subject. Since clinical medicine was not my forte Dr Frain-Bell spoke to Professor Hill who readily agreed to take me on his ward rounds. I also used to clerk emergency admissions at night. On one occasion a middle-aged woman was admitted with congestive cardiac failure. I did a routine smear study and to my surprise I found a picture of myeloid leukaemia. As I was showing the smear to the registrar, who should walk into the lab but Professor Hill, though it was 11 pm, since it was his admission day. He complimented me on my unexpected finding - an unforgettable moment in my life. As President of the Royal College of Physicians of Edinburgh his signature is affixed on my membership diploma, which I have carefully preserved.

Though the authors have mentioned the commissioning of the dialysis unit and the paediatric cardiology departments during Professor Hill's tenure at the DRI, they have not mentioned the starting of a superb photobiology unit by Dr Frain-Bell, the second of its kind at that time in the UK, for which Professor Hill gave his unstinted support.

His kindness, his dedication and his clinical demonstrations will never fade from my memory.

Patrick Yesudian, Professor of Dermatology (retired)

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## PAINTING OF MAISTER PETER LOWE

Sir, I am sure that I am not the first to draw your attention to the fact that copies of the same portrait of this gentleman were published in the two most recent issues of the *Proceedings*, namely in the October 1999 issue (Vol. 29, p. 338) and in the February 2000 issue (Vol. 30, p. 63). Curiously, in one of these portraits, Maister Lowe is shown facing to the left (Vol. 29, p. 338) and in the other he faces to the right (Vol. 30, p. 63).

As one of these is clearly incorrect, I thought that it would be appropriate to see which way he was facing in Tom Gibson's book entitled *The Royal College of Physicians and Surgeons of Glasgow: a short history based on the portraits and other memorabilia* (Edinburgh: Macdonald Publishers; 1983). In this, Maister Peter Lowe is facing towards the left of the illustration as shown in the article by Buchanan and Gately (Vol. 29, p. 338).

I also noted that the Latin statement from the *Lanercost Chronicle* (*Chronicon de Lanercost)* cited by Buchanan (Vol. 30, p. 70) was incorrect, and should have read *mortuus est dominus*... as correctly cited in the following article by myself and MacLennan (Vol. 30, p. 79), rather than *mortuns est dominus*...

Professor MA Kaufman (retired)

ATRIAL FIBRILLATION

Sir, With regards to Clarkson and Lip's paper[1] on the subject of atrial fibrillation; one of the most exciting developments in chemical cardioversion of atrial fibrillation and in prevention of relapse of this arrhythmia is the recognition that non-dihydropyridine calcium channel blockers (NDCCB) such as verapamil and diltiazem, respectively, can be successfully co-prescribed with amiodarone, either to enhance the likelihood of successful cardioversion by a significant amount ($p = 0.01$),[2] or to prevent relapse in refractory cases (with 82% success rate in those co-prescribed NDCCB vs. 23% success rate in those solely treated with amiodarone).[3]

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LETTERS TO THE EDITOR

Letters to the Editor are welcome providing they do not exceed 300 words in length. The Editor's decision on publishing or rejecting any letter is final, and the right to edit correspondence is reserved.