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We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included. Titles of papers are not, however, included in the correspondence section.

McIlroy, the media, and the macabre

SIR,—There would be no end to correspondence if the hospitality of your columns remained open indefinitely. New facts and some afterthoughts tempt us, however, to offer a further contribution to the McIlroy saga (14 April, p 973).

May we first stand corrected by Dr Alex Sakula, who pointed out that Raspe was not buried in Killarney and who drew our attention to his most interesting account of the original Baron von Munchausen.¹

For those whose reading is confined to the pages of your journal we confirm that McIlroy is alive and—as usual—not very well. He is not happily settled in an old people's home in Birmingham, as reported when the correspondence was closed (12 May 1979, p 1278) and as echoed in the national press. After entertaining several reporters he upped and left. Then, via Inverness, Glasgow, Belfast, and other places in Ireland, he has made his intermittently hemiparetic way back to England, where he is still, unbelievably, gaining admission to hospitals. In Cornwall he posed as a disabled sailor—collecting, en passant, both sympathy and sweaters. At Queen Mary's Hospital, Roehampton, his arrival generated a detailed circular, from

which it clearly emerges that not all hospital administrators (or, alas, all doctors) read the *BMJ*.

A year ago we reported that McIlroy had been admitted to 68 hospitals. The total now tops 80. Physicians have written to us expressing regret at their omission from the list of those duped. McIlroy's Amended Guide to NHS facilities now includes substantial sections on St Thomas's, the Central Middlesex, the West Middlesex, and the Westminster (in London); St James's, Leeds; the Royal Infirmary, Chester; the General Hospital, Birmingham; the Royal Northern Infirmary, Inverness; Lagan Valley Hospital, Lisburn; Ards Hospital, Newtownards; Tehidy Hospital, Cornwall—and the Falmouth Seaman's Hospital.

Three questions still remain unanswered. Firstly, why are these patients constantly on the move, despite attempts to help them? Great efforts were made over McIlroy in Birmingham, and they seemed (for a short while) to have succeeded—but something set him off again. Secondly, how should we treat such cases? We must concur with a recent comment² that the medical profession seems singularly inept in this field. Has anyone a

constructive suggestion? None has emerged, to our knowledge, in the 28 years since Munchausen's syndrome was first defined.

Our third question is sociological rather than medical. Why did a single report produce such reverberations? Requests for reprints have spanned Europe—from the National Hospital, Queen Square, to the Krakow Institute for Mother and Child (via the Zoological Institute of the University of Hamburg). Serious journals (from staid *Scientific American* to “with it” *World Medicine*) deemed McIlroy's take worthy of comment. The Canadian Broadcasting Company alerted its listeners from Vancouver to Newfoundland. *Time* magazine carried the story even further afield. From the *Shepherd's Bush Gazette and Post* to the *Straits Times* (Singapore) the popular press had a field day. We can only speculate whether the *Thunderer*, if thundering, would have picked up the trail.

Within the profession, was it that there was grudging admiration for a worthy opponent, for the man who kept winning against all odds? Outside it, is there a national—nay international—gratification when the carefully nurtured mystique of medical expertise is itself systematically eroded by a mere con-

sumer, and an amateur to boot? There is an unspoken feeling that doctors should be infallible and much lay criticism was voiced that so many had proved so credulous. Beneath the veneer of condemnation, is there widespread sympathy for McIlroy—as undoubtedly still lingers for the train robbers? In an era of mediocrity (and of theatrical recession) is the sheer technical competence of the feat silently applauded?

And could there be other, even less mentionable, motives? Does the average person (and even the average subeditor) experience an ambivalent shudder as he or she thinks of being at the receiving end of 48 lumbar punctures? How much sadomasochism underpins public interest in the McIlroy myth? "Heroic" behaviour on this scale clearly engenders much morbid fascination, as shown by the many letters we have received.

Last April we were concerned lest attempts by newspapers to track down our patient reach the stage of harassment. The motives of the media when confronted with the macabre or the unexpected may be highly ambiguous. How often is privacy invaded when the search for copy masquerades as a concern for the public good? May we plead that, so far as the lay press is concerned, McIlroy now be left alone? Hounding him into the ground would, in the words of Johnson's epitaph for David Garrick, "eclipse the gaiety of nations." It would certainly impoverish the medical (if not the public) "stock of harmless pleasure."

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¹ Sakula A. *J R Coll Physicians Lond* 1978;12:286-92.

² Bass C. *Lancet* 1979;iii:366-7.

The clinical chemist and the future

SIR,—Responding to Professor K G M M Alberti's letter (2 February, p 329) I am under no illusions; I am well aware that my iceberg (15 December, p 1581) is backed by millions of pounds' worth of investment in numerous commercial companies and I cannot stop tests moving from the laboratory environment to wards, intensive care units, health centres, patients' homes, and perhaps beyond. However, my grass roots view is very different from Professor Alberti's and before I succumb I would like to make some more comments.

The DHSS does indeed perform evaluations but until now it has only rarely investigated kits. It has just produced a detailed kit evaluation protocol which is about to be used for the first time. However, in this protocol ward side room kits were specifically excluded. This is a serious omission. Even if evaluations do occur the results are published in *Hospital Equipment Information* and sent to area health authorities. Apart from one or two HEIs that have reached me I have to request copies when I know they contain information I require. How will the DHSS ensure that information reaches users of side room kits? Another aspect is that the evaluations are performed only on production items; hence the situation where numerous meters have been sold and are in use in the field before an evaluation appears. Finally, just how much power is available to the DHSS to use against a product it considers unsuitable, and should this power be increased in view of future developments?

Professor Alberti defines the future of the clinical chemist as servicing the necessary machines, performing quality control, and meanwhile developing new tests and performing more complex assays back in the laboratory. This is fantasy for most of us. To service a clutch of ward instruments would take a good deal of time (and who orders consumables?). Quality control is a here-and-now procedure, not six hours before and after the test in question: and where does the money come from for all the fancy work? Returning to glucose estimations, our routine work load costs less than £200 per year (using a commercial kit, dare I say?). A saving of this amount would hardly support a research project.

I would next like to comment on the definition of deaths as a challenge the clinical chemist should take on in his search for foolproof techniques. The South-west clinicians I referred to in my first letter were faced with two results: one they had obtained and one from the laboratory. Since the laboratory ones did not fit their clinical impressions they were ignored. This is a challenge I am unprepared to accept and unqualified to deal with. Another point is that "foolproof" does not and perhaps cannot apply to diagnostic kits and equipment. Some companies are indeed selling on the basis of "suitable for use outside laboratories" ("no problem"), but these have a mirror image of more responsible manufacturers who will state that skill is required to maintain reliability. Who is right?

Finally, there are factors that will profoundly influence the situation that are as yet in the background. The DHSS Interim Advisory Committee on Safety in Clinical Laboratories is just about to publish and its recommendations will be used as a guide for the officers of the Health and Safety Executive. Side room testing does not escape from the safety procedures that apply to main laboratories and responsibility for this aspect will have to be assigned. Other interested parties currently absent from the scene are the ASTMS and NALGO, the strong unions of the medical laboratory scientific officers. I await their response with interest.

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*.*This correspondence is now closed.—ED,
BMJ.

Prophylaxis against febrile convulsions

SIR,—It is difficult to interpret the findings of Dr Sheila J Wallace and Mr J Aldridge Smith (9 February, p 353) without knowing (a) how often and at what times of the day their patients were taking the medication and (b) at what times blood was taken for estimation of phenobarbitone or valproic acid. The statement that "compliance was significantly better" in children given phenobarbitone is not explained: did the authors define compliance in terms of serum or plasma drug concentration or in terms of medicine taking? Compliance is influenced by the number of doses to be taken each day and by the pharmaceutical form of the drug, as well as by the incidence of side effects and other factors. Did it differ between children given tablets and those given liquid preparations?

A more fundamental problem concerns the authors' statistical analysis. They compare six-month periods of treatment or no treat-

ment, using the χ^2 test. Some children contributed more than one six-month period, and the data are therefore not independent. The χ^2 test is applicable only to independent data, and this makes it necessary either to recalculate the data using numbers of children rather than numbers of six-month periods or to present them in some other way.

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SIR,—It was a good idea to study the effect of therapy aimed at preventing febrile convulsions in the presence of the provoking stimulus, fever. In their paper (9 February, p 353), however, Dr Sheila Wallace and Mr J Aldridge Smith not only failed to show that the drugs they used were of value but did not mention the dangers, including fatal consequences, of prophylactic valproic acid (sodium valproate).¹⁻⁴

Readers should know that there is no universal agreement that prevention of febrile convulsions is a worthwhile aim,⁵ except in the special case of early childhood meningitis.^{6,7} If many febrile seizures are simply convulsive syncope^{8,9} an excellent prognosis would not be surprising. Any proposed therapy must be balanced against this generally good outlook.⁵

On closer inspection of this paper it is clear that the reduced incidence of febrile convulsions per febrile illness is not related to either of the drugs used in the treated children. If phenobarbitone or valproic acid were effective in themselves, then seizures would be less frequent at "optimal" or therapeutic blood concentrations than at suboptimal levels. The authors found that with "optimal" phenobarbitone levels there were six fits in 45 six-month periods in which fever occurred (13.3%). With suboptimal phenobarbitone levels the rate was eight fits in 58 six-month periods (13.7%). Similarly, with "optimal" valproate levels the recurrence rate was 5/39, and 6/35 when the valproate was suboptimal. Neither of these differences is significant.

This lack of a dose or blood level effect for either drug suggests that the treated groups had a different outcome not because of the antiepileptic drugs themselves but (as hinted in paragraph 1 of the discussion) through unrelated aspects of management such as cooling and the use of antipyretics. In support of this is the excessive number of episodes of fever in the untreated children. The 24 children on no treatment had between them 100 six-month periods in which fever occurred, while the 84 children on phenobarbitone or valproate had only 84 periods in which fever occurred. This difference is very highly significant ($\chi^2 = 27.5$, $p < 0.0001$).

If the drugs recommended were useless but harmless, it would be excusable to recommend them as an aid to parents' peace of mind. Phenobarbitone has potential hazards,^{10,11} but it has been with us for a long time and disasters from its use are not to be expected. Valproic acid (sodium valproate), however, is a newcomer, and good though it is in certain epilepsies the list of side effects steadily increases and in no way can it be regarded as "relatively free" from them.

The commonest complaints are of nausea, vomiting, and abdominal discomfort,¹ of which pancreatitis is now established as a known cause.^{1,2} One may ignore other effects³ such as anorexia or obesity, sedation or excitement,