A physician's eyewitness report in Iraq

Sir—Recently I returned to visit my alma mater in Iraq—the University of Mosul's Medical School. On the basis of what I had heard about the dire need for medical supplies there, I took with me a container with a balanced supply of medicines sufficient for about 100 patients for one month. The medicines were donated by pharmaceutical companies, physicians as myself, and Jordanians.

In all, I visited seven medical centres throughout Iraq. What I saw in terms of suffering and deprivation will stay with me for a very long time. The worst thing that can happen to an Iraqi nowadays is to become ill. Very basic medicines sufficient for about a month. The price will be many times more than their salary. Faced with a near total collapse of their country's health infrastructure, Iraqi physicians often resort to diagnosing cases without the necessary laboratory tests. The medicines they prescribe are often the very few that are available in local pharmacies.

To be done because of the lack of anaesthetics and surgical sutures. If available, an X-ray film is used for 3 patients at one time.

I find it difficult to decide who is most hard hit by this widespread shortage—elderly people, children, hypertensives, epileptics, diabetics, or the ones who suffer from an acute surgical condition for which surgery cannot be done because of the lack of anaesthetics and surgical sutures. If available, an X-ray film is used for 3 patients at one time.

The patients I encountered in Iraq were not consulted when Iraq invaded Kuwait in 1990. They should not pay the price for their government's policies. These Iraqi patients seem to have chosen to die quietly with pride, but the rest of the world, especially the medical community, should not stand idly by. I should like to invoke the principles of our civilisation to stop this cruelty against a whole nation. Our pledge to save human lives and to care for and love the sick should motivate us to save human lives in Iraq. All medical and humanitarian agencies and physicians around the world should lend a hand to the patients of Iraq. They desperately need such assistance now.

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Authorship inflation: a trend reversed

Sir—With the growing pile of publications, several surveys suggest a constant rise in the number of coauthors.1,2 This continuing rise of contributors increases the chance of authorship inflation. As shown by the Pearce/Chamberlain case from St George's Hospital, London, in which authorship was awarded to one who had not contributed intellectually, this might increase the likelihood of fraud.3,4 A paper investigating the number of authors for Lancet articles suggested that the mean authorship escalated from 1-3 authors per major article in 1930 to 4-3 in 1975. Moreover, there was a striking acceleration of this increase in the years just before 1975. To see whether this trend is continuing, the number of coauthors to Lancet articles was examined for 1975 to 1994.

Articles were retrieved electronically via MEDLINE and were selected by the keyword journal-article for publication type. This selection criterion excludes all papers published as letters to the editor and reports appearing in the news and bookshelf section. The dataset was loaded onto a wordprocessing programme and names were counted. Anonymous reports were omitted from the study. Because MEDLINE truncates the number of authors at 10, such papers were examined individually. In the 19 years studied, the mean number of authors showed a steady increase, from 3-18 in 1975 to 4-16 in 1990 but then a decrease to 3-99 in 1994. The number of papers with more than 10 authors showed a steady increase from none in 1975 to 36 in 1994. The difference between mean authorship in 1975, as reported by Strub and Black,1 is probably attributable to different selection criteria. Strub and Black looked exclusively at major articles (presumably publications appearing in the articles section), but such reports only reflect a small part of publications in The Lancet. Thus, although the growth of mean authorship is stable, the number of papers with more than 10 authors is rising.

Complex clinical questions often need input from a wide range of disciplines, resulting in a large number of potential authors. In multicentre studies, which typically involve many collaborators, agreement about authorship when individual cooperators are not credited is difficult to achieve. But in such reports participation solely in the acquisition of the data does not justify authorship. In a study of 12 articles in a general peer-reviewed journal, only 51 of 84 authors fulfilled possible and definite criteria for authorship, suggesting that at least a third of authors did not contribute substantially to the intellectual content of the paper.1

This lax view on authorship is worrisome, but some solutions can be offered. First, the contributions of separate coauthors to multiauthor papers should be assessed more critically as to whether individuals' efforts qualify for authorship. The decrease in mean authorship nevertheless suggests that investigators can adhere to the criteria for authorship. Furthermore, reports of studies involving many collaborators could be made on behalf of a joint group.
Viscous hearing loss

Sir—Acute sensorineural hearing loss is well recognised in renal failure. The causes include recurrent infection, underlying immune-mediated disease, or side-effects of drugs such as aminoglycosides and loop-diuretics, but usually the hearing loss remains unexplained.1 We report on a patient with acute sensorineural hearing loss associated with blood hyperviscosity due to polyclonal gammopathy. Despite progression of renal failure a return to normal blood viscosity was accompanied by recovery of hearing.

A 69-year-old woman with progressive renal failure secondary to glomerulonephritis was admitted for evaluation of acute unilateral hearing loss. Audiometry confirmed the hearing loss of 40 dB over frequencies of 0.5 to 4.0 kHz and an air-bone gap of 5 dB. Her serum creatinine (4.0 mg/dL) and blood urea (56 mg/dL) were raised. The haematocrit was 34.8% and she had a raised serum protein of 9.1 g/dL (normal 6.2–8.2) with a polyclonal γ-globulin peak of 31.1% (12.25%), and an albumin/globulin ratio of 0.69 (1.2–2.3). Viscosimetry (dropping ball technique) showed her blood to be abnormally viscous (1178 ms [normal 850–975]). The hearing loss persisted despite prednisolone (250 mg with rapid tapering by 25 mg daily) over 10 days, while renal function worsened. Haemodialysis was started on day 17 for control of fluid overload and incipient pulmonary oedema.

Despite the worsening renal function audiometry on day 20 revealed an improvement of hearing function by 20 dB. At that time the blood viscosimetry measurement was 1102 ms. Serum total protein was 8.1 g/dL with 27.4% γ-globulins and an albumin/globulin ratio of 0.92. During the next 3 weeks her hearing returned to normal, as did viscosimetry (950 ms) and the serum protein (7.4 g/dL), γ-globulin (24.2%), and albumin/globulin ratio (1.24). Intermittent haemodialysis was continued.

The internal ear is supplied by an end-artery, so rheological abnormalities resulting in increased blood viscosity are likely to play an important part in the pathogenesis of hearing loss.2 Hearing impairment secondary to hyperviscosity may be underdiagnosed yet it carries a good prognosis if treated. Further studies of the relation between blood viscosity and hearing loss may help toward specific preventive strategies in patients at risk of viscous hearing loss.

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Hyponatraemia after rehydration with sports drink

Sir—A 44-year-old boy was admitted to our unit acutely ill with fever, vomiting, and, predominantly, diarrhoea. He was passing frequent, small, loose stools, and was thought likely to have an acute gastroenteritis. Examination suggested that he was about 3% dehydrated and an oral rehydration solution (Gastrolyte) was prescribed. Over the next 36 hours his diarrhoea remained severe (he was subsequently found to have both salmonella and cryptosporidium in his stool) and his oral intake was less than intended. Because the dehydration was thought clinically to have worsened, his urea and electrolytes were checked and his sodium was found to be 124 mmol/L. Inquiry on the ward revealed that because the child had not liked the taste of Gastrolyte he had been allowed to drink Powerade, a sports drink, in the belief that such drinks would contain enough electrolytes to replace his losses.

Table: Electrolyte and sugar content of two oral rehydration solutions (ORS) and two sports drinks

<table>
<thead>
<tr>
<th>Content</th>
<th>Liquid</th>
<th>WHO ORS</th>
<th>Gastrolyte</th>
<th>Powerade</th>
<th>Gatorade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na (mmol/L)</td>
<td></td>
<td>0</td>
<td>60</td>
<td>5</td>
<td>13</td>
</tr>
<tr>
<td>K (mmol/L)</td>
<td></td>
<td>20</td>
<td>20</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>Glucose (g)</td>
<td></td>
<td>22</td>
<td>18</td>
<td>40</td>
<td>18</td>
</tr>
<tr>
<td>Sucrose (g)</td>
<td></td>
<td>0</td>
<td>0</td>
<td>40</td>
<td>45</td>
</tr>
</tbody>
</table>

The table shows the electrolyte and sugar contents of Powerade, another sports drink (Gatorade), Gastrolyte, and the oral rehydration solution recommended by WHO. Although the electrolyte content of sports drinks may be suitable for replacing losses during sporting activity, it is entirely inadequate for the purpose of rehydration in the setting of the electrolyte losses associated with an acute gastrointestinal illness. The high sugar content of the sports drinks may also be too high for the damaged gut, resulting in a worsening of the diarrhoea due to an osmotic effect.

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Early diagnosis of Duchenne muscular dystrophy

Sir—Marshall and Galasko (March 4, p 590) have appropriately emphasised the persistent difficulty of delayed diagnosis in Duchenne muscular dystrophy (DMD). They do not tell us the mean age at which the diagnosis was made. The timing is especially relevant for genetic advice because the earlier the diagnosis is made the greater is the opportunity of providing accurate counselling and, importantly, the possibility of offering prenatal diagnosis in subsequent pregnancies.

In our regional paediatric neuromuscular clinic the average age of diagnosis of 51 boys with DMD was 4.5 years (range 3 months to 8.5 years); in four of these families, further affected siblings were born before the diagnosis was established in the older child. Early diagnosis and the identification of high-risk pregnancies to prevent subsequent affected children is the predominant (and some would argue only) justification of screening of newborn babies for DMD.1 In the absence of a universal neonatal screening policy it is, as Marshall and Galasko correctly point out, imperative that this invariably fatal disorder is considered as early as possible.2

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