1982, 1391 (24%) were in New York City, where surveillance again provides data difficult to obtain elsewhere. Of these, 1391 infants, all but 20 still under investigation were born to mothers with AIDS (11%), at high risk by injecting drugs during pregnancy (54%), or by being the partner of a man at high risk (27%). The main clinical features were protozoal and bacterial infections. 90% of babies were black or Hispanic, and 63% lived in Brooklyn, the Bronx, or Manhattan. Many more, born saperative, did not get AIDS.

Horton and your correspondents Murphy and Mulcahy (April 15, p 988) generalise that AIDS is increasing faster in women than in men. To imply that this is so in all women denies to those few at uniquely high risk the special attention that they need, while ringing alarm bells everywhere for enormous numbers of women who are not and need never be at risk.

Gordon T Stewart
Glenavon, Clifton Down, Bristol BS8 3HT, UK

Authorship inflation: a trend reversed

Sir—With the growing pile of publications, several surveys suggest a constant rise in the number of coauthors. The 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. 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 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 coauthors are not credited is difficult to achieve. But in such reports participation solely in the acquisition of funding or collection 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. This lax view on authorship is worrisome, but some solutions can be offered. First, the contributions of separate coauthors to multi-author papers should be assessed more critically as to whether individuals' efforts qualify for authorship. The decrease in mean coauthorship 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,
instead of listing each separate contributor as an author. This style of reporting is developing in recent issues of The Lancet.

J P H Drenth  
Department of Medicine, Division of General Internal Medicine, University Hospital St Radboud, 6500 HB Nijmegen, Netherlands


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 3 dB. Her serum creatinine (4-0 mg/dL) and blood urea (56 mg/dL) were raised. The haematocrit (34-8%) and she had a raised serum protein of 9-1 g/dL (normal 6-2-8-2) with a polyclonal -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% -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), -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 relationship between blood viscosity and hearing loss may help toward specific preventive strategies in patients at risk of viscous hearing loss.

Lorenz C Hofbauer, *Armin E Heufelder  
Medizinische Klinik, Klinikum Innenstadt, Ludwig-Maximilians-Universitat, Ziemssenstrasse 1, D80336 Munich, Germany


Hyponatraemia after rehydration with sports drink

Sir—A 4½-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 haemoglobin 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>
<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>90</td>
<td>60</td>
<td>5</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>K+ (mmol/L)</td>
<td>20</td>
<td>20</td>
<td>7</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Glucose (g)</td>
<td>22</td>
<td>18</td>
<td>40</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>Sucrose (g)</td>
<td>0</td>
<td>0</td>
<td>40</td>
<td>45</td>
<td></td>
</tr>
</tbody>
</table>

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

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.

T S Hornung  
Department of Paediatrics, Western Hospital, Melbourne, Australia

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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