We can offer no obvious explanation for the discrepancy between our findings and those of Rosenberg and colleagues. However, we believe that the issue has to be clarified, and the validity of the suggested role of CD44 variants in UC should be re-examined.

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Invasive pneumococcal disease in HIV-1 infected patients

SIR—Gilks and associates (March 16, p 718)[1] have reported that HIV-1 infected patients in sub-Saharan Africa are at a very high risk for invasive pneumococcal disease early in the course of HIV-1 infection. This article appeared while we were evaluating a prospective series of patients with bacteraemia at our hospital.

From June 1, 1993 to June 1, 1994, 624 episodes of bacteraemia were recorded; 35 (5·6%) occurred in patients with HIV-1 infection. Bacteraemia was clinically significant in 26 patients (74·3%), whereas nine episodes were thought to be due to contamination. There were five women and 21 men (mean age 32 [SD 6·1] years; range 24–52 years); 18 were intravenous drug users, six were homosexual men, and two had acquired HIV-1 by heterosexual contact; 17 had comorbidity (chronic hepatitis 12, malignant blood disease 3, and solid tumour 2). 23 episodes of bacteraemia were community-acquired, and three hospital-acquired. All episodes were monomicrobial, and there were no deaths due to these infections. The most common causative pathogens were: *Streptococcus pneumoniae* (8), *Salmonella enteritidis* (5), staphylococci (5), and enterococci (3). *S pneumoniae* was responsible for more than 30% of all cases of clinically significant bacteraemia in HIV-1 infected patients. In addition, HIV infection represented the main underlying disease in 28·6% of all pneumococcal bacteraemias diagnosed. No patient with pneumococcal bacteraemia had been previously diagnosed as having tuberculosis. The primary focus of infection was pneumonia in six patients, sinusitis in two, whereas occult bacteraemia was diagnosed in another patient. All those with pneumococcal bacteraemia were or had been intravenous drug users, and their mean CD4 cell count in the 3 months before diagnosis was 292 (SD 158)/μL (range 87–476), whereas that of HIV-infected patients with bacteraemia caused by other bacteria was 97 (82)/μL (range 24–345) (p=0·0003). According to the 1993 expanded CDC surveillance definition, three (37·5%) of the patients with pneumococcal bacteraemia had AIDS, whereas 16 (88·9%) of those with non-pneumococcal bacteraemia had AIDS (p=0·02).

The pneumococcus is a significant pathogen in HIV-1 infected patients,[2,3] and among our patients it was the first cause of bacteraemia, usually of pulmonary origin. Typically, it causes disease at early stages of HIV infection, and especially among intravenous drug users. This has been observed by others,[1,4] and probably reflects an increased exposure of this group of patients because of low standards of living and poor hygiene. By contrast, other bacteria (such as *Campylobacter, Salmonella,* and gram-positive cocci) usually cause disease in advanced HIV infection, sometimes related to instrumentation or indwelling vascular catheters or venous ports, and do not show preference for intravenous drug users.

Invasive pneumococcal disease in HIV-infected patients is preventable, especially in the early stages of HIV disease, when a protective response against serotypes represented in the 23-valent vaccine can develop.[5] This immunisation—which is recommended on all HIV-1 seropositive adults in developed countries—should be used on a larger scale to avoid morbidity and mortality associated with pneumococcal disease in those with viral immunodeficiency.

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Measurement of urinary iodine concentration

SIR—Urinary iodine excretion, especially iodine concentration in spontaneous urine samples, is the most widely used biochemical indicator for the assessment and monitoring of iodine status.[1,2] Furthermore, in the evaluation of renal, urinary, and metabolic disorders multi-parameter urine test strips are commonly used under field conditions as well as in routine laboratories.

In a study to compare urinary iodine concentrations in spot and timed 24 h urine specimens from the same children (n=100, age 3–6 years), iodine analysis[3] was performed with (spontaneous samples) and without (24 h samples) prior dipping of a urine test strip (Combur 8, identical to Chemstrip 8, Boehringer Mannheim Corporation, Mannheim, Germany). Spot urine samples (one per child) were collected 1–3 weeks before collection of a second specimen, ie, a timed 24 h urine sample in each child. Initial urine testing with Combur 8 in the spot samples served as rapid noninvasive biochemical health check.

Normally, average urinary iodine concentrations of spontaneous and 24 h urine samples from the same region are almost undistinguishable. As shown in the table we found urinary iodine concentrations in the dipped spot urine samples which were on average 10-fold higher than those measured in the non-dipped 24 h urine samples.

Laboratory checks on each individual test patch of the dipstick (all patches were separated before they were briefly

Urine samples	n	Median	Mean (SD)	Range
Dipped	100	75·32	88·10 (69·13)	5·90–413·70
Non-dipped	100	6·81	8·59 (8·70)	0·58–84·45

Table: **Urinary iodine concentration (µg/dL) measured in spot urine samples ("dipped with urine test strips") and 24 h specimens ("non-dipped") from the same children**

immersed in distilled water or pooled urine samples) revealed that iodine contamination was exclusively caused by the patches "blood" and "glucose". Both patches produced increases in iodine concentration readings corresponding to 45–50 µg/dL. Consultation with the manufacturer yielded the information that both of these patches have an iodate-containing nylon mesh attached in order to minimise interference with strong reducing agents such as ascorbic acid. The manufacturer further confirmed that this iodate is readily soluble and that the resulting interference with urinary iodine quantification observed by us has not received attention hitherto.

Perhaps, the findings reported here can explain—in addition to the well-known (major) causal factors of ingestion and topical application of iodine containing drugs—why extremely raised urinary iodine concentrations are frequently reported.[4,5] In future to avoid this form of iodine contamination multi-parameter urine testings should be performed in separate aliquots of (freshly voided) urine samples.

We acknowledge support by grants from the Forum Schilddrüse e.V.

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DNR orders in acute stroke

SIR—Landi (March 30, p 848)[1] states that the recommended Canadian guidelines for do-not-resuscitate (DNR) orders in patients with acute stroke "imply a threshold for quantitative futility greater than 5%". However, his argument is based on survival rates for stroke patients in general rather than for those who have received cardiopulmonary resuscitation (CPR). There are few data regarding survival and neurological outcome after CPR in patients with acute stroke, probably because most stroke patients judged at high risk for death receive DNR orders. In three large studies in acute hospitals,[2–4] none of 46 patients with recent stroke survived to discharge following resuscitation, but the 95% confidence intervals for survival based on these results are wide (0–7·7%).

There are several reasons why patients with acute stroke are unlikely to do well following resuscitation. Many deaths in the weeks after a stroke are associated with brain swelling and coning; cardiac arrest in such patients is usually terminal. Even where cardiac arrest results from arrhythmia, it seems probable that patients with recent focal brain damage will be especially vulnerable to the effects of global cerebral hypoxia and ischaemia due to cardiac arrest. Additionally most stroke patients are managed on general or rehabililtation wards, where delays in initiating resuscitation further reduce the likelihood of a successful outcome.[2]

It may never be possible to support with data guidelines for DNR orders in patients with acute stroke, because so many will not be considered for resuscitation. In any event, decisions regarding resuscitation do not depend totally on outcome data but inevitably reflect the values and prejudices of physicians and of society. Thus, it is appropriate that DNR guidelines will differ between different countries. We suggest that DNR orders are generally appropriate in acute stroke patients with any one of the criteria suggested by Alexandrov and colleagues (severe stroke, life-threatening brain damage or major comorbidity).[5] However, individual patient factors must be taken into account in making a decision, and changes in clinical condition should lead to reconsideration of DNR orders.

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Body temperature and infection in acute stroke

SIR—Reith and colleagues (Feb 17, p 422)[1] convincingly show that in acute human stroke, body temperature is associated with stroke severity, infarct size, mortality, and outcome. The challenging question is, what causes the rise in body temperature in acute stroke—preceding or simultaneous infection or cerebral tissue necrosis?

In a recent case-control study, we identified infection within 1 week before ictus as an important and independent risk factor for ischaemic stroke (odds ratio 4·5; 95% CI 2·1–9·7). Certified or probable infection was diagnosed in 26% of our patients (n=197). Upper respiratory tract infection and purulent bronchitis were the most frequent diagnoses followed by pneumonia and urinary tract infection. Similarly to Reith, we detected a positive association between infection and stroke severity.[2,3] Reith and co-workers showed an infection in 16% of their patients, and they diagnosed only pneumonia and urinary tract infection. In our opinion, they probably underestimated the prevalence of infection before and simultaneous to stroke, especially upper respiratory infection and bronchitis. What were their criteria for diagnosis of infection? On the basis of a higher prevalence, the impact of infection on infarct size and outcome after stroke may be more important than is shown by their analyses. We feel that it cannot be concluded that cerebral tissue necrosis possibly causes raised body temperature; it is more likely that an undiagnosed infection was the cause of raised body temperature in many cases. Reith and colleagues mention that no patient received steroids, but provide no data about aspirin and other antipyretic drugs.