patients, and can also contact our hospital information services.

Muramoto seems concerned with Jehovah's Witnesses' theology, but his comments do little to advance effective non-blood medical management or to enhance respect for individual autonomy. Malyon[5] has discussed these issues in detail.

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1 Muramoto O. Jehovah's Witnesses and blood transfusions. *Lancet* 1998; **352:** 824.
2 Rosengart TK, Helm RE, DeBois WJ, et al. Open heart operations without transfusion using a multimodality blood conservation strategy in 50 Jehovah's Witness patients: implications for a "bloodless" surgical technique. *J Am Coll Surg* 1997; **184:** 618–29.
3 Trouwborst A. Acute hypervolaemic haemodilution to avoid blood transfusion during major surgery. *Lancet* 1990; **336:** 1295–97.
4 van Iterson M. Systemic haemodynamics and oxygenation during haemodilution in children. *Lancet* 1995; **346:** 1127–29.
5 Malyon D. Transfusion-free treatment of Jehovah's Witnesses: respecting the autonomous patient's rights. *J Med Ethics* 1998; **24:** 302–07.

Advertising claims for *The Lancet*

Sir—I was entertained by one claim—"Only one journal keeps you on track throughout your career in medicine"—and one implied claim—"Whose judgments are always reliable?"—in the advertising material for *The Lancet* enclosed in the journal.

Could we have some more information about the study? What populations were involved, what were the sample sizes, what other journals were considered, what were the statistical analyses, and what were the results? The absence of an adequate description of the work makes it difficult to judge quality. I suspect that this study might not meet the criteria required of evidence-based medicine. I also suspect that, if *The Lancet* were a drug, then a letter from the Medicines Control Agency might be on its way.

Should not advertisements for *The Lancet* meet the standards required of papers and of other advertisements in the journal? Or is *The Lancet* making a subtle protest against the occasional lunacies of the thought police? I hope the latter.

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The leprosy of Henry VII: incarceration or isolation?

Sir—German King Henry VII (1211–42), eldest son of Emperor Frederic II, died in 1242 and was buried in the cathedral of Cosenza (Calabria, southern Italy). An exploration of his precious Roman sarcophagus in November, 1998, revealed a skeleton, in partial anatomic connection, of a vigorous man, aged 30–35 years and 1·72 m high.

The palaeopathological study showed severe deformity of the left rotula, by juvenile fracture of the knee, certainly causing claudication. Chroniclers report that the nickname of Henry VII was "the lame". The facial bones revealed: total resorption of the anterior nasal spine with exposure of cancellous bone and partial cortical "capping" (figure); smooth remodelling of the inferior margins of nasal aperture; bilateral, extensive fine pitting, with remodelled subperiosteal new bone, of the nasal surface of the palatine; and confluent pits and erosions on the oral surface in the mid-zone of the palatine processes. These facial findings are patognomonic of the *facies leprosa*[1] or rhinomaxillary syndrome[2] of leprosy.

The feet showed bilateral thinning of the distal diaphyses of the fourth metatarsals and the proximal phalanxes, with typical resorption of the metatarsophalangeal joints, and severe femoral and tibial periostitis, findings also present in leprosy.[3] We conclude that this was a case of lepromatous leprosy; the first to be diagnosed by osteoarchaeological methods in Italy.

It is well known that Henry VII rebelled against the father but was defeated in 1235. After his submission, he was confined in the castles of southern Italy and committed suicide, by a fall into a ravine, in 1242, after 7 years of imprisonment.[4,5] With this discovery we now know that Henry VII was affected by a severe form of leprosy. This disease certainly started some years before his death and the disfiguring conditions must have obliged him to a forced isolation, until his dramatic suicide. On the basis of this scenario, Frederic II appears not only less cruel as a father, but also to be absolved from the grave suspicion of his son's murder.



Total resorption of the anterior nasal spine with exposure of cancellous bone and partial cortical "capping"

Research funded with European funds POP M.4.4-Calabria Region.

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1 Møller-Christensen V. Leprosy and tuberculosis. In: Disease in ancient man, 1983: 129–38.
2 Andersen JG, Manchester K. The rhinomaxillary syndrome in leprosy: a clinical, radiological and palaeopathological study. *J Osteoarcaeol* 1992; **2:** 121–29.
3 Aufderheide AC, Rodriguez-Martin C. The Cambridge University of human paleopathology. Cambridge: Cambridge University Press, 1998.
4 Kantorowicz E. Kaiser Friedrich der Zweite. Düsseldorf und München: Verlag Helmut Küpper Vormals Georg Bondi, 1976.
5 Abulafia D. Frederick II. A medieval Emperor. London: Penguin Press, 1988.

DEPARTMENT OF ERROR

Trimethoprim and fasting plasma homocysteine—In this Research letter (Dec 5, p 1827) by Y M Smulders and others line 7 of the second to last paragraph should read "Although the effect on DHFR is probably most important, trimethroprim may be involved in other steps of homocysteine remethylation or trans-sulphuration." The last paragraph should have read:

The clinical implications of our findings may be important. It is possible that the temporal relation between hyperhomocysteinaemia and thrombosis is shorter than that for atherosclerosis. Although a short course of trimethoprim can probably do no harm, an increase in the risk of thombosis in susceptible people is theoretically possible. The practice of prescribing long-term (prophylactic) trimethoprim should be reassessed in patients with a high cardiovascular risk profile, cardiovascular disease, a history of thrombosis, or who are being treated for hyperhomocysteinaemia. In six patients who completed the full 2-week study period, we assessed the methylenetetrahydrofolate reductase (MTHFR) genotype by PCR testing for the C to T mutation at nucleoside 677, which generates so-called "thermolabile MTHFR". One patient was homozygous for this mutation (TT genotype), one was homozygous wild type (CC), the remainder were heterozygous (CT). The increase in homocysteine in response to trimethoprim was 191% in the TT subject, 79% (median) in the CT subjects, and 40% in the CC subject. Although it concerns only a few observations, we speculate that inter-individual differences in the susceptibility to the effect of trimethoprim on homocysteine levels are partially explained by MTHFR polymorphism.

Differences between asthma exacerbations and poor asthma control—In this Article by H Reddel and colleagues (Jan 30, p 364) under the subheading "Peak expiratory flow analysis" of the Methods section the coefficient of variation should have been stated as being calculated as daily SD/mean.