When comparing the recent report by Olsen *et al*[3] with the original paper by Carlsen *et al* I noticed differences in the graphs. Figure 1 of the original paper contains data for only 31 of the 61 publications listed in the table: 30 data points are missing. Furthermore, the difference in the circles' areas is greater than expected: the ratio of the maximum to the minimum number of subjects in the studies, expressed as a logarithm, is 4·3, but the difference in the areas of the circles is far larger (fig 1a). Using the data given in the original paper's table, I redrew the figure (fig 1b). The overall impression is quite different.

I wonder about the reason(s) for these mistakes. As this paper had a considerable impact not only in the scientific community but also in the lay press, it is difficult to comprehend why these severe errors have been overlooked both before and after publication.

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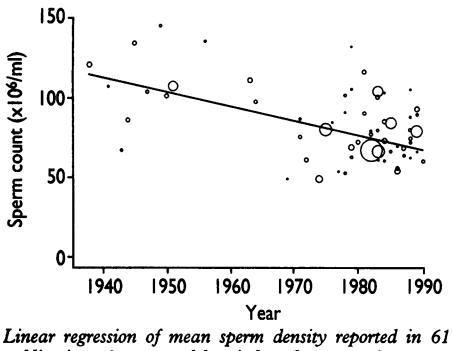
1 Carlsen E, Giwercman A, Keiding N, Skakkebaek NE. Evidence for decreasing quality of semen during past 50 years. *BMJ* 1992;305:609-13.
2 Brake A, Krause W. Decreasing quality of sperm. *BMJ* 1992; 305:1498.
3 Olsen GW, Bodner KM, Ramlow JM, Ross CE, Lipshultz LI. Have sperm counts been reduced 50 percent in 50 years? A statistical model revisited. *Fertil Steril* 1995;63:887-93.
4 Bromwich P, Cohen J, Stewart I, Walker A. Decline in sperm counts: an artefact of changed reference range of "normal"? *BMJ* 1994;309:19-22.

Authors' reply

EDITOR,—As Alexander Lerchl points out, figure 1 of our overview indicating that sperm concentrations have decreased during the past 50 years is deficient. During the final preparation for publication, for reasons that we cannot trace, some of the points were omitted. The regression analysis in the paper is unaffected by this: the new regression line (weighted by the number of subjects) had a slope of $-0{\cdot}934\times10^6$/ml per year (SE 0·157; $P<0{\cdot}0001$), and that line was correctly included in our original figure. A better impression of the regression analysis is provided by the figure in this letter, in which the areas of the circles are proportional to the number of subjects in each publication. There is no reason for Lerchl's scepticism.

Lerchl quotes several criticisms of our paper but omits our detailed and specific responses as well as the subsequently published empirical evidence, which points in the same direction as our paper.

Specifically, Lerchl quotes Brake and Krause, who, on the basis of our data, claimed that sperm concentration had significantly increased since 1970. In fact, Brake and Krause made a mistake in their calculation: the increase they quoted is non-significant ($P=0{\cdot}36$). Lerchl quotes Bromwich *et al*, who offered a speculative, elementary statistical argument with no empirical basis or verification. Lerchl fails to quote our earlier detailed comments on this theoretical exercise.[1,2] Lerchl finally quotes the recent report by Olsen *et al*, who also did not add new empirical evidence: they performed various unsurprising reanalyses of our data, all of which agreed about a significant decline in sperm concentration. We have submitted detailed comments on these reanalyses elsewhere.



Linear regression of mean sperm density reported in 61 publications (represented by circles whose area is proportional to number of subjects in study), each weighted according to number of subjects, 1938-90

Lerchl omits to refer to the additional empirical evidence that has been published. Auger *et al* (who were originally motivated by serious scepticism about our original report) studied 1351 healthy men volunteering to donate sperm in one clinic in Paris between 1973 and 1992.[3] Carefully separating age effects from cohort effects (year of birth), they documented a highly significant decrease in sperm count of 2·1% per year (from 89×10^6/ml in 1973 to 60×10^6/ml in 1992) and concomitant decreases in the percentages of mobile and normal spermatozoa. Three additional, shorter reports have been published, also based on data from one clinic and all with similar conclusions.

In a recent international effort the temporal trends in semen quality were viewed in a broader context.[4] There have been similar temporal increases in the incidence of testicular cancer and frequently of hypospadias and cryptorchidism, and geographical covariation of several of these symptoms as well as male breast cancer has been documented. In our view it would be irresponsible to disregard this evidence, even if the link to possible determinants is far from definitively established.

Although Lerchl points out a (qualitatively unimportant) deficiency in figure 1 of our paper, we hope that this will not delay a dedicated, wide ranging research effort to clarify these issues.

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1 Keiding N, Giwercman A, Carlsen E, Skakkebaek NF. Importance of empirical evidence. *BMJ* 1994;309:22. [Commentary on: Bromwich P, Cohen J, Stewart I, Walker A. Decline in sperm counts: an artefact of changed reference range of "normal"? *BMJ* 1994;309:119-22.]
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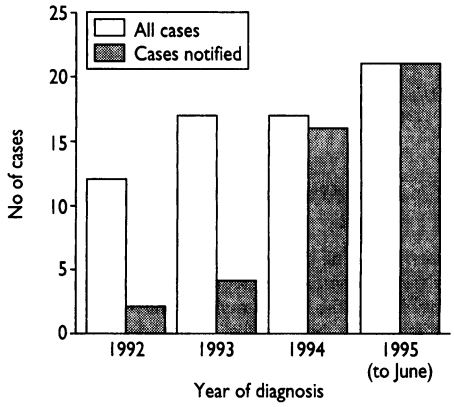
3 Auger J, Kunstmann JM, Czyglik F, Jouannet P. Decline in semen quality among fertile men in Paris during the past 20 years. *N Engl J Med* 1995;332:281-5.
4 Male reproductive health and environmental oestrogens [editorial]. *Lancet* 1995;345:933-5.

Reasons for increased incidence of tuberculosis

Audit suggests that undernotification is common

EDITOR,—In her editorial Janet H Darbyshire suggests that undernotification of tuberculosis, particularly in association with HIV infection, is still common.[1] If sufficiently widespread, undernotification could result in underestimation of the incidence of tuberculosis, particularly in patients coinfected with HIV, with considerable public health implications. We recently audited notification of tuberculosis in patients known to be infected with HIV who were attending our hospital.

A database on all patients with mycobacterial infection was established by searching microbiology, histopathology, and clinical computerised records systems. Case notes were then examined for all patients. Patients were considered to have tuberculosis on the basis of a positive result of culture of a specimen from any site or either histological or radiographic changes compatible with tuberculosis and a response to standard antituberculous treatment. This database was then cross referenced with a record of notifications for the whole hospital. The figure shows the results.



Number of cases of tuberculosis diagnosed and notified in patients with HIV infection, 1992-5

Tuberculosis was considerably undernotified in 1992. The reasons for this were not clear from this audit, but the appointment of a clinical nurse specialist who had specific responsibility for notification of, and contact tracing in, cases of tuberculosis and HIV infection led to a substantial improvement in the rate of notification. This suggests that clinicians' concerns about patient confidentiality were not the prime reason for undernotification. In addition, a considerable increase in the numbers of cases of tuberculosis in patients also infected with HIV has been seen this year. Although the number of notifications of tuberculosis from our hospital has risen, from 99 in 1992 to 60 in the first six months of this year, the proportion of patients with HIV infection has increased from 17% to 32% over the same period. This seems to be due to increased screening for HIV infection in patients with tuberculosis. We have thus shown that although undernotification of tuberculosis in patients with HIV infection occurs, improved notification may also lead to increased recognition of coinfection with HIV.

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1 Derbyshire JH. Tuberculosis: old reasons for a new increase. *BMJ* 1995;310:954-5. (15 April.)

Large immigrant population may have confounded study

EDITOR,—N Bhatti and colleagues present an interesting analysis of changing rates of notification of tuberculosis based on national notifications and local data from Hackney.[1] The findings are interpreted as suggesting that the national increase is largely due to socioeconomic factors that have affected the white population and established ethnic minority communities to a similar extent. The authors suggest that recent immigration has made only a small contribution to this increase. The study's findings do not justify these conclusions.

As quoted in the paper, markers of socio-

economic deprivation are highly correlated with the proportion of the population from ethnic minorities. Ethnic group is also a strong risk factor for tuberculosis. As such, ethnic group is potentially an important confounder of the relation between tuberculosis and social deprivation, which was not accounted for in this analysis. The authors state that the increase in rates of tuberculosis in Britain is confined to the most socially deprived areas. This is potentially misleading because the geographical distribution of the increase could reflect the ethnic composition of the populations rather than population levels of social deprivation.

The analysis of local data shows that the number of notifications increased in the groups classified as white or of Indian subcontinent or West Indian origin. The assertion that the sizes of these groups in Hackney remained stable over the study period is said to be based on comparison of census data for 1981 and 1991. The 1991 census was the first to include ethnic group as a variable, so it is unclear how changes in the sizes of the populations were ascertained.

Nearly half of the increase in notifications in Hackney occurred among newly established refugee and immigrant populations. Estimates of the size of these populations are likely to be highly inaccurate. Newly established populations are said by the authors to be unusually large in Hackney even when compared with those in other socioeconomically deprived boroughs. For this reason, immigration is thought likely to have played only a small part in the national increase. As we are not told the numbers and ethnic mix of refugees in other socioeconomically deprived districts it is difficult to predict how important their contribution to the increase in notifications may be.

We believe that caution should be used in interpreting the results of the national ecological study and in extrapolating the results from Hackney to other socioeconomically deprived districts. This analysis has provided valuable information about the epidemiology of tuberculosis in Hackney, but different factors may be important in other districts.

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1 Bhatti N, Law MR, Morris JK, Halliday R, Moore-Gillon J. Increasing incidence of tuberculosis in England and Wales: a study of the likely causes. *BMJ* 1995;310:967-9. (15 April.)

The 10 commandments of accident and emergency radiology

EDITOR,—A H Troughton and I Kendall[1] and T F Beattie and colleagues[2] comment on our article giving 10 commandments of accident and emergency radiology.[3] As we stated in the introduction, the article was written for inexperienced senior house officers working in accident and emergency departments. The authors of the letters either did not read this or are unaware that, commonly, inexperienced accident and emergency doctors work without direct supervision.[4]

In answer to Troughton and Kendall's comments, we wish to point out the following. Rules 1, 2, and 3 emphasise the need to assess the patient first and order a radiograph only if an underlying abnormality is considered. Radiographs of joints with long bones should not be obtained if the joints are clinically normal. The article states the need to have "an accessible library of normal radiographs in the accident and emergency department as well as reference books that show normal variants."[3] Radiographs must be obtained before foreign bodies are removed because only in this way can the most appropriate anaesthetic and operative procedure be chosen.

In contrast with what Beattie and colleagues imply, the Royal College of Radiologists' recommendations state, "The one possible exception [for comparison views] is the opposite elbow when there are strong clinical signs of fracture with no radiological evidence and no available radiologist." No further radiographs are required while the patient is in the *x* ray department if the initial radiograph is normal or obviously abnormal. Rule 9 emphasises that inexperienced doctors should seek experienced advice when a radiograph does not look quite right but no specific diagnosis can be made. Beattie and colleagues' statement that "the diagnosis of subtle fractures around the elbow joint will not necessarily change the clinical management" is a dangerous concept for inexperienced senior house officers. In our experience this can be interpreted as, "If you do not see an abnormality it is not important." Both statements rely on an experienced clinician viewing the radiograph.

Owing to lack of space, several points that we wished to incorporate in our article had to be deleted. They included the comment that variation in the alignment of joints is important to note (when comparing two sides) because it may obscure abnormalities. The subsequent publication, the *ABC of Emergency Radiology*, includes this comment.[5]

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1 Troughton A, Kendall I. Accident and emergency radiology. *BMJ* 1995;310: 1605. (17 June.)
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Differences in mortality after fracture of hip

Doubt remains over anticoagulant prophylaxis for deep venous thrombosis

EDITOR,—Anticoagulant prophylaxis may reduce the incidence of venous thromboembolism in hospital, but C J Todd and colleagues' study does not dispel doubts about its overall effect on mortality.[1] There was no significant difference between the survival rate at 90 days (82·2% at the four hospitals that routinely gave such prophylaxis (to 79·5% of patients)) and 82·0% at the other four hospitals (prophylaxis given to only 16·1%) despite the fact that 62·3% of patients in the former hospitals had surgery within 24 hours (favourable to outcome) compared with only 50% in the latter ($P<0·01$, χ^2 test). Hospital 4, which gave prophylaxis to the highest proportion of patients (91%), showed joint lowest 90 day survival (76%). In hospitals 5 and 8, in which only 10·5% of patients were given prophylaxis, 83% survived despite 4% dying of pulmonary embolism. None of the 13 patients diagnosed at necropsy as having fatal embolism was taking anticoagulants, but we wonder how many of these patients had recognised contraindications,[2] especially other conditions rendering them particularly prone to thromboembolism. Haematoma with subsequent infection is a worry with anticoagulants, and wound infection occurred in 9·8% (25/256) of patients in the hospitals that routinely gave anticoagulants compared with 4·9% (15/304) in the others ($P<0·01$).

There is a danger of relying on anticoagulants in hospital and neglecting to teach patients and carers about mechanical prophylaxis, including postures to increase the velocity of femoral vein blood,[3] the avoidance of bad postures, and the value of leg movements, since the risk of thrombosis continues after discharge from hospital.[2] Either infection or venous stasis would predispose to thromboembolism after discharge: did all 82 patients who died undergo necropsy?

Another factor that can affect outcome is the packed cell volume: lower values indicate lower viscosity (a value $\leq 0·33$ discourages thromboembolism[4]) and better tissue oxygenation than at higher values. Thus it would be interesting to know whether thromboembolism and survival correlated with differences in policy concerning transfusion of red cells or haemodilution. The report by the Thromboembolic Risk Factors Consensus Group can be criticised for omitting to mention posture or packed cell volume.[5] The decision whether to give anticoagulant treatment to a patient should take into account many factors, including the risk of haemorrhage as well as the risk of thrombosis. For many patients, mechanical methods to increase the peak velocity of femoral vein blood combined with haemodilution (for example, not replacing red cells lost) could be safer overall, especially if monitored by routine postoperative venography or colour-duplex scans and if clinically important thromboses are treated. Raising the legs moderately can enhance the blood velocity[3] and is easily continued after discharge. We agree that more research is needed, particularly large scale trials of alternatives to anticoagulant treatment.

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1 Todd CJ, Freeman CJ, Camilleri-Ferrante C, Palmer CR, Hyder A, Laxton CE, *et al.* Differences in mortality after fracture of hip: the East Anglian audit. *BMJ* 1995;310:904-8. (8 April.)
2 Kakkar VV, Cohen AT, Edmondson RA, Phillips MJ, Cooper DJ, Das SK, *et al.* Low molecular weight versus standard heparin for prevention of venous thromboembolism after major abdominal surgery. *Lancet* 1993;341:259-64.
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4 Duruble M. Prophylaxie des thromboses veinouses profondes. *Phlebologie* 1982;35:143-72.
5 Thromboembolic Risk Factors (THRIFT) Consensus Group. Risk of and prophylaxis for venous thromboembolism in hospital patients. *BMJ* 1992;305:567-74.

Casemix factors may not have been considered sufficiently

EDITOR,—The results of C J Todd and colleagues' audit report on 90 day mortality after admission to hospitals in East Anglia with fractured hip seem to suggest an impressive survival advantage if a patient is admitted to one particular hospital (hospital 6).[1] The authors state that this is probably due to factors associated with the care provided at that hospital.

How can the authors be sure that other casemix factors did not account for the observed differences in 90 day mortality? The favourable "process" measures reported for hospital 6 (low rates of pressure sores and wound infections and the early mobilisation of patients) may have reflected excellent care but could equally have been a result of less sick patients being admitted in the first place and this not being detected with the casemix measures