scoring systems may be valuable in determining which patients require referral to the regional trauma centre. The injury severity score, however, is not an appropriate system for triaging patients prospectively as definitive scoring requires accurate operative or necropsy anatomical data, which cannot be obtained either at the roadside or during initial resuscitation, but is of most value in retrospective audit. The revised trauma score provides a guide based on physiological variables (systolic blood pressure, respiratory rate, and Glasgow coma scale), and its value for prospective use to ensure that patients receive treatment at the correct facility is proved.

Although the department studied by Dr Kinny and Mr Jones was small by United Kingdom standards, with 21 000 patient attendances each year, there were 50 deaths from trauma over a period of 12 months. There is no detailed analysis considering whether any of these deaths were potentially avoidable, but the 30% preventable death rate from trauma shown elsewhere in the United Kingdom[2] possibly applied to north Wales. Outcome may be influenced by an improved trauma system based on a regional trauma centre, which, in addition to dealing with acute injury, must also maintain standards of prehospital care throughout the catchment area and provide facilities for intensive care and rehabilitation. Papers based solely on data on mortality from trauma fail to consider the impact of trauma centre management on reducing morbidity and allowing early return to full function; such management may not be provided elsewhere.

The total number of admissions to a trauma centre will be greater than predicted by Dr Kinny and Mr Jones as there will inevitably be patients who, after full evaluation, are deemed to have been referred inappropriately. In the United States there is concern if these patients constitute less than 10-15% of the total as this implies that access is denied to other patients who should have been referred. Patients with major trauma represent a small minority of the total number of attendances, and the volume and nature of trauma dealt with at the district general hospital level will still be more than adequate to maintain interest and morale.

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1 Kinny SJ, Jones DAH. Trauma service requirements in a district general hospital serving a rural area. *Br Med J* 1990;**300**:504-8. (24 February.)
2 Anderson ID, Woodford M, de Dombal FT, Irving M. Retrospective study of 1000 deaths from injury in England and Wales. *Br Med J* 1988;**296**:1305-8.

SIR,—Dr S J Kinny and Mr D H A Jones have found that very few patients with trauma need to be transferred from a district general hospital in a rural area to a regional trauma centre.[1] We have compared trauma services in rural Britain and France and found that district general hospitals in Britain offer a much greater depth of experience than is available at district level in France, where regional centres are well established.[2] For example, in south west France, away from the regional centre in Bordeaux, some 87 oral and maxillofacial surgeons in private practice treat an average of only four patients with jaw fractures annually, whereas in South Western Regional Health Authority all 14 consultants in the specialty treat at least 50 such patients each year. The need for concentration of services at subregional level in France, as occurs in Britain, is clear, though the optimum distribution of British regional or supraregional trauma centres to serve rural and urban populations has yet to be established.

It is inappropriate, however, to organise services only on the basis of European or American experience. Not only does Britain generate fewer patients with major trauma than equivalent populations elsewhere,[1,2] but systems of private health care, which are not noted for their control of manpower facilities or continuing education in rural areas, need regional centres to manage a great deal of cases of minor trauma as well as cases of major trauma. Market forces may determine numbers of specialists in a particular area in relation to demand for elective surgery, but the need for trauma services is very different, particularly in poorer areas where accidents and assaults are more likely.[3] Surely a lesson from international comparisons and from the findings of Dr Kinney and Mr Jones is that, supplemented by regional centres, district health authority trauma services in rural Britain are a jewel in the crown of the NHS and should be used as a model elsewhere in Europe and in the United States.

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1 Kinny SJ, Jones DHA. Trauma services requirements in a district general hospital serving a rural area. *Br Med J* 1990;**300**:504-7. (24 February.)
2 Timoney N, Saiveau M, Pinsolle J, Shepherd JP. A comparative study of maxillo-facial trauma in Bristol and Bordeaux. *J Craniomaxillofac Surg* 1990;**18**:74-7.
3 Shepherd JP. Violent crime in Bristol: an accident and emergency department perspective. *British Journal of Criminology* (in press).

SIR,—We commend Mr S J Kinny and Mr D H A Jones for their excellent paper on trauma services requirements in a district general hospital serving a rural area.[1] This particular form of audit allows an in depth analysis of many of the problems related to management of trauma. We agree with their observations that the demand for acute general and thoracic surgery in patients with trauma is uncommon, but when required it is needed urgently. They have identified that in their hospital they had four multiply injured patients (with an injury severity score ≥ 16) in one month and that they transferred three patients for further management elsewhere. Their conclusion that trauma centres are not necessary therefore seems inappropriate.

In addition, the area of the country that their study covered is one of the least densely populated areas in the United Kingdom, with little industry and no fast roads. The situation is obviously different in a busy urban area in the midlands with fast roads and plentiful industry. Trauma centres seem to have a considerable role in such an environment. To identify the size of the problem and the number of patients with trauma who have given us cause for concern and thus required special attention we did an analysis of patients with trauma who were admitted to our medical centre.

The centre provides services to the health districts of Nottingham, the northern part of Leicestershire, the eastern part of south Derbyshire, and the southern part of central Nottinghamshire. Its "home" population size is about 800 000, for which it is the local and only accident department. Because it is the base for a regional neurosurgical service it not only attracts patients with head injuries from its own area (these are primarily referred to a consultant neurosurgeon) but also accepts patients with severe head injuries from other districts. With regard to patients with orthopaedic trauma it provides a service for the home population (patients are primarily referred to an orthopaedic surgeon) but because of the special interests of some of the orthopaedic consultants it accepts patients with severe orthopaedic trauma from other districts. The patients analysed were allocated to the most appropriate group, although many had both neurosurgical and orthopaedic injuries, and there was no duplication of cases. An analysis of all patients admitted to the intensive therapy unit after suffering trauma who had an injury severity score of ≥ 16 allows a better understanding of the problem (table).

Although these figures include 13 patients who were injured in the M1 plane crash (who have been included in the home population figures: two with head injuries and 11 with orthopaedic trauma), they do not include children admitted to the paediatric intensive therapy unit and those patients admitted only to the wards at the hospital and not to the intensive therapy unit.

Currently the channelling of patients to trauma centres is not routine, but it is already happening to a lesser or greater extent. We believe that the development of trauma centres will change the pattern of referral for only a limited number of patients who are likely to benefit appreciably from the skill that will be concentrated in such centres. It is our view that this is in the best interests of the severely injured patient. These patients, however, are expensive, with regard to both the beds they utilise and the facilities they require, and this needs to be considered carefully in the future. We are pleased to note that the Department of Health has taken positive action in exploring further the concept and practicalities of introducing trauma centres in England and Wales.

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1 Kinny SJ, Jones DHA. Trauma services requirements in a district general hospital serving a rural area. *Br Med J* 1990;**300**:504-8. (24 February.)

Leukaemia and lymphoma among young people near Sellafield

SIR,—Although Dr Martin Gardner and colleagues have done a fine epidemiological study of increased leukaemia cases around nuclear installations[1]; their explanation, that the fathers' occupational exposure to radiation is associated with this increase, does not seem to be congruent with several biological and radiobiological principles.

It is implied in the explanation that a dominant mutation is being induced in the fathers' sperm and that this then specifically leads to leukaemia on a probabilistic basis.

A very high frequency of this class of mutations inducing leukaemia would have to be imagined. If there are about 4000 male workers and about 10 000 progeny are sampled, then five excess cases

Number and length of admissions of patients with trauma to intensive therapy unit from 1 January 1989 to 31 December 1989

	Head injuries		Orthopaedic injuries	
	Patients from home population	Patients from other districts	Patients from home population	Patients from other districts
Total No of patients	20	28	37	5
Mean stay in intensive therapy unit (days)	4·4	6·32	9·24	11·4
Total No of bed days	88	177	342	57

of leukaemia would yield an estimated frequency of 0·05% (compared to an average spontaneous frequency of 10^{-5} per locus).

Ionising radiation is considered a "democratic" agent for inducing mutations, and a specific class of mutations would be difficult to explain. If radiation is still "secular" it would induce other mutations as well. A virtual genetic meltdown (to use Bob Haynes's term) would be expected.

Part of the difficulty of inducing a specific class of mutations may be overcome by assuming certain breakage events in the chromosome(s)—for example, a breakage-fusion-bridge cycle—but this is likely to put that sperm at a competitive disadvantage because it then would carry a large deletion.

Valerie Beral has correctly pointed out[2] that increased leukaemia is not detectable in the progeny of the survivors of Hiroshima-Nagasaki bombings. These studies, taken together, would implicate "something else" for the raised incidence for leukaemia in Seascale.

Latent periods for inducing leukaemia (somatically) in bombed populations from Hiroshima and Nagasaki are known. Do the germinally induced leukaemias have similar latent periods? Are they similar to the leukaemias not induced by radiation?

Perhaps there are counterarguments or flaws in the foregoing; I would like to hear these.

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1 Gardner MJ, Snee MP, Hall AJ, Powell CA, Downes S, Terrell JD. Results of a case-control study of leukaemia and lymphoma among young people near Sellafield nuclear plant in West Cumbria. *Br Med J* 1990;**300**:423-9. (17 February.)
2 Beral V. Leukaemia and nuclear installations. *Br Med J* 1990; **300**:411-2. (17 February.)

SIR,—In the study of Professor Martin J Gardner and colleagues[1] on leukaemia and lymphoma among young people near Sellafield no attention was given to the paternal age of the radiation workers. In a recent survey of sporadic hereditary retinoblastoma we observed a "paternal age effect"—that is, we noted in the offspring of older fathers a considerably increased risk of hereditary tumours (D J Derkinderen *et al*, unpublished work). So if the incidences of tumours in the offspring of radiation workers are to be compared with those in the general population the effect of the paternal age should be discounted.

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SIR,—If irradiation can cause leukaemia in a child as a result of radiation damage to the father's sperm—that is, by causing a dominant mutation—the consequences for understanding carcinogenesis in general outweigh any application to a particular occupational hazard. Professor Martin J Gardner and colleagues did no more than claim that paternal irradiation explains statistically the occurrence of four cases of leukaemia.[1] They took the greatest pains to avoid epidemiological biases, but their statistical tests depended on accepting annual records of occupational radiation exposures at face value as if they truly represented radiation doses to parental testes and seminal vesicles.

When trying to relate annual exposure records more closely to conception Professor Gardner and colleagues halved the annual record and called that the radiation dose during the six months before conception. This is valid only if paternal exposures were uniform month by month during the year. Moreover, sperm have a limited life time; they continue to be made all the time, and those irradiated four to six months before conception of a child must represent a very small proportion of the sperm in the ejaculate responsible for the conception. Even sperm made between two and three months before the conception will have formed a far smaller proportion than the sperm made during the month before. A much more critical assessment is needed that takes account of the biology of conception to support the claim that it is an irradiated sperm that caused each excess case of childhood leukaemia at Sellafield.

It is obligatory, when reporting annual occupational exposures to radiation to make an allowance for missing records (for example, loss of a film badge). The missing record must be given the value corresponding to the annual dose limit—that is, one thirteenth of the dose limit for 52 weeks if the film badges are changed every four weeks. In most circumstances now the maximum monthly dose for a radiation worker is such a "nominal dose," a dose it was most unlikely he ever received. It is forbidden by regulation to replace the missing record by any more realistic assessment, such as the average of preceding and succeeding film badge readings. When the annual dose limit is 50 mSv, as it has been recently, and as 150 mSv (as it was in the 1950s), such a four week nominal dose would have been about 4 mSv and 12 mSv respectively. Thus the dose groupings used by Professor Gardner and colleagues to show an increased risk of childhood leukaemia with increase in exposure during the six months before conception (1-4 mSv, 5-9 mSv, and >10 mSv; table VI) are critically dependent on whether the recorded radiation exposure of the father of a child with leukaemia was inflated by a dose he never had.

Professor Gardner and colleagues now have the detailed occupational dose records and will be able to dispose of these methodological uncertainties. Did no referee draw the authors' attention to uncertainties that deserved mentioning?

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1 Gardner MJ, Snee MP, Hall AJ, Powell CA, Downes S, Terrell JD. Results of a case-control study of leukaemia and lymphoma among young people near Sellafield nuclear plant in West Cumbria. *Br Med J* 1990;**300**:423-9. (17 February.)

AUTHORS' REPLY,—Dr N K Notani gives an interesting discussion of the genetic aspects associated with our findings, on which we would also be interested in comments from others. We recognise that if our result was causal and occurred through a genetic pathway it would imply greater effects than predicted by previous estimates. We would point out, however, that we did not give "the explanation" but suggested "some explanations" and in our overall conclusions did not specify any preferred mechanism.

We did note, though, as well as Dr Valerie Beral, that our findings did not concur with those from Japan. We offered one thought on how the nature of the radiation exposure differed that was related to an interval of time between the bomb and the child's conception, and Dr Beral offered others; we would suggest that these be considered carefully. It would, in addition, be interesting to see an updated analysis from Japan of leukaemia in children of parents exposed to radiation before conception that included the revised lower dose estimates and was by year of birth. Other Japanese findings, of course, also do not concur with a raised incidence of childhood leukaemia after prenatal exposure to *x* rays.

In relation to latent periods, interestingly, the age distribution at diagnosis of the children with leukaemia born to fathers with a radiation record at Sellafield before their child's conception was lower than that of the other children with leukaemia. For example, five of the eight (62%) who were born to fathers who had been exposed to radiation had been diagnosed as having leukaemia when they were under 5 years of age compared with nine of the 38 (24%) children with leukaemia whose fathers had not been exposed. The table gives more detail. This suggested difference in age is in the direction that has been predicted in general if any mechanism involved were to contain a germline component.[1]

Number of children with leukaemia by age at diagnosis and recorded paternal exposure to ionising radiation at Sellafield before conception

Age (years) at diagnosis	Father with radiation record		
	Yes	No	Unknown
≤4	5	9	3
5-9	2	11	0
10-24	1	18	3

Test for trend in proportions of fathers with radiation record by age gives $\chi^2 = 4{\cdot}62$, df = 1, p = 0·03.

In reply to Drs J W Koten and W DenOtter, we did give in our paper some results on the relative risk of childhood leukaemia in relation to paternal age at birth, showing an increase in children of fathers over the age of 40 years of somewhat more than 50 per cent; but there was an even greater increase with maternal age, which was therefore discussed in more detail. The average age at their child's birth, for example, of the four fathers of children with leukaemia in the highest radiation dose groups that we used (≥100 mSv total or ≥10 mSv during the six months before conception) was 34 years (mothers 32), with one father (and one mother) being over age 40 compared with the average for their matched control fathers of 30 (mothers 27). The average age of the four fathers exposed to lower doses was 35 (mothers 33), again one father (and one mother) being over age 40; and for the remaining 38 fathers of children with leukaemia in our analysis the average age was 30 (mothers 27).

Dr R H Mole makes some reasonable comments about our estimation of paternal radiation exposure in the six months before conception in terms of the time period considered and the apportioning of annual doses. We accept these and had already planned to revise the analysis to examine a shorter period—probably two months—and to use the original dose records. What was reliably available to us for initial analysis was the annual dose estimates from British Nuclear Fuels, which had been checked by the National Radiological Protection Board,[2] and these were considered appropriate by colleagues whom we consulted. Of course any misclassification suggested by Dr Mole could equally apply to control fathers as much as to fathers of children with leukaemia and would be expected to bias the relative risks towards unity. Moreover, his comments do not affect our analysis of total radiation dose before conception.

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1 Wheldon TE, Mairs R, Barrett A. Germ cell injury and childhood leukaemia clusters. *Lancet* 1989;i:792-3.
2 National Radiological Protection Board. *Radiation dose histories at British Nuclear Fuels plc, Sellafield*. Chilton: NRPB, 1986. (NRPB M136.)

SIR,—In her editorial[1] on the study of Professor Martin J Gardner and colleagues on leukaemia