Sole cause cot deaths, 1988-90. England and Wales by regions. Figures are rates per 1000 live births

Year	England and Wales	Regions														
		Northern	Yorkshire	Trent	East Anglian	North West Thames	North East Thames	South East Thames	South West Thames	Wessex	Oxford	South Western	West Midlands	Mersey	North Western	Wales
1988	2·0	1·7	2·1	1·6	1·6	1·8	1·7	2·5	2·2	2·5	1·8	2·8	1·82	1·7	2·2	1·7
1989	1·7	1·8	2·4	1·3	1·4	1·8	1·3	1·5	1·7	2·3	1·3	2·2	1·8	1·6	1·8	1·7
1990	1·5	1·8	1·8	1·1	1·9	1·2	1·1	1·6	1·3	1·7	1·3	1·6	1·6	1·3	1·8	1·4

Censuses and Surveys has published figures on cot deaths[4] from 1971 to 1989, including those for the postneonatal period, but its figures include all death certificates with "any mention" of cot death: in some a cause will later be found, and once these deaths have been relocated the numbers of only cot death are published.[5] These numbers are not, however, divisible routinely into regions and districts. Another of the office's publications—VS3—does this; initially it included all deaths in the first year but since 1986 it includes only the postneonatal period. So now all the information wanted is on the one form.

An example of the use of VS3 is shown in the table. The sole cause cot death rate has fallen 25% from 1988 to 1990. In two regions—Northern and East Anglian—there was no fall; in 12 the fall was 18-32%; and in one it was 44%. This was the South Western region, and the fall is near to that of the four districts, three of which were in South Western region. The table suggests that information emanating from Bristol got to the rest of the country by word of mouth before the national advice went out at the end of 1991. Although some of this spread was by the media, most was probably by midwives and nurses.

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Neonatal mortality since 1935

SIR,—R K Whyte's suggestion that the heightened neonatal mortality found in the United States and the United Kingdom during 1951-80 reflected the impact of nuclear bomb tests[1] can be supported as follows.

Since in addition to the underweight infants who died between 1950 and 1980 around 10 times more survived with conditions known to be associated with low birth weight such as lowered immune competence we should now find in the United States and United Kingdom several millions born in those years with abnormally high mortality as they are increasingly exposed to sexually transmitted infections. This is indeed seen to be the case from two recent reports which noted increased mortality of men aged 25-44 since 1983 in both the United States[2] and the United Kingdom.[3] For example, the percentage of all deaths accounted for by men and women in this age group had been falling steadily in the United States, from 11·3% in 1940 to 5·4% in 1983, but has since risen to 6·6% in 1989, representing an increase since 1983 of 22%. According to the *United Nations Yearbook*, the corresponding percentage for Britain rose from 2·42% in 1983 to 2·61% in 1988, or by only 8%, which is consistent with the greater distance from the Nevada test site. In western Germany, south of the drifting Nevada fall out, the percentage for the 25-44 age group continued to fall, from 3·54% in 1983 to 3·10% in 1988, a drop of 12%.

Further evidence tying this increase in mortality to fall out can be found for the United States. The percentage share of all deaths from internal causes for those aged 35-44 fell from 3·23% to 2·57% representing cohorts born well before the start of bomb tests in 1945. But from 1983 to 1988 the cohorts in the 35-44 age group increasingly included those born in the early bomb test years and the percentage rose to 3·55% in 1988, an increase of 38%. In 1988 those aged 35-44 were born in 1938-47. The case is even clearer for the age group 25-34, whose percentage share nearly doubled from 0·99% in 1970 to 1·80% in 1988; the cohorts represented had been born during 1948-57. This hypothesis will be further tested if the anomalous rise in the percentage share of mortality for the 25-44 age group peaks in the early 1990s and then falls, when the full impact of bomb fall out on early infant development will be played out.

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SIR,—What would cause an estimated excess of 40 000 neonatal deaths in England and Wales from 1951 to 1980 and 280 000 excess deaths in the United States from 1955 to 1978? The cause should be a "modified single cause with discrete onset and extinction."[1] Certainly not oxygen deprivation or Cross's oxygen restriction hypothesis,[2] as R K White has convincingly shown.

An explanation to consider is the increase in registration during the '50s of neonates weighing under 2500 g. (Infants weighing under 1500 g account for just over 1% of live births in the United States and over 50% of the neonatal mortality[3]; over 30% of babies born under 1500 g are black infants.[4]) Birth weights have been regularly recorded in the vital statistics since 1950. These records show that the rate of low birth weight (<2500 g) for white infants with weight stated increased from 6·6% in 1950 to 7·2% in 1966, a 9·1% increase. This increase coincided with the excess deaths reported by Whyte. From 1966 to 1980 the rate fell to 5·7%, a 22% decrease. In 1989 the latest data available, the rate of low birth weight was 5·7%.[5]

David investigated the increase in non-white babies born under 2500 g from 1950 to 1967 and attributed the 32% increase that occurred to more complete reporting as more births occurred in hospitals.[6] This underreporting ceased during the '60s when hospital deliveries for non-whites became universal. Increased registration of black neonates under 2500 g in New York can also explain the finding by Bolton and Cross that the failure of death rates to improve in New York state, especially in New York City, was observable among only the low birth weight infants.[7] In New York state about 20% of newborn infants are black,[4] while in New York City over one half are black.[8] By 1980 the rate of low birth weight for black neonates in the United States had fallen to 10·7%; it had increased to 13·5% by 1989.[5]

Whyte's study helps open the way to reassess oxygen administration in the nursery. Greater flexibility and individualisation of oxygen concentrations could result in less aggressive management and less injury from barotrauma. In addition, if increased registration of low birthweight infants can explain the increased neonatal mortality of the '50s and '60s it can also explain why the rates of low birthweight infants remain as high as they are despite a fall in fertility rates and the efforts of medicine, philanthropic organisations, and government programmes to reduce these rates.

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Laparoscopic cholecystectomy

SIR,—Laparoscopic cholecystectomy is perceived by many as a great advance in the treatment of gall stones. It is without doubt here to stay, but as the demand is "patient led" the advantages to the patient must be objectively analysed. Are the reservations about this procedure voiced by Lord Smith[1] justifiable in the United Kingdom? Is the incidence of bile duct injury with its associated morbidity as high as he suggests?

In an attempt to answer this question we conducted a telephone survey of 10 hospitals in one region in south of England. During the twelve month period November 1990 to November 1991 a total of 264 laparoscopic cholecystectomies were performed. There were five bile duct injuries. Of these, four involved complete division of the common bile duct and one incomplete. The incomplete division was repaired over a T tube and the patient made an uncomplicated recovery from the immediate surgery. Of the four others, one was treated by end to end primary repair of the common bile duct, two by hepatodochojejunostomy en Roux performed, and the fourth, being diagnosed postoperatively, by delayed hepatodochojejunostomy en Roux as a secondary procedure. The patient with the division who underwent primary repair suffered a stenosis after operation