by the authors are prerequisites for any major collaborative audit, and the enthusiasm with which general practitioners in Manchester have accepted the task of improving their standards in the care of diabetes, in collaboration with consultant colleagues, suggests great achievements ahead. We have similar schemes for antenatal care, hypertension, and radiology as well as for facilitating audit in individual practices. Of course, as the authors identified, this work raises educational needs, and we are addressing these.

My argument with Berwick and colleagues is that, in the longer term, quality and outcomes as assessed by patients will be seen as clear objectives and that formal audit, though the main tool, may not be all that medical audit advisory groups find themselves asked to do. Already practices are asking for help with organisation and management as well as audit. Perhaps by the end of the decade we shall be restyling ourselves medical quality management groups.

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2 Berwick DM, Enthoven A, Bunker JP. Quality management in the NHS—II. *BMJ* 1992;304:304-8. (1 February.)

SIR,—In discussing quality management D M Berwick and colleagues emphasise the importance of studying the processes by which health care is delivered as well as the content of such care.¹ The multidisciplinary approach of medical or nursing audit, though appropriate in other contexts, is of limited use in this. We should examine how well the different professional and functional components of service delivery fit together as a whole for patients.

In these respects the Audit Commission's recent reports concerning the organisation of inpatient care in acute hospitals are disappointing.^{2,3} Readers are left struggling to bring together and make sense of two analyses which are not comparable and are based on different units of organisation: the ward for nurses and the bed for consultants. There seems to have been little attempt to deal with these difficulties either in the design of the studies or in the reports.

Two separate programmes of audit will now go ahead, which, if they follow the commission's recommendations, will ignore crucial issues about the boundaries and relations between the roles, organisation, and coordination of nursing and medical care on acute wards. This will do little to encourage fundamental questions about the costs and benefits of perpetuating these differences.

My recent experience of studying the role and management of junior house officers brought home the relevance of total quality management to the design of high quality care.⁴ Ward sisters and general managers as well as preregistration house officers, consultants, and other staff groups were interviewed. Without such a design we would probably have missed the point that patients may sometimes be dangerously exposed by the misalignment of the different units of work organisation of nurses and doctors and that this misalignment can contribute considerably to the work problems and problems of supervision of junior house officers as well as waste resources.

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Reducing risks in the sudden infant death syndrome

SIR,—In their editorial on the sudden infant death syndrome D P Southall and M P Samuels did not consider an alternative hypothesis: that the risk of sudden infant death increases in proportion to the amount of time an infant spends asleep out of parental earshot.¹

I worked for two years as the paediatrician in a hospital in Zimbabwe that served an urban black African population of about 300 000. About a fifth of the population was under 5 years old. Ours was the only hospital in the town. The only legal way of registering the death of a child was through the hospital mortuary. In two years neither I nor the single police officer who interviewed the parents of all children brought in dead ever heard of a "cot death"—of an infant put to sleep well and found dead. African colleagues told me that they could not think of any cultural reason why a story of sudden unexplained infant death might be withheld by parents. Black Zimbabwean infants almost invariably sleep with their mothers, at least until they are 6 months and often until 1 year.

Sudden infant death may result from trivial embarrassments—upper respiratory tract infection, overheating, regurgitation and aspiration, and so on. It may begin with unremarkable episodes of distress, which, if they had been witnessed, might have been simply attended to and thought little of.

My hypothesis is consistent with the finding of a lower incidence of the sudden infant death syndrome in overcrowded communities.² A small questionnaire study among women attending an antenatal clinic in Birmingham found that 22% of Asian babies were put to sleep on their backs compared with 3% of white babies. It also found, however, that 98% of the Asian babies slept in the same room as their parents for the first year, 34% in the same bed.³ Only 65% of white infants slept in the same room as their parents.

Existing databases on the sudden infant death syndrome should be interrogated with respect to this hypothesis, and prospective studies should be designed to address it. If the hypothesis were true a simple piece of advice—that babies should sleep next to their parents for the first six months of life—might make an important contribution to reducing the incidence of the syndrome. I have no doubt that parents whose baby is even trivially unwell should be advised to let the baby sleep with them.

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1 Southall DP, Samuels MP. Reducing risks in the sudden infant death syndrome. *BMJ* 1992;304:265-6. (1 February.)

2 Lee NN, Chan YF, Davies DP, Lau E, Yip DC. Sudden infant death syndrome in Hong Kong: confirmation of low incidence. *BMJ* 1989;298:721.

3 Farooqi S, Perry IJ, Beevers DG. Ethnic differences in sleeping position and in risk of cot death. *Lancet* 1991;338:1455.

SIR,—The falling incidence of the sudden infant death syndrome in Avon in 1990 and 1991,¹ which paralleled a reduction in prone sleeping practices, is further evidence that supine or lateral posture during sleep reduces the risk of sudden infant death. However, we seek reassurance that sleeping position was the only risk factor to vary over the study period.

An important mechanism underlying the sudden infant death syndrome is failure of respiratory control at a vulnerable stage of development: more a "physiological syndrome" than a disease in the accepted sense. Disturbance to this delicate equilibrium might upset the regulation of breath-

ing, leading sometimes to death. Epidemiological risk factors are somehow linked with destabilising influences to breathing, and by avoiding or modulating these the risk of death can be reduced.

We wonder whether some of these influences can be found in a baby's immediate environment. The practice of placing infants to sleep on their own for relatively long periods, a common practice in Western countries has few historical or cultural parallels. Babies left alone, particularly at night, might lose external sensory stimulation which may help stabilise breathing patterns. Experimental work with mothers and infants cosleeping in sleep laboratories is showing how patterns of breathing may interact.² Alertness of the babies' carers to early symptoms of illness might also be blunted.

In Hong Kong, where the sudden infant death syndrome is very uncommon,³ babies sleep lying supine but are never left alone. In Britain sudden infant death is much less common in Asian families than in white or Afro-Caribbean families.^{4,5} Asian babies are also more likely to be placed to sleep on their backs and in their parents' bedroom.⁶

Any fall in the incidence of the sudden infant death syndrome is welcome. However, we share some of Southall and Samuels' caution that we should not be too hasty in attributing the improvement solely to changes in sleeping posture.⁷ Any intervention programme which attempts to reduce the incidence of the syndrome has to consider the broad range of infant care practices which could also reduce risk.

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Monitoring cot death rates

SIR,—The postneonatal mortality rate for England and Wales ranged between 4.0 and 4.5/1000 live births in 1978-88. In 1989 it fell to 3.7/1000 and in 1990, which is the latest available, to 3.3/1000. Presumably this was due to a fall in numbers of cot deaths, which are responsible for a half of all postneonatal deaths.

Stanton reported a 50% fall in the cot death rate of Scarborough district after parents were advised not to overwarm their small infants (the "Keep cool, baby" campaign).¹ Wigfield and others reported a similar reduction in four other districts by advice against letting the infant sleep in the prone position.² Now the Foundation for the Study of Infant Deaths and the Department of Health recommend both procedures—supine sleeping and not overwarming.³

To monitor the effect it is important to have accurate figures on the cot death rate. Since most cot deaths occur between 1 and 4 months of age and neonatal deaths are now part of the perinatal mortality rate it is best to concentrate on postneonatal mortality only. The Office of Population

	England and Wales	Regions														
Year		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 deaths¹ 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 tests¹ 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 States² 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 mortality³; 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 Smith¹ 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 hepatochojejunostomy en Roux performed, and the fourth, being diagnosed postoperatively, by delayed hepatochojejunostomy en Roux as a secondary procedure. The patient with the division who underwent primary repair suffered a stenosis after operation