	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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1 Whyte RK. First day neonatal mortality since 1935: re-examination of the Cross hypothesis. *BMJ* 1992;**304**:343-6. (8 February.)
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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

and was subsequently referred to St George's Hospital for biliary reconstruction.

The incidence of bile duct injury at open cholecystectomy is around 1:300 in the United States. Therefore an incidence of 1:53 after laparoscopic cholecystectomy, as found in our survey, represents a considerable increase. Interestingly, of the five surgeons reporting injuries, only two had performed fewer than 20 laparoscopic cholecystectomies. If these injuries are to be put down to the learning curve then either this particular learning curve is much longer than is generally recognised (which has immense training implications) or the operation, as performed at present, is inherently more dangerous than open cholecystectomy. Probably it is a combination of these factors.

Our telephone survey was small and consequently may not be representative of the experience of the entire country, but our figures are comparable to the American experience. If these figures are representative then whether this operation is inherently more dangerous than open cholecystectomy must be considered. This requires comprehensive audit and honesty in reporting. If, alternatively, this increased incidence is reflecting the learning curve then laparoscopic teaching programmes must take this into account.

Minilaparotomy cholecystectomy has recently been proposed as a safer alternative to laparoscopic cholecystectomy.² At present this variation of cholecystectomy is practised mainly by committed biliary surgeons who are unlikely to damage the bile duct whatever the method used to remove the gall bladder. Whether or not the procedure will be safer than laparoscopic cholecystectomy when practised by general surgeons at large is difficult to predict.

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SIR,—In suggesting that it is "dangerous if not unethical to accept any new treatment . . . without putting it to the ultimate test—a randomised controlled trial," J N Baxter and P J O'Dwyer imply that surgical operations in current use have been subjected to this ideal scrutiny.¹ As supporters of the clinical trial we wish that this were so. Sadly, examination of the general surgical operative repertoire suggests that this is not the case. Most operations performed by general surgeons have not been accepted into clinical practice as a result of controlled clinical trials for reasons which one of us has elaborated elsewhere.² Worse, when a trial has been performed its conclusions are often ignored. Two randomised trials of highly selective vagotomy versus cimetidine both found in favour of surgery,^{3,4} yet despite this, elective surgery has all but disappeared in favour of treatment with H₂ receptor agonists.

It is unlikely that a controlled trial comparing laparoscopic with minilaparotomy cholecystectomy could be sustained for long enough to show a difference in mortality. In the series reported by Cuschieri *et al* there were no deaths in 1236 patients, and in the audit reported by Dunn there were two deaths in 1653 (0.12%).^{5,6} Similarly, bile duct injury seems unlikely to discriminate between the two techniques now that the lessons of the learning curve have been learnt. Voyles reported no bile duct injuries in 500 patients,⁷ and in our own hospital series there have been no major bile duct injuries after laparoscopic cholecystectomy in over 500 consecutive patients. The criteria used to compare these operations should therefore be postoperative analgesia requirements, postopera-

tive hospital stay, and time to return to work. Our own hospital series has shown a progressive reduction in all of these variables as experience is gained, and it seems inconceivable that minilaparotomy cholecystectomy could prove superior in these areas in any trial.

Laparoscopic cholecystectomy remains unchallenged in one all important respect. It has introduced general surgeons to therapeutic laparoscopy, which seems destined to bring the same benefits to the rest of the gastrointestinal tract as it has already brought to cholecystectomy. Rather than be distracted by learning and testing the technique of minilaparotomy cholecystectomy, surgeons should devote themselves to learning and improving laparoscopic techniques, for it already seems certain that the future of abdominal surgery lies in laparoscopy.

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SIR,—It is reassuring that the debate regarding minilaparotomy versus laparoscopy for removal of the gall bladder continues.¹ Less encouraging is J N Baxter and P J O'Dwyer's proposal of a randomised controlled trial to settle the issue. The opportunity to undertake this trial was lost some two years ago when laparoscopic cholecystectomy was still being evaluated in Britain.^{2,3} The almost universal adoption of the laparoscopic approach to the gall bladder over the past two years has been largely patient driven, as a result of media exposure and of surgeons' preference.

Two other factors have fuelled the laparoscopic revolution. The instrument makers have responded quickly to a sudden demand, resulting in large expenditures for the units using laparoscopy. Hospitals have been quick to fund this innovative approach for fear of losing patients to alternative providers.

These factors make it impossible to undertake a randomised controlled trial. Because of the expectations that now exist patients will not accept random allocation to laparoscopic or open treatment, albeit by mini-incision.

The use of the randomised controlled trial in surgery has been criticised.^{4,5} However, alternatives do exist and are being evaluated. One of these is comparative audit, a process which requires large numbers of non-selected participants. This approach may be well suited to studies comparing alternative treatment methods that are considered established practice.⁶ Such a study is currently being undertaken by the comparative audit service of the Royal College of Surgeons of England.

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Hazards of tests of growth hormone secretion

SIR,—A Shah and colleagues point out the dangers of insulin induced hypoglycaemia as a pharmacological test of growth hormone secretion.¹ But neither they nor C G D Brook in his editorial² mention a safer, non-invasive investigation for those children thought to be deficient in growth hormone: estimation of overnight urinary growth hormone excretion. Prepubertal children secrete growth hormone predominantly during slow wave sleep at night. Although only approximately 0.1% of the growth hormone in the serum appears in the urine, an immunoradiometric assay exists to detect this amount.³

An early morning collection of all the urine produced overnight is transported to the laboratory in a plain plastic bottle. The volume of urine produced is measured. A sample of this is then preserved by the addition of reagents and frozen for storage until analysis. This is a simple procedure that can be performed in any hospital biochemistry laboratory. The specimens can be transported frozen in dry ice to the nearest suitably equipped endocrine laboratory for analysis.

Walker *et al* have investigated children with short stature and shown that they produce significantly lower overnight urinary growth hormone concentrations than normal children.⁴ Comparison of overnight urinary growth hormone excretion with peak serum response to provocation testing with either clonidine or insulin also showed a highly significant correlation. Those children who were deficient in growth hormone on provocation testing had urinary growth hormone concentrations outside the normal prepubertal range.

In view of its non-invasive nature, its low cost, and the lack of need for admission to hospital urinary growth hormone analysis is a screening test that should be more widely advocated.

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SIR,—We were interested to read in D A Price's letter that Manchester growth clinic avoids using insulin tolerance tests.¹ Such a policy does not eradicate the hazards of pharmacological tests of growth hormone secretion as one of the deaths reported by Shah and colleagues occurred after a glucagon stimulation test.² Certainly, interpreting any test of growth hormone secretion, either physiological or pharmacological, is problematical.³ Moreover, growth velocity is a more accurate predictor of response to growth hormone treatment.⁴

The product licences for growth hormone state that it is for use in growth failure in children due to deficiency of endogenous growth hormone secre-