Though these figures do not show absolute accuracy in data collection, they suggest that the quality of the data is much better than the reputed norm for data collected routinely in the NHS. We believe that a system of data collection based close to the clinical team and well integrated with the process of care (both attributes of the system in North West Thames) offers a better opportunity for accuracy than a centralised system covering a whole hospital. Although it is inappropriate to consider database analysis as some sort of replacement for prospective research, we believe that our results show that routine data collection can be made sufficiently reliable for such studies to be of value to clinicians.

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1 Thorpe-Beeston JG, Banfield PJ, Saunders NJStG. Outcome of breech delivery at term. *BMJ* 1992;305:746-7. (26 September.)
2 Griffiths M. Outcome of breech delivery at term. *BMJ* 1992;305:1091. (31 October.)
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EDITOR,—We are surprised that only one of the correspondents commenting on the paper by J G Thorpe-Beeston and colleagues¹ pointed out a major error in the paper: analysis should have been based on the intention to treat.² Thus the neonatal death rate for vaginal delivery should be grouped with that for emergency caesarean section. Assuming, as do Anne C Deans and colleagues,³ that the neonatal death in the group who had caesarean section occurred after an elective operation, the excess of deaths consequent on an intention to deliver by the vaginal route was 3.3 per 1000, in line with our estimate of 2.4 per 1000.¹

The main conclusion of our decision analysis was, however, that a policy of elective caesarean section for breech presentation might not necessarily increase maternal mortality and morbidity. This is because maternal death and serious morbidity are more common after emergency surgery than elective surgery by a ratio of between 4 and 2 to 1.⁴ If all the emergency caesarean sections included in Thorpe-Beeston and colleagues' series were for failed trial of vaginal delivery (that is, none of these operations were performed for breech presentation in labour) their rate for failed trial of vaginal delivery was 52%. This is well above the 30% "break even" point we calculated at which maternal mortality associated with trial of vaginal breech delivery might equal mortality associated with elective caesarean section.

Several correspondents argue that vaginal breech delivery with a poor outcome that was not supervised by a senior doctor should be excluded from analysis.⁵ Apart from agreeing that such births should be adequately supervised, we contend that the value of the analysis lies in the fact that it reflects current clinical practice.

Many correspondents criticise the use of routine data and several acknowledge that a randomised controlled trial of sufficient size is impractical, but none suggest a way forward.⁵ We believe that the use of routine data, for which structured questions are asked, is valuable, and validity can be high if the data are used in the day to day management of patients.

The purchaser-provider split has potential for improving the quality of data, but purchasers will have to collaborate if the uniformity of data collection shown in this study is to be achieved. Without this investment, audit and research will suffer.

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1 Thorpe-Beeston JG, Barfield PJ, Saunders NJStG. Outcome of breech delivery at term. *BMJ* 1992;305:746-7. (26 September.)
2 Deans AC, Allman ACJ, Steer PJ. Outcome of breech delivery at term. *BMJ* 1992;305:1091. (31 October.)
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5 Correspondence. Outcome of breech delivery at term. *BMJ* 1992;305:1090-2. (31 October.)

Risk stratification for open heart surgery

EDITOR,—Samer A M Nashef and colleagues recommend use of the Parsonnet index for assessing results of cardiac surgery in Britain.¹ Though this scoring system might well produce reasonable ranking with respect to risk of death, the actual predicted risks given by the system may be unreliable.

Nashef and colleagues grouped the predictions made by the index according to the classes good, fair, poor, high, and extremely high, and their data allow observed and expected deaths to be calculated (table). There is a 42% reduction

Numbers of deaths observed and expected by Parsonnet risk group

Risk group	Observed deaths	Expected deaths by Parsonnet index
Good	7	8.0
Fair	10	15.8
Poor	10	19.1
High	3	8.2
Extremely high	6	11.2
Total	36	62.3

($p < 0.001$) in observed compared with expected deaths. Put simply, either Nashef and colleagues have achieved a considerable reduction in mortality compared with that predicted by the Parsonnet index or the index substantially overstates risk. Inspection of Parsonnet *et al*'s paper¹ suggests that the index overstates risk: it is derived in an unconventional and unclear way that does not use multivariate analysis, and the agreement between its predictions and outcomes is evaluated with an inappropriate measure of correlation.¹

Numerical risks obtained from the Parsonnet index should not be taken literally and certainly should not be used for audit.

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1 Nashef SAM, Carey F, Silcock MM, Oommen PK, Levy RD, Jones MT. Risk stratification for open heart surgery: trial of the Parsonnet system in a British hospital. *BMJ* 1992;305:1066-7. (31 October.)
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EDITOR,—The mortality in Samer A M Nashef and colleagues' study of risk stratification for open heart surgery looks impressive compared with the predictions of the Parsonnet system¹ but may simply reflect the inherent inaccuracy of the model. The Parsonnet model has several weaknesses, not least that it fails to take into account important advances in cardiac surgery over the past five to 10 years. Furthermore, the model is

based on clinical data from a small cohort of American patients, who are not necessarily representative of the usual case mix in Britain.

Notwithstanding these problems, the concept of collecting risk stratification data on a national basis is already well developed. At the Royal Postgraduate Medical School we are collecting data on risk factors from six major cardiac surgery centres in Britain. The intention is eventually to involve every cardiac centre in Britain, with each contributing such data to a national cardiac registry for every patient referred for open heart surgery. This project has the support of both the Society of Cardiothoracic Surgeons of the United Kingdom and Ireland and the Department of Health.

An initiative called PANECAN—the pan-European cardiovascular network—has already been established to extend risk stratification comparisons across Europe. The project will use specialised software, the patient analysis and tracking system, to link centres to a central pan-European registry. The programme runs in line with proposals set out in both *The Health of the Nation* and European Community directives.

Initial comparisons of the Parsonnet model with bayesian analysis, an alternative risk stratification model,^{2,3} suggest that bayesian analysis may be more accurate and offers the advantage that surgeons can compare local results against aggregated data. Bayesian analysis also offers the opportunity to look at other key performance indicators in cardiac surgery, such as length of stay, long term survival, and risk of reoperation, all adjusted for severity of illness and comorbid conditions.

The importance of risk stratification extends well beyond simply identifying clinical risk at operation: it could be used for prioritising selection from waiting lists, advising referring doctors and patients of the expected outcome of different treatment options, analysing resource use, and identifying and promulgating the most effective clinical protocols both nationally and internationally.

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1 Nashef SAM, Carey F, Silcock M, Oommen PK, Levy R, Jones MT. Risk stratification for open heart surgery: trial of the Parsonnet system in a British hospital. *BMJ* 1992;305:1066-7. (31 October.)
2 Edwards F. The theorem of Bayes as a clinical research tool. *Surg Gynecol Obstet* 1987;165:127-9.
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4 Edwards FH, Albers RA, Zajchuk R, Graeber GM, Barry M. A quality assurance model of operative mortality in coronary artery surgery. *Ann Thorac Surg* 1989;47:646-9.

EDITOR,—Samer A M Nashef and colleagues show¹ that the Parsonnet risk stratification system² can be applied to a British cardiac surgical practice. At King's College Hospital we have used the Parsonnet system prospectively since April as part of a computer based audit system. Analysis of our current data on 398 patients confirms Nashef and colleagues' assertions, mortality in the five groups, by ascending risk, being 3.3%, 5.9%, 14.1%, 19.1%, and 41.2% respectively.

The Parsonnet system has the advantage of simplicity. It is based on clinical data about which there can be little difficulty in interpretation and can be calculated rapidly in an outpatient clinic or at the time data are entered into a computer system. (A simple computer program calculated our scores automatically.) Risk scoring has several advantages. It can be used in audit to assess the results of an individual surgeon or unit against a general standard, but, more importantly, it allows a comparison of results between different centres

where the populations of patients may be very different. This is done by comparing the results in each risk group rather than mortality for individual operative techniques. When the Parsonnet system is used for audit scoring must be done preoperatively to eliminate any bias in interpretation when the outcome is known. Our only reservation about Nashef and colleagues' study is that it was retrospective.

If purchasing authorities are to interpret data on outcome properly some form of risk stratification is necessary. Moreover, the same risk scoring system should be used by all the provider units to allow accurate comparison. Other risk scoring systems are available,³ but the Parsonnet system is simple and has now been shown by Nashef and colleagues and our experience to be applicable to the British population. We advocate its use by other British cardiac surgical centres.

On a practical, clinical level, risk scoring has allowed us to better prepare patients and their relatives for high risk surgery. Risk scoring is also useful for planning admission and operation lists and consequent admission to the intensive care unit.

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1 Nashef SAM, Carey F, Silcock MM, Oommen PK, Levy RD, Jones MT. Risk stratification for open heart surgery: trial of the Parsonnet system in a British hospital. *BMJ* 1992;305: 1066-7. (31 October.)
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Propofol infusion in children

EDITOR,—T J Parke and colleagues report on five children with upper respiratory tract infections in whom metabolic acidosis, lipaemia, and fatal myocardial failure occurred after use of propofol.¹ Propofol was given in an oil-water emulsion equivalent to 10% Intralipid, providing 1.8-2.4 g intravenous lipid/kg daily—within the range used in parenteral nutrition. These case histories raise the question whether sick ventilated babies or young children react adversely to intravenous lipid infusions, with resulting metabolic acidosis.

To address this, a large multicentre database on preterm infants was examined.² Preterm infants commonly develop severe respiratory disease and require prolonged intravenous lipid treatment. In 75 preterm infants who received over 1.8 g Intralipid/kg daily (usual target 3.0 g/kg daily) base excess was assessed as the mean value for each of four periods: (1) birth to the start of intravenous lipid treatment; (2) the start of treatment to the age when intake reached 1.8 g/kg daily; (3) from then until Intralipid was stopped; and (4) from stopping Intralipid to the end of intravenous feeding. In period 3 the lipid intake was in or above the range

achieved by the five children reported on by Parke and colleagues. The proportion of babies ventilated fell from 87% to 26% from periods 1 to 4. Base excess in 266 control infants who were ventilated but never given Intralipid was compared with that in the cases at all four periods (ventilated controls had fallen to 23 and 15 by periods 3 and 4).

The table shows that the pattern of decreasing base deficit in babies receiving Intralipid was the same whether they were ventilated or not; and in ventilated babies the pattern was the same whether they received Intralipid or not. This was also found when babies were categorised according to the number who had metabolic acidosis (base excess > -8 mmol/l; not shown in table). Thus of 75 babies receiving Intralipid, the number who had metabolic acidosis fell from 17 at period 1 to 11 at period 2, and 4 at period 3, despite an increasing lipid intake.

During period 3, 11 babies receiving Intralipid had lipaemia on one or more days; interestingly, in these babies the mean base excess was 0.9 (SD 3.3) mmol/l, compared with -2.7 (3.6) mmol/l in non-lipaemic infants, providing no support for an association between lipaemia and acidosis.

Preterm babies requiring total parenteral nutrition are a high risk group. Indeed, nine died during or within 48 hours after lipid infusion. Yet none had the pattern of increasing acidosis and lipaemia seen in the fatal cases reported.¹

Our findings therefore do not indicate that the lipid component of propofol, at the dosages given, should be a risk factor for metabolic acidosis in sick ventilated babies. Nor do our data show associations between metabolic acidosis, lipaemia, and death in highly vulnerable, ventilated preterm infants usually receiving more intravenous lipid than those given propofol. Possibly in the cases reported by Parke and colleagues there was an imbalance in the intravenous fuels given; isolated lipid infusion without adequate carbohydrate or protein would be unphysiological (though this aspect was not described). If propofol was responsible for the adverse events reported I suggest that the lipid component itself, if given appropriately, should not have been contributory.

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1 Parke TJ, Stevens JE, Rice ASC, Greenaway CL, Bray RJ, Smith PJ, *et al*. Metabolic acidosis and fatal myocardial failure after propofol infusion in children: five case reports. *BMJ* 1992;305: 613-6. (12 September.)
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Malnutrition and diabetes mellitus

EDITOR,—Andrew B Swai and colleagues' conclusion that in Tanzania "diabetes [mellitus] is not more common in the most undernourished [based on body mass index] members of the [adult] population, and that it is much less common than in well nourished Western populations,"¹ will come as no surprise to those of us who have worked for many years in tropical Africa. But for the

authors to compare their result with data obtained in children who have recovered from kwashiorkor (the most severe form of protein-energy malnutrition) several years previously, in whom impaired intravenous glucose tolerance has previously been shown,² is, to say the least, misleading.

Severe pancreatic damage has been clearly shown in children who have died of kwashiorkor or its associated infections,³ and it would be surprising if endocrine and exocrine elements were to resolve completely after recovery from this disease. Where is the evidence in Swai and colleagues' paper to support their contention that these "functional and structural changes are [not] sufficiently severe to lead to permanent diabetes"? Though the aetiology of chronic calcific pancreatitis (which also gives rise to diabetes mellitus) has not yet been delineated, suggestions that this is also a corollary of childhood kwashiorkor⁴ have certainly not been satisfactorily dismissed; any plausible aetiological hypothesis must take into account the fact that this syndrome can occur in children as young as three years, and kwashiorkor must surely remain high on the list.

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1 Swai AB, Kitange HM, Masuki G, Kilima PM, Alberti KGMM, McLarty DG. Is diabetes mellitus related to undernutrition in rural Tanzania? *BMJ* 1992;305:1057-62. (31 October.)
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Needs and demands for ophthalmology services

EDITOR,—J H Sheldrick and colleagues' analysis of demand for ophthalmic services illuminates patterns of current use of services but may not fit the applications they suggest.¹ While demand incidence can be an important indicator of health need for acute conditions causing pain or sudden sensory loss, it is an unreliable guide to the ability of patients with chronic disorders to benefit from intervention. Those who might consider following the authors' advice to plan a minimum ophthalmic service on this basis should be aware of the limitations of the approach.

It is unsafe to concentrate on patients presenting for attention. Patients' knowledge of the potential benefits of intervention, perceived availability, and accessibility of services all affect demand. The pattern of presentation may be independent of the severity of handicap or professional judgment of the ability to benefit from intervention. Similarly, patients who fail to present to one service may make demands on another. Wormald *et al* clearly showed the difference between need and demand.² In their population survey in inner London general practitioners were aware of less than half the eye disease detected. Demand incidence, therefore, is an unreliable measure of need as it may exceed, equal, or underestimate ability to benefit.

Using demand incidence as a measure of need for health care also equates severity of illness at presentation with the threshold for intervention. For one major eye disorder, cataract, the diagnostic threshold that the authors adopt (6/9) is appropriate for screening but rarely for operation. In 76% of patients visual acuity is 6/18 or worse when they enter the waiting list for operation.³

Although minimising avoidable visual handicap is important, this must be balanced against the risk of unnecessary intervention. Over 85% of those identified with early cataract are elderly⁴ and may not survive for their cataract to reach a severity sufficient to warrant surgery.

In our assessments of the need for cataract

Base excess in babies given Intralipid who were and were not ventilated and in control babies who were ventilated but not given Intralipid

Period of assessment	Days post partum (medians)	Mean (SD) base excess (mmol/l)		
		Given Intralipid, not ventilated	Given Intralipid, ventilated	Not given Intralipid, ventilated
1: Birth to start of Intralipid	0-10	-6.6 (4.1)	-5.8 (3.0)	-5.5 (3.1)
2: Start of Intralipid to 1.8 g/kg daily	11-14	-3.2 (3.2)	-4.0 (3.8)	-4.8 (5.4)
3: 1.8 g/kg daily to end of Intralipid treatment	15-27	-2.6 (3.9)	-2.0 (3.8)	-3.0 (4.1)
4: End of Intralipid treatment to end of intravenous regimen	28-40	-0.5 (3.4)	-0.2 (4.9)	-0.2 (4.8)