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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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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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)

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 kwashiorkor[4] 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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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 elderly[4] and may not survive for their cataract to reach a severity sufficient to warrant surgery.

In our assessments of the need for cataract

extraction we have used the prevalence of cataract in the population and the methods to which the authors refer to derive estimates of the incidence.[5] We adjust for ethnic origin, the discrepancy between diagnostic and operative thresholds, attrition by death, and the level of contraindicated operations. Our estimates also suggest that unmet need for cataract extraction exists but they are unaffected by factors such as knowledge of, or access to, services that may distort demand incidence among elderly people.

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EDITOR,—By using a defined population base and considering both hospital and primary eye care services in Nottingham J H Sheldrick and colleagues have made considerable advances on previous published work on the pattern of presentation of eye disease to health care services.[1] The study would have been further enhanced, however, by consideration of the role of the high street optician. It is not clear whether everyone referred by optometrists to the hospital eye service during the study was included. Although some reference is made to asymptomatic disease picked up by screening by the optometrist, it would have been interesting to see the impact of this route of referral presented separately.

Though appreciating the value of the work accomplished, we disagree with the authors that demand incidence and demand episode rate are useful variables on their own for the planning of eye health care services. Although the rationale for such measures—that only patients presenting for medical attention create work for medical services—seems to be common sense, it ignores the fact that one of the most potent influences on demand is actually supply of services. As A Ralph Rosenthal points out in his accompanying editorial,[2] supply is changing dramatically with the introduction of new technology. For example, the visual acuity thresholds at which cataract surgery is recommended have dropped over the past 10 years and will change more with the introduction of phakoemulsification. We have calculated that this will create over 80 000 potential extra cases of cataract surgery in North West Thames region alone (unpublished data).

Thus planning services based on activity has left purchasing health authorities in the position of watching their waiting lists for cataract surgery get longer, while contracts based on demand calculated from activity are exceeded. Even allowing for the known age related distribution of demand shown in Sheldrick and colleagues' paper does not alleviate this problem.

If we really wish to plan eye health services rationally we need to develop a model incorporating information on the structure of the population and projected demographic change, estimates of the incidence of visually disabling eye disease, and determinants of uptake of services. At present we can only guess at the parameters of this model. Properly focused epidemiological and health services research—in particular, a well designed longitudinal study—providing estimates of incidence is urgently needed.

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EDITOR,—J H Sheldrick and colleagues' paper[1] and A Ralph Rosenthal's accompanying editorial on the demand for ophthalmic services[2] will be welcome ammunition to ophthalmologists seeking resources. Some of the equipment cited by Rosenthal as being desirable, however, will be nothing more than a pipe dream for those eye units whose financial masters do not even allow for the provision of proved technology already in widespread use, such as automated perimetry.

In my view the implications of excimer lasers differ greatly from those of other new modalities; there is more to this than "perfection of techniques" and "about £200 000 per machine"—more even than training staff and finding suitable space for such equipment in existing, often cramped, accommodation. The introduction of a new treatment for a potentially large market with a visually non-threatening condition must on no account be allowed to divert resources from the already overburdened provision for patients with non-refractive disorders. It must therefore be funded additionally, and justifying any source of finance other than that in force for the few lasers already in private use—namely, direct payment by the patient/client—may be difficult, certainly as far as low ametropia is concerned. Strategically located excimer units staffed by experts could thus emerge, providing also a service for the NHS to non-paying patients with certain other disorders for which excimer lasers hold promise.

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Paradoxical bronchoconstriction and salmeterol

EDITOR,—Brian H Davies[1] disputes the use of the word "paradoxical" in my and colleagues' report of bronchoconstriction after the use of salmeterol by metered dose inhaler[2] and our view that salmeterol has a slower onset of action than salbutamol.

I believe that our use of "paradoxical" in the title is appropriate in that the response expected after inhalation of a bronchodilator is bronchodilatation. Acute bronchoconstriction, be it due to the active drug or to other constituents in the formulation, might therefore reasonably be viewed as a paradoxical response to the medication. The same terminology has been used in previous reports of this phenomenon[3] and is currently used in Allen and Hanburys' prescribing information.

More importantly, Davies is mistaken in believing that salbutamol and salmeterol share the same time of onset of action. I am unaware of the "accumulated evidence . . . in many comparative studies" to which he alludes. It seems at odds with Boyd *et al*'s finding of a median time to 15% improvement in forced expiratory volume in one second of 7 minutes for salbutamol metered dose inhaler and 14 minutes for salmeterol metered dose inhaler ($p=0{\cdot}04$),[4] and of a median time to peak bronchodilatation of 60 minutes for salbutamol and 150 minutes for salmeterol metered dose inhaler ($p=0{\cdot}036$).[5]

Information on these and the other single dose studies (which similarly show a differential onset of action) is available on request from Allen and Hanburys' medical information department, which has confirmed the slower onset of action of salmeterol (personal communication). This is consistent with our findings. Thus the view that bronchoconstriction induced by the vehicle may be unmasked is justified.

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Health effects of organophosphate sheep dips

EDITOR,—Virginia S G Wiseman and colleagues write of the adverse effects of organophosphate sheep dips on health.[1] Organophosphate insecticides have been used in crop production and livestock husbandry worldwide for over 40 years. The symptoms of human toxicity and the treatments are well recorded. Over the past three years the safety of workers using organophosphate sheep dips has received much publicity in the farming media. The Health and Safety at Work Act and Control of Substances Hazardous to Health Regulations require farmers to guard against chemical and biological hazards on farms.

In Britain sheep dips are classed as veterinary medicines and must be licensed under the Medicines Act 1968. Every label carries detailed advice on protective clothing and advice to doctors on treatment. This is regularly reviewed by the Veterinary Products Committee (on behalf of the Ministry of Agriculture, Fisheries and Food), the Health and Safety Executive, and the manufacturers of sheep dips—all of whom are members of the National Office of Animal Health.

There are 95 000 sheep farms in the United Kingdom. Last autumn dipping was compulsory and involved roughly 300 000 people. Murray and colleagues report 34 cases of exposure to organophosphate dips, of which three were accidents; the remaining 31 cases include 15 cases in which patients were not wearing protective clothing as instructed. Details of the 16 others are not reported. We share Murray and colleagues' concerns. The fact that half of the patients reported on had taken no safety precautions after great publicity may seem surprising but is supported by reports from the Health and Safety Executive and companies.

This year both the Health and Safety Executive and the National Office of Animal Health have commissioned trials from the Institute of Occupational Medicine to investigate dipping practices and protective clothing. This will be reported to the Ministry of Agriculture, Fisheries and Food; thus even more detailed advice on pro-