CORRESPONDENCE

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Organ donation

SIR,—The recent articles by Mr N J Odom are to be applauded.[1] If they were standard reading the yield and quality of organs for transplantation would be improved. We must, however, comment on points made in the second article, "Logistical disincentives to organ donation."

Mr Odom states that in Northern Ireland the rate of heart and liver donation has been extremely low. He also suggests that "required request" might improve the number of organs for donation. We wish to consider these points in turn.

In 1988 Northern Ireland had the second best rate of donation for kidneys in the United Kingdom (23·1 per million population a year), just behind South East Thames region (24·7). This was a fairly typical year. In providing kidneys for donation we do somewhat better than most other regions in the United Kingdom.

With regard to the relative frequency of heart and liver donation we can speak only for our own unit, which normally provides roughly 70% of the kidneys harvested in Northern Ireland. Before 1987 there was no protocol for offering organs other than kidneys for donation. Since then this has been corrected. In the two years up to the spring of 1989, 33 of the 42 patients with a diagnosis of brain stem death became organ donors.[2] Medical contraindications (two), family's refusal to give consent (five), and failure to request donation (two) account for the nine other patients. This compares favourably with the performance of an intensive care unit in a specialist transplant centre.[3] When organs were not requested (two cases) it had been judged that the relatives would not give consent for organ retrieval and that a request for donation would further alienate the family.

Twenty two of the patients donated only kidneys. The other 11 were suitable for multiple organ donation, and from them eight hearts and 11 livers as well as 22 kidneys were offered for donation. If repeated nationally this would be equivalent to 250 livers and 200 hearts available for transplantation each year. Only 10 of these 19 organs were actually harvested. The reasons for not harvesting them were lack of beds in the intensive care unit or staff at the transplant centre, lack of supplies of blood, lack of availability of a transplant surgeon or retrieval team, and lack of a suitable recipient. Many of these points were mentioned in a previous letter from our unit.[4]

Bodenham *et al* found that lack of permission by the coroner was a common cause of failure to retrieve organs.[3] In our series the coroner did not withold permission in any case.

We think that these data make two points. Firstly, Mr Odom's comments regarding liver and heart donation in Northern Ireland are no longer applicable. Secondly, although required request may improve the yield of organs in units that deal with potential donors infrequently, it will have no benefit in units such as our own where every effort in this regard is already being made.

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1 Odom NJ. Organ donation. *Br Med J* 1990;**300**:1571-5. (16 June.)
2 Lavery GG, Coppel DL, Johnston JR, Lowry KG. An audit of organ retrieval—could we do better? *Journal of the Irish College of Physicians and Surgeons* (in press).
3 Bodenham A, Berridge JC, Park GR. Brain stem death and organ donation. *Br Med J* 1989;**299**:1009-10.
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Surgery for glue ear

SIR,—It is pleasing to see further British studies of methods of treatment for glue ear,[1] but I was concerned by the conclusions drawn from such doubtful material. Dr N A Black and colleagues state that children were admitted for surgery for bilateral glue ear, a necessary requirement when one is randomising treatments between two ears in the same patient—for example, unilateral myringotomy and grommet insertion against no treatment. But 44% of patients in this group had dry ears when myringotomy was performed. Does one presume that grommets were inserted into normal middle ear clefts in these cases and likewise through the four treatment groups, in which it seems that only between 56% and 76% were found to have glue or serous fluid in the middle ear. If so I would not concur with the authors' belief that this clinical management is fairly typical of otolaryngological practice in England and Wales in the 1980s.

Though it is accepted that this study does not address the many children aged less than 4 who have chronic glue ear, in whom audiometry is unreliable, and very young children, in whom it is impossible, the authors' choice of audiometry as a principal end point is unsatisfactory. Most studies would require impedance measurements and tympanometry together with otoscopic examination by a validated observer and a hearing assessment to provide more accurate end points for evaluating treatment. This being the case, and considering also that children with gross nasal obstruction requiring adenoid removal were excluded from the study, adenoidectomy may have been shown to have had a more important effect than Dr Black and colleagues found.

Of course the object of surgery for glue ear is to restore hearing. Nevertheless, if restoration by a grommet is effective for only six to 12 months and if within two years 45% of the patients treated by only a grommet require revision surgery compared with 19% of cases treated by adenoidectomy follow up for more than two years may confirm that adenoidectomy is far from irrelevant. The need for readmission to hospital (even on a day basis) to reinsert a grommet under general anaesthesia in more than twice the number of cases within two years than for adenoidectomy may constitute a cost ineffective practice with appreciable overall morbidity. Our own studies in which truly bilateral cases of glue ear were analysed in a similar way show very similar readmission rates for both treatments.[2,3] Further study of children selected for adenoidectomy on the basis of age and size of the postnasal airway rather than on a random basis showed that improvement in clearance of middle ear effusion one year postoperatively increased from 40% in the random group to 70% in the selected group.[4]

I share the concern of Dr Black and colleagues about the indications for surgery for glue ear and the number of these procedures carried out in this and in other countries, but I do not believe that the design of their study, in which a quarter to half of the ears treated did not have glue at the time of operation, allows them to make the somewhat sweeping statements in their discussion, particularly in relation to adenoidectomy for bilateral glue ear in children.

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1 Black NA, Sanderson CFB, Freeland AP, Vessey MP. A randomised controlled trial of surgery for glue ear. *Br Med J* 1990;**300**:1551-6. (16 June.)
2 Maw AR. Chronic otitis media with effusion (glue ear) and adenoidectomy: a prospective randomised controlled study. *Br Med J* 1983;**287**:1586-8.
3 Maw AR, Herod F. Otoscopic, impedance and audiometric findings in glue ear treated by adenoidectomy and tonsillectomy: a prospective randomised study. *Lancet* 1986;i: 1399-402.
4 Maw AR. Adenoidectomy and adeno-tonsillectomy for otitis media with effusion (glue ear) in children: a prospective randomised controlled study. London: University of London, 1986:105. (MS thesis.)

SIR,—"There are lies, damned lies and statistics." Whereas I agree with Dr N A Black and colleagues that the numbers in their trial are adequate for statistical analysis as defined in the paper,[1] it must be stated that the numbers in each group are too small to allow important conclusions to be reached.

Several points have not been adequately addressed. Firstly, the paper is entitled "Surgery for glue ear" but the study includes children with thick and thin fluid and even air in their middle ears. It seems to have been assumed that the fluid in both ears will have the same consistency, but this is often not the case. Indeed, a wide range of

consistencies exists, from serous to tenacious glue. In addition, no attempt has been made to relate the outcome to the presence, absence, and type of fluid.

Secondly, the reduction in the need for surgery after adenoidectomy is surely of great value to the patient and to the family, and the statement that "children who had undergone an adenoidectomy were less likely to have further surgery, but this was not surprising as it is usually possible to undergo an adenoidectomy only once" is idiotic. In addition, as in each group 52-70% of the patients had moderate or severe nasal symptoms it is not surprising that parents were more satisfied in the adenoidectomy group.

Thirdly, how abnormal is an abnormal tympanogram? How long before surgery were the audiogram and tympanogram obtained? With an abnormal tympanogram in 73-95% of the patients how can a 30% incidence of dry myringotomies be explained? This was obviously a carefully conducted study, but the results and conclusions are invalidated by a failure to consider the points mentioned above.

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1 Black NA, Sanderson CFB, Freeland AP, Vessey MP. A randomised controlled trial of surgery for glue ear. *Br Med J* 1990;**300**:1551-6. (16 June.)

AUTHORS' REPLY,—Your correspondents raise points relating to three aspects of our study: the selection of patients, preoperative assessment of the middle ear, and assessment of outcome.

Mr A Richard Maw criticises us for including children in whom glue was not present at operation. Although this is quite a high proportion of dry taps, most otolaryngologists report occasionally finding dry ears despite careful preoperative investigation. In addition, many surgeons insert grommets in dry ears on the basis of the child's history. In any case inclusion of such children did not preclude assessment of the outcome of surgery on the basis of preoperative measures of severity. Mr Maw correctly points out that by excluding children with gross nasal obstruction we are unable to comment on the effectiveness of adenoidectomy in such cases. We agree and have refrained from doing so. In practice such children make up a small proportion of all those undergoing surgery for glue ear.

Turning to the preoperative investigations of the middle ear (which were mostly performed the day before surgery), Dr I D Bottrill and Mr J A S Carruth are surprised by the lack of specificity of audiometry and typanometry. This is not a new finding.

Finally, as regards outcome we are unclear as to Mr Maw's view. He states that our choice of audiometry as a principal end point is unsatisfactory but goes on to say that the object of surgery for glue ear is to restore hearing. We subscribe to the second view.

Contrary to the comment of Dr Bottrill and Mr Carruth, we stated that outcome was related to middle ear content as well as several other variables. Only two variables, however, showed useful predictive power—audiometry and middle ear content. Given that the middle ear content is confirmed only at operation, it is of little or no practical decision making value to a clinician. As regards parental satisfaction, this applied to the parents' view of their child's hearing not nasal symptoms.

We may, as Dr Bottrill and Mr Carruth suggest, be idiots but we think it more likely that a child will undergo an adenoidectomy if one has not previously been performed. It is not clear from their opening comments whether they accept basic statistical concepts. If they do then they will appreciate that randomised trials are based on the expectation that the randomly assigned groups will be similar at a group level. No assumption need be made about one person or, in this trial, about one ear.

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SIR,—We congratulate Dr N A Black and colleagues on providing a further interesting study of the treatment of glue ear in the United Kingdom.[1] Yet to see this study in perspective one must look at several related points.

Firstly, the basic assumption that hearing gain is the sole aim of treatment for glue ear is invalid. It is well known that a proportion of patients referred for specialist treatment of glue ear may develop tympanic membrane atelectasis and adhesive otitis media.[2] This condition must be regarded as precholesteatomatous, and in our experience 6·5% of ears with grommets will ultimately develop this form of chronic suppurative otitis media.[3] At the 15 year follow up our controlled study also confirmed that tympanosclerosis of the tympanic membrane was commoner in the ears with grommets than in those without. Thus, advocating the use of grommets indiscriminately for hearing loss in glue ear, as do Dr Black and colleagues, is worrying. We would hope that most doctors managing such children would not offer surgery on the basis of only a pure tone audiogram, often done in a busy children's ear, nose, and throat clinic, but take into account the speech and language development noted by the parents and the reading and learning abilities noted by the children's teachers. These subjective data are of greater importance than the audiogram. A child of lower than average intelligence will do badly with a slight hearing loss, whereas a child with above average intelligence will do quite adequately with a moderate conductive hearing loss related to glue ear. Thus the first child warrants surgery and the second does not.

Many studies in the past, including ours and that of Dr Black and colleagues, use the Shepard grommet for middle ear ventilation. Ninety per cent of Shepard grommets are extruded by nine months whereas 90% of reuter bobbin or Shah grommets remain in situ for 15 months.[4] The histopathology of glue ear includes a metaplasia of the middle ear mucosa with large populations of goblet cells and formation of seromucinous glands.[5] Ventilation of the middle ear would probably be required for longer than six to nine months to ensure complete reversal of this metaplasia.

Finally, the parental view that children who had had adenoidectomy were subjectively better than those who had not may be accounted for by the fact that children tend to gain weight and thrive following such surgery.[6] Snoring improves and breathing through the mouth is easier after adenoidectomy, and thus the parents will construe that the child is healthier and report greater satisfaction with the treatment.

Glue ear is very difficult to treat, and we think that many more well designed trials, particularly with large numbers of children, are justified throughout the United Kingdom as the treatment is still open to some degree of conjecture.

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1 Black NA, Sanderson CFB, Freeland AP, Vessey MP. A randomised controlled trial of surgery for glue ear. *Br Med J* 1990;**300**:1551-6. (16 June.)
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ABO blood group and ischaemic heart disease

SIR,—Dr P H Whincup and colleagues found that ischaemic heart disease in British men occurs significantly more commonly in people of blood group A than blood group O and that this related significantly to serum cholesterol concentrations, but cholesterol was the only association of note that they observed.[1]

In 1964 a study of 411 normal people showed that the plasma concentration of factor VIII is affected significantly by blood group,[2] people of blood group A or B having an average of 8% more factor VIII than people of blood group O. Blood transfusion services have repeatedly observed and reported significant differences in the factor VIII content of plasma and cryoprecipitate prepared from such plasma according to blood group.[3]

Given these findings, the higher serum cholesterol and plasma factor VIII concentrations in people of blood group A than in those of blood group O may summate and thereby contribute to a greater atherogenic potential. ABO antigens are oligosaccharides, the A and B antigens having an extra saccharide unit to the O unit (*N*-acetylgalactosamine and galactose respectively). They are distributed in all tissues, including endothelial cell membranes, as all cells possess the relevant saccharide transferases. The oligosaccharide synthesis mechanism may be related somehow to the synthesis or secretion of clotting factor VIII or perhaps influence the activity of the molecule by affecting its glycosylation.

The oligosaccharide antigens are bound to both protein and lipid precursor substances and may well be influenced by the cholesterol or other related steroid content of cell membranes. More investigation of these two intriguing findings may show a further interaction of plasma and cellular factors in the process of atherogenesis.

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Management of menorrhagia

SIR,—Dr Adam L Magos alluded to the potential financial benefits to the NHS of introducing hysteroscopic methods of endometrial destruction in place of hysterectomy for the treatment of menorrhagia.[1] We recently assessed the cost of performing a hysterectomy and an endometrial resection in patients with menorrhagia (table). The costings are based on 108 patients who had a hysterectomy for menorrhagia in 1988, on