

practice but rather a distillation of the best in existing hospital care and is applicable to all patients. The problem is not simply how to achieve a coordinated development of hospice provision but more importantly—how is the model of excellence pursued by the hospice movement to be spread throughout the health service?

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SIR,—I note with interest and concern the comments on hospices in the BMA Annual Report of Council (31 March, p 8). The launching of "Help the Hospices" shows the inherent weakness of the independent hospices—namely, difficulty in ensuring regular and satisfactory funding. Many hospices are within the NHS, and although subject to financial constraints they continue to function in a successful way with their future assured.

I hope that this new national organisation to help the hospices, now keenly supported by the board of science and education, will not jeopardise the future of the hospice movement by encouraging dichotomy between the independent and NHS hospices.

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SIR,—Some of the points that Dr Tony Smith makes are very relevant—particularly those concerning the planning for future hospices, where there is clearly a requirement for more rational organisation. It seems inappropriate, however well intentioned, that a local community raises the money for a building only then to hand it over to an inadequately funded health service. There may be no real consultation or understanding of the requirements and availability of staff or appreciation of other methods of terminal care. The location of such hospices, inside or outside the NHS, also requires urgent consideration together with the need for training staff. The role of the cancer charities has been important in this respect.

Those who were instrumental in setting up the hospice movement would agree with many of Dr Smith's comments but would take great exception to the statement: "If the era of well intentioned amateurism is to be succeeded by hard headed professionalism." It is a pity to confuse some of the attempts now being made to establish hospices with the pioneering and highly professional approach of those who were there at the beginning. The substantial research and teaching commitment on dying, bereavement, symptom control, pharmacology, and nutrition testify to this approach.

For the past eight years a forum for such discussions has been provided at a "think tank" held several times a year at St Christopher's Hospice in London to develop and extend the questions which Dr Smith has raised. This group, comprising most of those involved in developing terminal care services, has discussed many issues including alternatives to hospices—for example, the development of hospital support teams,¹ which Dr Smith did not mention. If Dr Smith's concern for amateurism in medicine is a real one, and I believe it to be an important issue, then there are others more deserving of

attention than those who have developed hospices.

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¹ Bates T, Clarke DG, Hoy AM, Laird PP. The St Thomas's Hospital terminal care support team: a new concept of hospice care. *Lancet* 1981;i:1201-3.

Obstruction of the fetal urinary tract: a role for surgical intervention in utero?

SIR,—Only one of the eight cases of fetal urinary tract obstruction whose natural history was described by Mr D F M Thomas (17 March, p 858) is alive and normal. From this he concludes that intervention before delivery for such anomalies is not at present justified. His experience is similar to that at Northwick Park. Only one of the five cases of complete urinary tract obstruction that I reported (11 February, p 459) is now alive; case five died 27 weeks after delivery because of pulmonary hypoplasia. Elsewhere intervention has been more successful: of the eight cases summarised in table II (p 460) five survived, and although not all subsequent drainage of the fetal urinary tract has been successful¹ there have been successes.²

Mr R R Turnock and others (21 April, p 1234) agree that the case for intrauterine intervention is yet to be established since benefits to renal function are difficult to prove and not all dilated urinary tracts require treatment. Review of the data available suggests that while not every case requires or would benefit from intervention criteria are emerging which help to differentiate those for whom it would be reasonable to advise drainage of the urinary tract.

Intervention is not likely to improve the prognosis for the following cases: (a) unilateral obstruction unless the distended ureter or kidney is compressing appreciably other structures³ or appears to be precipitating premature labour⁴; (b) intermittent or partial obstruction where there is a normal amount of liquor⁵; and (c) when there is no renal function.

Accepting the difficulties in diagnosing obstruction and of establishing that the fetus is otherwise normal, the surgeon might consider intervention when: (a) the obstruction is discovered before 16 weeks since renal and pulmonary damage or arrest of development may then still be reversible or may be ameliorated⁶; (b) the fetus was seen to be normal on ultrasound at 16-20 weeks and the obstruction is discovered before 36 weeks. Later in pregnancy than this the lungs are likely to be sufficiently developed for the baby to survive delivery, but drainage in younger fetuses may prevent further renal deterioration or even increase parenchymal size as can happen in the neonate whose distended urinary tract is decompressed⁷; and (c) where there is marked oligohydramnios even if the fetal bladder appears to empty.

The fetus whose obstruction is first diagnosed after 20 weeks is a real dilemma. Many of these have died of pulmonary or renal maldevelopment, but there have been survivors²—presumably those whose urinary tracts were not obstructed by 20 weeks. Lung development may be satisfactory, but in the absence of amniotic fluid this is difficult to assess. Ultrasonic measurements of their chest wall dimensions may possibly show those with pulmonary hypoplasia, but fetal breathing movements are not of prognostic value since they can be present even if lung development is pathological.

The value of criteria such as these, and of the modes of treatment, can be evaluated best by follow up of all cases treated. Since the condition is rare and numbers are small the existence of an international registry facilitates the accumulation and critical analysis of the

results—details can be obtained from Dr F A Manning, Department of Obstetrics, Gynecology, and Reproductive Sciences, 59 Emily Street, Winnipeg, Canada R3E OW3. The more information that becomes available the quicker will answers appear to uncertainties such as whether treatment of an affected twin is in the best interests of the healthy sibling. The more cases which are recorded the better will we be able to advise parents whose fetus has an obstructed urinary tract.

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- ¹ Goodlin RC, Anderson JC, Gallagher TF. Relationship between amniotic fluid volume and maternal plasma volume expansion. *Am J Obstet Gynecol* 1983;146:505-10.
- ² Shalev E, Weiner E, Feldman E, Sudarsky M, Shmilowitz L, Zuckerman H. External bladder-amniotic fluid shunt for fetal urinary tract obstruction. *Obstet Gynecol* 1984;63:315-45.
- ³ Vintzileos AM, Nochimson DJ, Walzak MP, Conrad FU, Lillo ML. Unilateral fetal hydronephrosis: successful in utero surgical management. *Am J Obstet Gynecol* 1983;145:885-6.
- ⁴ Kirkinen P, Jouppila P, Tuononen S, Paavilainen T. Repeated transabdominal renocenteses in a case of fetal hydronephrotic kidney. *Am J Obstet Gynecol* 1982;142:1049-52.
- ⁵ Diamant MJ, Fine RN, Ehrlich R, Kangaroo H. Fetal hydronephrosis: problems in diagnosis and management. *J Ped* 1983;103:435-40.
- ⁶ Manning FA, Harman CR, Lange IR, Brown R, Dector A, MacDonald N. Antepartum chronic fetal vesicoamniotic shunts for obstructive uropathy: a report of two cases. *Am J Obstet Gynecol* 1983;145:819-22.
- ⁷ Gonzales ET. Annual meeting of the Section on Pediatric Urology. *Pediatrics* 1983;71:449-52.

*.*This correspondence is now closed.—ED, *BMJ*.

Maternal plasma volume and disorders of pregnancy

SIR,—Dr C W G Redman quoted us out of context (31 March, p 955). The missing part of the sentence was: "Accepting the concept that plasma volume expansion is a hallmark of normal pregnancy and that maternal failure of expansion of plasma volume is associated with pregnancy complications," which preceded Dr Redman's quote: "A major goal of antenatal care should be an expansion of plasma volume in pregnant women."¹

It is rare for an appreciably hypovolaemic pregnant woman to have a truly normal pregnancy, and I believe this relation is more than a coincidence. For a decade we have been attempting to test this hypothesis in a properly designed study. Besides our inability to find funding, or a suitable population we have had no acceptable technique to expand permanently plasma volume in hypovolaemic pregnant women other than to encourage good prenatal diet, rest, and tranquility. For ethical reasons alone these standards of prenatal care could not be manipulated.

We have, however, recently tried "head out immersion," apparently the only practical method of achieving plasma volume expansion in primates.² Pregnant women stand in warm water up to their shoulders and perform mild exercises for one hour two or three times a week. After monitoring more than 200 immersed pregnant women we have been encouraged with this expansion technique, and the subjects have also very much enjoyed the "baths."³ Although I can find nothing in print, I have been told that "bathing" was a popular technique of treating mild toxemia in

England. Perhaps as in so many other obstetric matters the British already have the answer.

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¹ Goodlin RC, Dobry CA, Anderson JC, Woods RE, Quaipe M. Clinical signs of normal plasma volume expansion during pregnancy. *Am J Obstet Gynecol* 1983;145:1001.

² Epstein M. Renal effects of head-out water immersion in man. *Physiol Reviews* 1978;58:529.

³ Goodlin RC, Engdahl Hoffman KL, Williams NE, et al. Shoulder-out immersion in pregnant women. *J Perinatal Med* (in press).

Treatment of end stage renal disease

SIR,—Dr N F Jones and others (31 March, p 992) show clearly the deficiencies in services for treatment of end stage renal disease in the United Kingdom. They state: "In 1982 no fewer than 14 other European countries treated more patients with renal failure per million population than did the United Kingdom. . . ." Against this background of general deficiency there is a wide variation between individual regions both in terms of patients treated and in terms of staffing. In some regions the service is similar to that of the poorest European countries.

We work or have recently worked in by far the worst provided region—both in facilities and in staffing at both consultant and junior level. The 3m people of the South West Thames Region have a totally inadequate service, and in 1981 only 114 persons (38 per million population) were being treated by dialysis, transplantation, or both, whereas the average for the UK was 142.9 per million population. This deplorable state of affairs is currently being concealed in some official publications—for example, *Renal Services*¹—by combining the information gathered in South West Thames with that of South East Thames.

Even South East Thames, which by comparison seems at first sight to be well provided with staff and facilities, was able to take on only 140 patients in 1979, a rate of 40 per million population.¹ This take on rate of South East Thames region, high for the UK and including patients from South West Thames, is still less than the national acceptance rate for seven of the 32 countries whose data are given in the 1983 report of the European Dialysis and Transplant Association.² There is clearly an unmet need, therefore, even in South East Thames.

By considering South West and South East Thames together there is the implicit assumption that the shortfall in South West Thames is covered by South East Thames. This has never been supported by any data, and inspection of available information for the end of 1980 shows that the patient stock (total dialysis and transplant patients) for South East Thames was 900. A total of 155 (22%) of the 719 patients for whom the home address was known resided in South West Thames (unpublished data, South East Thames Regional Health Authority).² These 155 combined with the 108 South West Thames patients being treated in South West Thames gives a total of 263 patients, a very small number indeed. Most patients being treated by dialysis units in South East Thames actually reside in South East Thames, which is not surprising since it is known the number of patients with end stage renal disease being treated bears a direct relation to the staff and facilities available,³ a state of affairs that probably holds wherever there is an inadequate service. A similar relation probably holds for the patients of South West Thames as general practitioners and non-renal consultant physicians are naturally reluctant to refer patients for consideration for dialysis, transplantation, or both, when they know facilities are lacking.

When the concept of dividing the nation into regions was devised it was never intended that patients should be forced because of lack of facilities in one region to travel for treatment to another. This process in patients with end stage renal disease incurs trouble, inconvenience, and cost—both to patients and the NHS. We believe it would be shortsighted of South West Thames Regional Health Authority to continue to deprive the 3m individuals in South West Thames of the facilities to which they are entitled. There has been a consistent failure to provide adequate funding for the treatment of end stage renal disease, and the present funds are only a fraction of those available in the other three Thames regions. Since the present arrangements result in considerable savings to South West Thames Region (as compared with others) we ask what alternative services these substantial sums are spent on?

The concept of a double region is counter-productive since it allows the lack of service in the South West Thames Region to lie hidden from view. The state of affairs in South West Thames was recognised in 1975 when, as now, it was the most underprovided region as far as facilities for end stage renal disease are concerned.³ How many more reports will have to be produced before the necessary action is taken?

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¹ South East and South West Thames Regional Health Authority. *Renal services*. London: South East and South West Thames Regional Health Authority, 1982.

² Wing AJ, Broeyer M, Brunner FP, et al. Combined report on regular dialysis and transplantation in Europe. *Proc Eur Dial Transplant Assoc* 1983;20:5-75.

³ Anonymous. Distribution of nephrological services for adults in Great Britain. *Br Med J* 1976;iii:903-6.

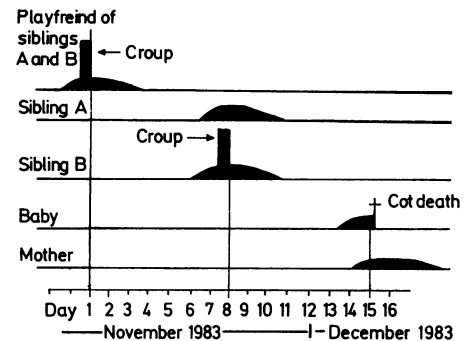
Acute stridor in a preschool child

SIR,—The leading article by Professor A D Milner on stridor in the preschool child (17 March, p 811) prompts us to describe a recent death investigated during our routine inquiries into all postperinatal deaths in Sheffield. We hope that Professor A D Milner will expand his advice on the infectivity and management of epidemic spasmodic croup.

Our case presented as a cot death. He was found dead at 10 am one Monday morning in December. He had been unwell during the previous day with upper respiratory tract symptoms. Necropsy showed no specific cause of death. There was rhinitis, a lymphocytic infiltration in the trachea, a small area of necrosis over the vocal chords, and influenza B virus was isolated from the lung cultures. The history from the police was unremarkable, and death was registered as "unexpected death in infancy."

Home visits are made after all deaths, and one of us visited the parents, by appointment, one very bleak evening in January and spent over 1½ hours talking with them. They told us the following story (fig).

Two weeks before the baby's death the two year old child of a close friend of the mother's had epidemic spasmodic croup. The emergency doctor called during the night, recommended steam and advised taking the child to the family doctor the next day. The mother asked if the condition was infectious and understood him to say that croup was not. The next day the croup had disappeared, and the child had an upper respiratory tract infection which was confirmed by her own doctor. This child



Pattern of local epidemic of spasmodic croup related to the cot death. Dark areas indicate upper respiratory symptoms.

and both siblings of the baby who eventually died continued to play together.

Exactly seven days later, after about one day of mild upper respiratory tract symptoms, the elder sibling of the child who died developed spasmodic croup. The mother and friend treated the child as before, and did not call a doctor. The next day that child was taken to his family doctor, where an upper respiratory tract infection was diagnosed. The other sibling in the family had a mild upper respiratory tract infection at the same time. One week later the mother noticed some difficulty in her baby's breathing. The next morning he was found dead in bed with the mother. The mother developed a mild upper respiratory tract infection on the day that the child died, which continued for several days. The father had no respiratory tract symptoms. The health visitor who looked after both of these families confirmed this story of croup but had not thought it worth mentioning.

The epidemiology of spasmodic croup within this compound family unit and the findings of influenza B virus at necropsy suggest that the association between the croup and the death was not fortuitous. There are, however, no adequate morbid anatomical findings to explain the baby's death.

This is a rare history to obtain in a child presenting after a cot death. Cot deaths have many causes, often complex, but the epidemiological history in this case would not have been found but for our local follow up study. These parents were quite convinced that two doctors had told them that croup was not infectious and, while alarming, was not to be worried about. That was why a doctor was not called to the second child.

Should parents be told to call a doctor to all cases of croup? The child that died was the only baby under 6 months in this group. If a spaced transmission of croup had been recognised before this baby's death, could something have been done about it? What advice should be given about spasmodic croup in children attending playgroups and nursery schools?

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* * * Professor Milner replies below.—ED, *BMJ*.

SIR,—The pattern of illness described by Professor Emery and Dr Taylor suggests a spread of infection with a seven day incubation period. The parents were concerned because they had understood from their doctors that croup was not infectious. Acute croup represents a particular response to a viral infection which can produce a number of