acceptable" must be rejected. Any method of delivery that increases perinatal morbidity is open to challenge, particularly when that method is directly responsible for a perinatal mortality rate of 35 per 1000.

In their second paper Drs Chiswick and James ignore the role of the active management of labour in avoiding the type of complications leading up to Kielland's delivery. Several papers have been published which conclusively demonstrate that these problems are avoidable.[3-5]

None of the correspondents appear to recognise the essential difference between intracranial haemorrhage due to trauma and that due to asphyxia. Tentorial tears, which cause subdural haemorrhage, have been shown to be 11 times more common when forceps have been applied, with or without intrapartum hypoxia,[6] and virtually all cases of gross intracranial haemorrhage occurring during labour are due to trauma.[7] In contrast, asphyxial intracranial haemorrhage in the mature infant is characteristically subarachnoid and petechial.

Finally, in the broader context of the role of Kielland's forceps in modern obstetrics my experience convinces me that they have no place. This personal experience is based on exposure to about 12 000 deliveries where labour was actively managed and Kielland's forceps unavailable (at the National Maternity Hospital, Dublin). The papers from Drs Chiswick and James support this conviction.

P BOYLAN

Institute of Obstetrics and Gynaecology,
Hammersmith Hospital,
London W12

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[5] O'Driscoll, K, and Stronge, J M, *Australian and New Zealand Journal of Obstetrics and Gynaecology*, 1975, **15**, 1.
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SIR,—We were interested to read Drs Malcolm L Chiswick and David K James's articles on Kielland's forceps (6 January, pp 7 and 10). Their study comprised a group of 86 cases; we have examined the notes of 75 cases of delivery by Kielland's forceps carried out in this unit in the last 24 months. None of our patients had epidurals. We found certain interesting differences in the results and perhaps in our conclusions.

We detected no difference in the delayed onset of respiration (allowing for epidural) or the subsequent finding of transient irritability in the child. However, we were rather surprised that in their study the incidence of injury (facial or brachial palsy or cephalohaematoma) was 15·1% of patients, whereas in our study the incidence was only 6·7%. There were no stillbirths or neonatal deaths in our study whereas there were three in theirs.

In view of the indications for using Kielland's forceps, the complications in our study are acceptable. It would be unfortunate if Kielland's forceps were to develop a bad reputation for the second time in their life. Carefully used with the correct indications, with adequate safeguards so that the obstetrician can change his mind and carry out a caesarean section where necessary, these instruments remain of considerable value.

Perhaps in these litigenous times we should be using trial forceps in theatre much more frequently than in the past.

MICHAEL BURKE
CHRISTOPHER WOOD

Preston Hospital,
North Shields, Tyne and Wear

Retinal haemorrhages in infancy

SIR,—Last year you reported an innocent cause of extensive retinal haemorrhages in infancy, after a 2-month-old baby had been discovered as a near-miss case of cot death (4 February 1978, p 281). The problem was to decide if these haemorrhages were caused by hypoxia or the trauma of resuscitation, as such lesions normally provide important evidence of child abuse.

Since then I have looked for retinal haemorrhages in cases of sudden unexpected death in infancy before the necropsy. Such haemorrhages are frequently visible, provided that the cornea has not become too opaque. In all the cases in which they have been observed subsequent examination has shown the typical distribution of petechiae in the thoracic organs. No evidence has been found of the rare viral encephalitides which can produce retinal haemorrhages.[1]

This work was funded by the DHSS.

A N STANTON

Child Health Unit,
Princess Mary Maternity Hospital,
Newcastle upon Tyne

[1] Holzel, A, and Tobin, J O'H, *Lancet*, 1967, **2**, 723.

Cryptorchidism: a renewed plea

SIR,—Most surgeons with a paediatric practice will agree with Mr M C Bishop and Mr R H Whitaker (10 February, p 407) that too many boys present late for orchidopexy and that our aim should be to place both testicles in the scrotum by the time a boy starts school. I would make two practical suggestions.

Firstly, the true undescended testicle will not be in the scrotum at birth so a careful postnatal examination will identify all children requiring supervision. Just as discovery of a clicking hip leads to an appointment with the orthopaedic surgeon, so discovery of a testicle outwith the scrotum should lead to an appointment with the surgeon who would, in the course of time, be responsible for orchidopexy should it be required. The importance of the finding should be explained to the parents and an appointment given to them for the surgical clinic in six to nine months' time. This policy would not result in any major increase of work load in outpatients and should lead to a sharp improvement in quality of care.

The postnatal examination is an opportunity not to be missed because the next time that all children are examined is at school, in their 6th year, and it is clear, as Mr Bishop and Mr Whitaker point out, that all boys with true maldescent are not then directed to a surgeon. Only 21% of our patients have an orchidopexy before 5 years of age, and 32% have the operation between 5 and 7. This still leaves 47% who have their orchidopexy between their 7th and 13th birthdays, which is much too late. There seems to be a case for a specific examination of the time and method of referral of boys in whom maldescent is found at the school examination.

Secondly, operative technique can make a contribution to earlier operation. It is not easy by the standard Bevan approach to mobilise an impalpable incompletely descended testicle from around the deep abdominal ring of a boy of 2 or 3 years and place it in a good position in the scrotum, so there is a natural tendency to defer this operation until the boy is older. Using the abdominal extraperitoneal approach, however, which has recently been described,[1] the separation of the hernial sac from the testicular vessels and the vas becomes comparatively easy, and a greater length of the testicular vessels can readily be freed. Thus we have operated on boys aged 4, 5, 8, 10, 12, and 16 weeks with troublesome inguinal herniae as well as maldescent, and the operation proved to be simple and was successful both immediately and on review at one year. I believe this approach can make a useful contribution to earlier operation.

PETER F JONES

Royal Aberdeen Children's Hospital,
Aberdeen

[1] Jones, P F, and Bagley, F H, *British Journal of Surgery*, 1979, **66**, 14.

SIR,—May I strongly support the plea which Mr M C Bishop and Mr R H Whitaker make (10 February, p 407)? There is no evidence that any significant descent occurs after about 3 months of age in the full-term baby boy. The diagnosis of the deformity should be made in the first three months of life and is therefore within the province of the general practitioner and the paediatrician.

Parents are usually anxious to co-operate and if an operation to bring the testis into the scrotum is not done between the 3rd and 6th months (possible in some 30% of cases) a yearly visit to the surgeon should be made. He can then plan to operate before the age of 5 years.

The importance of a recorded diagnosis made in infancy is that it eliminates the endless confusion of trying to make an accurate diagnosis in a boy aged 5 to 12 years, when spontaneous retraction of one or both testes is so common. If it is known that both testes were in the bottom of the scrotum at birth then the high testis in later years must be retracted even though the examiner's fingers cannot draw it down. It will "drop" as puberty approaches.

C G SCORER

Gerrards Cross,
Bucks

Guar crispbread in the diabetic diet

SIR,—Professor David J A Jenkins and others (23-30 December, p 1744) have again demonstrated reduction in glycosuria and a small but significant reduction in fasting blood sugar when guar gum was added to the diets of diabetic patients over a five-day period. The long-term effects of such supplementation are, however, less clear cut and, although a reduction in insulin dosage and glycosuria occurred over an eight-week period, there was no change in body weight and blood glucose concentrations were apparently not estimated.

In a study which we have just concluded (to be reported in full elsewhere) we studied the effect of adding either guar gum, bran, or

placebo to the diet of 22 obese, poorly controlled diabetic outpatients. Over a three-month period, there was no significant reduction in body weight or of fasting or postprandial blood glucose concentration. The type of patient studied differed, as Jenkins *et al* had only two patients significantly above the desired body weight; but obese, poorly controlled patients would have been expected to benefit from any long-term effect of fibre additive. We would suggest that optimistic reports of the use of fibre supplementation in the therapy of diabetes mellitus should be confirmed by careful long-term studies.

M COHEN
F I R MARTIN

Department of Endocrinology,
Royal Melbourne Hospital,
Melbourne, Australia

Conservation surgery for laryngeal cancer

SIR,—Although I fully agree with your leading article (11 November, p 1318) that early vocal cord tumours are best treated with radiotherapy, I disagree with your statement that with conservative surgery the cure rate for small supraglottic tumours (presumably T1 and early T2 N0 lesions) is higher than with radiotherapy. Regrettably, you have failed to mention one of the largest series of supraglottic cancer treated by irradiation. This is the series from the Fondation Curie in Paris. It is an unfortunate omission, since this is one of the most prestigious radiotherapy centres in the world, where treatment of laryngeal cancer was pioneered several decades ago. In its recent studies[1 2] (its series is one of the largest in the world), which have been published in English, the absolute and determinate survivals at three years were 80% and 87% respectively for these early stages. The preservation of the voice was excellent in over 90% of the patients. It should be pointed out that conservative surgery can be performed only on a very select and favourable group of early cancers, whereas such a selection does not apply for patients referred to the radiotherapist. Furthermore, because of the high incidence of occult nodal metastases in supraglottic cancers, the neck nodes bilaterally have to be treated electively with radiotherapy, even if conservative surgery has been performed.[3 4]

You have quoted the results from centres in New York, Texas, St Louis, etc, but not from Paris, which is a stone's throw from London—or is it? I hope that you will be able to correct this unfortunate omission in your editorial.

N A GHOSSEIN

Department of Radiology,
Albert Einstein College of Medicine,
New York

[1] Bataini, J P, *et al*, *Cancer*, 1974, **33**, 1253.
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Typhoid fever

SIR,—Your leading article on typhoid fever (27 January, p 213) comments on isolation of *Salmonella typhi* from the blood stream for up to 10 days after the start of treatment and that there is no apparent explanation for this. In fact, the presence of *S typhi* in blood clot of patients on treatment with chloramphenicol can be demonstrated much later than this. We have seen it in patients after 17 days' treatment and it is probable that such isolation may be obtained at any stage of illness in spite of therapy.[1]

The reason almost certainly relates to the intracellular location of the organism in reticuloendothelial cells and to their ability to survive there and reinvade the blood stream periodically. We have shown that such intravascular shedding occurs in typhoid carriers and indeed may be the only manifestation of the carrier state.[2] Such isolations may also be made from patients with no previous history of clinical typhoid fever.[2] Both in this group and in patients with clinical relapse during the acute disease release of intracellular organisms will result in positive blood cultures. In the latter group sufficient organisms are released to provoke clinical signs and symptoms. The nature of the host-parasite relationship in typhoid fever is such that phagocytic uptake and release of organisms probably occurs continuously during the acute disease. Blood sampling will then detect those organisms that have just recently been released and which have not yet been destroyed or inhibited by antibiotic. Once removed by antibiotic or by antibody and complement mechanisms they are replaced from the reticuloendothelial sites. It has also been shown that both penicillin and streptomycin are capable of stimulating phagocytic uptake of bacteria[3] and it is possible that antibiotic treatment may increase the potential reservoir of organisms in reticuloendothelial sites capable of reinvading the blood stream.

K C WATSON

Central Microbiological Laboratories,
Western General Hospital,
Edinburgh

[1] Watson, K C, *American Journal of Tropical Medicine and Hygiene*, 1957, **6**, 72.
[2] Watson, K C, *Lancet*, 1967, **2**, 332.
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Beta-blockers, blood sugar control, and renal function

SIR,—I was interested to read the paper from Dr A D Wright and his colleagues comparing metoprolol and propranolol in diabetics (20 January, p 159). I would agree that on their evidence there is no strong reason for choosing a cardioselective beta-blocker from the point of view of the minor differences in hyperglycaemia associated with their use. However, I think that they should have emphasised more the benefits of cardioselectivity in the recovery from insulin-induced hypoglycaemia, the evidence for which they review in their introduction.

My main purpose in writing is to confirm their observation of a greater deterioration in renal function during treatment with a non-selective compared with a cardioselective beta-blocking drug. We have recently completed a study comparing propranolol and atenolol in the treatment of 15 patients with essential hypertension in an open crossover trial. In doses producing equal reduction in blood pressure the creatinine clearance fell from $162 \pm 9{\cdot}4$ (SE) l/24 h in the untreated state to $132 \pm 8{\cdot}5$ l/24 h during propranolol treatment ($P < 0{\cdot}01$), but only to $152 \pm 13{\cdot}9$ l/24 h during atenolol treatment (P 0·05). The reduction in function during propranolol treatment was significantly greater than that during atenolol treatment ($P < 0{\cdot}0125$). It is well known that non-selective beta-blocking drugs reduce creatinine clearance,[1-3] and it is of interest that there is now evidence that both of the most cardioselective drugs available cause less reduction in renal function. These differences in effect on renal function are, of course, of little importance in essential hypertension, but may be important in patients with renal disease.

R WILKINSON

Department of Medicine and Nephrology,
Freeman Hospital,
Newcastle upon Tyne

[1] Nayler, W G, *et al*, *American Heart Journal*, 1967, **73**, 207.
[2] Schirmeister, J, *et al*, *Arzneimittel Forschung*, 1966, **16**, 847.
[3] Ibsen, H, and Sederberg-Olsen, P, *Clinical Science*, 1972, **44**, 129.

Mechanisms of sudden death

SIR,—Your leading article (23-30 December, p 1734) suggested that platelet embolism could be the precipitating factor for sudden death. I think this must be rarely so. Roberts has shown that, with few exceptions, neither sudden death nor myocardial infarction occur until two of the three main coronary arteries are narrowed by 75%.[1] It seems unlikely that platelet embolism would be delayed until this critical stage had been reached. Moreover, if platelet embolism were to blame, sudden death would occur more often during exertion or stress rather than at rest. An explanation more directly related to the degree of coronary narrowing is required.

Roberts argues that blood stasis from reduced perfusion is the initiating mechanism for ischaemia leading to heart attack. For ventricular fibrillation then to occur, the work of Lubbe *et al*[2] suggests that critical metabolic gradients must develop between well and poorly perfused myocardium. If fibrillation does not occur and ischaemia should persist, myocardial infarction could develop with or without thrombosis, depending on duration of survival.[1]

The question now arises: what causes the reduced perfusion? Given the prerequisite of 75% coronary narrowing of two vessels, which are presumably rigid and virtually incapable of dilatation in response to demand, elementary principles of physics should apply. Quite small falls in blood pressure could reduce coronary perfusion critically and predispose to a heart attack if collateral circulation is stretched. It may be relevant that the following circumstances are associated with a poor prognosis: (*a*) when blood pressure falls as a result of exercise in patients with known ischaemic heart disease[3 4]; (*b*) After recovery from myocardial infarction, if the blood pressure remains more than a few mm Hg lower than its preinfarction level[5 6]; (*c*) When the blood pressure falls over the months or years following recovery from myocardial infarction.[7]

It may also be relevant that smokers have a lower blood pressure than non-smokers,[8-10] which in the practolol trial persisted for about a year after giving up smoking.[10] An increased mortality also persists for some time after giving up smoking. Finally, heart attacks often occur during sleep when oxygen requirements are minimal but when blood pressure is at its lowest.

Blood pressure reduction may, of course, be an expression of existing cardiac damage, as in the first three studies cited above.[1-3] But