the possibility, notwithstanding the conjectural opinions reported by Scarisbrick, McLennan, and McNalty, that the fact of Henry Carey's survival to maturity at all postulates (on the face of the undoubted evidence relevant to the *non-surviving births* to the King) a further theory that he may actually have been the legitimate son of Sir William Carey and Mary Howard.

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[1] Leslau, J, *Ricardian*, 1978, **4**, 2.

Intraoperative management of phaeochromocytoma with sodium nitroprusside

SIR,—We read with interest the article by Dr Peter Daggett and others (29 July, p 311) and would agree with their conclusion that sodium nitroprusside "is a useful hypotensive agent [in the operative management of] patients with a phaeochromocytoma." We would, however, be grateful for an explanation of the rationale behind their choice of 0·5 mg/kg as a maximum total dose of the drug, as they present no evidence in support of this figure.

Our own studies,[1] based on data from over 300 patients and many animal experiments in which plasma and red cell cyanide were monitored, lead us to conclude that 1·5 mg/kg can safely be regarded as a maximum dose for short-term infusion. The apparent arbitrary establishment of such a low limiting dose as 0·5 mg/kg could possibly lead to a lack of realisation of the full potential of this drug.

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[1] Cole, P V, *Anaesthesia*, 1978, **33**, 473.

**We sent a copy of this letter to Dr Daggett and his colleagues, whose reply is printed below.—ED, *BMJ*.

SIR,—The maximum dose of sodium nitroprusside (SNP) of 1·5 mg/kg recommended by Drs Krapez and Cole is the same as that suggested by Vesey *et al*[1] and is applicable to situations where prolonged hypotension is required. In the very particular circumstance of arteriography or operation in cases of phaeochromocytoma, however, episodes of hypertension are usually short-lived. In a 70-kg man the upper limit advised by Drs Krapez and Cole would allow the infusion of 105 mg of SNP "short term." Most phaeochromocytomas are excluded from the circulation within 1-2 h of starting surgery, and at this point the need to infuse SNP ceases. Thus at the outside the infusion would last for 120 min, and in order to utilise the 105 mg computed above the infusion would have to deliver 875 μg/min for the whole of this time. Vesey and Cole[2] have observed that the rate of infusion is probably a more important determinant of possible toxicity than the total dose infused, and rates in excess of 800 μg/min are regarded as hazardous.[3]

The preparatory α- and β-adrenoceptor-blocking regimen described in our paper makes it unnecessary to use doses of this magnitude. A dose of 0·5 mg/kg in our hypothetical 70-kg man is equivalent to 350 ml of a 0·01% solution, a volume which is seldom even approached in this situation. To recommend a larger dose would encourage administration at a potentially toxic rate and this could bring into disrepute a drug which we are agreed is invaluable.

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[1] Vesey, C J, *et al*, *British Journal of Anaesthesia*, 1976, **48**, 651.
[2] Vesey, C J, and Cole, P V, *British Journal of Anaesthesia*, 1975, **47**, 115.
[3] Roche Pharmaceuticals, *Nipride*, p 24. Welwyn Garden City, Roche Products Ltd.

Copper intrauterine devices in the abdomen

SIR,—Mr P J M Watney's paper (22 July, p 255) prompts me to report an experience with a Copper 7 intrauterine device which supports the belief that these devices, if in the abdominal cavity, should be removed as soon as reasonably possible.

The patient was referred when approximately 18 weeks pregnant in her fourth pregnancy. Two years previously she had had a Copper 7 intrauterine device inserted, and the insertion had been very painful.

The pregnancy continued to term and ended in a normal delivery. The device was not found at the time of delivery, either with the placenta and membranes or in the uterus. A plain abdominal *x*-ray showed the device to be lying above the level of the umbilicus. The patient had requested laparoscopic sterilisation six weeks post partum and this was undertaken, but the device could not be located with the laparoscope.

Formal laparotomy was undertaken and after a prolonged search the device was located high in the small-bowel mesentery, penetrating the wall of the superior mesenteric artery as it emerged under the body of the pancreas. It was removed with great difficulty; after the removal the intima of the vessel was bulging into the cavity left by the device. The wall of the artery was repaired with fine silk sutures and the patient made an uneventful recovery.

This case suggests that the peritoneal cavity's reaction to such a device is more violent than to a non-metal-containing device. In this case it could only have been a matter of time before erosion into the lumen of the superior mesenteric artery occurred with what could have been catastrophic consequences.

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Diflusinal and Stevens-Johnson syndrome

SIR,—Cholestatic jaundice associated with diflunisal therapy has been reported by Dr Jonathan S Warren (9 September, p 736). We would like to add Stevens-Johnson syndrome as a further probable side effect of diflunisal therapy.

The first patient was an amputee aged 25 treated for pain in her back and remaining knee with 250 mg of diflunisal twice daily. She had been taking Paramol 118 (paracetamol and dihydrocodeine tartrate) for at least three months, and 10-14 days after the addition of diflunisal to this regimen she became ill with involvement of skin, conjunctiva, and mucous membranes typical of severe Stevens-Johnson syndrome.

The second patient, aged 54, was receiving 500 mg of diflusinal twice daily for pain related to cervical osteoarthrosis. In addition she had been taking Ferrograd C (ferrous sulphate and sodium ascorbate) for six months and lorazepam for one month. Two weeks after starting diflunisal she had an exactly similar presentation to the first patient, with conjunctiva, skin, and mucous membrane involvement typical of severe Stevens-Johnson syndrome. In this patient there was also a transient elevation of aspartate and alanine transaminases and creatine kinase. Both patients had a mild thrombocytopenia and a striking feature of both has been the persistence of oral lesions.

Further work is in progress to elucidate the relationship between diflunisal and Stevens-Johnson syndrome in these cases.

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Changing advice on vaccination

SIR,—As a poor bemused general practitioner struggling to keep abreast of medical progress, I am at a loss to know what recent advances in knowledge prompted the current changes in vaccination procedures.

In 1972 we were told that in view of recent advances in immunological knowledge it was undesirable to immunise infants under 6 months old owing to the incomplete immunity conferred and the risks of side effects. This seemed logical and the revised schedule received general compliance.

In 1978 we are told by the same experts that, following recent changes in knowledge and policy it is now desirable to immunise 3-month-old infants. By what criteria was it judged that the benefits of incomplete immunity conferred on a 3-month-old baby outweigh the risks of possible brain damage, which I would assume to be highest in this age group? Is this new schedule a temporary measure brought in to combat the present pertussis panic and will the experts recommend a return to the old regimen next year? Having weathered the recent wave of anxious mothers asking whether or not their 6-month-old babies should be "done" against whooping cough and having recommended vaccination, I do not relish the prospect of indefinitely immunising the younger age group.

In addition, the Department of Health and Social Security now recommends, after 15 years or more of using oral polio vaccine, that we should discard the remnants of a vial after an immunising session. This raises two important points. Firstly, why has it taken all these years to publicise adequately something which should have been apparent in the early stages of any research programme? And secondly, if the remaining vaccine in a vial is so unreliable are the hundreds of babies and