gave rise to suspicion. The second category would be restricted to all sudden deaths in subjects over a certain age (around 40) in which there were no suspicious or complicating circumstances. A proportion of these, say 5%, would be selected randomly for full necropsy; the results would be the basis of a statistical analysis of the cause of death of the group as a whole. The remaining 95% of cases would be subjected to detailed external examination by a qualified pathologist. Should any significant abnormality be discovered—for example, petechial haemorrhages suggesting death by suffocation—the case would be recategorized into group 1 and a full necropsy performed. If no external abnormalities were present samples of stomach contents and a wedge of liver tissue could be rapidly obtained by the mortuary technician and submitted for toxicological screening at a forensic science laboratory. If a well-organized national system was adopted there is no reason why such tests should not be automated and take only a few days. In the interim burial would be possible but cremation would await clearance by the toxicological laboratory.

The potential reduction in the number of coroners' necropsies by this method would be very considerable in the country as a whole.—I am, etc.,

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Azathioprine in Psoriasis

SIR,—Following the report of azathioprine in the treatment of psoriasis by Dr. A. du Vivier and others (12 January, p. 49) I thought it would be of interest to report the possible myopathic effect of azathioprine in this disease. Myopathy is not uncommon in renal transplant patients on both azathioprine and prednisone¹ but myopathy due to azathioprine alone has rarely been reported.

A 62-year-old man who had had psoriasis for 16 years developed exfoliative dermatitis three months before a course of azathioprine. Systemic methotrexate had been stopped because of haemorrhagic cystitis. After a month on azathioprine 200 mg daily (2.4 mg/kg) he developed progressive weakness of the legs and difficulty in walking. There was marked grade 4 weakness of the deltoids and supraspinati with absence of arm jerks and similar weakness of the proximal leg muscles with depressed knee jerks, absent ankle jerks, and flexor plantar responses. The muscles were not tender and there was minimal wasting. There was no clinical evidence of peripheral neuropathy and the clinical impression of myopathy was confirmed on electromyography. At this time the patient was putting about 80 g clobetasol propionate 0.05% ointment daily on to his skin without occlusion. After stopping azathioprine and changing the clobetasol ointment to betamethasone 17-valerate 0.1% diluted 10 times, muscle strength returned to normal within three weeks.

Systemic absorption of local fluorinated steroids has not previously been reported to produce myopathy and it seems likely that in this patient azathioprine alone produced the myopathy.—I am, etc.,

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¹ *British Medical Journal*, 1966, 2, 1378.

Battering: Unfortunate Backlash

SIR,—Having for some years been concerned with battering parents and those suspected of abusing their child, I agree

wholeheartedly with Dr. J. W. Woodward's letter (9 March, p. 452). Several instances known to me illustrate that after much initial reluctance to recognize the syndrome there may now be a real danger of hasty and insensitive steps being taken against innocent parents by workers who act, or are asked to act, on uncorroborated assumptions.

The problem contains other elements than those of careful diagnosis and investigation. It happens that all too often both confirmed and suspected cases are not handled with the skill that is required if the aim of the intervention is to improve the parent-child relationship and not just to achieve some spurious short-term "protection" of the baby. In order to be able to help effectively workers require special training and a better understanding of the reasons why even loving mothers are driven to harm their baby.

It has to be appreciated that battering is not an unrelated component of behaviour, and it cannot therefore be evaluated and treated in isolation. The syndrome is only one manifestation of a whole complex of interrelated and not uncommon conflicts both in women and men who find it difficult to adjust to their role as parents. Many of the women who feel inadequate in face of their maternal responsibility deal with this by avoiding close contact with their baby in an attempt to protect it from their aggression. The vulnerability and the special emotional problems of the fundamentally insecure mother require that she be approached in a way which does not undermine her already weak confidence and self-esteem. Above all, nothing must be said or done to imply even the slightest suspicion of child abuse until there is irrefutable evidence of it. The merest hint of such a suspicion is bound to be sensed by the innocent mother as a sign that her maternal adequacy is doubted, and this happens at the precise moment when she needs encouragement and support. By not focusing on the issue of battering but by attempting to cope with problems and their roots in the context of the wider spectrum of the parent-infant relationship the danger of causing irreparable harm to emotionally vulnerable families is avoided.

Whatever form the management of such cases take, it should be based on the recognition that battering is only one form of child abuse and that the scars of emotional abuse, for which there should be an equal measure of concern from the helping professions, are no less crippling to the child.—I am, etc.,

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Parents of Battered Babies

SIR,—Dr. J. K. Sarsfield and Mr. A. C. Dowell (30 March, p. 637) are quite correct in their assertion that anomalies of family status have a bearing on the circumstances leading to baby battering. Our results on this are to be published in detail elsewhere,¹ but the importance of the points Dr. Sarsfield and Mr. Dowell raise merits immediate comment.

In our sample lack of family cohesiveness was an important precursor of baby battering. In over one-third of cases the biological father was absent from home and in half the mothers were living with some other man.

Half the mothers had married before the age of 20 years and nearly three-quarters had conceived premaritally—a combination particularly likely to lead to marital breakdown.² Such mothers also had strikingly more premarital pregnancies and illegitimate births when compared with the general population, suggesting that the inconvenience of an unwanted child was an important motive underlying baby battering. Short acquaintance before marriage, disharmony in child rearing, and dissatisfaction with their partner's handling of the child were other predictors of divorce³ found in our sample. These parents also had little contact with their own parents and relatives and thus lacked the stabilizing force of a kinship relationship.⁴ Considering that a significant proportion of our parents suffered all these marital adversities and that one-third of battered babies were already living with one natural parent only it may be predicted that a substantial number of such children will eventually grow up in broken homes and be at risk of social maldevelopment and physical, and possibly fatal, injury.

Because the similarities between baby battering and other forms of deviant behaviour are striking¹ and because innumerable studies of deviant behaviour have been remarkably unrewarding, not only in establishing causes but also treatment, it is my contention that if doctors are to prevent re-battering then this is one area where they must be authoritative, intrusive, and insistent. As with crime and delinquency, treatment is likely to be only a supporting exercise. True prevention must lie in the effective education of the next generation and in possible legal changes in child care.—I am, etc.,

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¹ Smith, S. M., Hanson, R., and Noble, S., *British Journal of Psychiatry*. In press.

² Rowntree, G., *Population Studies*, 1964, 18, 147.

³ Goode, W. J., in *Sociology of the Family*, ed. M. Anderson, p. 301. Harmondsworth, Penguin Books, 1971.

⁴ de Lissovoy, V., *Journal of Marriage and the Family*, 1973, 35, 245.

Coxsackie B Virus and Diabetes

SIR,—Dr. D. R. Gamble and others (3 November 1973, p. 260) recently reported the association of positive titre levels for Coxsackie B4 virus and diabetes mellitus, supporting their earlier findings.^{1,2} The significant association was in the 10- to 19-year-old subjects. The incidence of positive titres is high in the population they studied, and since from their data the onset of infection and the onset of diabetes cannot be analysed no cause and effect relationship can be deduced. A true association of onset of infection, persistence of titre levels, and onset of diabetes could be evaluated in a community without prior exposure to the virus which suffered an epidemic, as Hadden and others suggested.³

We recently studied such a community on the Pribilof Islands in Alaska, U.S.A. In the winter of 1967-8 an epidemic caused by Coxsackie B4 occurred in a previously unexposed population, as determined by pre-epidemic sera. Of the 116 persons with available pre-epidemic sera only two had positive neutralizing antibody titres (1:4,

1:16). After the epidemic 89 (77%) had a four-fold titre rise. Of those under 20 years of age 46 out of 50 had positive titres after the infection. Clinical illness occurred in over 50% of infected subjects.

Five years later a follow-up study was done using 75-g oral carbohydrate load glucose tolerance test. Serum neutralizing antibody titres and two-hour plasma glucose levels were measured. Of the 136 persons under 25 years of age (M.= 65, F.= 71) the highest two-hour plasma glucose was 133 mg/100 ml. Persistence of positive titre levels was also documented since 77% of these subjects had a positive neutralizing antibody titre level.

Even though it was in the young diabetic that Dr. Gamble and his colleagues were able to show the strongest association with positive Coxsackie B4 titres we could not show that Coxsackie B4 infection caused a single case of diabetes, though the attack rate in the 136 under 20 years of age at the time of the epidemic was extremely high.—We are, etc.,

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- 1 Gamble, D. R., et al., *British Medical Journal*, 1969, 3, 627.
- 2 Gamble, D. R., and Taylor, K. W., *British Medical Journal*, 1969, 3, 631.
- 3 Hadden, D. R., et al., *British Medical Journal*, 1972, 4, 729.

Serotonin and the Mesenteric Circulation

SIR,—Dr. I. M. Murray-Lyons and others (29 December, p. 770) reported some unusual cases of gangrene of the small intestine in patients with carcinoid tumours of the small bowel and with raised blood levels of 5-hydroxytryptamine (5-HT). They found proliferative changes in the elastic tissue in the wall of the mesenteric vessels. Anthony and Drury,¹ reporting similar changes in the elastica in cases with small bowel carcinoid, postulated that some substance or substances produced by the carcinoid tumour caused the changes in the elastica, which in turn compromised the lumen and contributed to bowel necrosis in some cases. This hypothesis has not been proved directly. The elastica lesions have not been reproduced experimentally and the theory does not explain the presence of the elastica lesions almost exclusively in the mesenteric circulation.

In a recent laboratory investigation in dogs blood flow was measured with an electromagnetic flow meter simultaneously in the thoracic aorta, in the superior mesenteric artery (S.M.A.), and in the renal artery. An intravenous injection of 5-HT (0.03 mg/kg) caused a reduction in flow, which was most marked in the S.M.A. (fig.). The reason for this specific sensitivity of the mesenteric vascular bed to 5-HT is not known. Nor can it be stated with certainty that the human mesenteric circulation responds to 5-HT in a similar manner. However, this observation does suggest an alternative hypothesis for the aetiology of the elastica changes in the mesenteric vessels—that a continuously raised level of 5-HT in the blood may lead to a chronic increase in re-

Effect of intravenous injection of 5-HT (arrow) on flows (ml/min) and blood pressure (mm Hg).

sistance in the mesenteric circulation. The sustained increased resistance causes the degenerative changes in the elastica in the branches of the S.M.A. that in turn contribute to ischaemic necrosis in some cases of small bowel carcinoid.—We are, etc.,

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- 1 Anthony, P. P., and Drury, R. A. B., *Journal of Clinical Pathology*, 1970, 23, 110.

Serotonin Metabolism in Hepatic Encephalopathy

SIR,—We read with great interest the paper by Dr. A. J. Knell and others (23 March, p. 549) on disturbed serotonin metabolism in hepatic encephalopathy. Our interest in the metabolism of serotonin (5-HT) was aroused by a patient with cirrhosis of the liver who excreted large amounts of 5-HT and its precursor 5-hydroxytryptophan (5-HTP) in urine.¹ The hepatic metabolism of 5-HT in experimental cirrhosis was characterized by decreased formation of sulphate conjugates, increased synthesis of glucuronide and conjugates,^{2,3} decreased uptake of 5-HT by the liver,³ and deranged enterohepatic circulation of metabolites of 5-HT.^{3,4} Metabolism of 5-HT in the mucosa of the small intestine was disturbed only in the advanced stage of the cirrhosis, when the mucosa lost its ability both to store and metabolize 5-HT.³

A raised concentration of 5-hydroxyindole acetic acid (5-HIAA) in cerebrospinal fluid might well be related to increased excretion of 5-HT and 5-HTP in urine. According to our preliminary observations the storage and metabolism of 5-HTP in the small intestine mucosa of cirrhotic rats is disturbed like the intestinal metabolism of 5-HT. Thus an increased efflux of 5-HTP into the portal circulation may result in an increased transfer of 5-HTP to the central nervous system, where it is metabolized through 5-HT to 5-HIAA. This mechanism might be involved in the pathogenesis of the observed increase of 5-HIAA concentration in cerebrospinal fluid in patients with hepatic encephalopathy.—We are, etc.,

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- 1 Mustala, O., Kekki, M., and Pentikäinen, P., *Annals of Clinical Research*, 1969, 1, 197.

- 2 Pentikäinen, P., *Annales Medicinæ, Experimentalis et Biologiæ Fenniae*, 1970, 48, 135.
- 3 Pentikäinen, P., Kekki, M., and Mustala, O., *Scandinavian Journal of Gastroenterology*, 1969, 4, Suppl. 4.
- 4 Kekki, M., Mustala, O., and Pentikäinen, P., *Gastroenterology*. In press.

Laparoscopic Tubal Ligation

SIR,—I was extremely interested in the letter by Dr. K. R. Greene and others (6 April, p. 54). Only three days before that date and quite independently I did my first laparoscopic Pomeroy sterilization by virtually the same method as they described. I would confirm that it seems simple and safe and that a search of the literature suggests that it has not been tried previously. In thin patients with a mobile uterus there would seem to be no need for two lower quadrant incisions because a single midline stab 2-3 cm above the symphysis pubis suffices. It would seem that the main hazards of the procedure are likely to be haemorrhage due to tearing of the mesentery of the Fallopian tube and, if one uses two stab incisions, accidental puncture of the inferior epigastric vessels by the operating cannula—a complication which should be avoidable if one remembers the surface markings of these vessels. Both complications should be excluded before withdrawing the laparoscope. Neither would be as serious as bowel burns and either would be an indication for laparotomy, for which consent should always have been obtained.

In due course I too hope to report a large series of patients having a laparoscopic Pomeroy sterilization. It would seem important to make an early and full assessment of a technique which requires only ordinary laparoscopy equipment, permits tubal ligation and transection, avoids the hazards of diathermy, and is surprisingly easy.—I am, etc.,

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- 1 Greenhalf, J. O., and Roberts, H. R., *British Medical Journal*, 1971, 3, 304.

Death during Dental Anaesthesia

SIR,—I have read with interest Dr. J. G. Bourne's letter on this subject (16 March, p. 516), but I do not feel that he has adequately emphasized the fact that a young, healthy person died needlessly. The patient had an unrecognized faint during the intravenous induction of anaesthesia in spite of the presence of the cardinal sign of pallor. In spite of this pallor and while in the sitting position in the dental chair pure nitrous oxide was given. The anaesthetic was administered by an operator anaesthetist, a practice which is not supported by the Society for the Advancement of Anaesthesia in Dentistry.¹

While tragic errors of judgement have been made by most of us in the course of our professional careers we must realize that the price of liberty is eternal vigilance. I routinely anaesthetize the dental patient in the supine position, but this is purely because of the trend in dental ergonomics. I have never shared Dr. Bourne's anxiety about the sitting position and I never hesitate to use this position if the patient has, for example, a hiatus hernia or an abdominal