chosen will influence the results. This type of debate exposes our basic lack of both techniques and data to tackle the question of objectives of the Health Service. I am sure Mr. Teeling-Smith would agree with these deficiencies; as an economist he will, I hope, join us in remedying our shortcomings rather than in indulging in polemic.—I am, etc.,

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Poisoned Children

SIR,—I was surprised to read that Dr. J. R. Sibert (26 October, p. 231) considers that either emesis or gastric lavage is effective as a means of emptying the stomach in children. The paper he refers to¹ contains no comparative studies on quantities removed by the two methods. The article does mention two small previous studies which showed that (1) chemically induced emesis in children is an inefficient and unreliable means of emptying the stomach and (2) gastric lavage as it is usually performed in children is even less effective. With emesis the average returns were only about 30% of what was considered to be present in the stomach.

A study I carried out on a series of 378 adults showed that with gastric aspiration and lavage an average return of about 80% of a known quantity of magnesium sulphate could be obtained by modifying the position of the patient and the technique. I know of no similar study in children but would suggest that research should be directed in this direction. In the fortunately uncommon situation, in which it is thought that a child has ingested a dangerous poison it should be appreciated that chemical emetics are unlikely to remove most of the poison contained in the stomach. While not disagreeing with the use of chemical emetics in the majority of these cases I would suggest it should be preceded by immediate gastric aspiration and lavage, using the left Simms position with the table tilted, head down. This is the most effective position in adults. A nasogastric tube with the child sitting upright and wrapped in a blanket is probably useless.

The low death rate from poisoning in children is not related to effective first-aid management but to the fact that children rarely take dangerous poisons. It is unlikely that we will be able to improve the efficiency of chemical emetics. There is, however, some prospect of improving the efficiency of lavage in children if research is directed in this direction.—I am, etc.,

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¹ Reid, D. H. S., *Archives of Disease in Childhood* 1970, 45, 428.

Skin Reactions to Beta-blockers

SIR,—I note with interest the letter from Dr. Constance M. Ridley (26 October, p. 229) regarding her experience of an exacerbation of psoriasis while treating two patients with practolol.

This spring I saw a similar episode whilst treating a man of 65 who had quiescent mild

psoriasis with Trasacor (oxprenolol). Like the cases cited by Dr. Ridley there was an intense, fiery, annular erythema and underlying oedema on the legs and soles of the feet occurring a few weeks after introducing oxprenolol, but taking at least eight weeks to clear completely following withdrawal of the drug. The lesions he had previous to taking the drug had been dry scaly patches on the shins, hands, and elbows which had been present in that condition for about 10 years.

The manufacturers of practolol have suggested that if there is a reaction to their drug then another beta-blocker should be substituted. In the light of the experience I have had such advice would seem to be unwise.—I am, etc.,

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Anaesthesia during Raised Creatine Phosphokinase Activity

SIR,—I find it difficult to draw any worthwhile conclusions from the paper by Drs. G. Owen and R. J. Kerry (12 October, p. 75), which is sadly lacking in scientific detail.

In the discussion it is noted that none of the patients developed haemoglobinuria or any hyperpyrexia. No mention is made at any stage in the paper of the technique employed for the measurement of haemoglobinuria, nor is there any mention of how temperature was monitored or for how long. It is not obvious from the paper how many patients had raised creatine phosphokinase (CPK) activity before therapy was instituted and how many had raised values as a result of therapy. It is well known that depolarizing muscle relaxants will produce an increase in CPK activity.¹ Innes and Strømme² found postanaesthetic CPK values of up to 40 times the normal limit after an apparently normal general anaesthetic, and they concluded that suxamethonium is the most important agent for the release of CPK from muscle during anaesthesia. The muscle activity of a modified convulsion from electric convulsion therapy probably also contributes to a rise in CPK activity. It is not clear whether the blood specimens for CPK estimations were taken under optimal conditions. Some investigators³ emphasize the need for a standardized technique of specimen collection for reproducible results.

The authors seem to think that general anaesthesia might be withheld from a patient found on routine testing to have a raised CPK activity. This is not strictly so. For some time anaesthetists have interpreted raised CPK levels with caution not only because normal values differ from one laboratory to the next but also because of much variation from one sampling to the next in the same patient. This variation is dependent on the amount of exercise during the preceding few days, the degree of tissue hypoxia imposed by a tourniquet on the arm from which the sample has been extracted, and errors in the measurement technique. The value of the resting CPK activity as a screening test for susceptibility to malignant hyperpyrexia has been questioned.⁴ The CPK activity is raised not only after exercise but also in certain diseases (in addition to psychotic illnesses) which are unrelated to malignant hyperpyrexia—for example, Duchenne muscular dystrophy,

polymyositis, hypothyroidism, acute myocardial infarction, gas gangrene, and conditions of muscle hypoxia or damage. Thus if CPK activity is used as a sole test of susceptibility to malignant hyperpyrexia many false-positive results will be obtained.—I am, etc.,

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- 1 Tammisto, T., and Airaksinen, M., *British Journal of Anaesthesia*, 1966, 38, 510.
- 2 Innes, R. K. R., and Strømme, J. H., *British Journal of Anaesthesia*, 1973, 45, 185.
- 3 Parikh, R. K., and Thomson, W. H. S., *British Journal of Anaesthesia*, 1972, 44, 742.
- 4 Ellis, F. R., et al., *British Medical Journal*, 1972, 3, 559.

Cervical Plasma Cell Population in Infertile Patients

SIR,—Dr. Rosalind A. Hinton's comment (19 October, p. 168) on the postcoital and other tests such as the sperm penetration test is most relevant. I was pleased she used the term "cervical hostility" rather than "immunological infertility." The former, as I am sure Dr. Hinton would agree, is much broader in that it includes the problem of mechanical hostility as well as the possibility of an "allergic factor." Linking our plasma cell positive cases with other immunological tests would be one of the next steps in developing this new approach to the investigation of immunological infertility.

There are a number of tests in use, the postcoital and sperm penetration tests being two of the less complex. At the outset of our work a negative postcoital test was used as a starting point for our investigations, particularly in conjunction with our normal "essential triad." We did find that there was an increase in the number of negative postcoital tests in those patients who had plasma cells containing IgA in their cervixes. However, in a personal review of the results of some 350 postcoital tests from one infertility clinic I was surprised to find that there were roughly the same number of negative postcoital tests in those patients who subsequently became pregnant as in those patients who remained infertile.

The important point is that postcoital tests must be carried out under strictly controlled conditions, and I feel that the margin of error is greatly increased by clinical impressions rather than more scientifically controlled studies such as those described by Kunitake and Davajan.¹ This is not to detract from the clinical use of the test but its limitations must be recognized. This applies even more to complicated techniques. The problems revolving around the sperm agglutination and sperm immobilization tests have continued for years, even before the work of Franklin and Dukes,^{2,3} and are well documented. As emphasized by Jeffrey and Parish,⁴ these more sophisticated tests require training and, more important, constant practice in the technique and interpretation of findings before the results can be considered valid.

In our work we touched on a number of other aspects of this relatively new field. Perhaps one of the more interesting and slightly puzzling discoveries was that we estimated the serum IgA in seven of the patients with plasma cells present in their cervixes and found it lower than normal in six when we might have expected to find

raised levels. I can only repeat that there is a need for much more work on this interesting line of research.—I am, etc.,

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- 1 Kunitake, G., and Davajan, V., *Fertility and Sterility*, 1970, 21, 706.
- 2 Franklin, R. R., and Dukes, C. D., *American Journal of Obstetrics and Gynecology*, 1964, 89, 6.
- 3 Franklin, R. R., and Dukes, C. D., *Journal of American Medical Association*, 1964, 190, 682.
- 4 Jeffery, W., and Parish, W. E., *Clinical Allergy*, 1972, 2, 261.

Retinitis Pigmentosa and Squint in Africans

SIR,—May I correct some inaccuracies in two recent leading articles of ophthalmological interest?

Despite what you affirm (17 August, p. 429), retinitis pigmentosa is an important cause of blindness in Africans in East Africa and is also quite as common in females as in males. I had occasion recently to examine the inmates of a girls' school for the blind and out of 35 pupils no less than eight had retinitis pigmentosa. Though accurate statistics are not available, it is the impression of a number of ophthalmologists in East Africa that retinitis pigmentosa is in fact rather commoner here than in Europe and the reason for this may be that it is common for marriages to take place between cousins.

It is equally untrue to say that "squints are extremely rare in coloured races" (17 August, p. 430). Squints are very common in East Africa and have been known for a long time. Most of the local languages, including Kiswahili, have their own words for squint and many are the old wives' tales connected with them. In the past because of the very high illiteracy rates little attention was paid to this cause of loss of sight, but it is now becoming important.—I am, etc.,

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Gianotti-Crosti Syndrome and Viral Infection

SIR,—Recently there has been some discussion on what constitutes the Gianotti-Crosti Syndrome (leading article, 9 March, p. 407). Gianotti now defines two syndromes—namely, the papular vesicular acrolocated syndrome and papular acrodermatitis of childhood, which has a similar rash but with coexisting acute hepatitis and hepatitis B antigen in the serum.^{1,2}

In the past four years specimens for virological examination were obtained from six patients (four females and two males aged between 1 and 7 years) who had the classic rash of Gianotti-Crosti syndrome. The illness ran a non-relapsing course of 14-21 days and lymphadenopathy was noted in two patients. Three serial faecal samples were obtained from all patients and inoculated into primary rhesus monkey kidney and HEp2 cell cultures. Adenovirus type 1 and adenovirus type 2 were isolated from the two patients with lymphadenopathy and echovirus type 9 was isolated from a third patient. Acute and convalescent phase serum

samples from all the patients were tested, using the complement fixation test, against mumps, measles, herpes simplex, lymphocytic choriomeningitis virus, psittacosis, *Rickettsia burneti*, and *Mycoplasma pneumoniae* antigens and using the rubella haemagglutination inhibition test. Significant antibody titres against the above antigens were not found. Hepatitis B antigen was not detected in any of the sera using the immunodiffusion test or the much more sensitive haemagglutination tests³ (Wellcome Reagents Ltd. and Organon Teknika).

Though there was evidence of a viral infection in three patients it is impossible to say if the viruses were responsible for the rashes. Some viruses are often associated with skin eruptions⁴ and it is possible that the rash of papular acrodermatitis of childhood or papular vesicular acrolocated syndrome is a cutaneous reaction to infection with different viruses. On the other hand, the rash may be unrelated to hepatitis B or any other viral infection. We consider that one cannot differentiate dermatologically between papular acrodermatitis of childhood and papular vesicular acrolocated syndrome and would suggest that the title Gianotti-Crosti syndrome be retained for both until the aetiology is known.—We are, etc.,

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- 1 Gianotti, F., *British Medical Journal*, 1974, 3, 169.
- 2 Gianotti, F., *Archives of Disease in Childhood*, 1973, 48, 794.
- 3 Cayzer, I., et al., *Lancet*, 1974, 1, 947.
- 4 Wenner, H. A., *Progress in Medical Virology*, 1973, 16, 269.

Radiofibrinogen Scans for Deep Vein Thrombosis

SIR,—Mr. W. W. Barrie and others (19 October, p. 130) make the interesting comment that 25% of patients with venous thrombosis have leg swelling and positive isotope scans and yet have no venographic abnormality. The answer can only be that there is stasis in the lymphatics. This will account for both the swelling and the presence of excess radiofibrinogen, which does normally exchange quite rapidly with the lymphatic system. Hence their comment on the need for investigation of increased vascular permeability has special significance.—I am, etc.,

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Spontaneous Primary Hypothyroidism with Exophthalmos

SIR,—I was interested to read Dr. J. D. Wiener's letter (12 October, p. 108) reporting a patient with ophthalmic Graves's disease preceding the spontaneous development of hypothyroidism. This sequence of events is unlikely to be as rare as the sparsity of case reports suggests. This reply is prompted by the recollection of a similar case in which there were some additional features of interest.

A 46-year-old woman was referred to hospital in March 1966 with ophthalmic Graves's disease. Protrusion of the left eye had been noticed for six or more years. A small goitre was present but she

was clinically euthyroid; she had moderate bilateral exophthalmos (L>R) with lid lag, lid retraction, and periorbital oedema but no ophthalmoplegia. Tests confirmed that she was euthyroid: P.B.I. 5.3 µg/100 ml; ¹³¹I uptake at 4 h 15%, 48 h 31%; PB¹³¹I 0.3/l. She was seen in the thyroid clinic at frequent intervals but no change observed. In September 1966 her husband was killed in an accident on holiday, but no clinical or laboratory signs of hyperthyroidism ensued. In November 1973 she was seen again for a "check-up." Her only symptoms were those of anxiety, occasional diarrhoea, and dislike of cold weather. Again she appeared clinically to be euthyroid, with a small goitre and bilateral ophthalmopathy. Investigations showed her to be hypothyroid: P.B.I. 3.0 µg/100 ml; T₄ resin uptake 124%; high basal T.S.H. with a marked response to T.R.H.—zero 35 mU/l., 20 min >64 mU/l., 60 min 53 mU/l. (normal <5 mU/l.); immunofluorescence test for microsomal antibodies positive, tanned red cell titre 1:250. Assay for L.A.T.S. was negative but assay for L.A.T.S.-protector gave an unequivocal positive response.

As Dr. Wiener points out, it is well known that ophthalmopathy may precede the onset of hyperthyroidism, but the development of hypothyroidism with no period of hyperthyroidism is harder to explain. This patient appears to have features of both Hashimoto's thyroiditis and Graves's disease and illustrates the close connexion between the two conditions, which has been suggested by studies of the autoimmune and genetic mechanisms. Of particular interest is the finding of L.A.T.S.-protector in this patient, which seems inconsistent with the view that L.A.T.S.-protector is a human-specific thyroid stimulator. However, it would be unwise to draw conclusions from a single case and it may imply that cytotoxic immune mechanisms have in this case limited the capacity of the thyroid to respond to thyroid stimulators. Of incidental interest is the failure of this patient to develop hyperthyroidism after acute severe emotional stress.—I am, etc.,

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Ethics and Halothane

SIR,—Dr. J. M. K. Spalding (9 November, p. 345) is wrong again on two counts. First, my letter (12 October, p. 101) was not concerned with beliefs but with facts which were clearly stated and should not need reiteration. The second count is more important. Dr. Spalding's claim that in a clinical trial "patients receiving repeated halothane anaesthetics would . . . be exposed for the purposes of experiment to something which has been suspected of causing illness and even death and the other group to something which has not been so suspected" is nonsense. It ignores the fact that every anaesthetic technique carries with it some risk of illness and even death, as witnessed by the medicolegal report on a death during dentistry published in the same issue of *B.M.J.* (p. 352). Indeed, if Dr. Spalding's "responsible and informed doctors" were to turn their attention to this much more serious problem they would earn the gratitude of patients and anaesthetists alike.

Thus Dr. Spalding's defence of the behaviour of certain ethical committees is based on a false premise and your leading article (7 September, p. 589) was fully justified in pointing out that such ethical committees should not themselves prejudge an issue so obviously unclear in an endeavour to protect patients from one speculative danger