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.
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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 tests[3] (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 eruptions[4] 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; ^{131}I uptake at 4 h 15%, 48 h 31%; PB^{131}I 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; T_3 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