if ^{131}I therapy is used in these cases the thyroid gland will be permanently damaged and hypothyroidism will inevitably result. The problem then lies in identifying this group of patients beforehand rather than trying to reduce the dose of ^{131}I . Hence, although a correlation between high thyroid antibody titres and post-radiation hypothyroidism has yet to be shown, it would seem wise not to treat these cases with ^{131}I , since they are likely to "burn up" their own thyroid tissue. Further clues may be provided by an examination of serum levels of long-acting thyroid stimulator.

On the other hand, radiobiological studies might suggest a different explanation. Following radiation mitosis may be delayed. Furthermore, latent cell damage and death may not be seen until long after irradiation, when the cell has subsequently entered one or more mitotic divisions. As time progresses the feedback stimulus of the failing thyroid gland induces the pituitary to release more thyroid-stimulating hormone. Under this stimulus further thyroid cells enter division and die. This process could become a vicious circle, with increasing hypothyroidism causing increased cell destruction. This would correlate with the clinical findings of some workers who report² that the development of post-radiation hypothyroidism is a continuing process, progressing at a rate of about 3% per annum. Again, it has been shown³ that for rats, at any rate, a dose of radiation which results in a reduction of thyroid function inevitably interferes with the reproductive integrity of the gland.

Although this may be an insoluble problem, it has been pointed out⁴ that the irregular distribution of iodine in the thyroid gland may mean that some cells receive sublethal damage and are capable of recovery.—I am, etc.,

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Midgut Malrotation in Adults

SIR,—The article on midgut malrotation in adults (30 March, p. 803) clearly brings out the fact that such a gross abnormality can be missed on "routine" barium-meal examination. This examination normally includes evaluation of the oesophagus, stomach, and a variable portion of the duodenum, depending on the radiologist and/or the standard procedure in the particular department.

Because of the high incidence of ascariasis in the local population seen in Ibadan, Nigeria, one and a half years ago, we began including a delayed abdominal film on all barium-meal examinations (about 30 per week). This film is taken 30–60 minutes after the completion of screening, depending on the rate of gastric emptying observed during screening. Since beginning this routine we have picked up five cases of un-

suspected intestinal malrotation in adults, aged 16 to 69, as well as other small-bowel lesions. Three of these patients had symptoms in keeping with malrotation, none had an ulcer or other pathology demonstrated at the examination. One had had recurrent symptoms since childhood and was relieved of these following surgery for incomplete duodenal obstruction due to malrotation with bands.

After this experience, and considering the fact that these patients may be misdiagnosed for many years and sent from doctor to doctor, it seems reasonable to suggest that, when a barium meal is done for non-specific or vague abdominal symptoms of long duration and no cause is demonstrated after a routine study, a delayed film should be taken so that malrotation, if present, or other gross small-bowel lesions will not be missed.—I am, etc.,

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Herpes Simplex Cervicitis

SIR,—Dr. R. R. Willcox (9 March, p. 610) is to be congratulated for his perception in detecting six cases of what appeared to be herpes simplex cervicitis.

In recent years a body of literature has developed in North America on the subject of herpes simplex cervicitis.^{1–3} This literature stresses that apart from any clinical signs or symptoms the most important finding, the one which enables the condition to be properly diagnosed, is the presence of "virocytes" in the cervical smears. These altered epithelial cells are often gigantic, and contain large multiloculated nuclei in which there may be inclusion bodies. Their appearance is striking (see Fig.).

Cervical smear. Herpes simplex virocyte. (Papanicolaou stain. $\times 600$.)

I was surprised, therefore, to read that in all of Dr. Willcox's cases the cytology of the cervical smears did not provide any assistance in making the diagnosis of viral cervicitis. I suggest the smears did contain virocytes, unobtrusive ones perhaps, but that they were not recognized for what they were.—I am, etc.,

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Cardiac Failure with Propranolol

SIR,—I would like to comment briefly on the observations of Dr. N. Conway and his colleagues (27 April, p. 213) on the production of cardiac failure in patients with chronic rheumatic heart disease receiving propranolol. It may be of some significance that Cases 1 and 4 (and possibly Case 3) suffered from aortic regurgitation, and Case 2 from mitral regurgitation. Such patients tend to require an increase in heart rate to maintain their cardiac output, and in my experience it is these patients who do badly with beta-receptor blockade, particularly if they have not been adequately digitalized. For this reason beta-receptor blockade should be used with extreme caution in patients with aortic and mitral regurgitation. As Dr. Conway and his colleagues point out, the slow heart rate produced by propranolol may well mask the need for an increase in digitalis therapy.—I am, etc.,

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Measles and Panencephalitis

SIR,—Readers of your interesting leading article (27 April, p. 189) on measles virus and subacute sclerosing panencephalitis may imagine, from the way in which you refer to the work on immunofluorescence, that American contributors to the conference in Bethesda did not appreciate the small but important contribution by British members of our profession. Fortunately this was not so. Dr. J. M. Adams, who has believed for many years that measles virus is the principal aetiological factor in related conditions,¹ came here to express his pleasure and enthusiasm at seeing his beliefs so strongly supported by the immunofluorescent and other studies published by Dr. J. H. Connolly and his colleagues.² In addition, Dr. Connolly was invited to attend the conference, entirely at the expense of his American hosts, in order to present further observations.³

The justification for this interest and generosity appears in Table IV in Dr. Lennette's paper.⁴ Three of the six patients were from this side of the water.—I am, etc.,

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Retinal Changes and Chloroquine

SIR,—Following the observation that retinal changes can occur as a side-effect of chloroquine treatment¹ many animal investigations have been done to elucidate the changes involved.^{2,3} The fundal changes have been described as resulting from centripetal migration of uveal pigment and degeneration of the rods and cones. Changes in ganglion cells have been observed,⁴ but the exact nature of these changes has remained obscure.

In this laboratory ultrastructural examination of the retinae from albino and hooded rats treated chronically with chloroquine has