sensitive to 5-FC (M.I.C. 0·05, M.C.C. 1 μg/ml). After 20 weeks' treatment, during which high serum and urine drug levels were maintained, *C. albicans* was isolated from the urine, apparently identical to the first isolate excepting that the M.I.C. and M.C.C. of 5-FC were now over 500 μg/ml. The first and last isolates were both reasonably sensitive to clotrimazole, which the child is now receiving.

As mentioned by Dr. Cartwright, we have been unable to induce the in vitro emergence of clotrimazole-resistant *Candida* or *Aspergillus* spp., nor in any of the cases investigated by us have we seen such emergence during prolonged clotrimazole therapy. Our repeated experience has been that the microbiologically active levels of clotrimazole in urine and serum rarely reach the required inhibitory concentration of this drug and almost never attain cidal concentrations, and we have therefore stated[2] our belief that prolonged treatment, often for several months, may be necessary to create a subinhibitory situation which discourages the fungus rather than decisively eliminating it. We are impressed by the rapid elimination of *Candida* after five days' clotrimazole therapy in Dr. Cartwright's case.

It would be unwise to discount either 5-FC or clotrimazole solely because of occasional anomalies and failures; many of our cases have responded very favourably to these drugs without toxic side effects, none of which we have seen in children, and we regard these systemic drugs as the safest therapy now available for the treatment of severe mycoses.—We are, etc.,

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1 Holt, R. J., *Proceedings 10th International Congress of Microbiology* (Mexico City), 1970.
2 Holt, R. J., Abstracts, *7th International Congress of Chemotherapy*, Prague, 1971.
3 Szybalski, W., *Science*, 1952, **116**, 46.

Preclinical Detection of Dystrophia Myotonica

SIR,—Your leading article (15 April, p. 124) on "Preclinical Detection of Dystrophia Myotonica" prompts me to refer to a chromosomal abnormality we have reported in the past.

In a study of 17 families with dystrophia myotonica[1] we found a high incidence of miscarriages and a wide range of congenital physical and mental defects such as cleft palate, hare lip, spastic paraplegia, congenital heart disease, congenital cataract, and mental defects. In view of this we made chromosomal studies of leucocytes of peripheral blood in members of the so-called dystrophic generation with the fully developed disorder and members of "the third generation"—that is, children of parents with fully developed dystrophia myotonica. The study was of seven patients, three with congenital physical defects. All showed a modal number of 46 chromosomes. In five of the cases a small number of cells also had an additional small acrocentric chromosome, similar to chromosomes 21, 22, and the Y chromosome.[2 3] The frequency of cells showing the additional chromosome ranged in different cases from no cells out of 52 to a maximum of seven cells out of 65. Although the additional chromosome was present at a low frequency in just five cases we suggest that they might be supernumerary chromosomes related to the disorder. The frequency was about five times greater in cells from the defective children born of mothers with the fully developed disease.

The low frequency in the peripheral blood leucocytes could be explained by somatic elimination, which results from the frequent nondisjunction of supernumerary chromosomes in somatic mitosis, and a more accurate indication of their presence might be anticipated in gonadal tissue.

It is possible the increased physical defects in members of the "third generation" is due to increased frequency of an additional chromosome.—I am, etc.,

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1 Caughey, J. E., and Barclay, J., *Australasian Annals of Medicine*, 1954, **3**, 165.
2 Fitzgerald, P. H., and Caughey, J. E., *New Zealand Medical Journal*, 1962, **61**, 41.
3 Caughey, J. E., *Symposium über progressiva Muskeldystrophie*, ed. E. Juhn, p. 280. Springer Verlag, 1966.

Payment by Colour

SIR,—I read Dr. A. B. Kazi's letter (27 May, p. 532) on the subject of pay by colour with great interest. While I regret the circumstances under which Dr. Kazi left South Africa and share his distaste for any system which discriminates purely on the grounds of colour, creed, or tribe, certain points raised by him should be answered.

As an informed, politically aware South African, Dr. Kazi must know that his figure of 11 or 12 black graduates per year is inaccurate, although the numbers are nonetheless pitifully small. In the past five years this school alone has produced 186 doctors, of whom 54 graduated in 1971.

The relative shortage of medical practitioners in African areas is not entirely the fault of the authorities. Most doctors prefer to practise, as Dr. Kazi did, in urban areas, and this is by no means unique to South Africa. The problem will eventually be solved by introducing a national health service for all races. Until then, anyone who wishes to give practical expression to his concern for this country's peoples may do so by working in African rural areas, hospitals, or medical schools.—I am, etc.,

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Treatment of Trigeminal Neuralgia

SIR,—While one realizes that this article (3 June, p. 583) is specifically referring to the action of "To-day's Drugs" little has been done recently to explore the possible causes and more permanent treatment of this cruel condition.

It has often been proposed that it can arise following dental extractions because the tooth nerve is not necessarily separated at the apex of the tooth. Remnant nerve tissue in a socket is not only a good suspect for a later causative neuroma, but is not radio-opaque. One usually searches for residual roots as possible sources, and cures have followed their removal—but it is not the root, it is the nerve within the root or left behind after removal of the root, which would be a direct cause.

Why has this not been tested out, when so many heroic post-ganglion resections have been resorted to at a later stage? Perhaps because in its earlier stages this condition lies in a no-man's land between dentistry and medicine, and finally finds its way to a specialist outside the dental area.

But it is quite simple to check (preferably early) whether any individual case can be caused as above. The posterior superior branch of the maxillary nerve enters the maxilla approximately 1 in (2·5 cm) above the posterior cervical margin of the last molar (and if it has gone it does not need a computer to find). It is a simple matter to block off this nerve with a local anaesthetic, then stimulate the trigger points, and see if they are still active. If not, then the cause is in the maxillary section, and thinking of more drastic action behind the ganglion seems akin to switching off the whole mains supply, instead of checking first the local fuses, especially as no harm can be done—and no drugs are required.[1]—I am, etc.,

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1 Drummond-Jackson, S. L., *British Journal of Clinical Practice*, 1959, **13**, 12, 867.

Feto-maternal Transfusion

SIR,—It is surprising to see no mention of Botha's paper[1] in Dr. O. A. Ladipo's excellent work (18 March, p. 721). Although the purpose of Botha's series was to reduce postpartum haemorrhage and shorten the third stage of labour, if more people had taken it up at the time who knows how many Rh-sensitized babies may have been prevented? Botha's work was awarded a special merit prize by the South African Society of Obstetricians and Gynaecologists.—I am, etc.,

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1 Botha, M. C., *South African Journal of Obstetrics and Gynaecology*, 1968, **6**, 30.

Hyperthyroidism

SIR,—The article by Dr. Pat Kendall-Taylor on hyperthyroidism (6 May, p. 337), while a reasonable review of the topic in general, made one very serious error. She refers to the indications for antithyroid drugs and cites as one "patients with severe exophthalmos, in whom it is desirable to produce a euthyroid state as soon as possible." Exophthalmos is not itself an indication for therapy, and severe exophthalmos may be made worse by vigorous antithyroid treatment. It is difficult to see why such a statement was made, especially since the reference cited draws exactly the opposite conclusion.—I am, etc.,

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1 Aranow, H., and Day, R. M., *Journal of Clinical Endocrinology and Metabolism*, 1965, **25**, 1.