water shrimps, and this certainly seems to have played a role in the 1961 outbreak of cholera in the Philippines.[5] Travellers to countries where raw fish and other fish products are commonly consumed should be made well aware of the potential risks.—We are, etc.,

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Publicity for New Drugs

SIR,—During recent years there has been an important change in the methods used by pharmaceutical firms to bring their products to the attention of hospital physicians. Approaches by persuasive representatives armed with free samples have become less frequent, and their place is being taken by exhibitions, film shows, and scientific symposia, designed to secure maximum publicity for some particular drug. During the past few months there has been a disturbing new development in this method of sales promotion. The proceedings of a scientific symposium on the effects of a new drug are widely circulated to the medical profession by the manufacturers who sponsor it, and the publication is illustrated by photographs of the contributors, side by side with strident advertisements for the drug itself. This must be extremely embarrassing for the contributors to the symposium, and one wonders whether their consent has been obtained for the reproduction of their photographs in this context.

Scientific symposia promoted by pharmaceutical companies have made many valuable contributions to medical knowledge, and it would be unfortunate if they were brought into disrepute by the irresponsible actions of a small minority of unscrupulous firms.—I am, etc.,

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Amoebic Colitis Exacerbated by Steroids

SIR,—The observation by Drs. S. R. Kanani and R. Knight (7 June, p. 613) that corticosteroids may precipitate an exacerbation of amoebic colitis is supported by observations in this hospital in Addis Ababa.

Among our Ethiopian patients with leprosy there are many with acute neuritis, those with paucibacillary leprosy being treated with corticosteroids. It is my experience that, when patients who are being treated with corticosteroids complain of abdominal pain, frequently trophozoites of *Entamoeba histolytica* are found in the stool, which were not found on admission to hospital. These exacerbations of amoebic colitis respond readily to standard anti-amoebic therapy, and are not an indication for cessation of steroid therapy.—I am, etc.,

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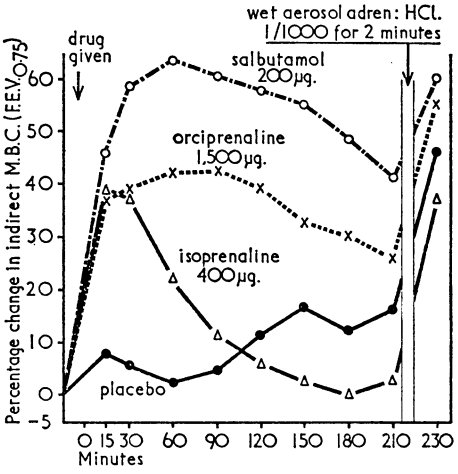
Beta-Adrenergic Stimulants in Asthma

SIR,—I was interested to read the report from Edinburgh by Dr. Y. F. J. Choo-Kang, and others (3 May, p. 287), comparing the effect of isoprenaline, orciprenaline, and salbutamol given by inhalation to patients with asthma. A comparison of similar doses of orciprenaline and salbutamol was carried out concurrently in this laboratory; the results, which were published in detail elsewhere,[1] confirm the Edinburgh findings except that in our hands the difference between the two drugs appeared to be slightly greater. Our time response studies are summarized in the Figure.



Percentage changes in $F.E.V._{0.75}$ after the inhalation of salbutamol, orciprenaline, isoprenaline, and placebo. The orciprenaline and isoprenaline were delivered by commercially available cartridges.

Orciprenaline, because of its longer action[2] and lower incidence of cardiovascular effects, has hitherto been preferred to isoprenaline in this clinic when an adrenergic aerosol is required. Since the above study suggested that salbutamol might represent yet a further improvement, this compound was investigated in other ways to assess its value in clinical use. I would like briefly to record the findings.

Thirty-seven patients were given pressure-packed salbutamol inhalers (100 µg. per puff) to use as an alternative to orciprenaline sulphate (750 µg. per puff) for routine self-administration. At the end of a month 25 stated that they preferred salbutamol to any adrenergic aerosol they had tried previously, five preferred orciprenaline, and seven had no preference. The distribution of preferences was thus very similar to that reported from Edinburgh.

Patients attending the clinic are routinely given a two-minute inhalation of a wet adrenaline tartrate aerosol (1 in 1,000 solution) from a Collison inhaler as a test for the degree of reversible airway obstruction at their weekly attendance. The forced expiratory volume is measured before, and five minutes after, this inhalation. For one month all patients received two puffs from a pressure-packed salbutamol aerosol (200 µg.) instead of adrenaline. A total of 241 patients were thus treated with salbutamol aerosol on at least four occasions. When the response was compared with that recorded after wet adrenaline during the months preceding and following, it was found that 169 patients responded equally to both agents, 21 responded better to the 200 µg. of salbutamol, and 51 to the two-minute inhalation of adrenaline.

These results indicate that salbutamol is indeed a very effective bronchodilator, since experience suggests that our two-minute inhalation of wet adrenaline aerosol produces something approaching the maximum improvement that can be achieved by adrenergic drugs in the individual patient on any given day.—I am, etc.,

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Type III Hyperlipoproteinaemia

SIR,—I have read Dr. P. Borrie's paper on type III hyperlipoproteinaemia (14 June, p. 665) with interest. While on the basis of the plasma lipid levels, the clinical features, and the response to treatment it is probable that most, if not all, of his patients do have this disorder, it should be emphasized that its certain diagnosis depends on the demonstration of beta-lipoprotein of abnormally low density by a combination of preparative ultracentrifugation and lipoprotein-paper electrophoresis.[1] Failure to do this may lead to type III being confused with other hyperlipoproteinaemias. The difficulties which Dr. Borrie alludes to in assigning cases to the various lipoprotein types may be resolved by strict adherence to the criteria laid down by Fredrickson *et al.*[1]

Dr. Borrie's description of the findings on electrophoresis in polyacrylamide gel might give the impression that the disease is characterized by a "greatly increased, very low density lipoprotein (pre-beta-lipoprotein) band"; when paper electrophoresis is employed[1] the plasma of type III hyperlipoproteinaemia usually exhibits the so-called "broad" beta band, and any pre-beta band due to normal very low density lipoprotein is incidental.

The genetics of the disorder are also worthy of mention. On the basis of their studies on 52 patients from 38 kindred, Levy and Fredrickson[2] suggest that it results from the inheritance of a pair of autosomal recessive genes. The two families described by Dr. Borrie, in whom pairs of siblings were found to be affected, are compatible with this view.—I am, etc.,

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