"Chronic bronchitis" is shorthand for a disease complex which changes as the individual ages. It may be largely asthmatic in childhood and adolescence, quiescent in early adult life apart from exercise-induced asthma and distended lung fields on x-ray, then with a period of both spasm and recurrent infection in middle life, and finally with varying symptoms of chronic respiratory insufficiency. The blue bloater and pink puffer are only different ends of the same spectrum, presumably related to the individual's tolerance of carbon dioxide retention. The amount of clinical or radiological emphysema may be very small, and it would be more rational to label such cases "chronic restrictive lung disease" rather than "chronic obstructive airways disease" when fibrosis is severe.

If medical students could think of bronchitis along such lines, more effort might be devoted to controlling infection in middle life, and less to the futile use of steroids and bronchodilators when the lung has become a bag of scar tissue.

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Antibiotics in respiratory infections

SIR,—There are certain statements in Dr I Gregg's article dealing with antibiotics in respiratory infection (Intake No 50, published by Abbot Laboratories Ltd, insert in *BMJ*, 9 July) which we feel require comment.

(1) "*Almost* (our italics) all strains of *Streptococcus pyogenes* have remained sensitive to penicillin." To our knowledge resistance to penicillin in this species has *never* been reported.

(2) The suggestion that ampicillin is more effective than benzylpenicillin against most strains of *Staphylococcus aureus* implies that ampicillin is active against penicillin-resistant strains of this organism. Resistance to penicillin in *Staph aureus* is dependent upon the production of β -lactamase, which is equally active against ampicillin, the use of which therefore could not possibly be indicated in the treatment of infections due to such strains.

(3) The suggestion that amoxycillin seems to be superior to ampicillin in the treatment of exacerbations of chronic bronchitis because of its ability to penetrate into bronchial secretions has never been put to the test in a controlled clinical trial and must therefore be regarded as conjectural.

(4) The author surely does not think that *Strep pyogenes* and *Staph aureus* are the commonest causes of bacterial pneumonia.

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Stability of measles vaccine

SIR,—Drs M D Coulter and B M Jones (9 July p 120) question the stability of measles vaccine and its distribution through the post. As part of the MRC Measles Vaccine Trial 1964 the stability of the vaccine in field conditions was investigated by this institute. Samples of vaccine used in clinics were returned to us in the post at the end of each

session. Vaccine was received from the 32 areas throughout England and Wales. Of the 45 samples tested, only one vial had a serious loss of potency. Samples "lost in the post" and "left out of the refrigerator" still retained adequate potency to stimulate immunity. However, vaccine does lose potency if not properly handled and we would like to join with Professor J A Dudgeon (2 July p 44) and Dr J K Anand (11 June p 1533) in stressing the importance of complying with the instructions issued by the manufacturer.

Drs Coulter and Jones quote Dr Christine L Miller (11 June p 1532) as saying "10% of vaccinated children have lost immunity 12 years later." I would not make this deduction from any of Dr Miller's statements. The protection rates reported for the trial ranged from 84% to 95% throughout the first five years.¹ If 90% of vaccinated children are still protected after 12 years it would indicate that the initial protection has been well maintained.

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¹ Measles Vaccine Committee of the Medical Research Council, *Practitioner*, 1971, **206**, 458.

ECT on television

SIR,—It seems that once again we have been subjected by the media to a biased piece of viewing, part of the ever-popular establishment knocking game in which professional expertise is denigrated. I refer to the "Panorama" programme of 18 July on electric convulsion therapy (ECT), an eminently safe and very widely used procedure which has saved countless lives (since depression, the treatment of which is its main function, is the chief cause of suicide). Of the countless grateful sufferers all over the world whose illness has been alleviated by ECT what were we shown? One woman with, I suspect, a long-standing neurosis who blamed her poor concentration (a common symptom of such an illness) on one course of ECT some years before—a youth who had suffered from an acute psychotic breakdown after taking LSD and had been effectively treated by ECT but complained that he hadn't signed a consent form.

Sensible people must surely realise that well-trained professionals are not going to continue administering a treatment for many years if it does not work. What would be the point? What a pity the BBC producer could not realise the same—but perhaps that would not have been so newsworthy. One could cite the same argument against appendicectomy—that not everyone does well afterwards and some have the operation unnecessarily. The example of psychiatrists' "foolishness" cited in the programme—that insulin coma therapy had now been discontinued despite its claim that it had been useful over many years—showed the lack of understanding of the subject very clearly. Insulin coma therapy was indeed effective but somewhat hazardous, and was replaced eventually by electroplexy for the very reason that the latter proved to be safer and more effective.

The Society of Clinical Psychiatrists is shortly to publish a report on "Public Relations in Psychiatry." As chairman of the study group which produced this report I have frequently been appalled by the way the mass media are able to present such biased pro-

grammes and are allowed to get away with it. Just who does advise them? We are strongly advocating the setting up of a public relations officer for psychiatry who can give official guidance and rebuttals where necessary to such bodies, but perhaps to hope that such advice would be heeded is vain.

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Diagnosing familial hypercholesterolaemia in childhood

SIR,—Dr D J Betteridge and his colleagues draw attention (9 July, p 127) to their paper¹ on the regulation of 3 hydroxy-3-methylglutaryl coenzyme-A reductase (HMG CoA reductase) activity in type II hyperlipoproteinaemia. We were aware of the article, which reports on measurement of the enzyme activity in fresh leucocytes from first- and second-degree relatives of an index patient with familial hypercholesterolaemia (FH), but we consider that their data failed to support the hypothesis that the diagnosis of FH can be made by measurement of HMG CoA reductase activity in the system they used.

This hypothesis can be tested only by identifying affected individuals by independent criteria. In the family studied there were no obligate heterozygotes, but the index patient and his brother (II₆), having tendinous xanthomata and hypercholesterolaemia, had a very high probability of being affected. In the remaining live members of the family serum cholesterol concentration was the only independent criterion available. The sister (II₄) had a serum cholesterol (8.58 mmol/l (331 mg/100 ml)) elevated more than two standard deviations above the expected mean for her age and therefore had a high probability of being affected. Only one of the offspring (III₁₁) of the probable heterozygotes tested (III_{6,7,8,9,10,11}) had a serum cholesterol above the upper limit quoted by the authors (5.9 mmol/l (228 mg/100 ml)).

In order to test the association of FH with HMG CoA reductase activity and to determine the discriminating power of the test it is essential to examine only the first-degree relatives of known heterozygotes all of whom have the same 1 in 2 chance of carrying the mutant gene. Thus of the individuals likely to be affected three (II₇ and 5 and III₁₁) had incomplete suppression and one (II₄) had normal enzyme regulation. Of the first-degree relatives of heterozygotes likely to be unaffected (II₃, I₁, II_{6,7,8,9,10}) four had normal enzyme regulation (I₁, II₃, III₅ and 6) and three had incomplete suppression (III_{6,9,10}). Only in the index patient and his relatives II₅ and III₁₁ were the cholesterol level and enzyme activity concordant in indicating a heterozygote. There appears to be no more than a chance association between the enzyme activity and cholesterol levels in this family.

Dr Betteridge and his colleagues mention in their letter that they have recently found discordance between HMG CoA enzyme activity and serum cholesterol levels in a man heterozygous for FH and suggest that this could be explained by heterogeneity of the mutant gene causing FH. This explanation cannot be used to explain the discordance in