they ate other things as well? What the experiment does not show was the effect on the teeth, and I feel that the whole pamphlet carefully avoids any reference to the harm done to teeth by pulpy bread and also, as seems likely, by a shortage of fluorine. There would seem little point in experimenting with the addition of fluorine to drinking-water if it can be shown that a better bread, as advocated by Sir James so long ago, could correct the deficiency.—I am, etc.,

Hitchin, Herts. G. C. PETHER.

New Treatment for Chronic Bronchitis

SIR,—A letter signed by Professor J. W. Crofton and his team (*Journal*, December 5, p. 1251), attacking methods of publicity used for the promotion of the sale of a bronchitis remedy, clearly refers to "lomutherapy." This letter has revived my intention, after a delay of some months, to seek space in your columns for personal observations on this treatment.

The theory that finely powdered bronchodilators and enzymes should be effective and helpful in chronic bronchitis seems reasonable, and the papers by Robinson *et al.*[1 2] in October, 1958, suggested strongly that this approach to the problem held considerable promise. When Benger Laboratories announced in March of this year that this form of treatment was available, I determined to try it out in a group of bronchitics who have so far failed to respond, to any extent, to all other forms of treatment. I find that this group of patients acts as a most convenient yardstick by which to judge the efficacy of any new remedy, as any benefit produced in such cases would indicate that the new preparation is a true advance in treatment.

Treatment with "lomupren" and "lomudase" was commenced, and in a few days I had some most unpleasant experiences, as four patients became much worse and two had to be provided with domiciliary oxygen cylinders to tide them over until the ill-effects of the treatment had worn off. Both had had lomupren only. Three with lomupren and two with lomudase had definite toxic effects, mainly consisting of weakness in the legs occurring within an hour or two after having an inhalation. In my experience even 20 mg. of isoprenaline under the tongue has never cause this type of side-effect, while each powder capsule contained only 0.1 mg. of isoprenaline. As the nature of the diluent powder used in the capsule was not stated, I suspected that this might be the cause. Benger Laboratories disclosed that this is dextran powder, while reference to the original papers quoted freely in support of their claims disclosed that Robinson *et al.*[1 2] used lactose powder.

The response of the patients in this small trial was assessed clinically, according to subjective statements, and objectively by means of spirometry. A bronchodilator effect could be demonstrated shortly after using the powder, but some complained that it was irritating and caused them to cough. Marked subjective and objective relief was obtained in one allergic asthmatic, but this effect was lost after about ten days. His statement was proved true by spirometry, and it was then shown that an ordinary 1% isoprenaline spray had as good an effect as the powder had had initially.

These findings prompted investigation of the original papers by Robinson *et al.* on which this preparation is based, and it became evident that there is no evidence that the preparation used by them bore any clear relation to the Benger's product. Robinson *et al.*[1] used a mixture of the enzymes desoxyribonuclease and chymotrypsin, 5 mg. of each diluted with lactose powder. They did not mention how active these enzymes were and only give the quantity by weight. On the other hand, lomudase contains 0.01 "Anson units" of chymotrypsin alone, so that it is not possible to know what the comparative activity of these enzymes may be. One cannot compare the activity of one enzyme standardized for activity against two enzymes of unknown activity used together. Repeated perusal of the article by Robinson *et al.* reveals no evidence that these enzymes were used separately at any time, so that it is not possible to know which one produced the results. The second paper by Robinson *et al.*,[2] referring to the use of inhaled isoprenaline powder, fails to reveal exactly what dose of isoprenaline was used diluted in lactose powder, as they mention "Approximately 0.15 mg. of 1–5 μ, or 0.3–0.5 mg. of a 1–10 μ, isoprenaline powder (passed through a 300-mesh sieve) diluted with lactose." A little later, mention is made of the possibility of tolerance to isoprenaline developing in those patients "who inhaled 4.5 to 15 mg. of isoprenaline daily for up to eight months." Even if each capsule contained 0.5 mg., these people must have been using from 9 to 30 capsules a day—if they had been taking lomupren or lomudase 45 to 150 capsules. Figs. 2 and 4 in this paper refer to the response to the "optimal dose of isoprenaline powder," but it is not mentioned what the dose was or how it was arrived at. The Benger preparation, lomupren, on the other hand, contains 0.1 mg. isoprenaline sulphate *B.P.*, and lomudase the same dose in combination with the chymotrypsin.

The inhalation appliance for the Benger powder (issued by them) bears no similarity to that which was described by Robinson *et al.*, and in use is apt to become blocked. Furthermore, I have had complaints from a local chemist that the capsules tend to become damp in stock.

The use of dextran as a diluent may cause development of allergy towards it, and I can find no trace of any work where dextran powder has been tried out either in man or animals to find out if it has any toxic effect or not. It seems incorrect that *all* the contents of the powder capsules should not be declared on the container, and it seems quite wrong to have thousands of people inhaling dextran when nobody knows what its long-term effects may be. We are all conscious of the effects of inhalation of many other industrial dusts, and I consider that the condition of the advanced bronchitic is bad enough without running the risk of any further deterioration from dextran inhalation. The only explanation for the weakness of the legs which occurred in five patients seems to be the dextran powder, as it and the isoprenaline are the only common factors between the two preparations, and isoprenaline could not have caused this effect in such a small dose.

"Lomutherapy" has been used by me in sixteen patients, and in no instance has it been shown to be more effective than an isoprenaline spray. In five cases there were toxic effects mentioned before, and definite worsening in four. I was conscious that the group of bronchitics might have been too severe to have been helped by any form of therapy, therefore seven cases of medium severity were included. These cases did not have toxic effects, but no advantage over isoprenaline could be demonstrated. One further bronchitic who became worse and had to be admitted to hospital has been encountered recently where lomudase had been given by the general practitioner. During the week he received this treatment his clinical condition steadily worsened and his doctor spontaneously suggested that the lomudase might have been responsible.

In view of my experiences I did not feel it justifiable to use this preparation any more, as I did not think it would be fair to the patients, and I communicated my views to the Benger Laboratories representatives. My analysis of the publications on which "lomutherapy" is based suggests that the good effects reported could have been due to the desoxyribonuclease, as reported by Salomon *et al.*[3] Benger Laboratories appeared to be so convinced that their preparation had already been proved by the work carried out by Robinson *et al.*[1 2] that no samples for clinical trial were available. I feel strongly that it is not correct to try out new preparations

at the expense of the National Health Service, but I was forced to prescribe these appliances before I found out that the preparation was no more useful than an isoprenaline 1% spray.—I am, etc.,

Derby Chest Clinic. H. MORROW BROWN.

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SIR,—The welcome letter and leading article in your issue of December 5 (pp. 1240 and 1251) rightly emphasize the singularly unconvincing evidence for the efficacy of "lomudase" and "lomupren." A recent article[1] based entirely on the subjective effects of these substances is equally lacking in reliability when trying to assess them. The method of sputum viscosity measurements employed by Robinson *et al.*[2] when investigating the effects of inhaling desoxyribonuclease and chymotrypsin are crude when compared with previously described methods.[3 4]

We have as yet no objective support for the assertion that the particles of either lomupren or lomudase reach the lower bronchi or bronchioles, where both spasm and accumulation of thick sputum are maximal. Another drawback of this treatment is the loss of efficiency of the "lomulizer" in emphysematous patients who have gross diminution of inspiratory power, which is by no means overcome by the use of the hand-bulb.

Apart from the question of publicity, with which I am in full agreement with you, Sir, and with Professor J. W. Crofton and his colleagues, we have still much to learn about the reduction of the viscosity of sputum in chronic bronchitis.—I am, etc.,

London, N.W.2. D. S. NACHSHEN.

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SIR,—I feel that a reply to the letter (*Journal*, December 5, p. 1251) by Professor J. W. Crofton and his colleagues is very necessary, especially in view of the discrepancy between his and my results with the "lomu" products. Professor Crofton, on his own admission, has treated several patients for obviously only a few weeks. I, however, have treated 35 patients, 16 for three months and over. I am hoping to publish at the end of this winter the results of a general-practitioner trial on the use of "lomupren" and "lomudase." However, the following is, by necessity, a very brief and incomplete résumé of my experiences with the "lomulizer."

My practice is in an industrial area of London which has a heavy dust-laden atmosphere. All the patients, prior to this treatment, had a long history of chronic bronchitis and/or asthma. In many cases they have been in hospital several times a year, and have been on continuous antispasmodic therapy for many years. An overall impression of the results so far has been one of astonishing success. Of the 35 cases, two have been complete failures, and the rest have shown very noticeable improvement, especially in the following four cases:

Case A.—Male, aged 40. In the last two years has been to work for a total of eight weeks. Has a long history of hospitalization. Since lomu therapy, has been to work continuously for four months at his original job, involving indoor and outdoor work.

Case B.—Male, aged 58. When first seen four months ago was thinking of retiring from his work (dustman) owing to recurrent attacks of chronic bronchitis. Rarely at work for longer than two weeks at a time. Since lomu therapy commenced has been back at work, and lost only one week owing to bronchitis.

Case C.—Male, aged 34. Recurrent attacks of status asthmaticus for years: treatment at the Brompton Hospital and the Central Middlesex Hospital. Had been told that nothing further could be done for him. Seen in status asthmaticus of 16 hours' duration. Over the course of 36 hours was given aminophylline, 54 min. (3.2 ml.) of adrenaline, 5 gr. (0.32 g.) ephedrine, aminophylline suppositories, sedation, etc. After one lomudase cartridge, sputum was liquefied and expectorated, and the asthmatic attack subsided.

Case D.—Female, aged 56. An unsolicited testimonial was afforded by the following paragraph in a letter concerning the examination of one of my patients by a chest physician. "At the commencement of the examination there were scattered rhonchi throughout the lungs, but these responded rapidly to an atomizer she had with her." The atomizer was the lomulizer.

Could it be that Professor Crofton and his colleagues failed either to continue therapy for long enough, or, more important, take sufficient time and trouble to explain the way to use the atomizer in a proper manner? I conclude with the hope that no general practitioner will be dissuaded from trying this new product by the remarkably incomplete investigations of Professor Crofton and his colleagues.—I am, etc.,

London, N.W.10. D. TILLEY.

Antibiotic Therapy in Chronic Bronchitis

SIR,—Two elderly chronic bronchitics are facing the prospect of another English winter. One is a man aged 63, still at work. The other is a woman aged 66, no longer working. In all other respects their cases are identical. Both are on continuous antibiotic cover, which costs the nation approximately £60 for six months' treatment (*Journal*, December 12, p. 1315). The patients derive equal benefit from treatment. In the case of the man, the cost of treatment is offset by the extra number of man-hours he is able to put in at work during the winter months. In the case of the woman, there is no corresponding financial advantage. Who is to tell me that I should withdraw treatment from the woman, and not the man? And when the man retires at 65, should I refuse him treatment the following winter?—I am, etc.,

Yaxley, Peterborough. CYRIL HART.

Recording Speech and Respiration

SIR,—Mr. L. R. C. Haward's statement (*Journal*, November 28, p. 1178) that I had found a throat microphone to be sensitive enough to permit respiratory sounds to be measured during playback rests on a misunderstanding, and I am not surprised that he had difficulties with this method of recording respiration. I used the throat microphone for obtaining a visual voice record while respiration is recorded through the usual microphone on to sound tape. The paper to which Mr. Haward refers[1] distinguishes between voice tracings obtained from the throat microphone which feeds speech *movements* into the polygraph, and "*inspirations* which were clearly audible on good-quality recordings." In connexion with the latter it gives reference to my