The vomiting ceased within 24 or at the most 48 hours, while the diarrhoea, which was of frequent, offensive, watery stools without blood or mucus, lasted two to three days, exceptionally four or more. In a few the onset was severe with marked prostration. One-fifth of the cases had no vomiting. Each case had a minimum of 3 stool cultures, 201 specimens in all being examined. No organism of the enteric, dysentery, or salmonella groups was found except in one case. This patient's illness lasted longer than usual—namely, 7 days. On the first, fourth, and eighteenth days from the onset her stool was negative; on the twenty-fourth day *Salm. typhimurium* was isolated. She was the only patient to have a further attack (not shown in these figures), which occurred two months after

the first, when her stools were again negative. Widal tests from this patient and 6 others on Sept. 17 were negative. The following organisms were also identified, the numbers in brackets referring to cases: Morgan's bacillus 1 (4), Morgan's bacillus 2 (1), paracolon-bacillus type (4), *B. faecalis alkaligenes* (1).

All cases were immediately isolated, discovery frequently depending entirely on the nurses' observation. In all but the infirmary ward strict isolation of individual patients could be enforced. Treatment was on the usual lines, with initial starvation and gradual return to normal of the diet by the fourth or fifth day. Attempts to increase the diet more quickly led to recurrence of the diarrhoea. Some cases were given phthalylsulphathiazole 3 g. daily without marked effect on symptoms or duration. However, sulphaguanidine and phthalylsulphathiazole were tried as prophylactics, as shown in the table and diagram. The results were encouraging.

Ward	Population	Type	No. of Cases	Duration	Prophylaxis	% Affected
1	90	Infirmary; aged and sick; overcrowded	37	July 17–Aug. 5	Phthalylsulphathiazole, 1 g. b.i.d. for 2 days	41
2	88	Refactory; overcrowded	15	July 25–Aug. 8	Phthalylsulphathiazole, 1 g. t.d.s. for 3 days	17
3	98	Overcrowded; patients out to work	10	July 24–Aug. 4	Sulphaguanidine, 2 g. t.d.s. for 2 days	10.2
4	33	Parole; not overcrowded	2	July 24–Aug. 16	None	6

When sulphaguanidine was given to all patients in Ward 3 the incidence of fresh cases stopped abruptly; one case appeared on the second day of prophylaxis, but her symptoms were mild and lasted only 24 hours. In Ward 1 phthalylsulphathiazole could be given to only a proportion of patients owing to low stocks. Two cases occurred on the second day of treatment and one the day following, all in patients who had been given the drug. Probably the dosage was too small. Fresh cases continued to occur in Ward 2 until phthalylsulphathiazole in a somewhat higher dosage was given to all patients.

An interesting point is that in Ward 4, where no measures beyond isolation of the patients affected were taken, only two developed gastro-enteritis. Here there was no overcrowding and the patients were out most of the day. In Wards 1 and 2 there was close all-day contact between patients, and in Ward 3 close contact all night and part of the day.

The course of the outbreak suggests that the initial cases were caused by one or more carriers from outside and that infection

thereafter spread directly from case to case, probably air-borne. Social functions were in abeyance, and so there was no contact between male and female patients. The sulphonamides appear to have a definite prophylactic value, but further trial would be necessary before a positive statement could be made. Similar claims were made for mepacrine⁵ which have not been borne out.¹ Even allowing their value, the incidence was directly related to the degree of overcrowding. The exceptionally high incidence in the infirmary ward may be explained by the difficulty of completely isolating so many, of "spotting" early cases among the totally demented by an already overworked nursing staff, and of controlling the excretions of the faecal incontinents.

The clinical course of the illness suggested a rapid invasion with most of the damage at the outset, which may explain why the sulphonamides were effective in prophylaxis but not in treatment.—I am, etc.,

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"Q" Fever

SIR,—I was more than interested in the article on "Q" fever by Drs. J. E. Caughey and J. A. Dudgeon (Nov. 1, p. 694). Well do I remember reading the article in question on "Primary atypical Pneumonia" which has turned out to be an epidemic of "Q" fever. The lesson to be learned from this is of prime importance in my opinion. To my mind it is a great pity that the term primary atypical pneumonia was ever introduced, because at the best it is vague, confusing, apt to be misleading, and falls readily from the lips of those who are satisfied with a diagnosis which usually depends on so-called typical radiological appearances in the lungs or, in their absence, on so-called typical findings on auscultation of the chest together with the general symptoms of a febrile virus infection, a white count usually below 10,000 per c.mm., and the occasional presence of a rash, splenomegaly, lymphadenopathy, etc. The presence of cold agglutinins in the serum is said to place the diagnosis beyond all shadow of doubt, but greatest stress is usually laid on the radiological findings and the physical signs in the chest.

Physicians who have much experience of febrile diseases, especially prolonged febrile illness in tropical and subtropical countries, realize how often there are present abnormal signs on auscultation of the chest, but these as well as positive radiological findings in the lungs do not necessarily result in a diagnosis of pneumonia of some sort—e.g., primary atypical pneumonia, "pneumonia," or "pneumonitis." Such cases, with or without evidence of radiological findings in the lungs and with a white-blood count of less than 10,000–12,000 per c.mm., are invariably investigated along the lines of a case of P.U.O., which includes blood films and thick drops for malarial parasites, blood cultures, repeated total and differential white-blood counts, appropriate agglutination reactions when facilities are available, and the other relevant investigations. I wonder how many cases of enteric-group fever, other septicaemias, and typhus-group fever, etc., have been diagnosed as primary atypical pneumonia or just "pneumonia."

I never make a diagnosis of primary atypical pneumonia until the last resort, as I believe that such a diagnosis should not be made until all relevant investigations have been carried out and all other relevant causes have been excluded. In this way errors in diagnosis should be lessened. Too often are certain febrile diseases excluded on the degree of the severity of the signs and symptoms or on the absence of some so-called typical sign or symptom. The converse is also true. This attitude is indicative perhaps of book knowledge and is characteristic of self-confidence in the inexperienced. Not infrequently I have seen physicians on clinical grounds alone exclude, with what appeared to be confidence and to their own satisfaction, such diseases as enteric-group fever, typhus-group fever, leishmaniasis, hepatic amoebiasis, and even plague, while later laboratory evidence revealed the true nature of the diagnosis. Surely it is about time that it was universally appreciated that specialist investigations are not just carried out to confirm a clinical diagnosis. Happily that day is at least passing—although not quickly enough—because so often such specialist investigations are necessary to make the diagnosis. Unfortunately some young physicians go to the opposite extreme

and carry out all sorts of specialist investigations before taking an adequate history or making a thorough clinical examination. No doubt the pendulum will settle. The ability to think of likely and not unlikely causes of an illness and their adequate exclusion is what is wanted and not just the ability to repeat verbatim textbook description of disease—a not uncommon failing.

What is pneumonia and what is primary atypical pneumonia? Surely there are different causes of pneumonia, some of which are known—e.g.: (1) Bacterial pneumonia, which may be due to pneumococci, staphylococci, streptococci, *B. tuberculosis*, *B. typhosus*, and *B. pestis*, etc. These are further differentiated by their sensitivity or otherwise to penicillin and/or sulphathiazole, etc. (2) Virus pneumonia due to known and unknown viruses—e.g., influenza pneumonia, psittacosis, so-called primary atypical pneumonia, etc. (3) Malarial pneumonia, pulmonary amoebiasis, pneumonia due to ascariasis and relapsing fever. (4) Pulmonary manifestations of yeasts and fungi. (5) So-called rheumatic pneumonia. (6) Lipoid pneumonia, and no doubt other causes of pulmonary pathology apt to be diagnosed as pneumonia or pneumonitis.

Each infecting agent may produce a typical or atypical picture, so why label one type of infection believed to be due to a virus as primary atypical pneumonia? Why not call it just a virus pneumonia, or, in the present state of our knowledge, why not just describe it as a case of P.U.O.? In cases so labelled exhaustive investigations are invariably carried out. Accordingly I would suggest that febrile illnesses of indeterminate origin should not be given what appears to be a definite diagnosis as "primary atypical pneumonia" would appear to be, otherwise encouragement would be given to the use of the term in cases of febrile illnesses with abnormal signs in the chest the origin of which is not known, vague, or uncertain. Well do I realize that many physicians to-day regard signs in the chest in a febrile illness of doubtful origin merely as a pulmonary manifestation of a generalized disease process and search assiduously for the cause. For them, as well as for those who leap to a diagnosis of "pneumonia" and to the rapid exhibition of penicillin and/or sulphathiazole, the paper of Caughey and Dudgeon should be invaluable.—I am, etc.,

B.A.O.R.

JOHN MACKAY-DICK.

BAL and Malnutrition of the Nervous System

SIR,—Dr. W. Russell Brain in his most valuable contribution (Nov. 15, p. 763) referred to Peters's work on BAL. May I state this had also caught my interest. Among those who gave so much help in West Africa one must mention the name of A. Clark. The writer met him on his arrival there and owes much to his guidance. It marked also a friendship which lasted till his death in New Zealand early this year. At that time I had come to the conclusion that the food cause of nutritional retrobulbar neuritis, with its associated conditions, was due to manioc ("gari").

In the therapeutic field naturally one was wholly dependent on knowledge gained from the biochemist. Neither nicotinic acid nor riboflavin had then been discovered. Clark was a biochemist, and his views greatly impressed me, for he advanced the theory that manioc, like sugar-cane in the West Indies (where H. H. Scott had described an identical syndrome, though of a more fulminating type), contained a cyanic glucoside, and this inhibited sulphur utilization by combining with it to form a thiocyanate and so prevent acute poisoning; but if the intake of protein itself was insufficient, then this condition was precipitated. I must add, however, for a number of reasons affecting one's later inquiries, I do not consider Clark's theory can be directly maintained.

I carried out a number of therapeutic trials with various sulphur derivatives, including intravenous sodium thiosulphate, but I found personally they all failed to influence the condition (see *W. Afr. med. J.*, 1937, 9, 35). But this is not a reason, for it was appreciated one was not really using an assimilable sulphur. Following extensive inquiries in other parts of the world, it had become clear that neither manioc nor sugar-cane could be wholly incriminated, as the syndrome could recur independently of either. Thus the Malayan dietary at Changi (where Landor and Pallister demonstrated this condition in 1935) showed the staple carbohydrate was rice. Again we know now, of course, part of the syndrome at least was due to an ariboflavinosis, though how much the neural lesions are due to that is problematical. So far as a food cause could be identified we had not advanced very materially from the old "Deeks theory" of excess carbohydrate-diminished protein intake, though one feels it is reasonable to add it had become possible to show that only certain carbohydrate sources of a specified type appeared more

directly involved—cf. manioc, sugar-cane, and "English" rice. Also there was no doubt of the protective value of certain protein foods.

In the therapeutic field by 1938 (Moore, *J. trop. Med. Hyg.*, 1939, 42, 109) it had also become possible to show autoclaved "marmite" (excluding vitamin B₁₂) cured the earlier case of nutritional retrobulbar neuritis just as formerly it had been shown to respond to ordinary marmite. Nicotinic acid had no appreciable effect. But I have always felt Clark had touched on part of the truth, and it is possible that, under conditions of dietaries already referred to, sulphur metabolism may be disturbed. I am not qualified to express any views as a biochemist, and one's own investigations have always been centred on clinical, therapeutic, and dietary survey of these cases. It seems also to the writer it is more than coincidental that arsenic can bear some comparative clinical relationship with these deficiency syndromes. Therefore it might repay us to test Clark's work further, and this supposition, by the use of BAL, which has been only recently discovered. It might prove of interest also to try BAL on swayback. Clark was extremely familiar with this animal-deficiency disease in New Zealand and referred to it in his arguments. (Here again he maintained the cause was a cyanic glucoside in the white clover, and he viewed cobalt as protective.)

Clark's work is published mainly in the *Trans. roy. Soc. trop. Med. Hyg.*, 1932, 26, 301; *Certain Aspects of Poisoning by Food Plants in Nigeria*, 1934-6; *J. trop. Med. Hyg.*, 1936, 39, 269; and from 1936 onwards in a series of articles in the *J. trop. Med. Hyg.* Admittedly the suggestion for therapeutic trial with BAL is conjectural, but to the writer it does seem worth while to explore in active cases, so far as is possible, every avenue which may present itself, provided we do not risk delay in effective treatment,—I am, etc.,

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D. FITZGERALD MOORE.

Battle Neurosis Treated with Leucotomy

SIR,—The article by Drs. William Sargent and C. M. Stewart (Nov. 29, p. 866) raises some interesting and perhaps controversial issues. There is no mention or speculation in the report as to the factors underlying the emotional conflict which manifested itself in neurotic symptoms. This does not, of course, mean that the psychodynamic viewpoint was not considered, but may it not be the absence of any discussion on this basis that tends to cause uneasiness among psychiatrists at reports of the more radical physical treatment of the psychoneuroses? This is not to say that there is no justification of the treatment of any particular case by such methods as electric convulsion therapy and leucotomy. Indeed, facilities for the treatment of chronic psychoneurosis are nowadays so limited that any method which produces results has to be considered. Nevertheless, as long as psychoneurosis is conceded to be the result of emotional conflict, are we not entitled to know or at least to inquire what are the pertinent factors in such cases?

In the case reported the pre-morbid personality gives indications of obsessional traits and might be designated an obsessional character. That the patient had a high morale is suggested by his leaving a safe civilian job for a highly combative one for which events proved he was ill-suited and which led to increasing tension and finally breakdown. One may speculate as to the subsequent history of this case had the war ended in November, 1944, at which time a slight wound had given him an honourable exit from battle. The real conflict which led to a persistence of his symptoms after that time may well have arisen from the feeling of failure in the high task which he had set himself. Intense guilt feelings may have been aroused from which the personality appears to have struggled wildly to escape by paranoid, hysterical, hypochondriacal, and depressive reactions.

"He always seemed unable to gain insight into his symptoms" might surely have been interpreted that the personality could not tolerate this insight, and in this lay no doubt the lack of success with abreactive and exploratory methods and the amelioration of the condition by sedation. Whether a more gradual and long-term psychotherapeutic approach such as psycho-analysis would have produced a better result is a matter for speculation. This might at least have succeeded in modifying the superego and enabled the personality of the whole to suffer less from feelings of guilt.

Might one not speculate as to whether prefrontal leucotomy does much the same thing as this but at some cost, not properly known at the present time, to the personality as a whole?