Correspondence

Letters to the Editor should not exceed 500 words.

Prevention of Tetanus

SIR,—I read with great interest the excellent contribution by Dr. A. O. Lucas and Dr. A. J. P. Willis on the prevention of tetanus (4 December, p. 1333). The circumstances in which wounds are sustained and the delay in getting patients to hospital for treatment are in many ways comparable with those that existed in this country in the early years of this century. Lucas and Willis have proved conclusively that tetanus antitoxin (A.T.S.) is of great value in the treatment of incipient tetanus where wounds are likely to have been heavily contaminated and have not received immediate surgical toilet in a population not yet sensitized to horse serum. They are careful to point out that what applies in Ibadan does not necessarily apply in Western Europe and North America.

A comparison of the incidence and mortality of tetanus in the two regions bears this out. Between 1959 and 1961 the incidence rate of tetanus in casualty-treated patients in Great Britain was approximately 0.1 per 10,000, and the mortality 0.024 per 10,000,¹ compared with an incidence rate of 2.3 per 10,000 and a mortality of approximately 0.7 per 10,000 in Ibadan. The mortality of A.T.S. in Great Britain, where over 30% of the population have received at least one dose of A.T.S., is in accordance with Laurent and Parish's² estimate of 0.05 to 0.2 per 10,000. Thus, while Lucas and Willis are correct in stating that, in Ibadan, "tetanus poses a greater risk than serum disease," in Great Britain serum disease is as serious a risk if not a greater one than tetanus. It is for this reason that I and my colleagues³ have advised again the indiscriminate use of A.T.S.

In discussions that have ensued since the publication of our paper the issue has incorrectly come to be represented as lying between the efficacy of A.T.S. or chemotherapy in the prophylaxis of tetanus. The true issue is between the efficacy of A.T.S. given on the first day of wounding and correct wound management and chemotherapy together given on the day of wounding. In 16 of the 20 tetanus cases in the Ibadan series treatment was inadequate by either regimen. When serum prophylaxis was the policy six of the seven patients who developed tetanus had not been given serum. When antibiotic prophylaxis was the policy 10 of the 13 patients who developed tetanus had received inadequate or delayed surgical toilet or chemotherapy. It has never been our view that tetanus can be prevented by inadequate surgery and delayed chemotherapy in contaminated wounds more than 24 hours old. Such patients must be assumed to have tetanus already, and they need surgery, often incision rather than excision, antibiotics, and human or equine antiserum.

Between November 1959 and March 1961, when the policy was to use antibiotic prophylaxis

in Ibadan, only three patients who later developed tetanus had been treated by surgery that the authors thought adequate and with penicillin on the day of wounding, and one of them died. When serum prophylaxis was the policy four patients were seen on the day of wounding and had the opportunity to receive A.T.S., but only one received it and all four died. The results as regards mortality in those treated by early surgery and chemotherapy compared with their "controls" treated under the A.T.S. regimen do not appear unfavourable.

Were I in Ibadan I would be anxious for the future when a substantial proportion of the population has become sensitized to A.T.S., the number of serious and fatal reactions increases, and subsequent injections of A.T.S. prove inadequate to treat tetanus in those who have been given A.T.S. on a previous occasion. It is to be hoped that they will succeed in immunizing a fair proportion of those at risk with toxoid before they enter an era when A.T.S. becomes less effective and more dangerous because of its previous indiscriminate application. Perhaps the most important lesson to be learned from the Ibadan series is that whatever programme of management is followed it must be executed properly or it will fail.—I am, etc.,

Sheffield 10.

W. J. W. SHARRARD.

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- 2 Laurent, L. J. M., and Parish, H. J., *Brit. med. J.*, 1952, 1, 1294.
- 3 Cox, C. A., Knowelden, J., and Sharrard, W. J. W., *ibid.*, 1963, 2, 1360.

SIR,—It was extremely interesting to read the paper of Dr. A. O. Lucas and Dr. A. J. P. Willis on the prevention of tetanus (4 December, p. 1333). They conclusively prove that there are "tetanus-prone areas" and that the same principle as recommended by Cox *et al.*¹ cannot universally be applied.

We here are in a fortunate position in not having seen a case of tetanus for the last eight years. For three and a half years we systematically used A.T.S. and had numerous, sometimes serious, complications. Since 1961 we have practically abolished A.T.S., but would not think that our "success" was due to surgical care and antibiotics alone, rather to the fact that the wounds had probably not been infected with tetanus bacilli.

In Britain the "tetanus prone wounds" are properly dealt with, with the result that, according to information from various centres, almost half of the cases of tetanus occurred in patients who either have not had a wound at all or did not notice it or did not bother to see a doctor, or infection occurred through a pre-existing lesion (chafed hands, varicose ulcers, etc.). Accordingly,

if A.T.S. and/or surgical care and antibiotics were 100% efficient they could reduce the incidence of tetanus only to half of the present figure.

If we also want to protect the other half, there is no alternative to active immunization of the population. That would cover all areas, "tetanus prone" or otherwise.—I am, etc.,

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REFERENCE

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Complications of Temporal Arteritis

SIR,—I would like to suggest temporal arteritis as the cause of the acute massive gangrene of the tongue of the 72-year-old woman in the case reported by Dr. Con Reed and Mr. M. J. Inglis (4 September 1965, p. 575), and that gangrene of the tongue, although extremely rare, may now be listed as another complication of this disease. The authors pointed out that in the course of her illness pulsations of the temporal and facial arteries disappeared. They suggested that "... the pains in the temples and face would appear to be manifestations of intermittent claudication of the temporalis and masseter muscles."

This case seems comparable in every way to that of the 82-year-old woman with gangrene of the tongue reported by Brearley and MacDonald¹ in 1961. In their case biopsy of the temporal artery showed changes compatible with temporal arteritis. I also believe that both the 74-year-old woman with gangrene of the tongue in the case reported by Howard and Cremin² and the 80-year-old woman with gangrene of the tongue in the case reported by Bergan³ had temporal arteritis. Unfortunately biopsy of the temporal artery was not performed in either case.

Temporal arteritis is a definite clinicopathologic entity, which I and my co-authors, Magath and Brown,⁴ were first to describe in 1932.

It is a subacute granulomatous stenosing arterial disease, largely confined to the carotid arterial system and occurs exclusively in older persons. Since 1931 at the Mayo Clinic the lumen of the temporal artery has been narrowed, usually severely, in all specimens showing temporal arteritis, but the artery itself has been thrombosed in only 10% of the instances. More than 200 cases of temporal arteritis have now been encountered at the Mayo Clinic, but gangrene of the tongue has not been observed in a single instance.

In dealing with a new disease entity one tends to wait for the development of the full-blown clinical picture before making a diagnosis. After the clinical picture has become well established one begins to work backward and to identify the disease in its less typical forms and its earlier stages. It was the appearance of the typically swollen, nodular, reddened temporal arteries which attracted my attention in the first two cases I observed in the spring of 1931. Had the temporal arteries not been so prominent

and thrombosed, biopsy of the temporal arteries would not have been carried out, and I would have missed the discovery of a new disease entity. In the original description⁴ my co-authors and I stated, "The two cases which form the basis for this report probably represent a new clinical syndrome the aetiology of which is still obscure. It [temporal arteritis] is apparently a focal localization of some systemic diseases. . . . At the present time no name can be given to the condition, but other cases will probably be found and the condition dignified by nomenclature." Our prediction that "other cases will probably be found" and reported proved to be true.

Complications

From the time I observed my first case of temporal arteritis in April 1931 until 1937 it did not occur to me that blindness would eventually prove to be the most serious complication of this disease. On 19 July 1937 I was confronted with a 70-year-old man who had experienced rather sudden loss of vision in both eyes two weeks previously. The basis for his blindness was obscure. In quizzing the patient and his wife I learned that at the time the patient became blind the temporal arteries were enlarged, swollen, and apparently thrombosed. He had been unable to wear his hat for a few days because of the swollen arteries. The acute phase of the temporal arteritis had subsided by the time I saw him. The temporal arteries were no longer enlarged or tender and pulsations were not present. Biopsy of the temporal artery revealed granulomatous giant-cell arteritis. This was the first case of blindness in a patient with temporal arteritis in the history of medicine. The patient remained completely blind, but otherwise enjoyed good health until shortly before he died in his eighty-first year. His was the seventh case of temporal arteritis which I studied at the Mayo Clinic. Some of my patients have lived more than 17 years after recovery from this illness.

The pain of intermittent claudication^{5,6} of the jaw deserves special comment. From 1931 to 1942 each of my 14 patients had complained of great difficulty in chewing, and it finally occurred to me over the next two years that the difficulty in chewing was, in reality, an exercise phenomenon, and that it represented intermittent claudication of the jaw. The pain occurs only during chewing and disappears with rest. It apparently does not occur in any other disease entity, and it is diagnostic of temporal arteritis.

I demonstrated this previously undescribed type of pain in the head and face at the staff meeting of the Mayo Clinic by means of motion pictures on 13 December 1944. In each case in which intermittent claudication of the jaw had been noted, thrombosis of both facial arteries had been observed.

It is my impression that temporal arteritis frequently goes unrecognized, and that there are hundreds of older people in America (perhaps in England too) whose vision could have been preserved and other complications could have been avoided if the medical profession were familiar with this treacherous disease. In 1955, when Dr. Magath and I demonstrated an exhibit⁷ on temporal arteritis for three days at the annual meeting of the American Medical Association, we were greatly surprised to learn that 90% of the physicians who visited the exhibit had never heard of temporal arteritis. It was the first time an exhibit on temporal arteritis had ever been displayed at a meeting of the American Medical Association. The early stages of the illness give no clue as to its nature. Only a high degree of suspicion on the part of the physician will lead to early diagnosis; and if appropriate treatment is instituted promptly

blindness and other complications may be prevented.—I am, etc.,

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- 4 Horton, B. T., Magath, T. B., and Brown, G. E., *Proc. Mayo Clin.*, 1932, 7, 700.
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- 6 — *Headache*, 1962, 2, 29.
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Thymectomy and Myasthenia Gravis

STR.—The letter in your issue of 1 January (p. 47) from Dr. M. C. Gill-Carey is interesting, but like most evidence derived from a single instance leaves us where we were. It is not wise to reason from the particular to the general. The erratic manifestations of myasthenia gravis, particularly in relation to thymomas, is notorious. Nevertheless, extended experience shows that the correct treatment for thymomas is thymectomy including the tumour, preferably preceded by accurate use of radiotherapy. Sometimes radiotherapy is so successful that no operation is needed. Some surgeons do not use radiotherapy, though my own experience demonstrated that operation can be made safer by suppression of myasthenic symptoms and that the end-results are better. Yet the prognosis is always very unfavourable in the presence of a thymoma. Occasionally among my patients removal of a tumour resulted in a complete recovery, only to be followed in a few weeks by relapse and death from irreversible myasthenia, nothing whatever being discovered post-mortem to account for it. The history of Dr. Gill-Carey's patient is somewhat similar to this, though the symptoms had not been seen before operation. Probably they would soon have developed in any event. I have known other patients whose tumours preceded the appearance of symptoms.—I am, etc.,

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Aspirin and Accidental Poisoning

STR.—Professor N. Kessel's appeal for discretion over the sale of large amounts of aspirin (4 December, p. 1336) merits wide support. Although directed towards suicides this advice is equally applicable to accidental poisoning.

Each year in England and Wales about 2,000 children are admitted to hospital with aspirin poisoning. The mortality is low, but the cases do give rise to much anxiety. Treatment is difficult, and the toxic effects sometimes delayed. The drug has practically invariably been taken in the form of junior flavoured aspirin. It is probably too much to hope that the sale of this form of aspirin might be prohibited—most mothers can disguise the bitter taste of the ordinary aspirin. A personal study of 115 cases of poisoning with junior aspirin over the last two years has shown that children (90% in the 2-4 age

group) practically always finish the bottle or packet, which usually contains 50 tablets (62 gr.—4 g.). A logical and practical preventive measure would be to forbid the sale of junior aspirins in packets of more than a dozen tablets. Even if the child swallowed all of these, 15 gr. (1 g.) is very unlikely to give rise to toxic effects.—I am, etc.,

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Occupational Dermatitis

STR.—As a chairman of medical boards for the Ministry of Pensions and National Insurance I used to struggle with Prescribed Disease 42, and I greatly appreciated Dr. F. R. Bettley's article "Diagnosis of Industrial Dermatitis" (4 December, p. 1340).

I agree that it would be better if injury benefit (which includes industrial disease) were paid at the same rate as sickness benefit. But this alone would not enable decisions on diagnosis to be postponed for the period of observation Dr. Bettley desires.

The Industrial Injuries Act was originally drafted to cover cases in which there was a specific "industrial accident," the exact date of which was known (e.g., hand trapped in machine), causing immediate absence from work (the injury benefit period), at the end of which time any disability remaining attracted disablement benefit assessed by a medical board and expressed as "percentage disability" (e.g., amputation of index finger—14%). There might also be entitlement to special hardship allowance if the disability necessitated a change of occupation, or to constant attendance allowance or unemployment supplement.

This system works well in the straightforward case, but it was then stretched to cover conditions where there is a variable constitutional factor (e.g., injury to a previously arthritic joint), and to the prescribed diseases where "date of accident" had to be interpreted as "date of development of the disease." This is usually given as the day the man first ceased work—clinically absurd but administratively necessary in order to determine the injury benefit period. So, because of the associated entitlements a firm diagnosis would still be demanded at an early stage even if sickness and injury benefit rates were identical.

When the man is back at work and receiving disablement benefit for P.D.42 he cannot draw injury benefit again for a second absence from work unless he can establish that he is suffering not from a "recrudescence" but from a "fresh contraction" of the disease. The clinical and administrative complexities now become extraordinary. If he has a "recrudescence" he has to claim "substantial and unforeseen aggravation" in order to have his disablement benefit increased. If he has a "fresh contraction" a situation arises where *on paper* he is apparently suffering simultaneously from two or even three attacks of the same disease.

Medical boards never, in my experience, make decisions on P.D.42 without a dermatologist's report, but if the report is indefinite (the dermatologist not being clairvoyant but