Correspondence

Letters to the Editor should not exceed 500 words.

Prevention of Tetanus

Sr,—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, compared with an incidence rate of 2.3 per 10,000 and a mortality of approximately 0.1 per 10,000 in Ibadan. The morbidity 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’s 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 colleagues 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 the 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 been “ 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 to 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,

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References

Complications of Temporal Arteritis

Sr,—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 MacDonald 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 Cremin and the 80-year-old woman with gangrene of the tongue in the case reported by Bergan 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, were first to describe in 1932.

It is a subacute granulomatous stenosing arterial disease, largely confined to the cranial 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 clinicopathologic 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...