CORRESPONDENCE

ANTIBIOTICS IN TONSILLITIS

Sir,

With reference to Dr M. J. Whitfield's comments (August *Journal*) on my recommendation for antibiotic use in all instances of tonsillitis (May *Journal*), two essential points require emphasis.

Firstly, Dr Whitfield has highlighted the confusion which exists between tonsillitis and sore throat, and I am fully in agreement with his statement that " uncritical prescribing of penicillin for all sore throats will result in poor medical care . . .". The recommendation refers to inflamed throats, as stated in the paper, and non-inflamed throats were excluded from the study. The non-inflamed or " normal " sore throat is a common feature in respiratory illness, occurring frequently in the adult with influenzal illness and fairly frequently in children with pyrexial respiratory illness, and does not require antibiotic treatment *per se*.

Concerning tonsillitis or pharyngitis itself, I acknowledge Dr Whitfield's reference to papers which I had not seen at the time of writing.

I believe, however, that it is wrong to call tonsillitis a disease of largely unknown aetiology. The inflamed throat is a recognisable clinical entity, associated with a β-haemolytic streptococcus in a good third, and with specific viruses in another third (Evans and Dick, 1964). It is the remaining third that represents the unknown element.

Studies done by Kaplan *et al.* (1971) and reviewed by Wannamaker (1972) have shown a convalescent rise in antibody titre in only about half the streptococcal incidence, suggesting that the other half may be a carrier state and not the cause of the illness. The significance of this finding relates to American concern about rheumatic fever prevention, and there is at present no certainty that *suppurative* complications would not occur in those without a rise. Further, it should not be overlooked that their finding concerns Group A streptococci only. Other Lancefield groups are associated with tonsillitis. Even if half the Group A incidence does represent a carrier state, this distinction cannot be made early in the illness, and to those without easy laboratory access it is academic.

I also think it wrong to quote either Evans and Dick (1964) or Gordon *et al.* (1974) as showing that penicillin did not shorten the clinical illness in throat infection. The former study was designed to show aetiology and related clinical features and was in no way, nor intended to be a clinical trial. The latter study included minor respiratory illness, and the absence of response to penicillin is hardly surprising. Merenstein and Rogers (1974) found that both culture positive and culture negative infections responded more quickly to penicillin than placebo, but the studies were done by nurse practitioners, and the authors conclude that some of the negative cultures may have been false.

However, the trial done by Chapple *et al.* (1956), does provide incontrovertible evidence that both streptococcal and non-streptococcal throat infections responded more quickly to penicillin than to placebo. Unfortunately though, their inclusion in the trial of some patients with otitis media renders their data on treatment-failures inconclusive. This study shows that penicillin does shorten recovery time, but why non-streptococcal infections also responded is at present unexplained.

Finally, while tender anterior cervical adeopathy may appear to correlate best with β-haemolytic streptococcal infection and an antibody response (Kaplan *et al*, 1971), it applies to Group A streptococci only, and the authors themselves indicate that this method of clinical differentiation is far from absolute. It is thus valueless in deciding how to treat an individual patient.

The original problem, as I argued in the May *Journal*, still remains. If the streptococcal incidence is to be treated at the time of presentation, one is obliged to treat all cases. The essential point at issue concerns the " if ". Does the β-haemolytic streptococcus justify treatment? I believe it does. Apart from the now very small risk of rheumatic fever, this organism is still a dangerous adversary, capable of causing death from septicaemia and capable of local spread. The point could be proven by further clinical trial, but I believe such a trial would be unethical. A reduced recovery time reflects the avoidance of local complications.

The inflamed throat needs to be seen in proper perspective. Of the whole complex of entities or components which constitute so-called upper respiratory tract infection (most of which do *not* require antibiotics), the inflamed throat is but a small part, and the unnecessary use of antibiotics in some of these cases seems acceptable.

My recommendation still stands.

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