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Epiglottitis in Adults

SIR,—In your leading article "Epiglottitis in Adults" (24 July, p. 204) you state that the causative organism of acute epiglottitis, in adults or children, is usually *Haemophilus influenzae* type B and that ampicillin is the antibiotic most naturally chosen for treatment. You add that "it may be wise to supplement this with cloxacillin in case the condition is *caused* (our italics) by a penicillinase-producing staphylococcus. . . ." This statement raises a point which we believe to be important, but inadequately appreciated, concerning the management of infections in which penicillinase-producing organisms are involved.

While agreeing with your recommendation that it may be wise to give cloxacillin with ampicillin, because of your use of the word "caused" we think our reasons may differ. Can *Staph. aureus* really cause acute epiglottitis? Although there are occasional reports in the literature of the isolation of this organism from the pharynx or larynx of patients with acute epiglottitis, there seems to be none of its isolation from a blood culture in this condition. In contrast, blood cultures positive for *H. influenzae* type B have been commonly reported.[1 2]

There must, therefore, be doubts about the causative association between *Staph. aureus* and acute epiglottitis but a case can be made, nevertheless, for adding cloxacillin to the treatment of any acute infection caused by a penicillin- or ampicilllin-sensitive organism in a site where penicillinase-producers may be present.

The possibility that penicillinase-producing organisms, though not themselves pathogenic in a given situation, might interfere with penicillin therapy directed against the true pathogen was predicted as long ago as 1946 by Fleming,[3] and a preliminary communication from our laboratory reported six examples of such interference, by enterobacteria, with ampicillin therapy of chronic *H. influenzae* bronchial infections.[4] We have subsequently studied this phenomenon in many more patients and the findings will be reported in due course.

Cloxacillin and methicillin are inhibitors of β-lactamase (the commonest type of penicillinase); they bind with it, but, being split only very slowly, release it again equally slowly. This property may be used therapeutically to prevent the destruction, by coincident penicillinase-producers, of penicillin or ampicillin directed against the true pathogen, and examples of patients in which this approach was adopted have been given in the report mentioned above.[4] Thus the addition of cloxacillin to ampicillin therapy of the type of infection mentioned in your leading article may be efficacious not because of the suppression of the (non-pathogenic?) *Staph. aureus*, but because of the inhibition of its penicillinase, which would otherwise destroy the ampicillin needed to suppress an underlying infection with *H. influenzae*. It is important to recognize the existence of such a mechanism, since one can envisage a situation in which penicillinase-producing *Staph. aureus* is isolated from a patient with, say, acute epiglottitis, leading to therapy with cloxacillin alone, which would of course have no effect on *H. influenzae*, if this were the true pathogen, and might have disastrous consequences.

The phenomenon of interference by one bacterial species with chemotherapy directed against infection by another does not seem to have been widely reported, but experience gained in this department of the management of chronic non-tuberculous respiratory infections suggests that it may occur quite commonly when more than one bacterial species is present at the site of infection. In addition to the patients mentioned above, in whom penicillinase-producers interfered with ampicillin therapy, we have seen a chronic bronchitic patient harbouring a strain of *Pseudomonas aeruginosa* which apparently destroyed chloramphenicol directed against an underlying haemophilus infection, and we believe that sometimes tetracycline can be destroyed by enterobacteria in vivo, although we have been unable so far to confirm this in vitro.—We are, etc.,

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1 Turk, D. C., and May, J. R., *Haemophilus influenzae—its Clinical Importance*. London, English Universities Press, 1967.
2 Gorfinkel, H. J., Brown, R., and Kabins, S. A., *Annals of Internal Medicine*, 1969, **70**, 289.
3 Fleming, A., ed., *Penicillin*, p. 88. London, Butterworth, 1946.
4 Maddocks, J. L., and May, J. R., *Lancet*, 1969, **1**, 793.

Bowlby's Disease?

SIR,—The interesting case described by Mr. R. A. W. McDowall (31 July, p. 290) with coexistent idiopathic cervical and mediastinal fibrosis raises once again the enigma of the association of those two rare diseases "Riedel's thyroiditis" and idiopathic mediastinal and retroperitoneal fibrosis. The association has been reported several times.[1-3]

The fibrosis around the thyroid is not usually contiguous with the mediastinal fibrosis, but those interested in this problem may wish to read an old case report by Bowlby.[4] The account includes a necropsy report. A "stony hard," infiltrative mass involved the thyroid and other structures in the neck and was contiguous with similar tissue throughout the mediastinum. Though its wide spread suggested to Bowlby a