CLINICAL NOTE

SOME NOTES ON STREPTOCOCCAL SORE THROAT

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STREPTOCOCCUS PYOGENES is a Gram-positive pyogenic organism which is a common cause of infective illness in man. It has a redeeming feature in that so far it has proved universally sensitive to penicillin.

Clinically the illness it causes may be divided into two groups, septic and non-septic. Upper respiratory tract infections such as otitis media, tonsillitis with or without a toxic skin rash, and pharyngitis are the most common presentations in the septic group, but skin infections, deep tissue infections, and even septicaemia occur. The non-septic illnesses include rheumatic fever and nephritis, both serious.

It is unfortunate that there is no classical syndrome diagnostic of this infection, even a febrile exudative tonsillitis often being due to other organisms or other disease, such as glandular fever. Most often infection presents as an upper respiratory tract illness however and it is with one aspect of this, namely the sore throat, that I wish to deal. Diagnosis of a streptococcal sore throat then, depends on clinical findings together with the taking of a swab and the subsequent bacteriological demonstration of the organism.

The recovery rates of *Str. pyogenes* from sore throats have varied in different series, where different definitions and criteria have been used to label the patients. Brumfitt and Slater in Army personnel, where systemic upset was also present, found 68 per cent of positive swabs out of 131 patients. In general practice in Australia, Gault found 25 per cent positives in 81 cases, while Holmes and Williams (with the criterion of fever) in one of Dr Barnardo's village homes, found 59 per cent positives in 568 cases. To assess the severity of the illness Brumfitt and Slater used seven factors:

1. Duration of sore throat—24 hours or less.
2. Highest temperature 101°F. or more.

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3. Considerable oedema of fauces.
4. Considerable erythema of fauces.
5. Moderate degree or greater of exudate on tonsillar fauces.
6. Moderate degree or greater of enlargement of lymph nodes.
7. Moderate degree or greater of tenderness of lymph nodes.

They found the percentage of positive swabs increased if four or more of these factors were present. There seems little doubt that the more severe the illness, the more likely one is to discover *Str. pyogenes* as the infecting organism and it will be present in 55 to 88 per cent of acute severe sore throats (Brumfitt, O'Grady and Slater).

For three months from 13 September 1962 I routinely took serum-coated throat swabs from all patients consulting me at the surgery who complained primarily of a sore throat. Thus the patients were ambulant and I took no cognizance of subsidiary symptoms or physical signs. Thirty-four out of 2,213 patients attending surgery (an incidence of 1.5 per cent) were swabbed. They comprised 13 males and 21 females ranging in age from 5 to 57. In seven of these the only organism reported was *Str. pyogenes* (one of these not being of the usual Lancefield Group A), while two others were reported as growing upper respiratory tract commensals together with a scanty growth of *Str. pyogenes* by Edinburgh University bacteriology department, using aerobic culture methods. One other swab was reported as growing a culture of coagulase-positive staphylococcus.

Nine out of 34, or a quarter of this series then had bacteriological proof of streptococcus in their throats. Only one of these patients had clinical tonsillitis (which usually presents with systemic manifestations, e.g. headache or vomiting and often necessitates a home visit), and there was little to differentiate one sore throat from another. The maximum duration of the symptom was two weeks and neither the degree of injection nor the presence of tender adenitis gave a reliable indication of the result of the swab. During the series and during the subsequent three months, I encountered no case of rheumatic fever.

Septic complications following an untreated streptococcal sore throat have been put at 10 to 17 per cent. This is most often a recurrence of the original symptoms or an otitis media but further invasion of any tissue is a possibility.

Nephritis usually arises only after type 12 infection and penicillin therapy may not prevent it. On the other hand, rheumatic fever can be prevented by such therapy, certainly if the streptococcus is entirely eradicated. The risk of contacting rheumatic fever following a streptococcal throat appears to vary widely. In an epidemic in a closed community of American airmen the risk was 3 per cent

(Rammelkamp). A leading article in *The Lancet* put the risk at 0.3 per cent among 608 untreated children in this country. In general practice in the absence of an epidemic, this would seem a more likely figure.

Outside hospital practice there have been no reports of a significant depression of denicillin-sensitive staphylococci with a resultant increase in resistant ones. All known *Str. pyogenes* are sensitive to penicillin, which as a result always shortens streptococcal illness and prevents all the complications with the exception of nephritis. Moreover, it is reasonable to assume that penicillin treatment of primary cases will prevent secondary ones from arising, as the disease is quite infectious (Jersild).

Unfortunately accurate diagnosis is impossible at the first consultation and this is the best time to commence treatment in an infectious illness which, whilst often of short duration, may be severe and carries a high risk of complications.

In this age of preventive medicine, I think there is a case for treating all sore throats *ab initio* with penicillin, preferably after taking a swab. If the swab grows *Str. pyogenes* an effort must be made to continue treatment for at least a week.

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