has appeared. The disease runs much the same course whether a cause for it is found or not. The history and clinical examination must concentrate on drugs, infections, and analysis of any other symptoms. The urine should always be examined for protein and red cells. Biopsy is seldom necessary to establish the diagnosis and does nothing to help establish the underlying cause, though immunofluorescence studies may be helpful in the future. Skin testing has not been shown to be of any value, though occasional positive tests with herpes virus and other antigens have been reported. When drugs are suspected to be causing it they are best avoided in the future irrespective of any laboratory investigation. The danger that re-exposure will lead to one of the more severe forms of erythema multiforme does not warrant even giving test doses. Serological evidence of a preceding infection—for example, mycoplasma—may be found in an appreciable number of cases, but by the time the result is available it has usually become somewhat academic.

Treatment is essentially symptomatic. The milder forms of the disease require little apart from reassurance, and local applications cannot be expected to be much more than placebos. The ability of systemic steroids to reduce the mortality of the Stevens-Johnson syndrome has not been proved, but there is no doubt that they can sometimes provide dramatic symptomatic relief, with a starting dose of prednisone 60 mg daily in a severe case. A systemic antibiotic is often combined to prevent secondary bacterial infection of the denuded area. Probably the most important part of treatment is skilled nursing. Recovery can be expected to be complete, with the possible exception of the more severe ocular complications.

Erythema multiforme sometimes recurs, but there is little that can be done to prevent recurrences apart from avoidance of offending drugs. Possibly in those cases that follow herpes simplex infection some success may attend early treatment of the herpes with idoxuridine.

Prophylaxis in Rheumatic Fever

A striking feature of rheumatic fever is the frequency with which it recurs, and the rising incidence of valvular heart disease with each recurrence has been clearly shown. In 1940 M. G. Wilson reported that 85% of her patients had had a recurrence within eight years of the first attack. C. B. Perry noted that 56% of his patients seen before 1939 had one or more recurrences, but after an intensive campaign for prophylaxis in 1955 there had been only one relapse among patients believed to be taking prophylaxis conscientiously. Florid rheumatic fever is now relatively rare and also less severe in the more economically advanced nations. Though this decrease in prevalence and severity began before the antibiotic era and has probably been associated with altered virulence of the streptococcus and improved social conditions, prompt and effective antibiotic therapy in group A streptococcal infections must have further lowered the incidence.

The changing clinical pattern of rheumatic fever, with minimal or no fever, often a non-migratory monarthritis, arthralgia, abdominal pain, or even carditis as the presenting feature, must be appreciated or it may be unrecognized and therefore untreated. A. R. Feinstein and Feinstein and M. Spagnuolo believed that recurrences of rheumatic fever tend to repeat the features of the first attack. They claimed that if the first attack did not affect the heart recurrences were also unlikely to do so, and they suggested that prophylaxis might be reserved for those who develop heart disease. Perry, however, was not so certain, and A. G. Kuttner and F. E. Mayer noted that 25% of cases did not follow a repetitive pattern. Nor does that take into account minimal carditis, which could easily be missed and which could be more severe in a second attack. Though the incidence of streptococcal infections tends to decrease with age the attack rate of rheumatic fever after an infection remains high in the susceptible.

Spagnuolo, B. Pasternack, and A. Taranta recently showed that symptoms of sore throat with fever were significantly associated with a high recurrence rate—up to 25% in children aged under 12 and 14% in adolescents—and particularly so when the interval since the preceding attack of rheumatic fever was short. Those with existing rheumatic heart disease and those who had had a number of previous attacks were also particularly susceptible to recurrences, but there was no significant association between recurrence and the rise in the antistreptolysin O titre. This means that prophylaxis is desirable in all children who have had rheumatic fever, while it is reasonable to instruct adults in a first attack without carditis to seek treatment promptly for any attack of sore throat.

Chemoprophylaxis, when decided on, should be with penicillin (penicillin V 120 mg twice daily) or sulphasalazine (0-5 g twice daily) by mouth, and should be given for a minimum of five years after an attack or indefinitely in cases of severe carditis. A sore throat occurring while on prophylaxis should be promptly treated with bactericidal doses of penicillin for 10 days. Spagnuolo and his colleagues stress the superiority in prophylaxis of a monthly injection of benzathine penicillin. The drawbacks of a monthly, painful injection have limited the use of benzathine penicillin in Britain. Allergic reactions to it are also relatively common. Oral prophylaxis conscientiously adhered to gives very reasonable protection, and, provided the severity and prevalence of rheumatic fever do not increase, there seems no reason to change British practice.

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