Medical treatment of acute episodes of rheumatic fever

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investigators have been unable to understand precisely the pathophysiology of the nonsuppurative sequels of streptococcal infection of the upper respiratory tract, thus making the clinical and the laboratory diagnosis of acute rheumatic fever very difficult. To establish an effective therapeutic regimen, it is essential to have a correct diagnosis, as well as an adequate understanding of the mechanisms of the disease. The difficulties in achieving such a correct diagnosis in rheumatic fever are many, mostly related to differential diagnosis of pure polyarthritis and the presence of recurrent carditis. From the clinical standpoint, the modified Jones criteria² are largely accepted, but are the source of a long list of criticisms. Laboratory tests also are not conclusive. Because of all these difficulties, therapy may frequently be given inadvisably, or insufficiently. My purpose, therefore, is to review the medical treatment of acute

¶ o date, and to the best of our knowledge,

Protocol for diagnosis

515.

At our institution, all patients with a possible episode of acute rheumatic fever are evaluated according to a protocol which includes a complete clinical and laboratory workup.

episodes of rheumatic fever. At the same time, I will

discuss some diagnostic methods, rarely mentioned,

which might permit clarification of the correct diagno-

The blood tests include complete counts of the different cells and measurements of erythrocytic sedimentation rate,

C-reactive protein, serum mucoprotein, serum protein electrophoresis, and streptococcal antibodies. The curve of levels of serum mucoprotein demonstrates the course of the inflammatory status, with a return to normal values demonstrating the suppression of rheumatic inflammation. We pay particular attention to the mucoprotein levels, and also to the cardiac uptake of gallium-67. Return to normal levels of mucoprotein is related to the absence of cardiac uptake of gallium-67.

The use of gallium-67 for diagnostic purposes began with the identification of rejection following heart transplantation.³ This experience was subsequently used to identify active myocarditis.⁴ To my eyes, rheumatic fever is just as great an immunogenic disease as is rejection after heart transplantation or active myocarditis. We are now using the same protocol for use of gallium-67 to detect cardiac involvement in acute rheumatic fever and, thus far, the results are very convincing.³ The diagnosis is completed with the electrocardiogram, chest x-ray and cross-sectional and Doppler echocardiography.

The other manifestations of acute rheumatic fever besides carditis usually disappear without sequels. Cardiac
involvement, in contrast, leaves important sequels in terms
of disease of the cardiac valves and in the myocardium.
Because of this, special attention is required to provide
appropriate treatment. An effective protocol for treatment,
therefore, must be established immediately in all patients
suspected of having cardiac involvement. Rare patients can
develop such severe valvar and myocardial involvement
that heart failure or cardiogenic shock may ensue and
treatment in the intensive care unit will be required.

On the other hand, some diagnostic doubts could remain in patients with borderline manifestations of acute rheumatic fever. In these cases, it is preferable to recommend bed rest until the laboratory and clinical evidence

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