Scarring in rheumatoid arthritis and rheumatic heart disease

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A scar may be defined as imperfect healing or restitution of less than or, sometimes indeed, more than before, usually symbolizing survival. In rheumatic heart disease the initial traumatic lesion of the valve cusp contact areas is first colonized by platelets and fibrin clot; these become gradually more and more fibrotic, partly by surface accretion of fibrin and its replacement by collagen, partly by deeper fibrosis, until we have this big mass of mitral stenosis. A very similar process is seen in lupus erythematosus. How much of this is due to the fibrin deposit associated originally with tissue damage (perhaps due to immune complex activation of complement) and how much due to primary fibroblastic activation associated in rheumatic fever with the original antigenic and presumably streptococcal stimulus we do not know. Current opinion sees Mel Kaplan's cross-reacting streptococcal antibody (Zabriskie *et al.*, 1970) attacking myocardial fibres, but mitral stenosis occurs long after the initial infection and ties up more closely with persistent antibody to group A capsular carbohydrate (Dudding and Ayoub, 1968). Is fibrin laid down faster than it is removed or is there a defect in removal, or is it colonized and collagenized by fibroblasts faster than usual? Or are there defects in its endothelial covering? Is the new collagen type III? Is it more rapidly cross-linked? Do 90 000 contractions per day affect stability? Is it undue tension that stimulates the fibroblast or in keloid? There are many more questions.

Turning to the joints, a major feature of the inflamed joint is the fibrin accumulation—a result of increased vascular permeability and also of continued movement. These fibrin clots may be free or attached. They rapidly become covered by hyperplastic synovium and incorporated into the membrane, thickening it and gradually getting collagenized. Fibrosis also occurs round blood vessels and, as a separate phenomenon, in the tough, fibrous capsule of the joint. This may be an adaptive change to altered joint mechanics and we know very little about it. It seems to be different from the deep fibrinoid nodules seen in rheumatic fever joints, which probably play a part in the development of Jacoud's syndrome deformities (Bywaters, 1975). There are two other types of scarring in rheumatoid arthritis. Firstly, pericarditis is common at all ages, with often some temporary acute collagen deposition subsiding with recovery but rarely constrictive. Pericarditis may occur (Bywaters, 1974) with gross collagenous thickening and shrinkage. This seems to be due to recurrent surface fibrin deposition and its rapid collagenization. Finally, there is the obliterative endarteritis of rheumatoid arthritis (Bywaters, 1957) associated with immune complex deposition in the arterial wall (Conn, *et al.*, 1972).

All these types of fibrosis are harmful and cannot so far be prevented, but we hope that this symposium will help to elucidate them.