Introduction

Fibrosis is essentially part of repair, and in the millennia preceding our own, when no one possessed antibiotics or corticosteroids or cytotoxic agents or immunosuppressive drugs, this process evolved as a non-specific reaction to injury alongside more specific immune reactions. Like anything else, it occasionally gets out of hand, and it is these pathological processes which we have chiefly in mind today. We are all grateful for the scarring of natural healing—even more so with the bomb outrages of today than with the face-slashing of a milder yesteryear. But our understanding of these natural processes is still imperfect; we need to know more about the normal process to understand what happens when it goes wrong. I was led in 1974 to propose this symposium to the Arthritis and Rheumatism Council because of my inability to understand the scarring processes in the heart and in the joints; it seemed obviously a much wider problem involving all areas and systems of the body. We know about its synthetic and its metabolic processes, its destructive potential when stimulated by practolol, and we have ever-increasing varieties of collagen and proteoglycan. Apart, however, from Bill Castor’s work and Prockop’s studies on proline analogues I can find very little about the mechanisms of abnormal fibroblastic activity in disease and how the fibroblast is turned on and turned off, although any Medlar’s search deluges you with references on fibroblast culture.

We used to think in simple terms that if an antigen were still present in the tissues and still antigenic the immunological reaction would lead to walling off of such material as you see, for example, in the liver around echinococcal cysts. Apart from the difficulty of finding and identifying antigens, this seems to me today too naive a view. It seems quite possible that sometimes something—and, in fact, usually somethings—can go fundamentally wrong with the basic repair process—either unmitigated inflammation or imbalanced fibrin accumulation, as in constrictive pericarditis, or undue and unregulated fibroblast repair. This, in my vision, was what this symposium would be about, and we have been fortunate in getting most of Britain’s top experts in the field. Symposia, in their new or their classical form, exist to form bridges or links or connections between ideas or between people in different fields of work, and I hope this one will succeed in doing so.

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