Foreword

Cardiovascular Comorbidities in Inflammatory Rheumatic Diseases

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Drs Karpouzas and Husni have assembled a series of articles that address one of the most important issues facing our management of systemic rheumatic disease patients in the modern era, the prevention and (hopefully) successful identification and avoidance of cardiovascular morbidity and mortality. In the 1960s and 1970s, patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) actually died directly from the heart and vascular complications of their disease. This has been reversed. Today, modern management consists of early institution of aggressive combinations of disease-modifying agents, and the disease complications we saw 50 years ago are relegated to museum findings, and our Fellows in training may never see them, fortunately.

But what do we have in return? Patients die today not from the disease itself but from their experience of cardiovascular complications resulting from immune-mediated vascular responses to the underlying inflammatory nature of RA and SLE as well as from the drugs that are designed to control that inflammation. What is disconcerting and certainly becoming more evident is that drug development (recent example, JAK inhibition) may in fact add to the burden of cardiovascular morbidity and mortality in certain individuals at risk. What this issue is designed to do is to bring into focus the work that is being done to develop risk models and to identify mechanistic studies that can not only provide acceptable low disease activity but also avoid the effects of clinical and subclinical inflammatory burden of disease. We are grateful to George and

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Elaine, who have identified these clinical and research gaps and the attempts to bridge them.

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