Vascular disease: It's not just the pipes

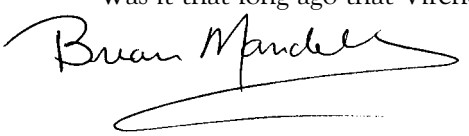
The management of patients with venous and arterial vascular disease keeps getting more complex, despite good studies on the intensity and duration of anticoagulation following deep vein thrombosis and on the therapy of abdominal aortic aneurysms (AAAs).

The pathophysiology does not seem to be getting clearer as more factors are identified. Factors linked to coronary artery disease now include C-reactive protein, rheumatoid arthritis, and systemic lupus erythematosus. Wegener granulomatosis and malignancy are linked with venous thrombosis, and antiphospholipid antibodies are linked with both venous and arterial thrombosis. Genetic factors known to contribute to hypercoagulability include mutations in the factor V, prothrombin, and antithrombin genes. Thus far, many of these factors seem to act together to increase the risk of venous thrombosis. I suspect there will be an equally interesting array of specific genetic factors that contribute to arterial disease, including myocardial infarction. These may be as complex as heterogeneity in cytokine promoter regions (controlling the expression of the acute-phase response and associated sequelae), or as straightforward as unique tissue metalloprotease activity (dictating progression of aneurysms).

But we do not yet know all the key players nor, more importantly, how to distinguish the direct mediators from the bystanders. And we have not yet even scratched the surface of the really tough question of why vascular disease localizes as it does. Peripheral arterial disease seems to be a marker for central arterial disease, but why does it progress to occlusive disease in only some patients, in some anatomic locations? Why don't abdominal and thoracic aneurysms coexist more frequently? How strong is the link between venous thrombosis and atherosclerosis? Why are upper-extremity venous thrombosis and occlusive arterial disease less common than lower-extremity disease?

On page 877 of this issue, Dr. AlMahameed and colleagues discuss AAAs—their epidemiology and progression and new suggestions for screening. On page 907, Dr. Federman et al select several controversies in the management of venous thrombosis and discuss some of the clinical trial data that have both clarified and obfuscated clinical decision-making regarding anticoagulation, ie, "How much for how long?"

Was it that long ago that Virchow's triad made it all seem so straightforward?



BRIAN F. MANDELL, MD, PhD
Editor-in-Chief