ATVB In Focus
Abdominal Aortic Aneurysms: Pathophysiological Mechanisms and Clinical Implications
Robert W. Thompson

With this issue of *Arteriosclerosis, Thrombosis, and Vascular Biology*, we begin a new series of brief reviews on the topic of abdominal aortic aneurysms (AAAs). Although aneurysm disease has often been relegated to somewhat of a secondary place in cardiovascular and atherosclerosis research, aortic aneurysms and dissections are quite common in the general population, and the risks of death from aneurysm rupture remain a significant clinical problem. Over the past decade, basic and translational research on this topic has led to an extraordinary increase in new insights, and aneurysm disease has received increasing attention as an important area of investigative focus. With the advent of recent initiatives to extend the breadth of cardiovascular research to noncardiac areas of vascular disease, better understanding of aortic aneurysms and their treatment have become of special interest to the vascular community. It is therefore our hope that the topic of aortic aneurysms and dissections will be of significant interest to the readership of *ATVB*, not only in its own right but also due to the many common features that exist between aneurysms and other problems in atherosclerosis, thrombosis, and vascular biology.

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The overall theme of this series will be to review the pathophysiological mechanisms that contribute to aneurysmal degeneration, and to critically consider how these mechanisms are related to other complicated forms of atherosclerosis. In addition, these reviews will highlight the potential therapeutic implications that new research findings might have for the management of aneurysm disease. For example, from both mechanistic and clinical perspectives, it is intriguing to consider that many of the pathophysiological processes involved in aneurysmal degeneration appear similar to those thought to mediate the processes of expansive arterial wall remodeling and the vulnerability of atheromatous plaques to rupture. Moreover, the use of new animal models of aneurysm disease and the ease by which AAAs can be detected and followed in patients both suggest the potential for rapid translation of research results toward clinical application. The reviews selected for this series will therefore extend from basic science topics relevant to the etiology and pathophysiologic mechanisms of AAAs, to translational clinical topics addressing potential pharmacologic management and optimal utilization of established surgical therapies.

The brief review by Powell et al is the first in this series of articles highlighting AAAs. Based on strong clinical evidence developed in recent large randomized clinical trials and their unique perspective in conducting these studies, the authors provide a state-of-the-art summary of current surgical management for small asymptomatic AAAs. These important investigations have helped to define the appropriate place of surgical/endovascular treatment versus imaging surveillance in patients with small AAAs, and they have illuminated the areas where broader therapeutic options are needed.

Future reviews in this series will focus on the use and limitations of experimental animal models of aortic aneurysm toward elucidating the pathophysiological and molecular mechanisms of disease, particularly in systems using genetically altered mice. Pathological mechanisms underlying the development and progression of aneurysmal degeneration will also be examined in detail, such as the role of chronic inflammation and local immune responses, oxidative stresses, extracellular matrix-degrading proteinases and their inhibitors, and impaired connective tissue repair in response to injury. Additional reviews will explore how the process of aneurysmal dilatation shares many features with the process of expansive remodeling in atherosclerotic arteries, how biomechanical forces unique to AAAs may have special relevance to disease progression, and how the development of AAAs involves inherited patterns consistent with genetic susceptibility. Finally, the development of novel therapeutic approaches to small asymptomatic AAAs, based on a better understanding of the pathobiology of aneurysmal tissues and pharmacological interventions with potential to suppress aortic degeneration, will also be examined. The subject of these reviews appears particularly timely, given the recent demonstration that not all patients with AAAs require surgical repair, the recognition that there is a pressing need for alternate forms of therapy, and the belief that new management strategies for patients with small asymptomatic AAAs may soon be on the horizon.

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