Editorial

Arteriosclerosis: Progress and Evolution

This issue marks the beginning of the fourth year of publication of the Journal. Since its inception, our understanding of arteriosclerosis has increased, and many of the new developments have been reflected in our published articles. The development of arteriosclerosis and its complications is a complex subject requiring a variety of approaches and a number of different disciplines. It is difficult to perform experiments that adequately encompass the factors identified as contributing to the development and progression of arteriosclerosis and its associated complications. At present, we believe that it is necessary to understand the biology of smooth muscle cells, the function and properties of endothelial cells, the nature and function of the connective tissues of the vessel wall, the impact of lipoproteins in the circulation together with the factors influencing their composition and quality, the process of lesion calcification, the interaction of blood with damaged vessel walls, and the problems of vasospasm, hemodynamic factors and thrombotic occlusions superimposed on arteriosclerosis.

The articles published in the Journal reflect developments in those disciplines contributing toward the study of arteriosclerosis. About 40% of the articles have been concerned with lipids and lipoproteins, and about one-half of these papers involved lipoprotein interaction with cells. Rapid progress has been made in our understanding of the composition of lipoproteins (particularly the apolipoproteins), their genetic control, and their interaction with cells.

It seems likely that, within the decade, an understanding of the molecular basis of the genetic defects in all or most of the familial disorders of lipid metabolism will be possible, presumably enhancing our understanding of how atherogenesis might be accelerated. Approximately 20% of the articles in the Journal have been concerned with epidemiologic and genetic studies. Further sophistication can be introduced in population studies as the nature of the genetic defects that contribute to the development of arteriosclerosis are more specifically defined.

The vessel wall has become increasingly accessible to newer techniques of study. Knowledge of arterial smooth muscle cells and their function has advanced considerably during the past decade, and now we are at the stage where the regulation of smooth muscle cell function at a molecular level can be more critically examined. Endothelial cells are being studied extensively in culture, and a more sophisticated understanding of their function and their interaction with other components of the vessel is evolving. Mechanisms of endothelial cell injury are still poorly defined, but an understanding of this process remains one of the keys to the prevention of arteriosclerosis. Nearly 19% of the articles published have related to the arterial wall, particularly vascular cell biology, and about 9% of the articles dealt with vascular biochemistry.

The clinical complications of arteriosclerosis are caused by mechanisms that include spasm and thromboembolic events. Platelets accumulating at injury sites and in thrombi may contribute to smooth muscle cell migration and proliferation. Blood flow affects the extent of thrombosis and possibly endothelial injury. Interest in this area is reflected by the fact that about 12% of the articles published were concerned with thrombosis and flow-related events.
The study of arteriosclerosis today involves molecular biology, cell biology, immunology, lipid carbohydrate and protein metabolism, rheology, and experimental pathology; pathobiology of cellular elements (such as smooth muscle cells, endothelial cells, platelets, polymorphonuclear leukocytes, and monocytes), of connective tissue constituents (such as proteoglycans, collagen, microfibrils, elastin, fibronectin, von Willebrand factor, and thrombospondin); genetics, epidemiology, nutrition, and clinical investigation. It is worth emphasizing that the purpose of this Journal is to provide a forum for the wide range of subjects that bear on the development of arteriosclerosis and its complications. Although the Journal's gradual and steady growth shows that it is possible to assemble articles from these diverse areas, the Journal will be fully successful only when it has achieved the maximum participation from the different disciplines involved in the study of arteriosclerosis and its clinical complications.

Since one key event in the development of arteriosclerosis is the interaction of blood constituents with injured vessels, a modification in the Journal title has been authorized by the AHA Publications Committee. Thus, to emphasize the relationship of thrombosis to the development of arteriosclerosis and its complications, the title of the Journal, effective with this issue, is *Arteriosclerosis: A Journal of Vascular Biology and Thrombosis*. This change does not imply that arteriosclerotic disease is less important to contributors and readers of the Journal. It is our hope that we will have a large proportion of high quality, scientific work concerned with vessel wall injury and thrombosis in relation to arteriosclerosis published in this Journal. We intend that the Journal will be an important forum for reports of the interdisciplinary studies so essential in unraveling the problems of this disease.

J. Fraser Mustard, Editorial Board
Edwin L. Blerman, Editor

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J F Mustard and E L Bierman

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