

Editorials

ON AUTHORS AND CONTRIBUTORS

IT is the responsibility of every person listed as the author of an article published in the *Journal* to have contributed in a meaningful and identifiable way to the design, performance, analysis, and reporting of the work. The specific requirements for authorship promulgated by the International Committee of Medical Journal Editors (ICMJE), which have been adopted by many biomedical journals, are posted on its Web site at <http://www.icmje.org>. By extension of this reasoning, it is logical that those who meet the criteria for authorship not be excluded as authors. However, our current editorial policy limits to 12 the number of authors whose names can be printed under the title of an article.¹ With advances in medical research, investigators with a broader range of skills than were required in the past are often needed to take new ideas from the bench to the bedside and to conduct large clinical trials. For this reason, we are modifying our policy on the number of authors we will list. Beginning with this issue of the *Journal*, we will no longer place a limit on the number of authors listed under the title.

As in the past, our policy is that all persons listed as authors must meet the ICMJE requirements for authorship. In keeping with the tradition of scientific trust, we do not ask that the specific contributions of individual authors be reported to us. It is instead the duty of the corresponding author to ensure that each of the authors listed meets the necessary criteria; before an article is published, we will require a written statement from the corresponding author to this effect. If we are concerned about the authorship of an article, we will discuss the matter with the corresponding author and, if necessary, request written documentation of authorship. The names of persons who have contributed substantially to a study but who do not fulfill the criteria for authorship will be listed in an appendix.

This change in policy applies to articles that contain original data, including Original Articles and Special Articles. In contrast, for review articles and editorials, in which we are seeking analysis and opinion from acknowledged experts in a field, we will, at our discretion, place limits on the number of authors.

We are indebted to the investigators whose research reports form the core of the *Journal* each week. Our change in policy will allow those who participate meaningfully in a research project to receive the credit they deserve.

JEFFREY M. DRAZEN, M.D.
GREGORY D. CURFMAN, M.D.

REFERENCES

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THE VALUE OF INFLAMMATION FOR PREDICTING UNSTABLE ANGINA

THE realization that atherosclerosis is, morphologically, an inflammatory disease was originally derived from studies of animal models. Early after the initiation of an atherogenic diet in animals, monocytes adhere to the vascular endothelium and accumulate in lesion-prone arterial sites.¹ This adherence of monocytes to the arterial surface is facilitated by the endothelial expression of surface proteins that are collectively known as adhesion molecules.² Adherent monocytes are enticed into the arterial intima and differentiate into macrophages with the help of such modulators of inflammation as monocyte chemoattractant protein 1 and macrophage colony-stimulating factor, which are produced locally by smooth-muscle cells. Resident macrophages then accumulate lipid to become foam cells and perpetuate the local inflammatory response.

In addition to its role in atherogenesis, inflammation is also involved in the later clinical manifestations of atherosclerosis. For example, the levels of circulating markers of inflammation such as C-reactive protein³ and serum amyloid A protein⁴ are higher in patients with unstable coronary disease than in those with stable coronary disease. Moreover, persistent elevation of C-reactive protein in patients with unstable angina is predictive of future myocardial ischemia and infarction.⁵ These clinical observations are consistent with histologic data demonstrating that sites of rupture of atherosclerotic plaque within the coronary arteries are characterized by collections of activated macrophages and smooth-muscle cells — findings that are indicative of an ongoing inflammatory response.⁶ Thus, “active” coronary artery disease is clearly associated with evidence of inflammation both systemically and at the level of the arterial wall. However, these data do not distinguish between inflammation that is a precipitating event in the clinical manifestations of coronary artery disease and inflammation that is a consequence of either recent myocardial ischemia or plaque rupture.

Fortunately, prospective studies have clarified this issue. In the Framingham Study, elevated levels of fibrinogen, an acute-phase reactant, were independently associated with future coronary events.⁷ Subsequently, data from large-scale population-based studies have demonstrated that increased circulating levels of numerous markers of inflammation, such as cytokines