Correspondence. Letters to the Editor must not exceed 400 words in length and must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Authors of letters selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication. Please submit three typewritten, double-spaced copies of the letter to Herbert L. Fred, MD, % the Circulation Editorial Office. Letters will not be returned.

Enhanced Inflammatory Response to Coronary Angioplasty in Patients With Severe Unstable Angina

To the Editor:

We read with interest the recent article by Liuzzo et al concerning the enhanced inflammatory response to PTCA in patients with severe unstable angina. They found that PTCA induced an increase in plasma interleukin-6 (IL-6) levels in patients with severe unstable angina, but IL-6 levels did not change after PTCA in stable patients.

We previously reported that plasma IL-6 levels become elevated in patients with acute myocardial infarction and that IL-6 mRNA is expressed in human atherosclerotic lesions. Therefore, we propose that the main source of plasma IL-6 in coronary artery diseases is the vascular tissue. Recently, we measured the levels of IL-6 and von Willebrand factor (vWF), a marker of endothelial injury, in the coronary sinus blood of patients with stable angina. Blood samples were taken from the coronary sinus immediately before and after and 4 and 24 hours after either PTCA (group A; n=52) or diagnostic coronary angiography (group B; n=7). Levels of IL-6 and vWF in the coronary sinus blood did not change in group B after coronary angiography. Conversely, in group A, the levels of IL-6 and vWF increased significantly 4 hours after the procedure from the baseline value of 3.34±0.56 to 8.20±0.63 pg/mL (P<0.01) and from 113±6.2% to 130±7.3% (P<0.01), respectively, and reached peak values at 24 hours (13.2±1.20 pg/mL and 139±7.4%, both P<0.01 versus baseline). Furthermore, there was a significant positive correlation between IL-6 and vWF levels in the coronary sinus blood at both 4 hours (r=0.38, P<0.01) and 24 hours (r=0.43, P<0.01), suggesting that the mechanism of increased IL-6 after PTCA is related in part to local vascular injury.

Although Liuzzo et al found an increase in plasma IL-6 levels after PTCA only in patients with severe unstable angina, we suspect that if blood samples were drawn from the coronary sinus rather than the peripheral vein, more definite conclusions regarding the involvement of inflammatory cytokines in the pathogenesis of acute coronary syndrome could be obtained.

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Circulation. 1999;100:e96
doi: 10.1161/01.CIR.100.19.e96
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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