Journal of the American College of Cardiology
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Published by Elsevier Inc.

Vol. 50, No. 25, 2007
ISSN 0735-1097/07/$32.00

CORRESPONDENCE

Letters to the Editor

Arterial Blood Pressure Gradient Across Vulnerable Plaque Might Increase Rupture

We read with interest the paper by Doyle and Caplice in the May 29, 2007, issue of the *Journal* (1). They describe the importance of neovascularization in vulnerable plaque. We have a theory as to a mechanical mechanism for rupture of a vulnerable plaque (Fig. 1). With normal blood pressure and normal velocity through an artery (A) and normal lateral blood pressure in the vasa vasorum (B), the pressure in the vasa vasorum is equal to the lateral pressure in the artery (A). However, with a 30% to 40% narrowing due to plaque formation (B), the velocity (C) through the narrowed lumen (D) is increased. According to Bernouli's Theorem, this increased velocity of blood produces a lower lateral blood pressure acting on the plaque (E). The pressure in the neovascularization in the plaque will be near that of the vasa vasorum and systemic pressure, thus a pressure gradient build up is created across the plaque that could rupture the plaque (E). Any increase in systemic pressure or increase in the narrowing of the lumen would further increase the velocity through the narrowed lumen (C). The pressure differential across the plaque would become even greater and be more likely to "blow the top" of the plaque. This plaque rupture mechanism would be another reason for actively trying to prevent transient increases in arterial blood pressure in patients with some coronary artery disease.

Many patients are known to have transient increases in arterial blood pressure caused by physical activity, anxiety, emotional excitement, and stress. A decrease in the arterial lumen can occur from vasospasm or intraplaque hemorrhage. Thus, there might be a variety of mechanisms that increase the velocity through the narrowed lumen leading to an increased pressure gradient across the plaque.

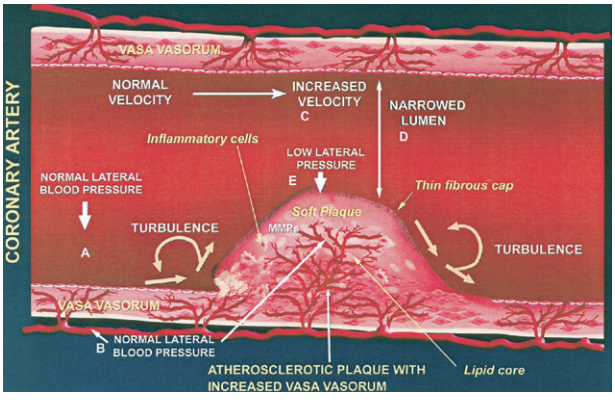

Figure 1 Mechanical Cause for Rupture of Vulnerable Plaque Owing to Increased Pressure Gradient

MMPs = matrix metalloproteinases.

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doi:10.1016/j.jacc.2007.08.049

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Reply

We thank Dr. Folts for his interest in our paper (1). The hypothesis that a pressure gradient between the parent vessel and the plaque microvasculature might contribute to plaque rupture is intriguing. One potential limitation of the hypothesis is the assumption that pressure in the plaque microvasculature will be near that of the vasa vasorum and that the latter is near that of systemic pressure. Under normal circumstances, one would expect that the intraluminal pressure within plaque microvessels would be somewhat lower than that of the systemic circulation, similar to the progressive step-down in pressure observed from systemic arteries to the capillary bed. To what extent this physiologic decrease in intraluminal pressure matches the drop in lateral blood pressure of the parent vessel beyond a tight stenosis is uncertain. We suspect that deficiencies in the integrity of the microvasculature remain the primary predisposing factor to intraplaque hemorrhage, but further studies of Dr. Folk's hypothesis would be of great interest.

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doi:10.1016/j.jacc.2007.09.023

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