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THE AUTHOR REPLIES:

We would like to express our admiration of the remarkable experiment by Gertz et al. (1) 35 years ago and agree with their findings wholeheartedly. Their animal study proved that endothelial damage and thrombus formation occurred just at the proximal site of partial obstruction without reduction in coronary flow based on the electron microscopy findings. They reported that for this reason, vasospasm could cause not only angina pectoris but also acute myocardial infarction (AMI) and furthermore suggested that the possibility that atherogenesis could start at the site of spasm because of endothelial damage.

In our study (2), which was an in vivo study, we presented optical coherence tomography findings in study subjects who had >90% diameter stenosis during spasm and had symptoms of angina accompanied by electrocardiographic changes. Nevertheless, we found thrombus in only 29% of subjects. However, a luminal irregularity suspicious for plaque damage was found in 61% of subjects even when thrombus was not found. It is postulated that the prevalence of thrombus in our study compared with thrombus observed in the animal study by Gertz et al. (1) can be underestimated due to the reason itself that the resolution of optical coherence tomography is much lower than that of electron microscopy. Certainly it is highly possible that the presence of thrombus could be lowered by endogenous thrombolysis as well as pre-treatment with aspirin, clopidogrel, and heparin. Although it may look as if the degree of coronary flow reduction during spasm is neither associated with the prevalence of thrombus nor with AMI clinically, this could be due to the small number of subjects (**Table 1**). We are in the process of finalizing our next study on the comparison of thrombus and plaque erosion at coronary spasm sites and nonspasm sites in patients with suspicious vasospastic angina using optical coherence tomography. In this study, all thrombi were observed in the areas with plaques, and thrombus was also observed at the nonspasm sites. In fact, however, thrombus was found 4 times more frequently at spasm sites than at nonspasm sites. Although endothelial damage and thrombus were found at the proximal stenosis sites due to arterial constriction in the animal study, the cause-and-effect relationship is not clear in the in vivo study. The effects that spasm has on the natural history of plaque are a future research subject of great interest to us.

TABLE 1 Prevalence of Thrombus in Vasospastic Angina

	Thrombus		
	Present (n = 23)	Absent (n = 57)	p Value
DS during spasm, %	91.6 ± 9.7	94.1 ± 9.3	0.290
DS after nitroglycerin, %	25.4 ± 22.5	23.3 ± 21.2	0.694
TIMI flow grade during spasm			0.757
0	8 (34.8)	16 (28.0)	
1	2 (8.7)	3 (5.3)	
2	2 (8.7)	9 (15.8)	
3	11 (47.8)	29 (50.9)	
AMI (n = 11)	5	6	0.487

Values are mean ± SD or n (%).

AMI = acute myocardial infarction; DS = diameter of stenosis; TIMI = Thrombolysis In Myocardial Infarction.

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Please note: Dr. Shin has reported that she has no relationships relevant to the contents of this paper to disclose.

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Characterizing the Hyperemia-Induced Mechanical Stress Acting on the Plaque

I read with great interest the article in *iJACC* by Choi et al. (1), who provided an innovative characterization of the hemodynamic force acting on individual plaques in a series of patients. By applying computational fluid dynamics in coronary computed