Editorial:
Coronary Spasm in Myocardial Infarction: Fact or Fiction?

William Ganz, M.D., C.Sc.

Patients with variant angina often develop a permanent coronary artery occlusion at the site of previous "spastic" closures. In this issue of Circulation, Gertz et al. demonstrate that a 50% focal constriction of a canine coronary or rabbit carotid artery with a suture thread produces extensive endothelial cell damage with thrombus formation. They propose that a similar chain of events may be triggered by coronary artery spasm in variant angina, leading to thrombotic occlusion at the site of spasm and myocardial infarction.

It can be questioned whether and to what extent a 1-hour constriction of a dissected coronary artery with a suture thread simulates the effects of a brief spasm on an intact artery. The mechanism proposed by Gertz et al. may be a plausible explanation for coronary thrombosis in variant angina with normal coronary arteries. Unfortunately, the evidence for such an occurrence is not unequivocal. Many cases of variant angina reported as having angiographically normal coronary arteries turned out on closer scrutiny to have had "minor luminal irregularities" or even stenoses up to 25%. Since coronary arteriography may often considerably underestimate the severity of a stenosis, one cannot rule out the possibility that the alterations of endothelial surface alone played a significant and possibly decisive role in the development of coronary thrombosis.

Most patients with variant angina have a severe, angiographically demonstrable lesion in the affected coronary artery. In the presence of a severe stenosis, drastic reductions in flow can occur with minimal reductions in the stenotic area. Such small changes in stenotic area may be produced by physiologic changes in arterial tone and do not require hypercontraction (spasm), as suggested by Gertz et al. Gensini et al. and Yasue et al. have reported spontaneous changes in coronary artery diameter. It seems unlikely that such minor variations in lumen diameter could produce endothelial changes as seen by Gertz et al.

Small decreases in stenotic area that cause drastic reductions in flow can also occur passively as a result of fall in distending pressure. Logan showed in isolated perfused human coronary arteries that a decrease in perfusion pressure leads to an increased resistance to flow across elastic, eccentric stenoses. Logan's findings were confirmed by Schwartz et al. in a canine model of elastic coronary stenosis. In light of these findings, one could speculate that the preferential occurrence of variant anginal attacks in the early morning may be related to the reduced arterial pressure between midnight and the morning hours. The cessation of attacks of variant angina during intraaortic balloon pumping in patients with a critical coronary stenosis also emphasizes the role of coronary distending pressure and makes it unlikely that coronary hypercontraction was the cause of anginal attacks.

Another possible cause for a passive change in the stenotic area is related to changes in arteriolar resistance distal to an elastic stenosis. Arteriolar dilation, whether the result of augmentation of myocardial oxygen demand or application of vasodilators, causes a fall in distal coronary artery and distending pressures and a decrease in stenotic area with increased resistance to flow. It is conceivable that the ST-segment elevation and temporary coronary artery closure during exercise in some patients with variant angina are manifestations of a passive collapse in an elastic coronary stenosis, secondary to a fall in distending pressure during the exercise-induced coronary arteriolar dilation. The fact that intracoronary injection of contrast medium, a potent arteriolar vasodilator, also produced coronary spasm in such patients further supports the possibility that a passive collapse, and not an active hypercontraction, was the underlying mechanism of occlusion and electrocardiographic changes. This explanation seems particularly plausible in cases in which ST-segment elevation and presumably coronary artery closure occurred after, rather than during, exercise. During exercise the elevated arterial pressure probably counteracted the effect of arteriolar dilation on the distending pressure and hence the stenotic area; after exercise the arterial pressure decreased whereas the coronary arteriolar dilation persisted.

It is important to realize that an elastic lumen is not a rarity: according to a study by Vlodaver and Edwards, in 200 coronary arteries from patients with severe atherosclerosis, 69% of the lumens were eccentric and presumably elastic. In 29% of the eccentric stenoses the lumen was slitlike, a shape readily missed by coronary angiography.

These alternative concepts of spastic phenomena involve small degrees of wall motion that seem unlikely to produce forces that would damage the endothelium.
Transient coronary artery occlusions may also be produced by platelet aggregation in small, stenotic lumens, according to some experimental studies. 26, 27

The study by Oliva and Breckinridge is often quoted as evidence for the presence of spasm at the site of coronary artery occlusion in acute myocardial infarction. These authors injected nitroglycerin into the completely or subtotally occluded coronary artery and succeeded in partially reopening or expanding the coronary lumen in six of 15 patients with acute myocardial infarction. The effect was relatively brief. Continued administration of nitrates was necessary to maintain the achieved patency. We have seen faint distal filling after intracoronary nitroglycerin in two of 20 occluded coronary arteries in patients with acute myocardial infarction. The effect lasted about 5–10 minutes. The effects described by Oliva and Breckinridge and seen in our study do not necessarily prove the presence of spasm or increased arterial tone. A portion of the coronary artery circumference may not have been firmly attached to the stenosing plaque and/or thrombus and was therefore capable of dilating in response to nitroglycerin, creating a lumen or enlarging the existing one. The brief duration of such a response (5–10 minutes) corresponds to the known duration of nitroglycerin effect on the large, conductance coronary arteries. 30

In 1940, Blumgart et al. 21 demonstrated that patients with ischemic heart disease have severe stenoses and obstructions in their coronary arteries. For decades the anatomic changes were considered the only factor in the manifestations of coronary artery disease, with no place for functional influences. The pendulum may be swinging too far in the other direction.

References


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