Coronary spasm has been felt to contribute to ischemic cardiac syndromes for nearly 80 years, however debate continues concerning its prevalence, contribution to angina, role in acute MI and its pathophysiology.

Coronary spasm should be differentiated from normal coronary vasoreactivity that can also contribute to angina in patients with high grade coronary lesions. Spasm is most commonly associated with variant angina, a distinctive clinical syndrome described by Prinzmetal nearly 20 years ago. The current estimates of the incidence varies from 5 to 7% of patients presenting with anginal complaints. However, it is also recognized that many patients with exertional angina and coronary artery disease have rest pain that cannot be adequately explained by severe coronary artery disease. In recent studies, rest pain may occur in as many as 60% of patients. Numerous studies by Maseri and others have shown that rest angina is not associated with a rise of myocardial demand, but rather a decline in coronary supply, as evidenced by a decrease in coronary blood flow and rise in coronary sinus lactate. While thrombus may play an etiologic role in some patients with unstable angina, it does not appear to be the mechanism of rest pain in patients with stable angina, as evidenced by the lack of effect of antiplatelet and anticoagulant therapy.

The incidence of recognized coronary spasm appears to be less, perhaps in part due to the widespread use of calcium antagonists. The incidence of positive ergonovine testing during coronary angiography at our institution has dramatically fallen from 20% to less than 5% over the past five years. Most commonly, coronary spasm occurs at sites of atherosclerotic plaques that may not be of hemodynamic significance. While spasm during coronary angioplasty was not uncommon five years ago (10%), it is rarely observed today (less than 1%). These observations may not reflect a true decline in the incidence of vasospasm, but merely indicate the effectiveness of calcium antagonists in treating patients with this problem.

The pathophysiology of spasm continues to remain unknown, while abnormalities of sympathetic and muscarinic receptors have been postulated, there was stronger experimental support for a loss of the protective role of the endothelium. This is consistent with the observation that spasm rarely occurs in the absence of atherosclerosis. Early atherosclerosis can result in the failure of the endothelium to produce one of several relaxing substances. Experimental studies by Cohen, Shephard and others have shown that direct vasoconstriction occurs from platelet aggregation. Serotonin, Epinephrine, Thromboxane and other substances when the endothelium was removed or damaged. Experimental data from our laboratory demonstrate that in the rabbit model of atherosclerosis, vasospasm occurs early in the development of lesions and disappears once high grade lesions are present. Vasospasm may also help promote the development of significant coronary stenoses.

Vasospasm leading to the syndrome of variant angina is less recognized in the United States than it was previously. However the role of vasospasm in precipitating angina in the presence of significant atherosclerotic lesions or in their absence is now well-documented. Current therapy with calcium antagonists has helped considerably in controlling this problem, although the mechanism of vasospasm remains unclear.