When, approximately two centuries ago, the syndrome of angina pectoris was first described and named, one effect was controversy, which has waxed and waned until the present time. However, from the very first, the clinical syndrome has emphasized pain—pain that was precordial, pain that signaled the onset of the syndrome with exertion, vanishing pain that signaled the offset of the syndrome with rest or certain medications. The growth of interest in the clinical syndrome led to differences of opinion on causation and mechanism. In the meantime, clinicians became familiar with the facts that pain associated with esophageal reflex, esophageal diverticulum, diaphragmatic hernia, and cervical arthritis, for example, might be difficult to distinguish from the pain of angina pectoris and that true angina pain might be aggravated by hyperthyroidism or by chronic cholecystitis.

In the late part of the 19th century and the early part of the 20th, knowledge about coronary artery disease in general expanded due to the results of pathologic and physiologic laboratory studies on myocardial infarction. The findings were in part applied to angina pectoris, but here the effects of coronary vascular spasm, and those of increased cardiac work despite limited blood flow were also invoked to explain the paroxysmal nature of angina pectoris. The matter of the pain was never settled satisfactorily, but an analogy was drawn between it and intermittent claudication in the legs (also never satisfactorily explained). In the case of the increase in cardiac work needed to explain the precipitation of anginal pain with exercise, there was a widespread assumption that a rise in pulse rate occurred at the onset of angina, despite the fact clinicians knew that it often did not occur, and physiologists have long known that a rise in cardiac rate did not necessarily cause a rise in left ventricular work. Nevertheless the cluster of processes depicted above was considered to explain satisfactorily the pathophysiology of angina pectoris.

However, recent studies with modern technology have shown that the currently popular scheme is not acceptable. A rise in cardiac rate is relatively infrequent at the onset of anginal pain.1 Most attacks of myocardial ischemia of the type that produces angina cause no pain2,3 and the physiologic accompaniments of painful angina are no different from those of painless angina pectoris even when the exercise test is positive.4 Silent angina is held by some to have an unfavorable prognosis,5 as well it might since the patient is deprived of a warning that he is doing too much. Unstable anginal pain exhibits diurnal variation that is not explainable physiologically.

The whole matter of the significance and origin of the pain of coronary disease has come into question. The history of pain is not definitive6 and the absence of pain in many cases of coronary artery disease is not explainable.7 Coronary artery spasm does not seem to be the cause.8,9 The possible correlation between the pathophysiology of coronary artery disease and the occurrence of paroxysmal exertional or other chest pain cannot be defined. Even more confusing is the fact that some patients (approximately 10 percent) have typical angina pectoris but normal coronary angiograms, and yet have hemodynamic responses to exercise typical of those with abnormal angiograms.10

The formulation of valid concepts of the relations between seemingly established pathophysiology of coronary artery disease and anginal pain is not in sight.

REFERENCES
2 Singh BN, Nademanee K, Figuras J, Josephson MA. Hemodynamic and electrocardiographic of symptomatic and silent myocardial ischemia: pathologic and therapeutic implications. Am J Cardiol 1986; 58:3B-10B

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Coronary Artery Disease and Anginal Pain (Mark D. Altchule)

VII EUROPEAN CONGRESS ON DISEASES OF THE CHEST
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For more information, contact: Dr. J. Horgan, Secretariat, VII European Congress on Diseases of the Chest, 44 Northumberland Road, Dublin 4, Ireland. Telephone 688244; telex 31098; telegrams to Congrex, Dublin.

4 Nademanee K, Intarachot V, Singh PM, Josephson MA, Singh BN. Characteristics and clinical significance of silent myocardial ischemia in unstable angina. Am J Cardiol 1986; 58:26B-33B
8 Araki H, Nakamura M. Diurnal variation of variant angina. Internat J Cardiol 1984; 5:402-05
9 Quyyumi AA, Wright CM, Mochus WJ, Fox KM. How important is a history of chest pain in determining the degree of ischemia in patients with angina pectoris. Br Heart J 1985; 54:22-6