In the last decade or two, the idea of coronary artery spasm as a factor in the production of symptoms has achieved great prominence. The modern story, however, properly begins half a century ago, with the observation of Riseman and Stern that the amount of walking that will precipitate angina pectoris in patients is highly reproducible when conditions are standardized with respect to ambient temperature. This has been corroborated, most recently by Khurmi et al., using the most modern technology.

Riseman's exercise test was designed only to (a) test the effect of medication on anginal pain and (b) distinguish diagnostically between the chest pain of exertional angina and the chest pain of esophageal disease (including hiatus hernia) and of cervical arthritis. It was not offered as a means of proving the presence or absence of coronary artery disease.

Although exercise is clearly important in the genesis of anginal pain, it is not the sole factor. Cold temperatures have a decisive part in the process. Accordingly, analysis of the effects of cold in exertional angina pectoris is important. Changes in pulse rate and arterial blood pressure during exposure to cold do not correlate with the occurrence of anginal pain. Studies of ventricular status can be made by means of radioisotopes, such as radioactive thallium, technetium, or gold. When such studies are made during exposure to cold without exercise, abnormalities in ventricle status or function occur in normal hearts as well as in hearts with coronary artery disease. The test does not reliably distinguish one heart from another, or patients with angina pectoris from those without it.

It is apparent from currently available data obtained by modern technology that when normal persons, patients with coronary artery disease, and patients with angina pectoris in the presence of normal coronary arteriograms, are studied by the latest technologies, the data show a great overlap, so great that exercise alone, or exposure to cold alone, do not reliably distinguish persons from one group from those in another. Yet the fact remains that exercise in the cold is an excellent way to standardize the precipitation of angina pectoris.

Studies reported by Freedberg et al. 40 years ago, presented important findings bearing on this matter. They showed that the local application of cold to the hand lowered the exercise tolerance of patients with angina pectoris, even in a warm room. The effect of the cold application was apparent after as little as ten seconds and never took more than 30 to 40 seconds to appear. The effect was not prevented by obliteration of venous return from the hand, but could be nullified by the prior application of heat or by the administration of nitroglycerine. The studies showed that some neurogenic factor, arising in chilled skin, lowered the exercise tolerance in angina pectoris. The findings suggested the occurrence of coronary arterial constriction as a mechanism. At that time, coronary artery spasm was generally not considered an important factor in the genesis of anginal pain; emphasis was given to the concept that pain was precipitated by the requirement during exercise for increased blood flow through organically narrowed coronary arteries. Today, owing to angiographic observations, we know that spasm is common in relation to the pain. We also know that approximately 10 percent of patients with angina pectoris have no detectable disease of the epicardial arteries. However, Blumgart et al., using an injection method in post-mortem studies, have emphasized the finding of at least one major coronary occlusion in patients with exertional angina pectoris.

The very concept of myocardial ischemia used to explain the symptoms of angina pectoris can no longer be considered free from ambiguity. Ischemia may occur at times in patients with the syndrome but produce no pain. At least as regards local myocardial damage produced by ischemia, factors other than blood flow have major importance. The mechanism whereby coronary arterial spasm causes anginal pain selectively in some persons is not

*Francis A. Countway Medical Library, Harvard Medical School, Boston.
Reprint requests: Dr. Altschule, Harvard Medical School, Boston 02115
known. Neither can the role of spasm in angina pectoris in general be defined. However, it is worth noting that triple or quadruple coronary bypasses, shown to relieve cardiac pain regularly, provide for a blood supply to much of the myocardium that can in no way be influenced by vasomotor phenomena.

The situation with regard to studies on patients with exertional angina pectoris may be summarized as follows: (a) exposure to cold before or during exercise lowers exercise tolerance in patients with the pain syndrome but not in other persons; (b) exposure to cold alone produces changes in myocardial status in both normal subjects and patients with coronary artery disease with or without the pain syndrome; and (c) electrocardiographic changes, and changes in ventricular status produced by exercise alone in normal persons, persons with angina pectoris with or without coronary artery disease, and patients with coronary artery atherosclerosis with or without anginal syndromes overlap to such a degree as to prevent, by themselves, reliable clinical evaluation. In short, of the phenomena thus far studied, only the production of angina pectoris by exercise in the cold is diagnostic.

Angina pectoris is a clinical syndrome whose anatomic and physiologic substrates cannot be defined precisely. A vast amount of gadget-oriented study has not solved the fundamental problems. However, we may anticipate that the widespread use of gadgets in angina patients will continue, since as Sir James Mackenzie remarked, "The seeming exactness of a mechanical device appeals much more strongly to certain minds than a process of reasoning."

**References**