Correspondence

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Angina Pectoris Without Chest Pain

To the Editor:

The article by Stern1 in the October 8, 2002 issue of Circulation raises an interesting and valid point, but he does not take it far enough. He comments, properly, on the many symptoms in addition to pain that may accompany myocardial infarction—ie, shortness of breath, overwhelming fatigue not present earlier, irregular heart beats, etc. This is certainly correct, but it is equally true of transient myocardial ischemia (or, more properly oligemia), a phenomenon that may or may not lead to the painful discomfort of classic angina pectoris.

The very term angina is unfortunate, because it connotes painful discomfort that for centuries after Heberden’s epochal insight was thought to be the only clinical manifestation of the pathophysiology—a misconception that persists to this day. Before monitored stress procedures, there was no objective way of demonstrating transient myocardial ischemia, so the solipsism of pain as the exclusive manifestation of transient myocardial ischemia persisted.

My colleagues and I, in the early days of monitored stress testing, carried out a study on a number of patients with documented coronary artery disease. Monitored stress testing was performed, and when significant ST deviation appeared, the patient was asked to describe the symptoms. Our study appeared in the American Journal of the Medical Sciences2 and was abstracted in the Yearbook of Cardiovascular Medicine and Surgery,3 but after that, it seemed to sink out of clinical percpetion.

The most common symptom at the time of ST change was dyspnea, followed by the sequence dyspnea pain. Isolated, typical anginal pain was documented in only 17.3% of patients. Paresthesias, anxiety, and other manifestations were present in many patients, and 10% were truly “silent”—the first documentation, by the way, of silent myocardial ischemia.

Transient myocardial ischemia, like myocardial infarction, produces a whole spectrum of symptoms arising in part from the fact that the pathophysiology induces transient congestive heart failure. Any experienced catheterizing cardiologist will attest that left ventricular end-diastolic pressure rises acutely during anginal pain. If the clinician is not aware of this fact, the phenomenon may be overlooked with tragic consequences.

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Stern claims1 that in 10% of cases the symptom of transient myocardial ischemia was “silent.” We performed a study in 1967–1968 of 100 patients who were monitored for signs of ischemia before and after exercise. We found that 10% of patients were truly “silent” and that 34% of patients demonstrated ischemia without a sensation of chest pain. This means that, in addition to the Braunwald1–3 “silent ischemia,” there exists a phenomenon of “silent chest pain.”

According to Stern, silent chest pain may never be definitively diagnosed unless the patient is monitored for significant ST deviation. However, in our study, 10% of patients had no chest pain, 19% had chest pain that was less than that for which they were hospitalized, and 65% had chest pain that was of the same or greater severity than had been experienced prior to the electrocardiographic change. Thus, silent chest pain can be and has been diagnosed in the absence of monitored stress testing.

Phibbs and his colleagues3 have done a great deal to bring this important phenomenon to the attention of the medical community. We agree with them that the phenomenon of transient myocardial ischemia and exercise-induced chest pain should not be overlooked with tragic consequences. If the clinician is not aware of this fact, the phenomenon may be overlooked with tragic consequences.

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