Angina Pectoris Without Chest Pain

To the Editor:

The article by Stern\(^1\) 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 Sciences*\(^2\) and was abstracted in the *Yearbook of Cardiovascular Medicine and Surgery*,\(^3\) but after that, it seemed to sink out of clinical apperception.

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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Response

I greatly appreciate the valuable response of Dr Phibbs to my Clinician Update on silent ischemia. Dr Phibbs’ aim was to widen this clinical entity, by stressing that it can be observed also during exercise testing, as he and his colleagues described in 1968,\(^1\) and that if its expression is other than angina in the left chest, for example, dyspnea, this should still be included in the definition of “silent ischemia.”

I fully agree with both amendments of Dr Phibbs and am even grateful to him that he expresses these views in connection with my contribution in *Circulation*.\(^2\) Recognition of the “atypical” presentations of ischemia is vital to the proper care of our patients, especially diabetics, the elderly, and other high-risk patients who have a tendency to exhibit atypical symptoms when suffering ischemia.\(^3\)

Now, after more than 200 years, it too late to fight against the expression angina pectoris; instead, we should always use it not to describe a symptom but to affirm the diagnosis of “chest pain due to myocardial ischemia.” That myocardial ischemia may have other expressions, notably dyspnea, is worthy of emphasis, even several decades after its description by pioneering investigators, such as Dr Phibbs and his colleagues.

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_Circulation_. 2003;108:e37
doi: 10.1161/01.CIR.0000084387.51590.EC

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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