Painless Myocardial Ischemia

The triad of anginal pain, ST segment change on the electrocardiogram, and the demonstration of the end products of anaerobic metabolism is recognized as the hallmark of myocardial ischemia. It appears that, of these indices, chest pain may be the least reliable. This suggestion is supported by the following observations: (a) that myocardial infarction may occur without pain in both diabetic and nondiabetic populations; (b) that ST segment depression following maximal treadmill exercise may occur in apparently asymptomatic individuals and, of these, a significant number will develop clinical manifestations of coronary artery disease within 2.5 years; (c) that ST segment changes without chest pain can be induced in patients with angina by atrial pacing, and (d) that patients with the variant anginal syndrome may experience ST segment elevation and arrhythmias both in the presence and in the absence of chest pain. It is to the latter group of patients that we now direct our attention. The report by Kossowsky and associates describes five such cases (ie, without chest pain). The above observations are not, after all, unique to the syndrome popularized by Prinzmetal and colleagues, for spontaneous ST segment depression without chest pain has been observed in patients with the more typical anginal syndrome.

The frequency and severity of chest pain are usually utilized as an index of the severity of coronary artery disease, and the relief of pain is regarded as an index of effectiveness of various therapeutic modalities. It is appropriate, therefore, to consider those factors which contribute to the production of chest pain in patients with ischemic heart disease. These include: 1) the presence of nervous tissues capable of responding to appropriate stimuli and of neuronal pathways capable of transmitting these impulses, 2) the presence of stimuli capable of exciting the neural tissues, and 3) the recognition of chest pain by the patient.

The heart is richly supplied by sympathetic nerve endings which are found throughout the myocardium and around the coronary arteries. The afferent sympathetics containing myelinated and non-myelinated fibers travel to the spinal cord via the sympathetic ganglia. A variety of stimuli, including manipulation and occlusion of the coronary arteries, the application of KCl to the ventricular surface, and the injection of bradykinins and catecholamines into the left atrium, are capable of stimulating these fibers. Furthermore, the intracoronary injection of bradykinins in the awake dog will elicit a response consisting of hypertension, hyperpnea, and vocalization. What is not well understood is the quantitation of these various factors. For instance, it has been shown that a minimum of 100 µg/ml KCl is required to excite the sympathetic afferents. However, it is not known whether the rate of accumulation of potassium alters this critical concentration. Nor is it known whether combinations of algesic substances alter the critical quantity of each individual substance. Furthermore, there is no quantitative information available regarding the number of sensory fibers which must be stimulated, or the number of discharges in the afferent fibers required, to produce a sensation of chest pain.

The possible clinical importance of these considerations is suggested by the observation that a majority of patients with angina pectoris and positive stress tests will have episodes of "painless" ST depression documented by ambulatory monitoring.

We found that the episodes of ST segment depression unaccompanied by chest pain tended to be of shorter duration and to be associated with a slower heart rate than those episodes which were accompanied by chest pain. We found no difference in the magnitude of ST segment depression and could not correlate pain or the absence thereof with the activity of the patient. These observations suggested to us that the patients might have been experiencing varying degrees of myocardial ischemia and that the less severe episodes might have been "painless." Another important determinant of chest pain is each individual's pain threshold and the influence of physical and psychic factors on this threshold. The well-known placebo effect of surgical and pharmacological interventions exemplifies the importance of these factors.

Thus, although typical anginal pain indicates the presence of myocardial ischemia, the absence of pain does not guarantee the absence of ischemia. The therapeutic modalities currently in vogue for the treatment of coronary artery disease, particularly coronary artery bypass surgery and the beta-adrenergic blocking agents, may be capable of altering the frequency and severity of chest pain without necessarily reducing myocardial ischemia. Both modalities might induce a significant placebo effect. In addition, pericarditis invariably follows coronary artery surgery and myocardial infarction occurs not uncommonly in the course of such surgery. Either of these complications might destroy sympathetic sensory endings or alter their sensitivity to algesic substances, thereby interfering with the ability of the patient to recognize myocardial ischemia. The beta blockers cause local anaesthesia and slow nerve conduction. These drugs might therefore decrease impulse transmission in the afferent sympathetics and obliterate chest pain without necessarily decreasing myocardial ischemia.

Because of these considerations, it behooves the physician to rely on parameters of myocardial...
ischemia which are more objective than chest pain. Of these, the simplest is exercise stress testing although other methods, such as ambulatory monitoring, atrial pacing or other types of stress testing, might be employed. This alternative is particularly important when evaluating the frequency of myocardial ischemia and/or assessing the response to therapeutic interventions which might alter any of the multiple factors contributing to the production and appreciation of chest pain.

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Pioneer of Viennese Operettas

Although he was one of its most eloquent practitioners, Strauss himself did not pioneer the operetta form in Vienna. That task fell to Francesco Ezechiele Ermengildo Cavaliere Suppé Demelli (1819-1895), who quite understandably condensed his name to Franz von Suppé. In 1860 he produced the first of his successful operettas, and many more followed before Strauss began to corner the market effectively about a dozen years later. Little of Suppé's vocal music is heard these days, but you can hardly escape the overtures. "Poet and Peasant," for instance, is a marvelous curtain raiser and still fun to hear and so is "Light Cavalry," without which half of the Westerns in Hollywood could never have been made. As for Strauss himself, he sometimes was ill at ease with the longer, more involved format of the operetta, but at his best he was, as usual, unbeatable. Die Fledermaus is a frothy farce, a fast and funny show with more whistleable tunes than anything you will find on Broadway today. Not far behind is another brilliant Strauss creation, The Gypsy Baron.


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