Prinzmetal’s Angina Pectoris

To the Editor:

A very interesting paper by Yasue and associates concerning the role of the autonomic nervous system in the pathogenesis of Prinzmetal’s angina pectoris appeared in the September 1974 issue of Circulation.

The authors report their ability to precipitate attacks of angina pectoris with the choline ester methacholine and were able to block this response by prior administration of atropine. Although the authors did not demonstrate coronary artery spasm in their patients at the time of coronary arteriography, the literature now contains enough reports of non-catheter-induced spasm in the large coronary vessels in individuals with this syndrome to support Prinzmetal’s original thoughts about the etiology of this unusual and interesting form of angina pectoris.

Aside from the obvious therapeutic implications of parasympathetic triggering of coronary artery spasm, it is quite possible that many episodes of coronary artery spasm which would otherwise be noted at the time of coronary arteriography are thwarted by prior administration of atropine which many cardiovascular laboratories give prior to injecting the coronary arteries in order to diminish bradycardia.

In view of this possibility it is our feeling that the routine use of atropine prior to coronary arteriography should be re-examined, particularly in patients with suspected variant form of angina pectoris.

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The authors reply:

To the Editor:

We appreciate the comments of Drs. Bennett, Galyean and Lehan in reference to our recent article.1 We agree with their opinion that atropine should not be administered to patients suspected of variant form of angina prior to coronary arteriography. Instead of atropine and nitroglycerin, which prevent coronary arterial spasm, we administer propranolol (40 mg orally) two to three hours before coronary arteriography in patients with variant form of angina because this drug increases coronary arterial tone.2 Coronary cinearteriography is then performed before, during and after the attack which is induced by the administration of either epinephrine (0.4–0.5 mg subcutaneously) or methacholine (10 mg subcutaneously) under the constant monitoring of electrocardiogram and blood pressure. By this method we were able to demonstrate severe spasm of a large coronary artery during the attack in four patients with variant form of angina. The spasm disappeared after the attack was relieved by the sublingual administration of nitroglycerin (0.6 mg). It is now established that Prinzmetal’s variant form of angina is caused by severe spasm of a large coronary artery as postulated by Prinzmetal and his associates.3

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References


Exercise and CHD

To the Editor:

Dr. Mann states (Circulation 50: 1283, 1974) that I have “misquoted the evidence bearing on occupational exercise and coronary heart disease as revealed by studies of the Finnish lumberjacks,” presenting in support prevalence data published by Dr. Karvonen in 1961. In 1970, on the basis of prospective observations which are more reliable than studies of prevalence, Karvonen wrote as follows (Circulation 41 and 42 [suppl I]: I-52, 1970): “It has been repeatedly claimed that a sedentary life or a low level of physical activity is a major risk factor for CHD, but the evidence is not very satisfactory. This question is examined for the Finnish material in table VI.8. The more sedentary and inactive Finns were not more
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