**LETTERS TO THE EDITOR**

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**Prinzmetal’s Variant Angina**

To the Editor:

I wish to take exception to some of the main conclusions expressed by Scherf and Cohen in their editorial on variant angina pectoris (Circulation 49: 787, 1974). They suggest that “Prinzmetal’s angina” is neither new nor unique; nor do they believe it defines a sufficiently homogeneous group of patients to be of practical clinical utility.

Of course there were cases of variant angina reported in the older literature before Prinzmetal and his colleagues made their initial report, but these were isolated cases. It was Prinzmetal and his co-workers, however, who collected from their own patients and prior medical literature the information which allowed them to make the first clinical description of the variant angina syndrome, and it was their description which called this condition a medical curiosity.

Despite the fact that most reports on variant angina in the past fifteen years have consisted of only small numbers of cases, exceptions to this generalization can be found, and important new information has been yielded. Scherf and Cohen are astonished at how rarely the level of the blood pressure during attacks of pain has been reported, but they failed to note the sophisticated physiological monitoring studies of Guazzi et al.; this study clearly demonstrated the absence of significant alterations in heart rate and blood pressure at the beginning of attacks of variant angina, distinguishing it clearly in this regard from classical angina.

The realization that variant angina can occur in patients with arteriographically normal or nearly normal vessels has been the result of too many reports to cite here. In addition, the arteriographic documentation of spasm of normal and diseased large coronary arteries during attacks of variant angina pectoris is occurring with increasing frequency across the country. This has important therapeutic implications.

It is quite clear now that Prinzmetal’s variant angina is due to intermittent coronary artery spasm. It is this pathophysiology of arterial spasm which makes variant angina unique, differentiates it from the classical angina of Heberden, and produces a homogeneous group out of what Scherf and Cohen consider a heterogeneous syndrome. With this in mind it is easy to understand why the clinical presentation of variant angina can be so varied. The attacks associated with ST-segment elevation usually occur at rest, but in a minority of patients can be precipitated with exercise. Classical exertional angina with ST-segment depression may coexist in those subjects who also have a high grade fixed coronary obstruction. Usually no inciting cause for attacks is apparent but there are isolated reports of patients in whom attacks could be reproduced by a variety of stimuli. Coronary arteriographic findings can range from normality to three vessel obstructive disease, but if abnormalities are present, they are usually focal and fixed stenoses are rarely severe enough to account for angina at rest. All these different clinical presentations are a result of the transience and intermittency of the spastic coronary artery obstruction.

Prinzmetal’s variant angina is more than just a medical curiosity. Increasing numbers of patients with it are being recognized. It is the first type of ischemic heart disease which has been documented as being due to coronary artery spasm and raises the probability that vasomotion of large coronary arteries plays an as yet unappreciated role in many patients with more classical forms of ischemic heart disease. Its presence stands as a challenge to physicians to uncover yet other disorders of autoregulation of the coronary circulation which may be responsible for perplexing clinical problems.

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**References**

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