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This Week's Citation Classic

Robinson B F. Relation of heart rate and systolic blood pressure to the onset of pain in angina pectoris. *Circulation* 35:1073-83, 1967. Dept. Medicine, St. George's Hospital Medical School, London, England]

Studies in patients with angina pectoris showed that the onset of pain under varying conditions could be consistently related to the level achieved by the product of heart rate and systolic arterial pressure. The rate-pressure product provides a useful clinical index of myocardial work. [The ***SCI***® indicates that this paper has been cited over 295 times since 1967.]

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"The early 1960s saw a sudden surge of interest in angina pectoris with the development of coronary arteriography by Mason Sones[1] and the emergence of the first b-adrenoceptor antagonist, pronethalol. I was fortunate to be involved through A.C. Dornhorst in the early clinical studies with pronethalol. The b-adrenoceptor antagonists had been the brainchild of J.W. Black[2] (since knighted by the Queen for his remarkable contributions to pharmacology) and he had been led to develop this new class of compound by the thought that selective inhibition of the effects of catecholamines upon the heart might provide a useful means of controlling the work and oxygen requirements of the myocardium in patients with angina pectoris. The role of the myocardial work load in determining the onset of angina was at that time far from clear (many problems remain today!) and this suggested to me the idea of investigating the relation between the two. Review of the physiological literature indicated that the heart rate and systolic pressure were probably the most important variables determining changes in myocardial oxygen consumption between rest and exercise and I therefore chose to use the product of the two as an index of myocardial work.

"The experimental work was carried out in the course of 1963 on the one day a week that I could keep relatively free for research. Each study involved continuous recording of intraarterial pressure during repeated bouts of exercise and other types of stress. The results led to the conclusion that the precipitation of angina normally resulted from an increase in the work of the myocardium, as measured by the rate-pressure product, to a critical level that was essentially fixed in each patient.

"It is pleasing to know that work I carried out nearly 20 years ago with a view to obtaining my MD has been accepted and is still referred to. The concept it put forward undoubtedly represents an oversimplification of the complex circulatory disturbances that lead to the onset of angina, but I hope, nevertheless, that it has proved useful as a way of thinking about the problem. Why has the paper been so frequently cited? Largely, I think, as a reference for the use of the rate-pressure product as a clinical index of myocardial work. When I originally decided upon these variables for my measure of myocardial work, I was strongly influenced by the fact that they could at least be easily measured and I had some reservations as to whether they provided the best available index. It was therefore with delight (and some relief!) that I learned that more recent work involving direct measurement of myocardial oxygen uptake in normal subjects had shown that the rate-pressure product is not only a valid index of oxygen consumption, but is a better predictor than some other indices that have been proposed."[3]

1. **Sones F M, Jr.** & **Shirey** E **K.** Cine coronary arteriography. *Mod. Cone. Cardiovasc. Dis.* 31:735-8, 1962.
2. **Black J W & Stephenson J** S. Pharmacology of a new adrenergic beta-receptor-blocking compound (nethalide). *Lancet* 2:311-14, 1962.
3. **Nelson R R, Gobel F L, Jorgensen C R, Wang K, Wang Y & Taylor H** L. Hemodynamic predictors of myocardial oxygen consumption during static and dynamic exercise. *Circulation* 50:1179-89, 1974.