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Literature Briefs

Myron B. Laver, M.D., Editor

Literature briefs were submitted by Drs. I. C. Andrews, R. Clark, A. Goldblatt, J. Levitt, W. Mannheimer, Briefs appearing elsewhere in this issue are part of this column.

Circulation

TACHYCARDIA AND CORONARY-ARTERY DISEASE This paper summarizes the hemodynamic consequences of maximal atrial pacing performed in conjunction with coronary-artery and left ventricular angiography in 41 patients. Thirty-one had coronary artery disease (CAD). Measurements included cardiac output (CO), mean femoral arterial pressure (LAm) and left ventricular end-diastolic pressure (LVEDP), from which stroke volume (SV) and stroke work (SW) were calculated. Left ventricular function (LVF) curves during incremental pacing were constructed. Ten normal patients paced up to 130 ± 6 SEM beats/min manifested no change in CO or FAm; LVEDP, SV, and SW declined, indicating that LVF remained normal. In the CAD group, the atrial rate was elevated to 122 ± 4 SEM beats/min. Angina pectoris occurred in 18 patients, and ST-segment changes (> 1 mm horizontal depression, lead II) appeared in five additional pain-free patients as evidence of ischemia. All patients were found to have angiographically demonstrable obstruction of more than one coronary artery. The ventriculograms were normal in only three patients in the CAD group. The ischemic signs at maximal pacing rates were invoked by the excessive myocardial oxygen demand. Interestingly, these signs were less common among patients with CAD and prior infarction, presumably due to the presence of scar tissue. Some patients with CAD had increased CO, FAm, and importantly, LVEDP (especially at the onset of angina, suggesting a decrease in LV compliance), which often climbed further with cessation of pacing. SV and SW were diminished, but LVF was abnormal in 17 patients and depressed in 16 of 18 who complained of angina. *(Linhart, J.W.: Atrial Pacing in Coronary Artery Disease. Am. J. Med. 53:64, 1972.)*