

Literature Briefs

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Literature Briefs were submitted by Drs. G. Battit, J. Bland, R. Clark, B. Dalton, A. Goldblatt, J. Jacoby, C. Rockwell. Briefs appearing elsewhere in this issue are part of this column.

Circulation

VEIN GRAFTS FOR CORONARY ARTERY DISEASE Saphenous vein grafts used for aorto-coronary bypass of occluded or stenosed arteries of eight subjects were examined at necropsy. The causes of death were described as cardiac (infarction and acute failure) in four cases, respiratory in two, gastric hemorrhage in one, and intestinal perforation in one. Postmortem coronary angiograms were made in seven cases. In all eight cases the vein grafts were sectioned transversely and longitudinally at different levels and the tissues were prepared for microscopic examination. The incidence and extent of thrombosis in the vein grafts (four cases) did not correlate with duration of postoperative survival, which ranged from two hours to ten months. Some of those who had survived longest had either organized, recanalized thrombosis or fresh thrombus formation. Phlebosclerosis, thought to have been pre-existing, was found in the grafts of the two shortest-term survivors. Both died of their heart disease.

Examination of the long-acting bypass grafts showed the following histologic changes: 1) thickening of the entire wall, mainly the result of intimal hypertrophy; 2) transient medial muscular hypertrophy succeeded by atrophy and fibrosis; 3) deposition of elastic fibers in the external vein wall. No aneurysmal dilatation was found in these specimens. (*Marti, M., Bouchardy, B., and Cox, J. N.: Aorto-coronary By-pass with Autogenous Saphenous Vein Grafts: Histopathological Aspects, Virchows Arch. Abt. A Path. Anat. 252:255-266, 1971.*)

ABSTRACTER'S COMMENT: This series was regrettably small. This report underlines the need for more information about selection of vein grafts, their intraoperative handling, and postoperative changes, particularly in view of the increasing number of such operations being performed.

ATRIAL PACING AND VENTRICULAR FUNCTION IN ANGINA PECTORIS

The study was undertaken to ascertain whether a decrease in left ventricular performance consistently accompanies the appearance of pacing-induced angina and whether a fast-acting glycoside, ouabain, can prevent the hemodynamic abnormalities associated with angina. Eleven patients, ages 30 to 58 years, were studied. Coronary arteriography demonstrated greater than 50 per cent occlusion of at least one major coronary artery. Hemodynamic measurements included the left ventricular end-diastolic pressure (LVEDP), left ventricular stroke work index (LVSWI), stroke volume index (SVI), and peak rate of rise of left ventricular pressure (peak dp/dt). All these values were obtained during a control period and following atrial pacing at rates 10 to 20 beats faster than the control rates. Heart rate was subsequently increased in increments of 10 beats/min until angina appeared. Angina appeared during pacing in nine of 11 patients, when the mean control heart rate of 72/min was raised to 146/min. Mean control LVEDP was 11.5 mm Hg and fell slightly (mean: 1.8 mm Hg) following administration of ouabain. Before ouabain, LVEDP fell as the pacing rate was increased and tended to rise again at the maximum pacing rate. The response after ouabain was similar, although the control values (before pacing) were slightly lower. It is of interest that LVEDP rose transiently immediately upon cessation of pacing. Cardiac output did not change significantly at any

time, but LVSWI fell consistently as heart rate increased. When the relation of LVEDP to LVSWI was evaluated to judge the performance of the heart as a pump, evidence of impaired function was found during or immediately after pacing in five of ten patients. Ouabain did not prevent the appearance of these changes. Before ouabain, dp/dt rose significantly with increased heart rate. Ouabain produced a significant increase in control values of dp/dt , but subsequent pacing was accompanied by no change. When the relation of LV peak dp/dt to LVEDP was used to assess the inotropic state of the heart, contractility was enhanced by both pacing and ouabain, but the effects of the two were not additive. The study indicates that in some patients with coronary artery disease LV performance is depressed when tachycardia is induced by atrial pacing. Thus, when the relation of stroke work to LVEDP was determined, unequivocal decreases in LV function were found in two of ten patients during pacing. On the other hand, when peak LV dp/dt was related to LVEDP, a decrease in LV function was demonstrated in only one of ten patients, and in six it appeared to be improved. Since angina represents a specific end-point in the relationship between myocardial oxygen re-

quirements and available blood flow, it is possible that the discrepancy between hemodynamic performance (*i.e.*, LV competence) and metabolic needs will depend on the extent of LV wall involvement in the ischemic process. (Higgs, L. M., and others: *Effects of Ouabain on the Left Ventricular Response to Atrial Pacing in Patients with Angina Pectoris*, *Amer. J. Cardiol.* 28:17-24, 1971.)

CARDIAC TAMPONADE Perforation of the ventricle and tamponade are possible complications seen during left-heart catheterization. In the authors' series it was recognized in eight of 20,000 procedures. Tamponade also followed percutaneous myocardial biopsies (incidence 12 of 286 procedures).

Because the ensuing situation is life-threatening, early recognition and treatment are necessary. Early signs include radiologic evidence of dye extravasation and acute changes in vital signs, *i.e.*, tachycardia and elevated venous pressure. Recommended treatment is thoracotomy and drainage of the pericardium. Pericardiocentesis is not recommended because the clotted blood cannot be removed. (Effler, D. B., Loop, F. D., and Spampinato, N.: *Iatrogenic Cardiac Tamponade*, *Arch. Surg.* 103:189-190, 1971.)