Myocardial preservation by topical hypothermia

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Cardiac operations are facilitated by a still and bloodless field provided by arrest of both mechanical cardiac activity and coronary perfusion. The magnitude of ischemic damage to the heart during periods of aortic cross-clamping without coronary perfusion is reduced substantially by lowering myocardial temperature and by sustained membrane depolarization with potassium ion. Topical myocardial hypothermia alone, using 0.9% saline at 3 to 4 C, is capable of consistently providing protective levels of left ventricular midwall hypothermia in the range of 15 to 20 C. Meticulous attention to details of local cooling, however, is necessary to avoid large swings or regional gradients in myocardial temperature. Cooling is initiated immediately after cross-clamping by lavage of the pericardial reservoir with 2 liters of cold saline and is continued by infusion of saline into the pericardial sac at 100 to 150 ml/min. Immersion of both ventricles in cold saline is enhanced by orientation of the operating table, and additional myocardial cooling is achieved by endocardial lavage during procedures involving cardiotomy. Low flow cardiopulmonary bypass (40 to 50 ml/kg/min) and systemic hypothermia in the range of 31 to 33 C minimize the warming effect of

collateral coronary circulation via mediastinal and bronchial vessels. Complete right heart emptying is assured by double venous cannulation. During a single cross-clamp interval, valve replacement(s), ventricular resection, and all distal coronary anastomoses are carried out.

Left ventricular intramyocardial temperatures decrease gradually over a 5- to 10-minute period after initiation of cooling, and in the anterolateral left ventricular midwall temperatures in the range of 15 to 18 C can be obtained consistently. Transmural temperature gradients (subepicardial-subendocardial) and regional variations in myocardial temperatures of 5 to 6 C, however, are usually present.

We have previously documented the superior clinical efficacy of topical hypothermia in comparison to continuous coronary perfusion with ventricular fibrillation and left ventricular venting for aortocoronary bypass grafting. The topical hypothermia technique was associated with significantly lower postoperative serum enzyme rises and myocardial infarction rates; there were no statistical differences, however, in operative mortality rates or immediate postoperative hemodynamic status. Further, though indirect, evidence for the effectiveness of topical hypothermia has been deduced from analysis of the patients undergoing aortic or mitral valve replacement over the past decade. In 741 consecutive patients undergoing aortic valve replacement (average cross-clamp time 62 minutes) and in 742 patients undergoing mitral valve replacement (average cross-clamp time 39 minutes) there was no significant correlation of ischemic interval with postoperative outcome (operative mortality rates 7.4% and 8.2% respectively), taking into account subcategories of patients defined on the basis of left ventricular function and associated coronary artery disease. No effects of cross-clamp duration on late postoperative survival were evident in patients discharged from the hospital.

Thus, our clinical experience supports the contention that topical myocardial hypothermia alone provides satisfactory myocardial protection during cardiac operations. Its primary advantages consist of simplicity and excellent operating conditions; recognized disadvantages include gradual, rather than immediate cessation of cardiac mechanical and electrical activity, as well as heterogeneous transmural and regional cooling.