Myocardial Perfusion After Repair of Transposition: Is It Worth the Switch?*

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Background. Transposition of the great arteries is a lethal congenital malformation and represents the most common cardiac cause of cyanosis in the neonate. Left untreated, up to 90% of infants with the condition will die within the first year of life (1). The prognosis for these infants, however, has been remarkably improved with the introduction of palliative and corrective surgical techniques. The major physiologic consequences of transposition of the great arteries are severe hypoxemia, acidosis and congestive heart failure (2). In the most common form of the malformation, the ventricles maintain a normal relation to each other (i.e., right-sided right ventricle and left-sided left ventricle [d-transposition]). However, the great arteries are transposed, resulting in parallel circulations. Systemic venous return is directed to the right ventricle and back to the systemic circulation through the aorta, whereas pulmonary venous return is directed to the left ventricle and back to the lungs through the pulmonary artery. Mixing of the two circulations must occur for oxygenated blood to reach the systemic circulation, or death ensues soon after birth. A major advance in sustaining the life of these infants occurred with the introduction of the balloon atrial septostomy procedure by Rashkind and Miller (3). This temporizing procedure allowed mixing of the parallel circulations across the atrial septum. Among the earliest and most successful surgical procedures was the physiologic correction of the circulation by redirecting the pulmonary and systemic venous returns to the appropriate ventricles using intraatrial baffles (Mustard [4] or Senning [5] procedure). This atrial switch operation has had great success in the early operative period, with postoperative survival rates of 90% and 80% at 10 and 20 years, respectively (6,7). Enthusiasm for this approach has been dampened somewhat because of concerns that the right ventricle may be unable to function successfully as the systemic ventricle for long periods of time (8,9).

Successful anatomic correction of transposition was first reported in 1976 (10). The procedure consisted of switching the great vessels to the proper ventricles and reimplanting the coronary arteries to the newly formed aorta. The morphologic left ventricle then becomes the systemic pumping chamber, for which it is better suited. Although technically demanding, the procedure is best performed during the neonatal period, before the regression of left ventricular mass as a result of the decreasing pulmonary vascular resistance that occurs after birth. Alternatively, the left ventricle can be conditioned to accept the high systemic vascular resistance if pulmonary banding is performed as an initial procedure. Concerns have been raised since the inception of the arterial switch procedure about the possibility that distortion or growth failure of the newly implanted coronary arteries may result in myocardial ischemia and, possibly infarction.

Present studies. The report by Hayes et al. (11) in this issue and that by Weindling et al. (12) in the February issue of the Journal provide important new information about myocardial perfusion after the arterial switch procedure. In both reports, myocardial perfusion was evaluated 5 to 6 years after surgical correction by use of technetium-99m sestamibi. In both studies, left ventricular perfusion abnormalities were found in roughly 25% of segments in nearly all of the patients. These defects were noncoronary in distribution, did not show significant reversibility (fixed) and often involved the apex. In the study by Weindling et al., no apical vents were used during the operation, which might explain the presence of these perfusion defects. The study by Hayes et al. is particularly noteworthy for the inclusion of two "control" groups. Myocardial perfusion was evaluated in children of similar age who underwent cardiopulmonary bypass procedures for correction of atrial or ventricular septal defects and in children with congenital heart disease who did not undergo surgical correction. Surprisingly, a high incidence of perfusion abnormalities was also found in the patients undergoing bypass procedures without manipulation of the coronary arteries, whereas myocardial perfusion was essentially normal in the patients who did not undergo surgical intervention. The findings in these latter two groups eliminate technical artifacts related to the imaging methodology as an explanation for the perfusion defects. The results strongly suggest that the cardiopulmonary bypass procedure resulted in the alterations in perfusion. Although the exact mechanisms are unknown, Hayes et al. suggest the possibility that microemboli during the bypass procedure may have resulted in small myocardial infarctions. This hypothesis is further supported by the finding of a trend toward a greater number of perfusion abnormalities in patients undergoing a second cardiopulmonary bypass procedure. Perfusion abnormalities have also been documented in patients after the arterial switch procedure using thallium imaging (13–15). In the study by Vogel et al. (15) none of 7 children undergoing primary arterial switch had perfusion or wall motion abnormalities, whereas 8 of 14 children undergoing a two-stage repair had perfusion defects. Seven of these eight children had regional wall motion abnormalities corresponding in location to the perfusion defects. The fact that the patients with the two-stage repair had defects is consistent with the possibility that the repeat cardiopulmonary bypass procedure may have increased the risk of microemboli. It is interesting that there were no regional wall motion abnormalities...
associated with the scattered perfusion defects in the study by Weindling et al. (12). Whether the time period after surgical correction (5 to 6 years) in the patients studied by Weindling et al. versus 2 years in the patients studied by Vogel et al. allowed some type of "compensation" of wall motion abnormalities by the remaining viable and functioning myocardium is unknown.

It appears that coronary artery manipulation and reimplantation do not result in late myocardial perfusion abnormalities, at least in those infants with successful initial results. What about the specificity of perfusion imaging for detecting problems with coronary flow when technical problems with the reimplantation procedure occur? It is likely that structural abnormalities at the origins of the transplanted coronary arteries will result in large, segmental perfusion defects and should be easily detected with current scintigraphic techniques. Previous studies with thallium have shown large perfusion abnormalities in the territories of occluded coronary arteries after the switch procedure (14).

The patients described in the studies by Hayes et al. and Weindling et al. were asymptomatic and had normal left ventricular function and normal exercise capacity and in general did not require any cardiac medication. These observations suggest that the scattered perfusion defects that were observed do not result in any functional compromise. Whether this excellent result will continue after longer periods of time is unknown. However, the results to date are impressive.

References


