Surgery for Transposition of the Great Arteries

DR. ALFRED BLALOCK pioneered palliative surgery for congenital cardiac deformities and also, in discussing years ago a report of an early series of open-heart procedures, anticipated a perplexity which has not only persisted but has progressed. This perplexity derives from the question of when cardiac surgery should be considered palliative, when curative, and when intermediate (that is, accomplishing various degrees of correction but less than true cure).

The first successful operations tended to be the simpler and the more nearly curative, such as closure of an atrial or ventricular septal defect. However, even these were not curative if significant pulmonary vascular obstructive disease had developed before operation. The more complicated procedures, such as repair of the tetralogy of Fallot, are less than curative in many instances, depending on the extent of deformity of the right ventricular outflow tract and pulmonary valve and the consequent degree of residual pulmonary stenosis or insufficiency.

It is not unexpected that the recently developed operations on the more complex congenital cardiac defects are likely to be less curative and more nearly palliative. This applies to conditions such as truncus arteriosus, in which a substitute artery and valve must be inserted and in which pulmonary vascular obstructive disease is common, and to complete atioventricular canal, in which atrioventricular valvular deformity usually causes some degree of postoperative mitral regurgitation. Similarly, the surgical treatment for transposition of the great arteries (TGA), as we now know it, must generally be considered to be midway between the palliative and curative ends of the spectrum.

With all the "plumbing" expertise of the modern cardiovascular surgeon, a truly curative operation for TGA would seem to be attainable, save for one problem. To divide the great arteries, rotate or contrapose them, and then reanastomose them is simple in concept and technic and would be totally corrective except that the coronary arteries then would originate from the pulmonary artery, a situation incompatible with life. The solution to this dilemma has not been found. Indeed, it may have been postponed by the availability of established palliative types of operations.

With knowledge of the unavailability of a curative operation for TGA, the concept of transposing the venous return within the atria was conceived and has become the accepted treatment. This approach was progressively developed by Albert, Senning, Merendino,
and Mustard. In the absence of a ventricular septal defect (VSD), pulmonary stenosis, or pulmonary vascular obstructive disease, the results from this procedure have become increasingly rewarding and the risks very low. We are concerned about the long-term performance of the right ventricle and tricuspid valve at systemic work loads, and this concern is reinforced by the occasional appearance and progression of tricuspid incompetence postoperatively. Because of this, the transposition of venous return (Mustard operation) cannot be considered curative.

If a ventricular septal defect is also present, the results are drastically less satisfactory, not just because the operation is technically more complicated but probably also because pulmonary vascular obstructive disease is usually present and severe; and the approach to the ventricular septum must be either through a ventriculotomy in the systemic (right) ventricle or through the delicate and important tricuspid valve. Early banding of the pulmonary artery in this situation, with subsequent debanding and intraatrial transposition of venous return, has been far less than reward- ing, not only because two major operative risks are involved but also because banding of the pulmonary artery in this situation often fails to prevent or reverse progressive pulmonary vascular obstructive disease.

The presence of congenital pulmonary stenosis does protect the pulmonary vasculature, but its configuration in the posteriorly placed left ventricular outflow tract makes it poorly amenable to adequate surgical relief. It is seldom the result of commissural fusion alone; it usually is associated with annular narrowing and subvalvular hypoplasia. Although the Rastelli-type operation has given new hope for the treatment of patients having TGA, VSD, and pulmonary stenosis, it is highly demanding from the technical standpoint and involves the limitations and uncertainties of a substitute right ventricular outflow tract and valve, whether this be a homograft or a synthetic aorta and valve.

The excellent review of a significant experience by Clarkson, Barratt-Boyes, Neutze, and Lowe, in this issue of Circulation, focuses and supports these views. These authors further suggest that by performing "corrective" operations earlier in life, the salvage rate can be materially improved, because the early attrition of infants having this condition can thus be avoided and probably also because pulmonary vascular obstructive disease can be minimized. They attribute the decrease in operative risk in infants largely to the use of profound hypothermia and circulatory arrest, although this point cannot be proved by their experience.

Certain principles of management for infants with TGA are emerging. All should undergo early balloon septostomy—at the time of initial cardiac catheterization—to improve mixing of the pulmonary and systemic circulations. Those with an intact ventricular septum who thereafter thrive and demonstrate adequate mixing by a resting arterial oxygen saturation of greater than 60% should be considered for the "corrective" operation of intraatrial transposition of venous return (as by the Mustard technic) some time during the second or, at latest, third year of life (ages 1 or 2 years). If the infant remains more cyanotic than this, the Mustard operation seems indicated even during the first months of life. Increasing experience should establish if this approach is preferable to early surgical creation of an atrial septal defect and later "correction" by the Mustard procedure.

A similar policy can be followed in the case of an associated ventricular septal defect except that, if severe pulmonary hypertension is present, very early operation is indicated because of the probability of rapidly progressive pulmonary vascular obstructive disease. In fact, if the pulmonary arterial pressure is at systemic levels, repeat catheterization at 6-8 months of age seems appropriate. Unless the pulmonary arterial pressure has decreased, closure of the ventricular septal defect and intraatrial transposition of venous return is advisable at that time. The poor results with early pulmonary arterial banding and later repair and "debanding" warrant such a trial with early "corrective" surgery.
Finally, for infants and young children who have a ventricular septal defect and pulmonary stenosis associated with TGA and who are severely incapacitated, excellent palliation is afforded by a systemic-to-pulmonary artery shunt, especially by the Blalock technic. This approach not only improves pulmonary blood flow but also imposes obligatory mixing between the two circulations and, because of this, affords excellent palliation even if pulmonary flow is calculated to equal or even double systemic flow. In such patients, "corrective" operation by the Rastelli method usually can be deferred until 5 years of age, at which time a low operative risk is attainable, as shown by the experience at the Mayo Clinic with some 40 Rastelli operations for TGA and pulmonary stenosis.

I must reiterate the hope that technics may yet be developed which will more nearly "cure" TGA than those more palliative methods used at present. In 1968\(^1\) a patient with TGA, a large ventricular septal defect, and overriding of the ventricular septal defect by the pulmonary artery (Taussig-Bing deformity) underwent correction by an intraventricular repair whereby the patch closing the ventricular septal defect spiraled in such a way that the left ventricle drained into the aorta and the right ventricle drained into the pulmonary artery. We subsequently have repeated this operation successfully on two other patients. Recently, Kawashima et al.\(^2\) of Japan have independently developed a similar technic. Following an evolutionary pattern, a similar intraventricular repair of the true TGA with VSD has been performed here on two patients with one success (as yet unreported), and laboratory testing of the technic by Puga (unpublished data) has given further encouragement to and technical improvement of this operative approach. Furthermore, the innovative surgical imagination should not neglect the earlier dream of a more curative repair by contraposition of the transposed great arteries.

Dwight C. McGoon

References


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DWIGHT C. MCGOON

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