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Discussion

Dr. Dwight C. McGoon (Rochester, Minn.): Dr. Creech, Gentlemen: We have long looked to Dr. Gerbode for his descriptions of technics and methods which have been valuable to us in adapting our own methods. In no areas have his results been more outstanding, in my opinion, than in this area of endocardial cushion defects.

Although we can compare our results reasonably favorably in the partial A-V canal group with his, so far as operative mortality is concerned, we, along with others, have noted a high incidence of residual mitral insufficiency following operation, using what I believe to be the same technic; so this is a question I would like to raise—why should this difference exist?

In the complete A-V canal group we have not been able to obtain the operative mortality rate which Dr. Gerbode has recorded. The three other groups, including our own, who reported results in this area in 1964 had a mortality rate which was nearly prohibitive, ranging from 63 to 75%. At that time Drs. Gerbode and Sabar reported one death in 13 such operations, if I interpret the data correctly.

Since 1964 we improved our results with a mortality rate in this period for the complete A-V canal of 25%; but still, taking the over-all results, ours are not really comparable to his, and one wonders again why this should be. I would ask him if he would have some trick or some lead to give us as to why this might be so different.

Since 1964 we have adopted a somewhat different technic for repairing the complete A-V canal. (Slide) Dr. Rastelli of our group has identified three different types of complete A-V canal. Fortunately, the most common is also the most ideal to repair, and in this situation the mitral portion of the leaflet is attached to the underlying septum by means of chordae. We repaired these defects entirely through the atrium, having abandoned the separate ventriculotomy, and placed a pericardial or Teflon patch along the right aspect of the ventricular septum below, leaving all of the mitral tissue, or this common leaflet tissue, on the mitral side.

In closing, I would again like to ask Dr. Gerbode if he has any lead as to why we have residual mitral insufficiency in a high incidence in the partial A-V canal group, and why our mortality rates in the complete canal group have not totally approached his. Thank you. (Applause)

Dr. Denton A. Cooley (Houston): Dr. Gerbode's results with surgical treatment of endocardial cushion defects have been better than ours. I suspect this may be due to a discrepancy in anatomic classification of the partial and complete types of A-V canal and to the large number of patients in our series less than 2 years of age who had severe or extensive canal defect.

During a 10-year period we operated upon 133 patients with endocardial cushion defects. Ninety-nine were partial canals (ostium primum with cleft mitral valve) with a 6% mortality. In 34 patients with a complete canal (atrioventricularis communis), surgical mortality was 56%. Mortality in patients less than 3 years of age was 91% and in those older than 3, it was 41%.

Because of our dissatisfaction with standard methods of surgical correction, we recently performed a more radical and definitive type of repair for complete A-V canal. The operation consists of excision of the remnants of mitral valve following which the atrioventricular septal defect is repaired with a Dacron patch and the mitral valve replaced by a low profile discoid prosthesis. The tricuspid valve is sutured when possible.

This operation was successfully accomplished in three patients ages 10, 4 and 13 years. The first operation was performed in August 1966, and the most recent only 2 months ago. We believe that this new radical repair may serve to improve the results of surgery for complete A-V canal. Undoubtedly a major cause of morbidity and mortality following the usual repair of the deformed mitral valve has been residual mitral incompetence and a prosthetic replacement provides the only dependable and predictable solution to this problem.

I will close by complimenting Dr. Gerbode and his associates on their outstanding results.

DR. HARRIS B. SHUMACKER, JR. (Indianapolis): Certainly the contributions which Dr. Gerbode has made have been most helpful to all of us. I should like to mention one very rare complication related to the repair of ostium primum defects; namely, persistent hemoglobinemia and hemoglobinuria. One young lady was readmitted to our hospital with marked anemia and mulberry-colored urine 8 months after surgical repair. Cardiac catheterization and angiography revealed moderate mitral insufficiency and an intact septum.

At the time of her second operation, it was apparent from digital palpation through the right atrial appendage that there were a number of jet-like points of mitral regurgitation. When the valve was inspected, the repair of the cleft was intact except for one area perhaps 2 mm. long. There were a number of small fenestrations in both leaflets and some of the leak occurred through the mitral orifice itself.

Her valve was excised and a ball valve prosthesis was inserted. Unfortunately, as was true in one of Dr. Cooley's cases, a block which necessitated the use of a pacemaker developed. During the first part of the operation her urine was mulberry colored and the free hemoglobin content 27.2 mg./100 ml. Toward the end of the operation, the urine was only mildly discolored and the hemoglobin 13.6 mg./100 ml. By 1:30 p.m. that afternoon, there was only 0.24 mg./100 ml. of free hemoglobin in the urine. Her urine has remained completely free of hemoglobin since and she has remained well.

DR. FRANK GERBODE (Closing): I would like to thank the discussants.

In reply to Dr. McGoon's question, I just don't know the answer. I'm sure he's a better surgeon than I, and therefore he should get better results.

The question of mitral insufficiency which he and others have raised—I mentioned that approximately 72% of our postoperative patients have systolic murmurs. There are very few of these who have systolic murmurs of mitral insufficiency who have dynamically significant incompetence. We have recatheterized many of these patients.

There were two patients who required valve replacements in the entire series. One was the patient with a large septum primum defect whom I mentioned previously. He had hugely dilated tricuspid and mitral annuli, and no other valve deformities. Both the mitral and tricuspid valves had to be replaced some years after the initial operation. The second patient was a 56-year-old man who had mitral valve replacement after a primary repair of a congenital cleft mitral valve which was not successful.

We haven't used prosthetic valves in small children. I have studiously avoided doing this, because I don't think in the long run this is going to be a satisfactory answer, although I recognize the necessity in some instances of having to do it. Our repairs have been satisfactory.

Heart block has been a major cause of fatality in some reported series. In our series we have had one complete and permanent heart block in the last 49 consecutive cases; this girl has a rate now of 50, and does not require a pacemaker. Thank you very much. (Applause)