Interrupted of the Aortic Arch: Experience in 17 Infants

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ABSTRACT Between April, 1965, and August, 1982, 17 children ranging in age from 2 days to 4 years were identified as having interruption of the aortic arch and operated on at our institution. There were eight type A interruptions, eight type B interruptions, and one type C interruption. Associated intracardiac anomalies were present in all but 3 patients. These 3 children, who all had type A interruption, underwent repair by mobilization of the aorta and end-to-end reconstruction. The other 14 children had initial palliative operations. The 2 patients in Group 1 had type A interruption with associated ventricular septal defect (VSD), and underwent subclavian artery-aorta anastomosis. In Group 2, the palliative procedure consisted of placement of a Dacron tube graft in 1 patient with type A interruption and associated VSD, and placement of a polytetrafluoroethylene (PTFE) graft, division of the patent ductus arteriosus, and banding of the pulmonary arteries in 11 patients—2 with type A, 8 with type B, and 1 with type C interruption. Ten children (71%) survived initial palliation, 1 of the 2 in Group 1 and 9 of the 12 in Group 2. In Group 2, 5 children had interruption of the aortic arch (4, type B; 1, type C) with associated VSD; among the 4 who survived palliation, 3 subsequently have had successful closure of the VSD and 1 is awaiting closure. Among the patients who had palliative procedures, there are 6 long-term survivors (43%). In the total series, there are 9 long-term survivors (53%).

We conclude that children with isolated type A interruption can undergo repair with mobilization of the aorta and end-to-end reconstruction, while children with interruption of the aortic arch and associated intracardiac anomalies can be palliated initially with PTFE reconstruction of the aorta, division of the patent ductus arteriosus, and pulmonary artery banding. Total repair is dependent on the severity of the associated anomaly. Patients with less complicated intracardiac anomalies, such as isolated VSD, have excellent long-term survival.

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Interrupted of the aortic arch encompasses 1.4% of congenital cardiac anomalies [1] and, if unrecognized or untreated, is associated with an 80% mortality in the first month of life [2]. Anatomical clarification of this defect was made by Celoria and Patton [3] in 1959 and was based on patterns of partial or complete failure of formation of the fetal aortic arches. Pathophysologically this was attributed to abnormal blood flows through intracardiac shunts that resulted in little blood flowing through the aorta. Without the stimulus of blood flow, the aortic arch fails to form. By this classification, three primary types of arch interruptions can be identified. In type A, there is interruption or atresia of the aorta distal to the left subclavian artery. In type B, the interruption is between the left carotid and left subclavian arteries. In type C, the interruption is between the innominate and left carotid arteries (Fig 1).

Medical therapy in general has a lethal outcome, and operative therapy has emerged as the primary form of treatment of this defect. Controversy has arisen, however, as to the optimum surgical approach. Whether initial palliation or attempted early repair should be undertaken remains an issue. Despite earlier diagnosis of the defect and earlier operative intervention, surgical mortality continues to be high.

We have reviewed our experience with this entity and suggest that immediate operative intervention be undertaken with the goals of reestablishment of aortic continuity, closure of the patent ductus arteriosus, and palliation of associated intracardiac defects if present. Operative repair of these defects then can be undertaken on an elective basis.

Material and Methods

Between April, 1965, and August, 1982, at the University of Oklahoma Health Sciences Center, 17 children were identified as having atresia or interruption of the aortic arch. There were 10 boys and 7 girls, ranging in age from 2 days to 4 years. The most common presentation was congestive heart failure with associated respiratory distress and poor feeding. None of the infants exhibited differential cyanosis. Two of them had associated DiGeorge's syndrome.

Eight of the children were identified as having interruption or atresia of the aortic arch distal to the left subclavian artery (type A). Eight had interruption between the left carotid and left subclavian arteries (type B), and 1 child had interruption between the innominate and left carotid arteries (type C) (Table 1). Associated intracar-
Coronary anomalies were present in all but 3 children; these 3 had type A interruption. Ventricular septal defect (VSD) was the most commonly identified intracardiac anomaly and was seen as an isolated defect in 7 of these infants—3 with type A, 3 with type B, and 1 with type C interruption. Other coexisting intracardiac defects included truncus arteriosus, transposition of the great vessels with tricuspid atresia and single ventricle, double outlet right ventricle with atrial septal defect, and complete endocardial cushion defect (see Table 1).

The 3 children with type A interruption and no intracardiac defects underwent repair at initial operation with mobilization and end-to-end reconstruction of the aorta; Patient 1 also had division of the patent ductus arteriosus. The remaining 14 infants had initial palliative operations. These patients were divided into two groups. The 2 patients in Group 1 (Nos. 5 and 7) had anastomosis of the left subclavian artery to the aorta; both had type A interruption with associated VSD. The 12 in Group 2 had prosthetic reconstruction of the aorta (in recent experience, with expanded polytetrafluoroethylene [PTFE] tube graft), division of the patent ductus arteriosus, and banding of the pulmonary arteries in those with increased pulmonary blood flow. Group 2 included 3 patients with type A interruption, 8 with type B, and 1 with type C (see Table 1).

Operative Procedure

Prior to operation, all infants with the suspected diagnosis of aortic arch interruption have biplane cineangiography to define the aortic anatomy and extent of associated intracardiac defects. Once the diagnosis is established, the infant is immediately taken to the operating room. Infants who have marked lower-extremity hypotension and acidosis are given a constant infusion of prostaglandin E₁, which is continued until the ductus is divided.

Operation is performed through a lateral thoracotomy on the side of the descending aorta. Once the chest has been entered, the pleura and pericardium are opened and the anatomy is identified. Ascending aorta, descending aorta, and ductus arteriosus are mobilized. If interruption is present distal to the left subclavian artery (type A), either mobilization of the aorta with end-to-end reconstruction or end-to-side anastomosis of the divided left subclavian artery to the aorta is performed. Then the patent ductus arteriosus is divided, and the ends are oversewn. Prosthetic reconstruction of the aorta is an alternative to use of the subclavian artery.

When interruption is between the left carotid and left subclavian arteries (type B) or between the innominate and left carotid arteries (type C), interposition of a PTFE tube graft is undertaken. In general, we have tried to use the largest graft that the aorta can accommodate. In neonates as well as infants less than 1 month old, this has been a 6 mm graft; an 8 mm graft was used in a 2½-month-old infant (Patient 13; see Table 1). The graft is anastomosed in an end-to-side fashion to the ascending aorta using a partially occluding clamp and continuous
Table 1. Initial Operative Procedures in 17 Infants with Interruption of the Aortic Arch

<table>
<thead>
<tr>
<th>Patient No., Age, Sex</th>
<th>Type of Interruption*</th>
<th>Associated Cardiac Defect(s)</th>
<th>Operations</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 3½ mo, F</td>
<td>A</td>
<td>None</td>
<td>Resection, end-to-end anastomosis, division of PDA</td>
<td>Survived</td>
</tr>
<tr>
<td>2. 2 yr, M</td>
<td>A</td>
<td>None</td>
<td>Resection, end-to-end anastomosis</td>
<td>Survived</td>
</tr>
<tr>
<td>3. 2 mo, F</td>
<td>A</td>
<td>None</td>
<td>Resection, end-to-end anastomosis</td>
<td>Survived</td>
</tr>
<tr>
<td>4. 4 yr, F</td>
<td>A</td>
<td>VSD</td>
<td>11 mm Dacron tube, division of PDA</td>
<td>Survived</td>
</tr>
<tr>
<td>5. 3 days, M</td>
<td>A</td>
<td>VSD</td>
<td>Left subclavian artery—aorta anastomosis</td>
<td>Died</td>
</tr>
<tr>
<td>6. 8 days, F</td>
<td>A</td>
<td>TOGV, tricuspid atresia, single ventricle</td>
<td>6 mm Dacron tube, division of PDA, PAB</td>
<td>Died</td>
</tr>
<tr>
<td>7. 3 mo, F</td>
<td>A</td>
<td>VSD</td>
<td>Left subclavian artery—aorta anastomosis</td>
<td>Survived</td>
</tr>
<tr>
<td>8. 4 mo, M</td>
<td>A</td>
<td>Truncus arteriosus</td>
<td>6 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>9. 2 wk, M</td>
<td>B</td>
<td>VSD, aberrant left subclavian artery</td>
<td>6 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Died</td>
</tr>
<tr>
<td>10. 2 days, F</td>
<td>B</td>
<td>VSD</td>
<td>6 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>11. 2 days, F</td>
<td>B</td>
<td>VSD, aberrant right subclavian artery</td>
<td>6 mm Impra (PTFE) tube, division of PDA</td>
<td>Survived</td>
</tr>
<tr>
<td>12. 1 mo, M</td>
<td>B</td>
<td>Truncus arteriosus, VSD</td>
<td>6 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Died</td>
</tr>
<tr>
<td>13. 2½ mo, M</td>
<td>B</td>
<td>VSD</td>
<td>8 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>14. 4 days, M</td>
<td>B</td>
<td>VSD</td>
<td>6 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>15. 4 days, F</td>
<td>B</td>
<td>Endocardial cushion defect, aberrant right subclavian artery</td>
<td>6 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>16. 14 days, M</td>
<td>B</td>
<td>Double outlet right ventricle, atrial septal defect</td>
<td>6 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>17. 8 days, F</td>
<td>C</td>
<td>VSD</td>
<td>6 mm Impra (PTFE) tube, division of PDA, PAB</td>
<td>Survived</td>
</tr>
</tbody>
</table>

*Type A = interruption distal to the left subclavian artery; type B = interruption between the left carotid and left subclavian arteries; type C = interruption between the innominate and left carotid arteries.

TOGV = transposition of the great vessels; VSD = ventricular septal defect; PDA = patent ductus arteriosus; PAB = pulmonary artery banding; PTFE = polytetrafluoroethylene.

Follow-up of all survivors has been obtained through periodic office examination. Repeat catheterization was done by two of us (J. D. R. and W. M. T.).

Results
All 3 infants with type A interruption of the aortic arch and without associated cardiac defects survived the repair procedure. Of the 14 infants who had initial palliative procedures, 10 (71%) survived the first operation—1 of the 2 infants in Group 1 and 9 of the 12 infants in Group 2.

There were no early deaths (within 30 days postopera-
Fig 2. Steps of palliative procedure: (A) Performance of lateral thoracotomy on the side of the descending aorta. (B) Identification of type of aortic interruption. (C) Interposition of polytetrafluoroethylene conduit end-to-side between the ascending and descending aorta. (D) Division and oversewing of the patent ductus arteriosus. (E) Banding of the main pulmonary artery.
Table 2. Patients Undergoing a Second Operation for Repair of Associated Cardiac Defects

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age at Second Procedure</th>
<th>Type of Interruption*</th>
<th>Associated Cardiac Defect</th>
<th>Operations</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>16 mo</td>
<td>A</td>
<td>Truncus arteriosus</td>
<td>Patch closure of VSD, valved conduit</td>
<td>Died</td>
</tr>
<tr>
<td>10</td>
<td>14 mo</td>
<td>B</td>
<td>VSD</td>
<td>Patch closure of VSD, removal of PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>13</td>
<td>2½ yr</td>
<td>B</td>
<td>VSD</td>
<td>Patch closure of VSD, removal of PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>14</td>
<td>1 yr</td>
<td>B</td>
<td>VSD</td>
<td>Patch closure of VSD, removal of PAB</td>
<td>Survived</td>
</tr>
<tr>
<td>15</td>
<td>3½ mo</td>
<td>B</td>
<td>Endocardial cushion defect</td>
<td>Patch closure of ASD and VSD</td>
<td>Died</td>
</tr>
</tbody>
</table>

*Type A = interruption distal to the left subclavian artery; type B = interruption between the left carotid and left subclavian arteries. VSD = ventricular septal defect; PAB = pulmonary artery banding; ASD = atrial septal defect.

Slightly) among those who survived the initial operation. The single survivor in Group 1 (Patient 7) remains well 6 years following that procedure. Although a small VSD was present at the time of diagnosis, follow-up catheterization showed a left-to-right shunt of less than 1.5:1. In addition, only a 25 mm gradient was found between the ascending and descending aorta.

One child in Group 2 (Patient 4) required revision of a Dacron graft 13 years after the initial palliative procedure. A moderate-sized VSD was identified at initial catheterization, although repeat catheterization 2 years after the second operation demonstrated a left-to-right shunt of only 1.3:1. There was a 20 mm pressure gradient between the ascending and descending aorta. Among the survivors in Group 2, 1 infant died six weeks after palliation with persistent congestive heart failure, pneumonia, and pulmonary hypertension. Five of the 7 survivors in Group 2 with major intracardiac defects have had a second operation for the repair of those defects (Table 2). Three infants with type B interruption and isolated VSD have had patch closure of the VSD and debanding of the pulmonary artery and have survived. One infant with type A interruption and type I truncus arteriosus had conduit repair of the truncus at 16 months of age but died in the early postoperative period. One infant with type B interruption and complete endocardial cushion defect had repair of the latter at age 3½ months but died following repair of the cardiac defect (see Table 2). Another early survivor, a male infant with type B interruption, double outlet right ventricle, and DiGeorge’s syndrome, died of pulmonary sepsis before repair could be attempted. Consequently, 6 of the 14 infants who had palliative procedures (43%) are currently living.

Follow-up of the Group 2 survivors has ranged from 4 months to 15 years. Currently all children are normotensive with palpable distal pulses and without signs of congestive heart failure. Repeat catheterization in these children has shown good flow across the PTFE graft and a gradient of 20 mm Hg or less between the ascending and descending aorta.

Comment

The high mortality associated with unrecognized and untreated interruption of the aortic arch is a culmination of several coexisting pathological states. Because there is obstruction to the flow of blood through the aorta, left ventricular afterload remains consistently high, resulting in early ventricular failure. Flow to the lower extremities is through a large patent ductus arteriosus in almost all instances; as pulmonary vascular resistance falls in the first days of life and as the ductus begins to close, flow to the lower extremities decreases, resulting in hypoperfusion of the kidneys and lower extremities. Finally, because most of the infants have concomitant cardiac anomalies, abnormal intracardiac shunting and increased pulmonary blood flow contribute substantially to early ventricular failure. Most of these infants come to operation with severe metabolic and hemodynamic derangements. Any hope for survival depends on surgical treatment as soon as the diagnosis is established.

Early reports describing surgical repair of this defect dealt primarily with older infants and emphasized initial reconstruction of the aortic defect with later attempts at repair of the intracardiac anomalies [4–6]. As advances were made in the surgical treatment of congenital defects, there were several reports of successful treatment of this lesion in the neonatal period utilizing cardiopulmonary bypass and, in some instances, hypothermic arrest [7–10]. In general, mortality for these procedures has remained high. As the majority of these infants are seen in critical condition, it has been our impression that early palliation provides the best chance of survival.

The anatomy of type A interruption of the aortic arch lends itself to reconstruction by either end-to-end anastomosis of the ascending aorta to the descending aorta or reconstruction utilizing the brachiocephalic vessels. In this series, the use of the subclavian artery–aorta
anastomosis as described by Blalock and Park [11] has been preferred. Although follow-up of this type of reconstruction has been limited to 1 survivor in our series, repeat catheterization has revealed only a 22 mm gradient from the ascending to the descending aorta. In type B and type C interruption, the distance between the ascending and descending aorta tends to preclude direct anastomosis. In these instances, a synthetic conduit has been employed. Early in the present series, a Dacron tube was used, but after reports appeared on the use of expanded PTFE grafts in infants for central shunts and other conduits, this became the preferred material for aortic reconstruction [12]. Arguments have been raised that the use of synthetic material necessitates future operation for relief of this artificial coarctation. Reports by Sturm [13], Braunlin [14] and their colleagues of the use of PTFE conduits in interruption of the aortic arch, however, have revealed no pronounced gradient across the conduit in at least 24 months of follow-up. Follow-up cardiac catheterization of the survivors in our series, which has ranged from 6 months to 2½ years, also has shown no major gradient across the PTFE graft. Examination has demonstrated the presence of palpable distal pulses in all survivors.

In infants with severe intracardiac shunting, increased pulmonary blood flow must be modified in order to prevent continued congestive heart failure as well as to decrease the risk of irreversible pulmonary hypertension. One infant in this series (No. 1) did not have pulmonary artery banding at the original operation. Postoperatively, she continued to exhibit signs of severe congestive heart failure with associated respiratory failure, and despite late banding of the pulmonary artery, she died with persistent failure and pneumonia. Postmortem examination revealed changes of marked pulmonary hypertension. For this reason, pulmonary artery banding has become a necessary addition to the initial palliative procedure.

Because blood flow to the distal aorta is dependent on flow through the ductus, closure of the ductus results in notable tissue hypoxemia, worsening metabolic acidemia, and ultimately death. The use of prostaglandin E1 to maintain ductal patency in cyanotic heart disease has been extensively described [15]. In cases of aortic interruption, return of pulsatile flow to the descending aorta with hemodynamic and metabolic improvement has been reported [16]. In view of this, we think that it is important to begin a constant infusion of prostaglandin at the time the diagnosis is established in those infants with signs of lower-extremity hypofusion and acidosis.

The fact that early survival was achieved in 71% of the infants in our series following palliation argues favorably for this approach. The major factor limiting both early and late survival in these infants, however, has been the presence of associated intracardiac anomalies. Four of the 5 infants who had type B or type C interruption and isolated VSD survived initial palliation; 3 have had subsequent patch closure of the VSD with removal of the pulmonary artery band, and the remaining patient is awaiting closure of the VSD. In this series, the infants with more complex intracardiac lesions either did not survive palliation or died following the second repair procedure.

The association of aortic arch anomalies and DiGeorge's syndrome has been reported previously [17]. Two infants (Patients 14 and 16) in this series were noted to have thymic aplasia and abnormal lymphocytes. Both survived initial palliation, and 1 survived subsequent repair of an associated VSD. The other infant, however, died of persistent pulmonary sepsis several months after palliation before attempts at repair of the associated cardiac defects could be done.

The current long-term survival of 53% for the entire series and 43% for those infants who had palliative operations compares favorably with other small series reporting the results of both palliation and early single-stage repair for aortic arch interruption. In view of this, we believe that our current approach is a good alternative to attempted early single-stage repair. The procedure is applicable to neonates and young infants, and it provides a period of growth prior to attempted repair of the associated intracardiac defects.

Addendum

The child awaiting VSD closure (Patient 13) has undergone total repair.

References


Discussion

DR. GEORGE G. LINDESMITH (Los Angeles, CA): I compliment the authors on their outstanding results in the management of this difficult anomaly.

In the last five years we have operated on 17 children for aortic arch interruption. Sixteen were less than 1 month of age, so this was a somewhat younger group than the series of Fowler and colleagues. All had intracardiac defects as well as patent ductus arteriosus.

Five children had type A arch interruption and 12, type B: there were no patients with type C interruption in this series. Interestingly, in the children with type B interruption, both subclavian arteries arose from the distal segment of the aorta in 5 patients and the right subclavian artery was retroesophageal.

Our repair included direct anastomosis in 6 patients. The subclavian artery was used for repair in 7 patients and Gore-Tex grafts, in 4. All survivors had concomitant pulmonary artery banding.

Thirteen patients survived the immediate surgical event, but 7 of them died later. Three deaths occurred more than 2 months postoperatively: 1 of apparent arrhythmia, 1 of reeking pulmonary problems despite adequate repair (this child had inadequate T-cell function), and 1 of severe pulmonary hypertension during attempted VSD closure. The last child mentioned was 1 of 2 patients who did not have banding at the time of initial repair.

Currently, we too favor the use of expanded Teflon grafts to bridge the arch interruption except in those few patients in whom the size of the subclavian artery is unquestionably adequate to provide satisfactory bridging of the defect. We agree that pulmonary artery banding should be accomplished at the time of repair if intracardiac lesions are producing increased pulmonary flow. We routinely use 1 mg per kilogram of body weight of heparin prior to clamping any vessels and during the anastomosis. Dr. Fowler, have you had any experience with the use of heparin in these situations?

In patients stabilized with prostaglandin prior to operation, we have elected to leave the ductus undivided. Prostaglandin is terminated electively after the patient becomes stable in the intensive care unit. In our experience, this has resulted in ductal closure. Would you comment on whether you have considered this maneuver?

Again, I compliment Dr. Fowler and his co-workers on their excellent series, and I hope that our current approach to this problem, which seems to approximate theirs, will allow us to obtain similar results in the next four to five years.

DR. JOHN G. JACOBSON (Loma Linda, CA): Dr. Fowler, I appreciate the work you and your colleagues did in compiling this large series of patients treated surgically for such a lethal condition. At our institution we have a different philosophy: we prefer to perform total repair of all anomalies and to avoid placement of nonovergrowing conduits. Applying this philosophy, my colleague, Dr. Leonard L. Bailey, and I have had experience with 9 newborn infants who had either type A or type B interrupted aortic arch complex. Six of these infants had what we term a simple interrupted aortic arch complex in which VSD, or ASD, or both were the associated intracardiac anomaly. Three of the infants had more complex intracardiac disease; 1 had dextrotransposition with VSD, another had a double outlet right ventricle, and a third had type II truncus arteriosus.

We have utilized bilateral thoracotomy, which gives adequate exposure for mobilization of the descending aorta to the level of the diaphragm, and this permits easy anastomosis of the descending aorta to the ascending aorta. All of these repairs were done with the patient under deep hypothermia and circulatory arrest. Five out of the 9 infants (56%) have survived. Four out of 6 of those with simple interrupted aortic arch complexes, that is, interrupted arch and VSD, have survived (67%). One out of 3 of the infants with complex interruption has survived (33%); this was a patient who had a Mustard procedure, VSD closure, and primary repair of the aorta. Four of the 5 patients have been reevaluated and show a growing aortic anastomosis.

Dr. Fowler, did the 3 type A patients with no associated intracardiac disease have any fibrous connection between the aortic arch and the descending aorta? And in the type C patient, was the ascending aorta adequate for anastomosis of the graft?

DR. ANTHONY L. MOULTON (Baltimore, MD): Dr. Fowler and his associates are to be congratulated on their results in this difficult group of patients. I wish to focus attention on the subset of 8 patients with type B interrupted aortic arch, and I echo the sentiments of the Loma Linda group in support of primary repair.

In this series, there were 5 patients with type B interrupted arch and an isolated VSD. One patient (20%) died during initial palliation with a prosthetic arch repair and pulmonary artery banding, but only 3 of the 4 survivors (60% of the initial group) have undergone definitive repair. A year and a half ago my colleagues and I presented our results with single-stage definitive repair of the lesion with resection of all identifiable ductal tissue, primary arch reconstruction, and closure of the VSD. In our experience, this has been achieved easily through a median sternotomy without sacrifice of any arch or intercostal vessels. In a collective series that included the early patients from the Loma Linda series, the initial mortality with this approach was 31%; the updated results from Dr. Bailey's group show further improvements. Fifty-eight percent of all patients are surviving two to ten years later, with evidence of growth of the reconstructed segment and gratifying long-term results.

Therefore, we continue to advocate primary definitive repair in the subset of patients with interrupted aortic arch and simple VSD to avoid the potential problems of a prosthetic graft and the well-known difficulties with banding of the pulmonary artery. In patients with more complex associated intracardiac lesions, our results with primary repair have been disappointing. In such instances, the staged approach outlined by Dr. Fowler and his associates probably still plays a role.
DR. JOHN W. BROWN (Indianapolis, IN): I compliment Dr. Fowler and his group on a fine presentation and a nice series.

We agree with the authors' staged approach to newborns with interrupted aortic arch, but we believe that our results leave much to be desired. We prefer a native arterial connection between the two segments of the aorta whenever possible. In 6 of our patients, this was impossible and we had to use a PTFE graft. We had 2 early and 2 late deaths in the PTFE group. These deaths were not related to use of PTFE grafts but to associated anomalies.

There are a few technical considerations in using PTFE that I think are important. One was conveyed to me by my colleague, Dr. Edward Bove of Syracuse, NY. A 5 or 6 mm Gore-Tex graft frequently will be outgrown, and a gradient will develop across it within two years. Whenever possible, we favor using an 8 or 10 mm Gore-Tex graft sewn to the spatulated undersurface of the transverse aortic arch. Unstented PTFE occasionally will kink at the apex of the chest; thus, we favor externally stented PTFE for interrupted arch repair.

When use of native artery seems desirable and the two segments of the aorta cannot be joined, we divide the left carotid or the left subclavian artery or both vessels. We have not had any complications with this approach. Both arteries can be sacrificed without neurological sequelae. This technique is most applicable to type A lesion in which both the subclavian and the carotid can be sewn together and swung down in the manner described by Blalock and Park. We also have used this method for repair of type B interruption. We have not observed any neurological deficits in this group of patients.

DR. FOWLER: I thank the discussants for their kind remarks.

Dr. Lindesmith, my colleagues and I also have had 1 late death related to immunological deficiency in a baby with thyroid aplasia and lymphocyte abnormality. I mention this to emphasize the known association between DiGeorge's syndrome and arch abnormalities; this should be kept in mind when one is dealing with these infants. In response to your other questions, we do not heparinize prior to placement of the cross-clamp and in general we discontinue the prostaglandin infusion at the time the ductus is divided rather than relying on closure of the ductus in the postoperative period by withdrawing prostaglandin.

Dr. Jacobson, there were no fibrous connections between the ends of the aorta in the infants with type A interruption. In the infant with type C interruption, the aorta was more than adequate to allow placement of a partially occluding clamp.

Dr. Moulton, we agree that arch interruption with isolated VSD is usually associated with better survival. In this series, there were 9 babies who had arch interruption and isolated VSD; of that group, 6 are long-term survivors.

Erratum

The Annals of Thoracic Surgery regrets that an error appeared in a recent article by Dr. James W. Battaglini and colleagues (Intractable Dysphagia following Placement of Angelchik Prosthesis for Reflux Esophagitis; Ann Thorac Surg 35:551, 1983). The preoperative barium swallow on page 552 was printed upside down.