SURGICAL MANAGEMENT OF NEONATAL COARCTATION

Between 1983 and 1994, 307 consecutive neonates underwent coarctation repair by a single surgical technique: extended end-to-end anastomosis. Mean age at operation was 13 ± 8 days. Isolated coarctation was present in 95 patients (group 1), 102 patients had associated ventricular septal defect (group 2), and 110 patients had associated complex intracardiac lesions (group 3). Aortic arch hypoplasia was present in 81% of the patients (62% in group 1 versus 85% in group 2 and 93% in group 3: $p < 0.001$). In 271 patients, the aortic arch reconstruction was performed via a left thoracotomy with normothermia (100% of group 1, 95% of group 2, and 72% of group 3); in the other 36 patients, undergoing one-stage repair or palliation of the associated lesion, it was performed via a midline sternotomy during a short period of deep hypothermia and circulatory arrest (5% of group 2 and 28% of group 3). Pulmonary artery banding was performed in 94 patients. Spontaneous ventricular septal defect closure was observed in 39% of the patients of group 2 operated on via thoracotomy. Early mortality rates in groups 1 (2%) and 2 (2%) were significantly lower than in group 3 (17%) ($p < 0.001$). There were 29 late deaths, all related to associated cardiac lesions or their subsequent repair. The overall total mortality was 16.9%. In group 3 this rate was significantly higher in patients undergoing two-stage procedures (47%) than in those undergoing one-stage repair (23%) ($p < 0.05$). All but 14 survivors were followed up for a mean of 61 ± 36 months. Actuarial survivals at 10 years were 98% in group 1, 94% in group 2, and 60% in group 3. The recoarctation rate was 9.8%, leading to 21 reoperations and three angioplasties without mortality. Patients with a more extended or severe form of aortic arch hypoplasia had a significantly higher risk of recoarctation ($p < 0.001$). Actuarial freedom from reoperation for recoarctation at 10 years was 93%. The findings of this study suggest that extended end-to-end anastomosis provides an adequate and safe repair of neonatal coarctation. Low recoarctation rate, owing to effective relief of the obstruction created by aortic arch hypoplasia and to complete resection of ductal tissue, freedom from major morbidity, and feasibility via both lateral and anterior approaches are the main advantages of the extended end-to-end anastomosis. Mortality is mainly dependent on the complexity of the cardiac associations. Successful management of the majority of the neonates with coarctation and associated ventricular septal defect is possible with repair of coarctation alone. One-stage repair of neonatal coarctation and associated complex heart defects (with indication for two-ventricle repair) by means of an anterior approach provides a better outcome than a two-stage repair. (J THORAC CARDIOVASC SURG 1995;109:663-75)

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The results of the surgical management of neonatal coarctation have constantly been improved since Mustard and associates¹ performed the first successful repair in 1953. The introduction of the preoperative use of prostaglandin E₁² and the development of a surgical technique providing an effective reconstruction of the entire aortic arch,³⁻⁶ often hypoplastic in symptomatic neonates with aortic coarctation,⁷⁻¹¹ have been extremely important. Since 1983 we¹² have tried to correct neonatal

coarctation by a single surgical procedure either via thoracotomy or via sternotomy (when repair of associated intracardiac lesions was attempted in the mean stage): resection and extended end-to-end anastomosis. The purpose of this paper is to review our 11 years of experience with aortic coarctation repair in 307 neonates treated by extended end-to-end anastomosis.

Patients and methods

Definitions. The *aortic arch* was defined as lying between the brachiocephalic artery and the ductus arteriosus and as comprising three segments: the *proximal transverse arch*, between the brachiocephalic and left carotid arteries, the *distal transverse arch*, between the left carotid and left subclavian arteries, and the *isthmus*, between the left subclavian artery and the ductus arteriosus. Anatomic features of the aortic arch were carefully evaluated by both the cardiologist at the time of diagnosis and the surgeon at the time of operation. The criterion for defining *aortic arch hypoplasia* was a diameter of proximal transverse arch, distal transverse arch, and isthmus less than, respectively, 60%, 50%, and 40% of that of the ascending aorta.¹³ Three types of aortic arch hypoplasia were defined according to the location: *distal aortic arch hypoplasia* (hypoplasia of distal transverse arch and isthmus), *complete aortic arch hypoplasia* (hypoplasia of the entire aortic arch), and *complex aortic arch hypoplasia* (very short proximal arch associated with a very long and hypoplastic distal transverse arch and isthmus).

With regard to the associated anomalies, any heart defect having a surgical indication was considered as a *complex intracardiac lesion* if other than a ventricular septal defect (VSD). *Early mortality* was calculated as the rate of deaths within the first 30 postoperative days or before hospital discharge. Any postoperative stenosis in the repair area giving a resting peak pressure gradient of greater than 20 mm Hg at Doppler was considered as a *recoarctation*.¹⁰ More precisely, it was defined as *residual coarctation* when the gradient was detected during the postoperative hospital stay or as *recurrent coarctation* when the gradient was detected after discharge from the hospital. Any postoperative *respiratory failure*, *left ventricular dysfunction*, and *blood hypertension* necessitating, respectively, ventilatory support, inotropic support, and medical treatment for more than 15 days were defined as *persistent*.

Patient population. Between 1983 and 1994, 307 consecutive neonates (≤ 30 days old) underwent surgical repair of aortic coarctation by extended end-to-end anastomosis at the Marie Lannelongue Hospital, Paris. Patients in whom coarctation of the aorta was part of a true hypoplastic left heart syndrome (class IV)¹⁰ were not included. There were 191 boys and 116 girls. Mean age, weight, and body surface area at operation were, respectively, 13 ± 8 days, 3.2 ± 0.6 kg, and 0.20 ± 0.02 m². The indication for operation in all cases was congestive heart failure. Ductal patency was maintained preoperatively with prostaglandin E₁ infusion in 221 patients (72%), 168 of them in the last 5 years. The prevalence of prostag-

landin E₁ therapy has increased from 40% in the period 1983-1989 to 96% in the period 1990-1994 ($p < 0.001$). Preoperative ventilatory and inotropic supports were given in 150 (49%) and 111 (36%) patients, respectively. A correct fetal diagnosis was obtained in 28 patients (9%). Cardiac catheterization was performed in 74 patients (24%); in the remainder, the diagnosis was made clinically and confirmed with two-dimensional echocardiography ($p < 0.001$).

The patients were divided by their additional cardiovascular defects into groups 1 to 3 (Table I). Group 1 ($n = 95$) included those with isolated coarctation, group 2 ($n = 102$) those with associated VSD, and group 3 ($n = 110$) those with associated complex intracardiac lesions.

Aortic arch hypoplasia was present in 248 patients (81%): 59 in group 1 (62%), 87 in group 2 (85%), and 102 in group 3 (93%) (group 1 versus groups 2 or 3: $p < 0.001$). All three types of aortic arch hypoplasia were encountered: distal in 169 patients, complete in 59, and complex in 20. Complete and complex aortic arch hypoplasia were observed significantly more often in patients with associated major intracardiac lesions ($p < 0.001$) (Table II).

Operative technique. Since 1983 all the neonates referred for coarctation repair underwent aortic arch reconstruction by extended end-to-end anastomosis. Two surgical approaches were used: left posterolateral thoracotomy in the third intercostal space in 271 patients (88%) and midline sternotomy in 36 patients (12%).

Extended end-to-end anastomosis was performed via thoracotomy in all patients of the group 1 and in those in groups 2 or 3 considered for intracardiac repair in a second stage (see Table I). The technique was previously reported by our group.¹² The arterial pressure was monitored by means of a catheter in the right radial artery. Care was taken to keep the patient normothermic during the procedure. The entire thoracic aorta including all its branches was widely dissected to allow an extensive mobilization all over, from the ascending aorta proximally to the level of the fourth set of intercostal arteries distally. The ductus arteriosus was primarily divided between two ligations to allow more easily free dissection of proximal transverse arch and ascending aorta. Intercostal arteries were rarely divided. Aggressive proximal and distal mobilization is mandatory part of this technique to avoid any tension at the anastomosis (Fig. 1).

A curved vascular clamp of appropriate size was used to crossclamp the proximal transverse arch as proximally as possible, including the left carotid artery, while checking unobstructed forward flow to the innominate artery by a radial arterial line. The left subclavian artery was clamped as well, except in seven patients with a complex aortic arch hypoplasia, in whom it was either ligated before part of the distal transverse arch was resected or used as a flap to augment the top of the distal arch. In all the patients ($n = 4$) with anomalous origin of the right subclavian artery (from the upper descending aorta), this was ligated and resected. Another curved vascular clamp was used to crossclamp the descending aorta at the level of the first-second set of intercostal arteries. After placement of both proximal and distal clamps, the coarctation was

Table I. Neonatal coarctation and associated cardiac anomalies: Type of surgical approach

Anomaly	No. (rate)	Thoracotomy No. (rate)	Sternotomy No. (rate)
Group 1			
Isolated coarctation	95 (31%)	95 (100%)	0
Group 2			
Coarctation with VSD	102 (33%)	97 (95%) (48 PAB)	5 (5%)
Single VSD	76	71 (22 PAB)	5
Multiple VSD	26	26 (26 PAB)	—
Group 3			
Coarctation with complex heart defect	110 (36%)	79 (72%) (46 PAB)	31 (28%)
TGA or DORV + VSD	59	35 (32 PAB)	24
TGA + IVS	12	9	3
UH	10	7 (7 PAB)	3
HLV	7	7	—
Aortic valve stenosis	6	6	—
Aortic + mitral valve stenosis	6	6	—
CTGA + VSD	4	4 (4 PAB)	—
CAVSD	3	3 (3 PAB)	—
Mitral regurgitation	2	2	—
Aortopulmonary window	1	—	1

VSD, Ventricular septal defect; TGA, transposition of the great arteries; DORV, double-outlet right ventricle; IVS, intact ventricular septum; UH, univentricular heart; HLV, hypoplastic left ventricle; CTGA, corrected transposition of the great arteries; CAVSD, complete atrioventricular septal defect; PAB, pulmonary artery banding.

widely resected, with removal of any ductal tissue. A longitudinal incision was made in the undersurface of the arch and extended proximal to the origin of the left carotid artery. The extension of this incision was more limited when aortic arch hypoplasia was absent (19%). An incision was then made in the posterolateral aspect of the descending aorta so that the diameter in the two aortic ends would be equal (Fig. 2).

The anastomosis was performed with continuous 6-0 polypropylene suture. It was begun proximally on the arch with all the loops of the posterior aspect inserted before the two clamps were approximated, the suture tightened, and the anterior aspect completed (Fig. 3). The mean crossclamp time was 22 ± 10 minutes (range 14 to 38 minutes) and the mean postoperative gradient was 5 ± 6 mm Hg (range 0 to 24 mm Hg).

Pulmonary artery banding (PAB) was performed as an additional procedure in 94 of 176 patients of groups 2 and 3 undergoing extended end-to-end anastomosis via thoracotomy (53%). In group 2, PAB was performed in all the patients with multiple VSDs and in just 31% of those with a single VSD. Spontaneous VSD closure was observed with time in 39% of the patients of group 2 operated on via thoracotomy (38/97).

Extended end-to-end anastomosis was performed through a sternotomy in five patients of group 2 and in 31 of group 3, undergoing one-stage repair or palliation of the associated lesion (see Table I). After the institution of cardiopulmonary bypass (CPB), the aortic arch was first reconstructed during a short period of deep hypothermia and total circulatory arrest according to the aforementioned principles. The mean circulatory arrest time was 31 ± 11 minutes (range 19 to 58 minutes) and the mean postoperative gradient was 6 ± 7.5 mm Hg (range 0 to 25 mm Hg). After completion of arch repair, the distal clamp

Table II. Distribution of the different types of aortic arch hypoplasia according to associated cardiac anomalies

Patient groups	Distal AAH (n = 169)	Complete AAH (n = 59)	Complex AAH (n = 20)
Group 1	51	8 (α)	0 (δ)
Group 2	56	23 (β)	8 (ϵ)
Group 3	62	28 (χ)	12 (ϕ)

Group 1, Isolated coarctation; group 2, coarctation with VSD; group 3, coarctation with complex heart defect; AAH, aortic arch hypoplasia. α (= 14%) versus $\beta + \chi$ (= 86%): $p < 0.001$; δ (= 0) versus $\epsilon + \phi$ (= 100%): $p < 0.001$.

and head vessel snares were removed and low-flow CPB was resumed. After deairing, flow was returned to normal and intracardiac repair followed.

In group 2, among the patients operated on via this approach, three had a very large VSD, two had a complex aortic arch hypoplasia, and one had both these anomalies.

In group 3, the anterior approach was used in 27 patients undergoing repair of transposition of the great arteries (with or without VSD) or double-outlet right ventricle (TGA-DORV) by arterial switch, in one patient undergoing aortopulmonary window repair, and in three patients undergoing palliation of univentricular heart with TGA and subaortic stenosis by arterial switch. Five of these patients had a complex aortic arch hypoplasia. Among the patients of group 3, the prevalence of one-stage repair has increased from 8% (5/61) in the period 1983-1989 to 53% (26/49) in the period 1990-1994 ($p < 0.001$).

Follow-up. Two hundred seventy of 284 hospital survivors (95%) were followed up for a mean of 61 ± 36

Fig. 1. Wide dissection and mobilization of the entire thoracic aorta after division of the ductus arteriosus.

months (range 3 months to 11 years). Follow-up data were obtained from the referring pediatric cardiologists. Routine echocardiographic studies, including two-dimensional echocardiography and Doppler assessment of pressure gradients, were carried out at varying intervals during follow-up. Patients with recoarctation underwent a complementary angiography.

Statistical analysis. Continuous data are presented as means $\pm$ standard deviation. Proportions are accompanied by their 95% confidence limits (CL). Statistical comparisons were evaluated by the χ^2 test and actuarial estimates were calculated with the Kaplan-Meier method.

Results

Mortality. The overall early mortality was 7.5% (23/307; CL = 4.5% to 10.4%). Early mortality rates in groups 1 (2%) or 2 (2%) were significantly lower than in group 3 (17%) ($p < 0.001$). The overall late mortality was 10.7% (29/270; CL = 7% to 14.4%), and significant differences still persisted between groups 1 (no late deaths) or 2 (4%) and group 3 (28%) ($p < 0.001$) (Table III).

In group 1, both early deaths occurred at the beginning of our experience and were caused by ventricular fibrillation after an acute declamping syndrome.

In group 2, one early death was due to low cardiac output related to biventricular obstruction in a patient with residual coarctation and PAB, and the other was due to congestive heart failure. In the same group all late deaths occurred at a second-stage VSD closure: one was related to an abnormal coronary artery injury, one was related to a "Swiss

Fig. 2. Resection of coarctation including all ductal tissue (A) and extended incision in the arch concavity (B) after placement of the clamps.

cheese type" multiple VSD closure, and two were sudden deaths. No deaths occurred among patients operated on in a single stage via sternotomy.

In group 3, only two early deaths were in relation to a residual coarctation; the other early deaths and all late deaths were in relation to the associated lesions or their subsequent repair.

The overall total mortality was 16.9% (52/307; CL 12.7% to 21.1%). In group 3 this rate significantly decreased from 49% in the period 1983-1989 to 29% in the period 1990-1994 ($p < 0.05$). In the same group, a significant difference was present as well between total mortality rates among patients undergoing two-stage (47%) and one-stage (23%) repair or palliation of the intracardiac defect ($p < 0.05$). Because the majority (87%) of the patients undergoing a one-stage procedure had a TGA-DORV repair, a comparison between one- and two-stage repairs in this last subgroup became mandatory: total mortality rate in patients undergoing two-stage repair (45%) was significantly higher than in patients undergoing one-stage repair (19%) ($p < 0.05$) (see Table III).

Survival. The overall actuarial probability of survival at 10 years after operation, including operative mortality, was $83\% \pm 4\%$. According to associated anomalies, 10-year actuarial survival rates were $98\% \pm 3\%$ in group 1, $94\% \pm 5\%$ in group 2, and $60\% \pm 9\%$ in group 3 (Fig. 4).

Fig. 3. The anastomosis is begun at the posterior aspect (A) and completed after the clamps had been approached and the suture tightened (B and C).

Table III. Mortality according to associated cardiac anomalies

Patients	Early mortality rate (no. of deaths)		Late mortality rate (no. of deaths)		Total mortality rate (no. of deaths)	
All ($n = 307$)	7.5%	(23)	10.7%	(29)	16.9%	(52)
Group 1 ($n = 95$)	2%	(2) (α^1)	0	(0) (α^2)	2%	(2) (α)
Group 2 ($n = 102$)	2%	(2) (β^1)	4%	(4) (β^2)	6%	(6) (β)
Group 3 ($n = 110$)	17%	(19) (χ^1)	28%	(25) (χ^2)	40%	(44) (χ)
Group 3						
1983-1989 ($n = 61$)					49%	(30) (δ)
1990-1994 ($n = 49$)					29%	(14) (ϵ)
Group 3						
Staged repair ($n = 79$)					47%	(37) (ϕ)
Primary repair ($n = 31$)					23%	(7) (γ)
TGA-DORV						
Staged repair ($n = 44$)					45%	(20) (η)
Primary repair ($n = 27$)					19%	(5) (λ)

Group 1, Isolated coarctation; group 2, coarctation with VSD; group 3, coarctation with complex heart defects; TGA, transposition of the great arteries; DORV, double-outlet right ventricle. α versus β or χ ; $p < 0.001$; α^1 versus β^1 or χ^1 , $p < 0.001$; α^2 versus β^2 or χ^2 , $p < 0.001$; δ versus ϵ , $p < 0.05$; ϕ versus γ , $p < 0.05$; η versus λ , $p < 0.05$.

Morbidity. Persistent respiratory failure occurred in 16 patients and persistent left ventricular dysfunction in 12. Fourteen patients having had an adequate coarctation repair had a persistent postoperative hypertension. Left phrenic nerve paralysis was detected in two patients who underwent additional PAB; in one it was reversible, but in the other it necessitated surgical diaphragm plication. Chylothorax was present in three patients, and one of them required thoracic duct ligation. One patient had thrombosis of the superior vena cava and required surgical management. Postoperative paraplegia or neurologic complications were not encountered.

Recoarctation. Thirty patients had a recoarctation (9.8%; CL 6.4% to 13.1%) after a mean time of 4 ± 3 months (range 1 to 11 months) from the repair. Recoarctation rates in patients operated on through a thoracotomy (10%) and through a sternotomy (8.3%) were not significantly different. All but one of the patients in whom a recoarctation developed initially had some form of aortic arch hypoplasia ($p < 0.001$).

Furthermore, recoarctation occurred in 12% of all the patients with aortic arch hypoplasia and in 2% of all those without aortic arch hypoplasia ($p < 0.05$). According to aortic arch hypoplasia types,

Fig. 4. Actuarial probability of survival according to associated cardiac anomalies. *CoA*, Coarctation; *VSD*, ventricular septal defect; *Complex CoA*, coarctation with associated complex heart defects.

Table IV. Influence of surgical approach, arch hypoplasia, and associated cardiac anomalies on recoarctation

Variable	Recoarctation no. (%)	
Thoracotomy	27 (10)	(α)
Sternotomy	3 (8)	(β)
Normal arch ($n = 59$)	1 (2)	(χ)
AAH ($n = 248$)	29 (12)	(δ)
Distal AAH ($n = 169$)	7 (4)	(ϵ)
Complete AAH ($n = 59$)	12 (20)	(ϕ)
Complex AAH ($n = 20$)	11 (55)	(γ)
Group 1 ($n = 95$)	3 (3)	(η)
Group 2 ($n = 102$)	13 (13)	(λ)
Group 3 ($n = 110$)	14 (13)	(μ)

AAH, Aortic arch hypoplasia; group 1, isolated coarctation; group 2, coarctation with VSD; group 3, coarctation with complex heart defects. α versus β , $p = \text{n.s.}$; χ versus δ , $p < 0.05$; ϵ versus ϕ or γ , $p < 0.001$; ϕ versus γ , $p < 0.01$; η versus λ or μ , $p < 0.05$.

recoarctation occurred in 4% of the patients with distal aortic arch hypoplasia, 20% of the patients with complete aortic arch hypoplasia, and 55% of the patients with complex aortic arch hypoplasia. Therefore patients with more extended aortic arch hypoplasia were at a significantly higher risk for recoarctation ($p < 0.001$). According to patient groups, recoarctation occurred in three patients in group 1 (3%), 13 patients in group 2 (13%), and 14

patients in group 3 (13%). Therefore patients with an associated major intracardiac lesion were at a significantly higher risk of recoarctation ($p < 0.05$) (Table IV). According to the timing of diagnosis, residual coarctation occurred in nine patients (2.9%; CL 1% to 4.8%) and it was attributed in four early cases to the lack of experience and in the remaining five to an extreme form of complex aortic arch hypoplasia. Recurrent coarctation was detected in 21 patients (7.8%; CL 4.6% to 11%); nine patients with recoarctation proximal to the anastomosis and therefore associated with an insufficient extension of the aortic arch incision, six patients in whom recoarctation was probably related to incomplete resection of ductal tissue, and six patients with a severe form of complex aortic arch hypoplasia.

The actuarial probability of freedom from recoarctation at 10 years was $90\% \pm 4\%$ (Fig. 5).

Twenty-four patients underwent successful reoperation ($n = 21$) or transfemoral balloon angioplasty ($n = 3$) for recoarctation 1 to 26 months (mean 8 ± 7 months) after the initial repair. Surgical procedures included extended end-to-end anastomosis in eight patients, extended end-to-end anastomosis plus subclavian artery resection in four, subclavian flap aortoplasty¹⁴ in four, extended end-to-end anastomosis plus reversed subclavian flap aortoplasty¹⁵ in three, and reversed subclavian flap aortoplasty in

Fig. 5. Actuarial probability of freedom from recoarctation and freedom from reoperation for recoarctation.

two. Of the 13 patients in whom recoarctation repair was performed by using subclavian artery resection or flap (anterograde or retrograde) aortoplasty, 10 patients (77%) had a complex aortic arch hypoplasia ($p < 0.02$).

There were no deaths or major complications at reoperation. Of the three patients who underwent balloon angioplasty, one later had an aortic aneurysm (33%).

Freedom from reoperation for recoarctation 10 years after the operation was $93\% \pm 3\%$ (see Fig. 5).

Discussion

Although the outcome of neonatal coarctation repair, as judged by mortality, morbidity, and recoarctation, has dramatically improved in the last years, optimal surgical management is still under debate. Main controversies are essentially two: the technique of choice according to the aortic arch anatomy and the surgical approach according to the repair of associated heart defects. The data presented in this retrospective study on the largest reported series of neonates undergoing coarctation repair revealed a number of interesting findings that specifically address such controversies.

Technique of choice. Some degree of aortic arch hypoplasia is often present in the neonate with

aortic coarctation.^{7, 9, 11} In the past, several autopsy reports emphasized the frequency of this association.^{8, 13, 16, 17} In this study aortic arch hypoplasia was present as an additional lesion in 81% of the patients. Significant prevalence of aortic arch hypoplasia in similar patients referred for operation has already been observed by us¹² and by others,^{18, 19} and probably it is a simple consequence of the increasing incidence of the presentation of neonates for coarctation repair after the introduction of preoperative prostaglandin E_1 therapy.²⁰ It can be inferred that most of the neonates with aortic coarctation and arch hypoplasia were seen in the past by the pathologist; now they are treated by the surgeon.¹² The presence of a hypoplastic arch usually increases the left ventricular obstruction created by the coarctation. It has already been emphasized that the extended end-to-end anastomosis is the surgical technique that relieves such an obstruction more effectively.^{3-6, 12, 18, 19, 21, 22} Furthermore, this procedure seems to be free from most of the disadvantages of the classic techniques: standard end-to-end anastomosis,²³ subclavian flap aortoplasty,¹⁴ and synthetic patch aortoplasty.²⁴ High prevalence of recoarctation was widely reported for neonatal coarctation repair by both end-to-end anastomosis^{10, 25} and subclavian flap^{20, 26, 27} or synthetic patch^{28, 29} aortoplasties. Recoarctation was

mainly attributed to growth failure of the anastomosis³⁰ or to an inadequate resection of the ductal tissue³¹ as regards end-to-end anastomosis, and to persistence and development of the coarctation shelf as regards subclavian flap or synthetic patch aortoplasties.³²⁻³⁴ Several histologic examinations showed the presence of a circumferential sling of ductal tissue extending from the ductus arteriosus and surrounding the aorta at the level of the coarctation shelf.^{8, 35, 36} Proliferation and constriction of residual ductal tissue, still active in the neonatal period,⁷ would make postoperative recurrence more likely.^{33, 37} Late aneurysm formation is another widely recognized disadvantage of subclavian flap aortoplasty³⁸ and synthetic patch aortoplasty.³⁹ With regard to subclavian flap aortoplasty, moreover, shortening and claudication of the left arm,^{34, 40, 41} gangrene of the left arm,⁴² subclavian steal syndrome,⁴⁰ and Horner's syndrome¹⁹ were reported as well.

Concerns about these complications and awareness of the increased prevalence of aortic arch hypoplasia stimulated the development of extended end-to-end anastomosis to provide an adequate and safe neonatal coarctation repair. In our opinion, for a normal aortic arch to be restored, three major key points should be respected: (1) a wide dissection and mobilization of the entire thoracic aorta, (2) a large resection of the coarctation including all ductal tissue, and (3) an incision in the arch concavity extended beyond the origin of the left carotid artery. Nonobservance of these points may increase the risk of recoarctation because of excessive tension on the suture line in the first case,¹⁰ molding of ductal tissue in the second case,^{10, 31, 37} and inadequate anastomotic size or insufficient relief of the obstruction present at the transverse arch level in the third case.^{5, 6, 12} However, when arch hypoplasia is absent or distal, a more limited arch incision can be sufficient to allow adequate repair.

In this study, recoarctation rate and freedom from reoperation for recoarctation at 10 years after the initial repair were, respectively, 9.8% and 93% $\pm$ 3%. Others reported similar results with extended end-to-end anastomosis,^{5, 18, 19, 21, 22} although their series did not contain exclusively neonates and actuarial estimates were not so extended. In our patients the occurrence of recoarctation was significantly influenced by the presence of aortic arch hypoplasia, and particularly by the complex type ($p < 0.001$). The association with a major intracardiac defect resulted in an increasing risk of

recoarctation as well ($p < 0.05$). This observation is not particularly surprising, because aortic arch hypoplasia was significantly more common and extensive in patients with an associated major intracardiac defect ($p < 0.001$). This concomitance seems to strongly support the hemodynamic underdevelopment theory for the pathogenesis of aortic coarctation.⁷

In view of the findings here presented, a more flexible approach can be advocated as regards complex aortic arch hypoplasia. This uncommon form of arch hypoplasia (7% of our patients), in which a very short proximal arch is associated with an elongated and narrow distal arch, very often was extremely difficult to repair. When there is virtually no proximal arch (left carotid artery arising very close to or as a branch of the innominate artery), it may not be possible to place the upper aortic clamp proximal to the entire hypoplastic segment without seriously compromising flow to the innominate artery during clamping. In this case with a so called "bovine" innominate trunk,¹¹ arch repair by an anterior approach with CPB and deep hypothermic circulatory arrest is certainly preferable. In this series 13 patients had such a lesion: eight underwent successful repair via sternotomy, and the remaining five, who were treated via left thoracotomy, all had a postoperative recoarctation. Furthermore, complex aortic arch hypoplasia often includes another anatomic variant, an extremely long distal arch. We think that in this case adequate repair requires the use of extended end-to-end anastomosis in addition to sacrifice of the left subclavian artery; this artery can be used as an antegrade or reversed flap to augment the top of the distal arch or it can simply be ligated to allow the resection of part of the distal arch. In this series 17 patients had this anatomic variant. Subclavian artery resection and flap aortoplasty were part of the initial successful repair in seven of these patients and were used in the reoperation for recoarctation of the remaining 10.

The potential for spinal chord ischemia (performing a long anastomosis) and brain damage (clamping most of the head vessels) could be of great concern with extended end-to-end anastomosis. However, in our patients these complications were not encountered. Other recent reports of similar series were also free from paraplegia or cerebral injury after extended end-to-end anastomosis.^{18, 19, 22} In our experience, continuous monitoring of adequate blood pressure by a right radial artery line during clamping and great care to keep the clamp time shorter than

40 minutes have been satisfactory measures to preserve the brain and spinal cord.

Percutaneous balloon aortoplasty has been proposed as a primary coarctation repair, but this technique would appear to have the same disadvantages as other surgical aortoplasties (subclavian flap or synthetic patch). Recoarctation may occur frequently,⁴³ because ductal tissue is not removed, and aortic aneurysm may develop.⁴⁴ Furthermore, vascular complications at the introduction site of the catheter may occur.⁴⁵ Although impressive results have been reported after balloon dilation for postoperative recoarctation,^{44, 46} we observed late aneurysm formation in a patient treated with this technique (33%). Since our experience with reoperation for recoarctation is free from mortality and major morbidity, surgery continues to be our treatment of choice for recoarctation.

In our patients extended end-to-end anastomosis was performed with continuous 6-0 polypropylene suture. Although superiority of absorbable suture has been advocated by some authors,^{5, 18, 21, 33, 47} a sudden unexplained death in a neonate treated with such a suture material was reported,²⁰ and others recently observed no difference in the prevalence of recoarctation between infants undergoing coarctation repair with either absorbable or nonabsorbable sutures.^{19, 33} Furthermore, satisfactory anastomotic growth was observed by us⁴⁸ and by others^{20, 37, 49} in neonates undergoing arterial switch operations with the same suture technique and material here reported.

Surgical approach. As regards the group of patients with coarctation and associated VSD, the lateral approach was the most frequently used in this series (95%). Encouraging results have been reported for complete repair by means of an anterior approach.⁵⁰⁻⁵³ We used such an approach for patients either with a single large VSD (infundibular type) or with severe complex aortic arch hypoplasia. Because spontaneous VSD closure has been observed frequently in this series (39%) and in others (80%),⁵⁴ in recent years we began to limit the use of PAB to the palliation of multiple VSDs.

In this study the most important risk factor for postoperative death was the association of complex cardiac lesions. Differences of early, late, and total mortality rates among patients with complex heart defects and patients with either isolated coarctation or associated VSD were highly significant ($p < 0.001$). Similar findings have been reported from several authors.^{12, 18-22, 29} Critical preoperative sta-

tus is another well-defined risk factor for early death,^{19, 20} although it is becoming less common. Intensive preoperative management (including prostaglandin E₁ administration, artificial ventilation, and inotropic support) and the increasing use of echocardiography in place of angiography were mainly responsible for the better outcome recently reported.^{18, 20, 29} They allow operations to be performed in patients in more stable hemodynamic condition. Other risk factors for death, such as very young age or small size at operation,^{20, 22, 33} often appear in conjunction with complex cardiac lesions.

In this series, the actuarial survivals at 10 years after operation for patients with isolated coarctation or coarctation associated with VSD or with complex cardiac anomalies were, respectively, 98%, 94%, and 60%. Kirklin,¹⁰ Vouhé,¹⁸ Van Heurn,¹⁹ Ziemer,²⁰ Harlan,²¹ Trinquet,²² and their associates reported actuarial survivals at 5 years ranging from 80% to 96% for infants with isolated coarctation, from 62% to 90% for infants with associated VSD, and from 15% to 43% for infants with associated complex heart defects. The improved results here presented have special significance because this series contains exclusively neonates, and it is by far larger than the others recently published. The major improvement in survival undoubtedly occurred in patients with associated complex anomalies. The changing policy with regard to surgical strategy has been largely responsible for such an improvement. In the past 5 years, one-stage repair via sternotomy for patients of this group increased from 8% to 53% ($p < 0.001$). During the same period mortality in this group decreased from 49% to 29% ($p < 0.05$). Furthermore, a significant difference resulted from comparison of mortality rates in patients treated by means of the primary (26%) or staged approach (46%) ($p < 0.05$). Interestingly, in the aforementioned series a large majority of the patients with complex heart defects were treated by means of a staged approach.^{10, 18-22} In consideration of the poor results obtained with this approach, a complete repair during a one-stage procedure through a median sternotomy has therefore been advocated.^{10, 19} Several reports have shown that aortic arch reconstruction with concomitant repair of the associated lesions is feasible and gives better results.⁵⁰⁻⁵³ In our series, this approach has been used mainly (87%) for patients with associated TGA or DORV (Tausig-Bing type). These anomalies were the associations more frequently encountered (65%). In patients with these associated anomalies, deleterious

effects of PAB and better outcome provided by one-stage repair have been recently reported by our group.⁵⁵ In this study comparison of mortality rates after one-stage and two-stage repair in these patients showed a significant difference ($p < 0.05$).

Among complex associations, it is possible to identify two other main categories of lesions: left-sided obstructive lesions (aortic stenosis, mitral stenosis, subaortic stenosis, hypoplastic left ventricle) and univentricular heart. In the first group, also termed hypoplastic left heart syndrome class II or III,¹⁰ the surgical management was associated in this series and in others recently reported^{11, 53} with high mortality, and indications for two- or single-ventricle repair mainly depend on the number of associations¹⁰ and on careful evaluation of many morphologic and functional parameters of the left side of the heart.⁵⁶ In patients with univentricular heart, a palliative procedure, in view of a subsequent Fontan operation, is always indicated. The choice of the palliation for patients with indications for single ventricle repair is a difficult one. Although PAB has been commonly used, concerns about deleterious effects on suitability of the Fontan operation, such as impaired diastolic function related to the myocardial hypertrophy generated by the PAB,⁵⁷ stimulated many surgeons to consider other options: the Damus-Kay-Stansel procedure, palliative arterial switch, and Norwood-type procedure.⁵⁷⁻⁵⁹ Regarding the Norwood procedure, a biologic patch-graft augmentation of the aortic end-to-end anastomosis⁶⁰ through an anterior approach appears to be an attractive alternative in some cases in which, with wide aortic dissection, arch reconstruction by means of direct anastomosis would be not feasible without any excessive tension. Complete relief of the aortic arch obstruction (mandatory in patients with left-sided obstructive lesions) and easy reproducibility are the major advantages of the ascending aorta/arch augmentation.⁶¹

In conclusion, we have described our 11 years' experience with the surgical management of neonatal coarctation. Because improvements in preoperative management (e.g., prostaglandin E₁ therapy, echocardiography in place of angiography) allowed an increasing number of neonates to be referred for coarctation repair, a higher prevalence of associated aortic arch hypoplasia in these patients was evident. Both operative mortality and recoarctation rates have been generally higher for coarctation repair in patients in this age group. Main reasons for this are the associations with aortic arch hypoplasia and complex heart defects. The findings of this study

suggest that extended end-to-end anastomosis safely provides an adequate reconstruction of the aortic arch in patients in this age group. The main advantage of this technique is the effective relief of left ventricular obstruction created by aortic arch hypoplasia. Furthermore, extended end-to-end anastomosis is associated with low operative mortality and low probability of recurrence, and it is applicable via both anterior and lateral approaches. Freedom from major morbidity, absence of foreign material, complete resection of ductal tissue, and preservation of the left subclavian artery are other advantages of this technique. In some cases with a severe form of arch hypoplasia, some sort of arch augmentation by means of a subclavian flap or a homograft patch could be preferable. Successful management of the majority of neonates with associated ventricular septal defect is possible with repair of coarctation alone. Early VSD repair or PAB should be considered in patients in whom medical management of postoperative congestive heart failure is unsuccessful. Indications for PAB should be limited to the palliation of multiple VSDs. Survival in patients with additional complex cardiac lesions is mainly related to the severity of the associated anomalies. One-stage repair of neonatal coarctation and associated complex heart defects (with indication for two-ventricle repair) by means of an anterior approach provides a better outcome than a two-stage repair.

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Discussion

Dr. William S. Blakemore (*Birmingham, Ala.*). On the illustrations you used a continuous suture. I can recall that shortly after World War II, Julian Johnson and a group of us worked in an animal laboratory using interrupted sutures at least on the anterior surface. We were also using silk as our continuous suture in the experiment.

Perhaps a more difficult and more pertinent question is this: Do you have any idea what causes the aortic arch deformities in this group of patients? I ask this because as we worked in a rural area in starting a program we thought we saw an extraordinary number of aortic arch deformities in the patients from the rural countryside. They had surface water wells. They planted corn up to the house, and the well was beside the house. We were concerned that herbicides or insecticides might be having an effect in the pregnant women, especially at the critical times of about 12 weeks. Do you have any thoughts about what might be causing these kinds of deformities?

Dr. William A. Gay, Jr. (*Bronx, N.Y.*). Have you had any untoward events from your angioplasty for the recoarctations? Have you had to operate on any of these patients

urgently? At what time after repair of a coarctation do you believe that it is safe to dilate the anastomosis?

Dr. Lacour-Gayet. I thank the discussants for their comments.

To answer the questions of Dr. Blakemore: Regarding the use of continuous suture, we feel quite comfortable with this technique because we have now performed 600 arterial switches in neonates using continuous suture with a nonabsorbable monofilament on the aortic anastomosis without observing stenosis. Regarding the difficult question on the etiology of aortic arch hypoplasia: It is clear that this hypoplasia takes place during pregnancy, and several hypotheses have been suggested, among them the malalignment of the conal septal. However, I can tell that

true malalignment VSD is not always associated with aortic arch obstruction, for severe arch hypoplasia can be seen in the absence of malaligned conal septum. Therefore, other causes still under investigation might be responsible for this anomaly.

To answer the question of Dr. Gay regarding the indications of angioplasty: We have not attempted to indicate balloon dilatation in native coarctation. For patients with recoarctation, the results obtained after balloon dilatation are good, as similarly observed by the group from Boston. The aorta is surrounded by fibrous tissue and we believe that the risk of bleeding is not very important. On the other hand, all reoperations for recoarctation in this series were achieved without mortality.

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