Transverse arch hypoplasia is an integral, albeit anatomically independent, part of neonatal coarctation of the aorta. Extended end to end anastomosis has been advocated to overcome arch hypoplasia. Numerous studies demonstrated the growth potential of the aortic arch following repair with confined extensivity. Limited information, however, is available on how rapidly the hypoplastic aortic arch adapts to post-coarctectomy circulatory conditions. The purpose of this study is to demonstrate that a hypoplastic aortic arch adapts to post-coarctectomy circulatory conditions, however, is available on how rapidly the hypoplastic aortic arch adapts to post-coarctectomy circulatory conditions.

**PATIENTS AND METHODS**

Thirty four consecutive infants (19 boys and 15 girls; mean (SD) age 13.1 (6.19) days (range 1–58 days); mean weight 2.8 (0.53) kg (range 1.1–4.6 kg)) underwent coarctation repair as primary operation at our institution, forming the patient group of this study.

Diagnoses were established by two dimensional Doppler echocardiography (Sonos 5500, 7.5 MHz, Hewlett-Packard, Andover, MA) using standard projections. Internal diameters of the distal transverse arch (distal to left carotid artery) and descending aorta (at the level of the diaphragm) were measured and this ratio was chosen to express the degree of transverse arch hypoplasia (a ratio of < 0.5 was considered hypoplastic). Patients were grouped as normal and hypoplastic arch groups according to arch ratio.

From left posterolateral thoracotomy the transverse aortic arch and its branches, arterial duct, and descending aorta were fully dissected. Having divided the arterial duct the coarctation was resected and the isthmus and distal transverse arch stump were opened to accommodate the diameter of the bevelled descending aorta. An anastomosis was then made with 7.0 or 8.0 non-absorbable monofilament sutures. In no case did the anastomosis reach proximally beyond the left carotid artery.

Systematic two dimensional Doppler echocardiographic assessments were made on the first, third, and fifth postoperative day, preferably by the same examiner. Colour coded Doppler flow mapping of the ascending aorta and descending aorta with no angle corrections were utilised. The left ventricle shortening fraction (LVSF) was determined from two dimensional guided M mode tracing of the left ventricle short axis. To estimate the contribution of the transverse arch to the descending aorta peak flow velocity (DAFV) in different circulatory conditions, any difference between descending and ascending aorta peak flow velocities (AAVF) was indexed to the LVSF.

**RESULTS**

Normal (n = 20) and hypoplastic (n = 14) arch groups could be formed using the ratio of the transverse arch and descending aorta diameters (0.88 (0.25) v 0.42 (0.06); p < 0.0001) at a cut off point of 0.5. Each group had nearly normal distribution and were matching in all other characteristics but bodyweight (3.67 (0.67) v 2.8 (0.07); p = 0.041) and associated atrial septal defect (4/20 v 14/14; p < 0.0001).

Impaired ventricular function (in emergencies LVSF 0.26 (0.04) v non-emergencies 0.39 (0.05); p < 0.0001) at presentation was the main indication for an emergency operation (within six hours of the diagnosis) in 15/34 cases. In no case did the end to end anastomosis reach proximally beyond the left carotid artery. All patients were extubated within 48 hours of the surgery with no right radial to femoral artery pressure systolic gradients > 15 mm Hg. No mortality/morbidity occurred in the series. Follow up was 14.1 (2.4) months (range 7–18 months). In two cases of obstruction at suture line level successful balloon dilations were performed postoperatively at 4 and 4.5 months respectively. All patients are symptom-free. Event-free survival of the arch repair is 94.11% (95% confidence limits (CL) 81% to 97%) at 18 months postoperatively.

At least three different measurements of postoperative two dimensional Doppler echocardiography were available for each patient. No regional wall motion abnormalities were observed and LVSF could be determined in every case. LVSF showed a gradual increase over the series. LVSF on the first postoperative day was lower in the hypoplastic group (0.39 (0.08) v 0.32 (0.07); p = 0.01). No descending aorta diastolic antegrade flow (diastolic gradient) was observed in any patient. All peak flow velocities but DAFV in the hypoplastic arch group showed an increase parallel to an improving LVSF over the entire immediate postoperative period. Conversely, DAFV in the hypoplastic arch group exhibited a descending pattern.

**Abbreviations:** AAFV, ascending aorta peak flow velocity; DAFV, descending aorta peak flow velocity; LVSF, left ventricle shortening fraction.
slopes (postoperative day 1, 2.73 (0.33) v day 3, 2.55 (0.35); p = 0.038; day 3, 2.55 (0.35) v day 5, 2.49 (0.29); p = 0.23).

Post-coarctectomy transverse arch remodelling was quantified by comparing normal and hypoplastic arch data of the DAFV and AAFV difference indexed to the LVSF. The difference between the two groups disappeared beyond the fifth postoperative day (fig 1).

**DISCUSSION**

Previous studies have demonstrated that transverse arch and descending aorta are independent variables, therefore a ratio of their diameter better signifies transverse arch hypoplasia than that of transverse arch/ascending aorta. We found an empiric cut off point between normal and hypoplastic arch groups at 0.5 with equal distribution of anomalies, excepting uniform association of atrial septal defect to hypoplastic group.

An arch incision extended beyond the origin of the left carotid artery was proposed as mandatory optimal reconstruction of the hypoplastic aortic arch. It is generally acknowledged that the transverse arch grows after coarctation repair so most centres now perform it with limited extenty. Postoperative remodelling is also recorded. The purpose of this study was to investigate the time frame within which it occurs.

We observed a uniform and significant increase of LVSF in the post-coarctectomy period. Initial LVSF increase may partly be explained by the disappearance of an obstacle in the arterial circuit, whereas a further increase can signify increasing cardiac output that is paralleled by higher AAFV and DAFV. LVSF and peak flow velocities are independent variables. We chose LVSF as a surrogate to index the change of cardiac output.

We hypothesise that the peak flow velocity difference between the descending and ascending aorta is caused by the convective acceleration that occurs in the transverse arch, and is determined by its diameter and compliance. The

**REFERENCES**


**IMAGES IN CARDIOLOGY**

Large coronary artery aneurysms following sirolimus eluting stent implantation

The patient underwent surgery. The operative findings showed pronounced inflammation and fibrosis around the area of proximal LAD with platering of tissues, which were very hard in consistency. No dissection of this area was done and the distal LAD was grafted using a left internal mammary artery. The patient is doing well and symptom-free after three months of follow up.