Detrimental Sequelae on the Hemodynamics of the Upper Left Limb After Subclavian Flap Angioplasty in Infancy

Jacques A.M. van Son, MD, Wim N.J.C. van Asten, PhD, Henk J.J. van Lier, MSc, Otto Daniëls, MD, Josef G. Vincent, MD, Stefan H. Skotnicki, MD, and Leon K. Lacquet, MD

The long-term effect of two surgical techniques for repair of coarctation of the aorta in infancy, namely, resection and end-to-end anastomosis (RETE) and subclavian flap angioplasty (SFA) on the blood supply of the upper left limb, was quantified by Doppler spectrum analysis of blood flow velocities in the left brachial artery at rest and during postocclusive reactive hyperemia. Twenty-three patients participated in this study: nine patients after SFA (median age, 8 years), 14 patients after RETE (median age, 8 years), and 10 control subjects (median age, 9.5 years). At rest, a highly significant decrease of blood flow velocities in the left brachial artery was measured in all patients of the SFA group compared with those of the RETE and control groups, as documented by various Doppler spectrum parameters: maximal frequency of advancing curve ($p=0.0001$), pulsatility index ($p=0.0005$), and resistance index ($p=0.039$). During reactive hyperemia, a moderate capacity of physiologic augmentation of blood flow velocities was observed in five patients of the SFA group. This capacity was marginal in two patients with complaints of claudication in the left upper limb during strenuous exercise, which can be related to the number of branches of the left subclavian artery ligated during operation. This study indicates that SFA in infancy may lead to compromised hemodynamics of the upper left limb with potential for symptoms of ischemia during exercise. (Circulation 1990;81:996–1004)

Subclavian flap angioplasty (SFA) was introduced1 as a technique to overcome reported high mortality and coarctation recurrence rates in neonates treated by resection and end-to-end anastomosis (RETE).2–4 In this operation, the left subclavian artery is ligated and transected distally at its thoracic outlet and subsequently is used to reconstruct the aorta, thus avoiding a circumferential anastomosis. This procedure gradually gained wide acceptance and was advocated as the operation of choice for coarctation in infancy.5–8 However, sacrifice of the left subclavian artery carries the disadvantage of impaired blood supply to the left arm. Although in the recent literature support has been lent to the concept that development of a ductal diaphragm after SFA at neonatal age may be more important in causing recoarctation than growth failure of a circumferential anastomosis after RETE,9–12 the detrimental effect on the upper left limb after SFA could be a second major determinant against selection of the SFA technique for repair of coarctation in infancy. The paucity of reports on the long-term hemodynamic effect of SFA in infancy on the development of the left arm prompted us to perform a hemodynamic study in order to quantify this effect by means of quantitative Doppler spectrum analysis of blood flow velocities in the left brachial artery in a group of SFA, RETE, and control patients.

Transcutaneous detection of blood flow velocities is possible with the Doppler shift principle, which refers to the alteration of frequency of backscattered ultrasound waves in proportion to the velocity of the erythrocytes. Recently, we recommended this technique as an accurate and reliable screening method for coarctation in infancy and childhood.13 Because Doppler flow velocity recordings are dampened distally to hemodynamically significant arterial stenoses, analysis of recordings from the left brachial artery should be capable of detecting the severity of impairment of left brachial arterial flow in children who have undergone ligation of the left subclavian artery.

From the Departments of Thoracic and Cardiac Surgery, Pediatric Cardiology and Statistical Consultation, University Hospital St. Radboud, Nijmegen, The Netherlands.

Address for correspondence: Jacques A.M. van Son, MD, Department of Thoracic and Cardiac Surgery, University Hospital St. Radboud, P.O. Box 9101, 6500 HB Nijmegen, The Netherlands.

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in infancy. Determination of the resting blood flow velocities of a compromised vascular bed may have limited diagnostic value because a significantly stenotic inflow system may still permit normal resting blood flow velocities; therefore, postocclusive reactive hyperemia was used in this study as an additional diagnostic criterion.\textsuperscript{14,15}

**Methods**

The study group consisted of 23 patients. Of these, nine had undergone SFA and 14 RETE in infancy. The control group consisted of 10 healthy age- and sex-matched children who were subjected to the same measurements. All patients were right-handed except one patient in the SFA group and one control subject.

Parents and children were questioned about symptoms relating to ischemia of the upper left limb both at rest and during exercise. In addition, inquiries were made about the presence of symptoms of subclavian steal syndrome and differences in temperature between both upper limbs. Operative reports were reviewed to determine the number of branches of the subclavian artery that was ligated at the time of subclavian division.

Physical examination included measurement of upper arm length (acromion-olecranon), forearm length (olecranon-radial stylos), and upper arm and forearm circumferences (maximum girth). Pulse volumes at the axillary, brachial, and radial arteries were assessed. Resting systolic blood pressures in both arms were measured in the supine position, using a sphygmomanometer and bidirectional 8-MHz Doppler ultrasound velocity detector. Pneumatic cuff sizes were chosen to cover approximately 75\% of upper arm length.\textsuperscript{16} The Doppler probe was placed at the antecubital fossa.

Blood flow velocities in the upper limbs were measured by Doppler ultrasonography. Simultaneously, the heart rate was documented by electrocardiography. Doppler signals were obtained from both brachial arteries, both at rest and during reactive hyperemia, using an 8-MHz bidirectional continuous-wave probe (Meda Sonics, Mountain View, California) with the patient in a supine position. The Doppler probe was placed at the antecubital fossa and was kept at an angle relative to the axis of the brachial artery that corresponded with optimal spectra with freedom from artifacts caused by vessel wall movement and venous flow. To produce reactive hyperemia, a sufficiently wide pneumatic cuff\textsuperscript{16} was placed around the upper arm, inflated 50 mm Hg above systolic pressure for 4 minutes, and then released. Doppler spectra were continuously recorded immediately after release of the pneumatic cuff until the velocity value returned to preocclusion levels. The spectrum with the highest velocity value was subsequently selected for analysis. The Doppler signals were processed by a real-time spectrum analyzer (model SA 8000, Radionics Medical, Scarborough, Ontario, Canada) and subsequently fed into a Digital MNC 11/23 computer on the basis of electrocardiographic triggering. Maximal frequency waveforms (MAX-curves) during the heart cycle were calculated from the spectra off-line by a sophisticated local algorithm.\textsuperscript{17} When the advancing and receding flow velocities are processed separately, two MAX-curves are derived from each spectrum. These indicate the maximal advancing and receding blood flow velocity as a function of time (Figure 1A). To describe the shape of the curves, four parameters were calculated: the maximal frequency of the systolic deflection (FMAX), the end-diastolic frequency (FDIA), the mean frequency during one heart cycle (FMEAN), and the maximal frequency of the receding curve (FMIN) (Figure 1B).\textsuperscript{18} The pulsatility index and resistance index were derived by combining different parameters.\textsuperscript{19–24} Both are dimensionless figures that are independent of the insonation angle.

**Statistical Analysis**

Quantitative data were analyzed with the Kruskal-Wallis, Student's $t$, and Mann-Whitney $U$ tests. Grouped data are given as median with
TABLE 1. Data at Rest and During Reactive Hyperemia for Subclavian Flap Angioplasty Group

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age at surgery (wk)</th>
<th>Age at study (yr)</th>
<th>Number ligated branches</th>
<th>Type of CoA</th>
<th>Presence ICD</th>
<th>SBP at rest (mm Hg)</th>
<th>Gradient RSA-FA at rest (mm Hg)</th>
<th>Doppler spectrum parameters</th>
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<tbody>
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<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>M</td>
<td>1</td>
<td>6</td>
<td>1</td>
<td>pre</td>
<td></td>
<td>95</td>
<td>15</td>
<td>FMAX (10)</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>14</td>
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<td>2</td>
<td>pre</td>
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<td>F</td>
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<td>4</td>
<td>pre</td>
<td>CAVCD2</td>
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<td>-10</td>
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</tr>
<tr>
<td>5</td>
<td>F</td>
<td>12</td>
<td>8</td>
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<td>para</td>
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<td>8</td>
<td>1</td>
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<td>6</td>
<td>2</td>
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<td>F</td>
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<td>8</td>
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<td>9</td>
<td>M</td>
<td>9</td>
<td>8</td>
<td>3</td>
<td>pre</td>
<td>VSD3</td>
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<tr>
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<td>90</td>
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<td>1.695 (0.98)</td>
</tr>
<tr>
<td>(IQR)</td>
<td>(16)</td>
<td>(2.5)</td>
<td>(1.5)</td>
<td>(18)</td>
<td></td>
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<td>(17.5)</td>
<td>(946) (1.09)</td>
<td>(2,261) (0.15)</td>
</tr>
</tbody>
</table>

LSA, left subclavian artery; CoA, coarctation of the aorta; ICD, intracardiac defects; SBP, systolic blood pressure; RSA, right subclavian artery; FA, femoral artery; FMAX, maximal frequency of advancing curve; PI, pulsatility index; RI, resistance index; L, left-handed; pre, preductal coarctation; para, para ductal coarctation; VSD, ventricular septal defect; CAVCD, common atrioventricular canal defect; 1, closed spontaneously; 2, repaired 1.2 years after subclavian flap angioplasty; 3, repaired 1.8 years after subclavian flap angioplasty; IQR, interquartile range.

*Patient with claudication in upper left limb during exercise.

LQI, low subclavian ischemia.

**Controlled autoregulation. Statistical significance was reached at p≤0.05.

Results

Two of the nine patients in the SFA group complained of claudication in the upper left limb during strenuous exercise. Both had noticed symptoms of unilateral fatigue in their upper limbs during swimming. All patients in the SFA group indicated that the upper left limb was colder than the right one. Symptoms of subclavian steal syndrome were absent. Children in the RETE group denied symptoms of upper left limb ischemia at rest or during exercise.

TABLE 2. Data at Rest and During Reactive Hyperemia for Resection and End-to-End Anastomosis Group

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age at surgery (wk)</th>
<th>Age at study (yr)</th>
<th>Type of CoA</th>
<th>Presence ICD</th>
<th>SBP at rest (mm Hg)</th>
<th>Gradient RSA-FA at rest (mm Hg)</th>
<th>Doppler spectrum parameters</th>
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</tr>
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<td>2</td>
<td>15</td>
<td>pre</td>
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<td>3</td>
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<td>11</td>
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<td>VSD1</td>
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<td>+10</td>
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<td>6</td>
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<td>para</td>
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<td></td>
<td>105</td>
<td>-20</td>
<td>3,009 (6.76)</td>
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<td>SAS5</td>
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<td>VSD1</td>
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<td>Median</td>
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<td>105</td>
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<td>3,185 (3.60)</td>
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<tr>
<td>(IQR)</td>
<td>(15)</td>
<td>(5.2)</td>
<td>(20)</td>
<td>(16.25)</td>
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<td>(1.902)</td>
<td>(2.98)</td>
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CoA, coarctation of the aorta; ICD, intracardiac defects; SBP, systolic blood pressure; LSA, left subclavian artery; RSA, right subclavian artery; FA, femoral artery; FMAX, maximal frequency of advancing curve; PI, pulsatility index; RI, resistance index; L, left-handed; pre, preductal coarctation; para, para ductal coarctation; VSD, ventricular septal defect; CAVCD, common atrioventricular canal defect; 1, closed spontaneously; 4, repaired 2.6 years after resection and end-to-end anastomosis; 5, repaired 2.1 years after resection and end-to-end anastomosis; IQR, interquartile range.
TABLE 3. Data at Rest and During Reactive Hyperemia for Control Group

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age at study (yr)</th>
<th>SBP LSA at rest (mm Hg)</th>
<th>Gradient RSA-FA (mm Hg)</th>
<th>Doppler spectrum parameters at rest</th>
<th>Doppler spectrum parameters during reactive hyperemia</th>
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<td>FMAX (Hz)</td>
<td>PI</td>
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<td>−15</td>
<td>4,856</td>
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<td>−10</td>
<td>6,347</td>
<td>5.93</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>9.5</td>
<td>118</td>
<td>−2.5</td>
<td>5,082</td>
<td>5.50</td>
<td>0.88</td>
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</table>

SBP, systolic blood pressure; LSA, left subclavian artery; RSA, right subclavian artery; FA, femoral artery; FMAX, maximal frequency of advancing curve; PI, pulsatility index; RI, resistance index; L, left-handed; IQR, interquartile range.

**Physical Examination**

The upper left arm was shorter than the right one in all patients of the SFA group (median difference, 1.1 cm; IQR, 0.2 cm; p<0.005) although this comparison fails to take into account the tendency for right-handed subjects to have a longer upper right extremity. There was no significant difference for the forearm. Median upper arm and forearm circumferences were, respectively, 0.4 cm and 0.2 cm less on the left side. One nonathletic patient in the SFA group had developed a 2.5 cm shortening of the upper left arm with substantial diminution in muscle mass, which had first been detected 2 years after operation (Table 1, case 3). In the SFA group, six patients were athletic, including both symptomatic patients. In the RETE and control groups, 11 and eight patients, respectively, were athletic.

All nine patients in the SFA group had diminished (four patients) or absent (five patients) pulses in the left arm at the axillary, brachial, and radial levels. The median systolic blood pressure in the left brachial artery in the SFA group was 90 mm Hg (IQR, 18 mm Hg) (Table 1), 105 mm Hg (IQR, 20 mm Hg) in the RETE group (Table 2), and 118 mm Hg (IQR, 22 mm Hg) in the control subjects (Table 3). A difference in the median systolic blood pressure in the right brachial artery in the three groups was not detected: 120 mm Hg (IQR, 32 mm Hg) in the SFA group, 120 mm Hg (IQR, 15 mm Hg) in the RETE group, and 120 mm Hg (IQR, 21 mm Hg) in the control group. The systolic blood pressure difference between upper right and left limbs in the SFA group varied from 30 to 55 mm Hg (median, 35 mm Hg). Both of the symptomatic patients in the SFA group had the lowest systolic blood pressure of the group.

**Electrocardiography**

During reactive hyperemia, the median heart rate increased from 75 beats/min (IQR, 8 beats/min) to 87 beats/min (IQR, 14 beats/min) in the SFA group, from 73 beats/min (IQR, 7 beats/min) to 83 beats/min (IQR, 12 beats/min) in the RETE group, and from 70 beats/min (IQR, 7 beats/min) to 79 beats/min (IQR, 10 beats/min) in the control group (p>0.05).

**Doppler Spectrum Analysis**

A monophasic spectrum in the left brachial artery, both at rest and during reactive hyperemia, was found in all nine SFA patients, whereas biphasic and triphasic spectra in the same artery at rest were observed in 13 of 14 RETE patients and in all 10 control subjects. The only patient in the RETE group with a monophasic spectrum in the left brachial artery had involvement of the origin of the left subclavian artery in a recurrent coarctation, as documented by angiography, resulting in right-left brachial and right brachial–femoral systolic pressure gradients of 35 mm Hg (Table 2, case 2). Except for this patient, we invariably documented loss of the reverse flow component and occurrence of a monophasic spectrum in the left brachial artery in the RETE and control groups during reactive hyperemia. Figure 2 depicts representative Doppler spectra of the left brachial artery at rest and during reactive hyperemia after SFA and RETE.

Doppler spectrum analysis revealed significantly decreased values for the maximal frequency of the advancing curve and the pulsatility and resistance indices in the SFA group, in comparison with the RETE and control groups (Table 4) both at rest and during reactive hyperemia. Figure 3 illustrates the differences in resistance index before and after reactive hyperemia in the SFA, RETE, and control groups. When the three Doppler spectrum parameters obtained from both brachial arteries at rest and during reactive hyperemia in the SFA group were compared, a highly significant difference in favor of the right brachial artery was detected (p<0.005).
two patients of the SFA group, adequate compensatory increase of the maximal frequency of the advancing curve and decrease of the pulsatility and resistance indices were observed during reactive hyperemia (Table 1, cases 1 and 6); in five patients, these parameters changed moderately under the same condition (Table 1, cases 2, 3, 5, 7, and 8); in two patients, only marginal increase of the maximal frequency of the advancing curve and absent-to-marginal decrease of the pulsatility and resistance indices were observed during reactive hyperemia (Table 1, cases 4 and 9, respectively). The latter two patients were symptomatic during strenuous exercise.

Discussion

Studies on the long-term effect of SFA in infancy have concentrated mainly on relieving coarctation of the aorta. A second potential drawback of this procedure, ligation of the left subclavian artery, has rarely been studied. Currarino and Engle reported that interruption of the subclavian artery in growing individuals usually causes a substantial diminution in the longitudinal growth of the long bones, diminution in the muscle thickness of the corresponding arm, or both. The latter finding has been confirmed by Lodge and associates. Upper limb flow studies following the Blalock-Taussig anastomosis, performed beyond infancy, revealed a significant shortening of the forearm on the operated side. Our data, however, support a report of shortening of the upper left arm after SFA in infancy. These different observations may have been caused by the difference in age of the children at the time of the operation because upper arm growth tends to predominate in younger children and lower arm growth in older children.

A highly significant decrease of blood flow velocities in the left brachial artery was measured in all patients of the SFA group compared with those of the RETE

<table>
<thead>
<tr>
<th>Table 4. p Values of Various Doppler Spectrum Parameters of Left Brachial Artery by Operation Type as Determined by Kruskal-Wallis and Mann-Whitney U Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doppler spectrum parameter</td>
</tr>
<tr>
<td>Mann-Whitney U Test</td>
</tr>
<tr>
<td>FMAX</td>
</tr>
<tr>
<td>PI</td>
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<tr>
<td>RI</td>
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</table>

RETE, resection and end-to-end anastomosis; SFA, subclavian flap angioplasty; FMAX, maximal frequency of advancing curve; PI, pulsatility index; RI, resistance index; NS, not significant.
and control groups, as documented by various Doppler spectrum parameters. This difference cannot be explained by the cardiac status of the patients in the SFA group because none of them was known to have a history of limited physical activity based on cardiac status. This contention is supported by the fact that during reactive hyperemia, a similar increase of heart rate in the three groups was measured.

A diminished extremity blood flow can be compensated for by a decrease in the peripheral vascular resistance. The resistance of the peripheral vascular bed is a microcirculatory adaptation to the metabolic demands of its surrounding tissue, by means of dilation of a large number of resistance vessels to the same extent. The level of this dilation appears to be one of the most important determinants of flow reversal. Our documentation of absence of reverse flow, already at rest, in all SFA patients supports our hypothesis that after division of the left subclavian artery in infancy, the peripheral vascular bed of the upper left limb is in a permanent state of maximal or submaximal vasodilation. It also explains the limited ability of these patients to further minimize the peripheral vascular resistance of the affected limb during reactive hyperemia, for vasodilation is already maximal or submaximal at rest.

We used the technique of temporary circulatory occlusion of the limb of interest as a controllable maximal vasodilatory stimulus, although exercise testing after coarctation repair is increasingly being used to evaluate aortic and subclavian artery physiology with increased cardiac output.

We prefer the former technique because we have some concern about reproducibility and quantification of exercise stress testing in young children.

The hemodynamic differences found in our study between the patients in the SFA group and the patients in the other groups appear to contradict those of Joyner and associates, who reported a similarity of blood flow values at rest and during maximal vasodilatory stimuli in the normal and surgically altered upper limbs of patients who had undergone division of a subclavian artery. They measured total blood flow in the affected arm, however, whereas we measured blood flow velocities in the left brachial artery. In the normal arm, during one heart cycle, the blood flow velocities are high during systole and approximate zero, or are even reversed, during diastole. In the surgically altered arm, the blood flow velocities fluctuate considerably less during the cardiac cycle. During systole, the maximal blood flow velocities are lower than in the normal arm, whereas even during diastole, blood flow velocities in anterograde direction remain detectable. However, the total anterograde blood flow during one cardiac cycle, which is determined by the combined blood flow in the brachial artery and its collaterals, may reveal no significant differences between normal and affected arms, as evidenced by Joyner's findings. Our findings show a considerable impairment of the hemodynamics in the surgically altered brachial artery.

In this study, we were unable to find a correlation between the presence of claudication and handedness, sex, time from operation to entry in the study group, and right brachial–femoral pressure gradient. However, our data suggest a correlation between the number of branches of the left subclavian artery ligated at the time of operation and the occurrence of symptoms. Both of the symptomatic patients had more than two branches ligated and had the lowest systolic blood pressure in the upper left limb. A third patient in the SFA group, who had developed a hypoplasia of the upper left arm, was not symptomatic. This patient, however, was nonathletic and, therefore, possibly asymptomatic because of his lifestyle.

The development of the collateral circulation, bypassing the left subclavian artery, mainly depends on the particular site of division of the subclavian artery, as this accounts for the degree of recruitment of stem and reentry vessels, which is essential for the immediate recovery of inflow into the left brachial artery. The magnitude of the pressure gradient across the collateral vessels subsequently modulates the degree of angiogenesis of midzone arteriolar vessels. The implication of these data is that minimizing ligation of branches of the left subclavian artery saves potential stem and reentry vessels and thus is beneficial for the recovery of inflow into the left brachial artery. Also, development of sustained hypertension, as may occur in coarctation repaired beyond infancy or in residual or recurrent coarct-
tion, should be prevented because it may result in arteriolar rarefaction with decreased collateral circulation to the affected arm.

We always ligate the vertebral artery because concern has been expressed in the past over the possibility of creating symptoms of subclavian steal when this is not done. Our data suggest, however, that if technically possible, the internal mammary artery, the thyrocervical trunk, and any additional branches of the left subclavian artery should be left undisturbed to avoid potential adverse sequelae on the hemodynamics of the affected arm.

In case of developmental abnormalities of the aortic isthmus, with a resultant short or absent isthmus, or in isthmic hypoplasia accompanying coarctation of the aorta, the orifice of the left subclavian artery may be narrowed by ductal tissue (Figure 4) or may be hypoplastic, respectively. This may limit the usefulness of the subclavian artery in an SFA procedure. Therefore, when this anatomic pattern is encountered, we prefer to perform a resection of the coarctation and the hypoplastic isthmus and an additional enlargement of the origin of the left subclavian artery. This is accomplished either by a short incision of its lateral wall and partial inclusion of its origin in the end-to-end anastomosis of the aorta, or, alternatively, by resection of its proximal obstructive lesion and reimplantation of the left subclavian artery proximal to the end-to-end anastomosis. The latter is preferred when ductal tissue is present at the orifice. When, in addition, the distal transverse aortic arch between the left common carotid and left subclavian arteries is hypoplastic, we enlarge this segment. Thus, adequate inflow in both the descending aorta and the left subclavian artery is restored.

Based on the aforementioned considerations, enlargement of the orifice of the left subclavian artery had been successfully performed in five patients of the RETE group, resulting in excellent pulsatile flow in the left brachial artery in all cases, as documented by Doppler spectrum analysis.

In one (athletic) patient of the RETE group in the earlier part of the series, in whom enlargement of the stenotic orifice of the left subclavian artery had not
been performed, substantially decreased blood flow velocities in the left brachial artery were measured. This patient was asymptomatic, despite his active lifestyle. Angiography showed a recurrent coarctation with a severe stenosis of the origin of the left subclavian artery, directly proximal to the end-to-end anastomosis, with collateral vessels bypassing the proximal left subclavian artery. The latter finding may explain the absence of symptoms in this patient.

In conclusion, we do not support routine performance of SFA for repair of coarctation of the aorta in infancy. This conclusion is based on the documentation of considerably decreased blood flow velocities in the left brachial artery at rest and during reactive hyperemia, with possible detrimental effects on the long-term development of the left arm, and the disadvantage of potential contracture of residual ischemic ductal tissue during the first 3 months of life. Creation of a satisfactory end-to-end anastomosis after resection of all ductal tissue with preservation of growth potential of the aorta and restoration of inflow into the left subclavian artery seems to be the most appropriate method of repair, both anatomically and physiologically.

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