

Altered Baroreceptor Function in Children With Systolic Hypertension After Coarctation Repair

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To determine whether altered baroreceptor function may contribute to systemic hypertension after coarctation of the aorta (C of A) repair, baroreceptor function was evaluated in 6 children with repaired C of A mild arm systolic hypertension. Data were compared with those from 7 normotensive control children with hemodynamically mild heart disease. Age at C of A repair averaged 9.9 ± 3.1 years (mean \pm standard deviation [SD]). Arm systolic pressure was 143.8 ± 2.9 mm Hg in the C of A repair group, compared with 118.3 ± 9.9 for control subjects ($p < 0.001$). At catheterization, steady-state sigmoidal baroreceptor function curves relating mean arterial pressure to R-R interval were derived by increasing

and decreasing mean arterial pressure with small injections of phenylephrine and nitroprusside. Compared with control subjects, the baroreceptor function curves of children with repaired C of A (1) are reset about a higher baseline mean arterial pressure (108.8 ± 6.6 versus 90.3 ± 8.6 mm Hg, $p < 0.01$), (2) have a decreased slope (7.9 ± 3.7 versus 17.4 ± 3.6 ms/mm Hg, $p < 0.001$), and (3) have a diminished R-R interval range (246.7 ± 81.5 versus 535.7 ± 97.2 ms, $p < 0.001$). Thus, in children with hypertension after C of A repair, the baroreflex is reset to an elevated mean arterial pressure level and has a diminished sensitivity to changes in arterial pressure.

Arm hypertension, at rest or during dynamic exercise, occurs frequently after repair of coarctation of the aorta (C of A).¹⁻⁵ In some patients, hypertension is clearly related to a residual or recurrent C of A.⁶ In others, however, those with little or no residual resting C of A gradient, the cause of postoperative hypertension is less clear. The baroreflex is intimately involved in blood pressure regulation, but its function in patients with repaired C of A has not been evaluated. Nevertheless, studies in dogs with experimental C of A^{7,8} have documented baroreflex alterations similar to those described in adults with chronic essential hypertension.⁹⁻¹¹ The purpose of this study is to evaluate baroreceptor function in children with repaired C of A who have mild arm hypertension.

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Methods

Six children, 5 boys and 1 girl, with good surgical repair of C of A (resting peak systolic gradient of < 25 mm Hg) and mild resting arm systolic hypertension (> 140 mm Hg) served as subjects of this investigation. A group of 7 normotensive children with hemodynamically mild heart disease served as control subjects. Pertinent clinical and hemodynamic data are presented in Table I. The children with repaired C of A ranged in age from 13.8 to 19.0 years, averaging 16.8 ± 2.0 years (mean \pm SD). The control children averaged 15.4 ± 4 years of age, which is not significantly different from that in the C of A repair group. The 6 children with repaired C of A had undergone repair of a discrete thoracic C of A at an average age of 9.9 ± 3.1 years (range 6.8 to 13.8). Before C of A repair, all children had resting arm hypertension (defined as systolic pressure at or above the 95th percentile for age and sex) averaging 146.8 ± 16.2 mm Hg. C of A repair was performed by end-to-end anastomosis in 4 children (Patients 3 to 6) and by patch aortoplasty in 2 (Patients 1 and 2). Immediately after surgical repair, 5 children (Patients 1, 2, 3, 5, and 6) had paradoxical hypertension requiring vigorous antihypertensive therapy for 2 to 3 days. The interval from repair to the present study averaged 6.9 ± 3.7 years. At the time of this study, no patient was receiving antihypertensive medication or digoxin. There were no associated hemodynamic lesions of significance. One patient (Patient 4) had also undergone repair of a ventricular septal defect with excellent

TABLE I Pertinent Clinical Data in 6 Children With Repaired Coarctation and 7 Control Children

| Patient | Diagnosis | Age (yr) | Age at C of A Repair (yr) | Resting Outpatient Data | | | | Treadmill Exercise | | |
|--------------------|------------------------|----------|---------------------------|-------------------------|-------------------|------|-------------------------------------|------------------------|--|---------------------------------|
| | | | | HR (beats/min) | Rt Arm BP (mm Hg) | | Rt Arm-Rt Leg Syst Gradient (mm Hg) | HR Peak Ex (beats/min) | Rt Arm Systolic Pressure (mm Hg) Post Ex | C of A Gradient (mm Hg) Post Ex |
| | | | | | Syst | Dias | | | | |
| Coarctation Repair | | | | | | | | | | |
| 1 | Patch aorto-plasty | 13.8 | 12.9 | 93 | 142 | 82 | 8 | 184 | 206 | 32 |
| 2 | Patch aorto-plasty | 18.2 | 13.8 | 76 | 145 | 70 | 6 | 202 | 210 | 86 |
| 3 | End-to-end anastomosis | 14.4 | 6.8 | 81 | 141 | 60 | 7 | 198 | 240 | 90 |
| 4 | End-to-end anastomosis | 19.0 | 11.9 | 85 | 146 | 80 | 0 | 182 | 220 | 40 |
| 5 | End-to-end anastomosis | 17.1 | 6.8 | 76 | 141 | 70 | 28 | 188 | 230 | 120 |
| 6 | End-to-end anastomosis | 18.3 | 7.2 | 71 | 148 | 85 | 2 | 189 | 234 | 83 |
| Mean | | 16.8 | 9.9 | 80.3 | 143.8 | 74.5 | 7.8 | 190.5 | 223.3 | 75.2 |
| ±SD | | ±2.0 | ±3.1 | ±7.8 | ±2.9 | ±9.4 | ±10.6 | ±7.9 | ±13.6 | ±33.2 |
| p value* | | NS | ... | NS | <0.04 | NS | <0.05 | NS | <0.001 | ... |
| Controls | | | | | | | | | | |
| 7 | PS, mild | 14.0 | ... | 84 | 115 | 76 | -2 | ... | ... | ... |
| 8 | TOF repair PS, mild | 19.0 | ... | 74 | 125 | 78 | 0 | 178 | 168 | ... |
| 9 | PS, mild | 20.2 | ... | 90 | 136 | 75 | -2 | ... | ... | ... |
| 10 | PS, trivial | 13.7 | ... | 75 | 112 | 60 | -5 | 200 | 144 | ... |
| 11 | AS, mild | 13.6 | ... | 82 | 108 | 78 | 0 | 188 | 140 | ... |
| 12 | VSD repair | 19.5 | ... | 78 | 122 | 76 | -2 | 180 | 140 | ... |
| 13 | AS, trivial | 8.1 | ... | 95 | 110 | 73 | 1 | 170 | 141 | ... |
| Mean | | 15.4 | ... | 83 | 118 | 74 | -1.4 | 183 | 147 | ... |
| ±SD | | ±4 | ... | ±8 | ±10 | ±6 | ±2.0 | ±11 | ±12 | ... |

* Coarctation repair group versus control group.

AS = aortic stenosis; AsAo = ascending aorta; C of A = coarctation of aorta; DescAo = descending aorta; dias = diastolic; HR = heart rate; NS = not significant at $p < 0.05$; PS = pulmonic stenosis; Rt = right; SD = standard deviation; syst = systolic; TOF = tetralogy of Fallot; VSD = ventricular septal defect.

results, and another (Patient 6) had trivial mitral insufficiency. No patient had aortic insufficiency or aberrant right subclavian artery.

Outpatient blood pressure was measured by auscultation in the right arm and by the use of a Doppler flow probe over the posterior tibial artery. Blood pressure cuffs of appropriate size, covering at least two thirds of the limb, were used on the right upper arm and calf. All reported blood pressure values are the average of at least 3 measurements.

Dynamic exercise testing was performed on a motorized treadmill according to the Bruce protocol.¹² All children exercised to physical exhaustion defined as inability to exercise further despite vigorous encouragement from the staff to do so. Leads II, aVF, and V₅ were recorded throughout the exercise study. Blood pressure was measured by auscultation in the right arm and by the use of a Doppler flow probe over the posterior tibial artery. Blood pressure cuffs of appropriate size, covering at least two thirds of the limb, were used on the right upper arm and calf. Simultaneous supine arm and leg blood pressure measurements were made at rest (reported values are the average of at least 3 measurements) and within 30 seconds after the conclusion of exercise.

After obtaining informed consent, all patients underwent cardiac catheterization to evaluate their coarctation repair site

and to determine a possible explanation for their resting and exercise hypertension.

During diagnostic cardiac catheterization, resting baroreceptor function was evaluated in each patient before angiography. All patients received light sedation with morphine sulfate (0.1 mg/kg, maximum 5 mg) and diphenhydramine (1 mg/kg, maximum 50 mg). The R-R interval was measured directly from lead II of the electrocardiogram, recorded at a paper speed of 100 mm/s. Arterial pressure was recorded from a catheter situated in the aortic arch proximal to the site of C of A repair. Mean arterial pressure (MAP) was determined by electronic damping and was recorded, together with phasic arterial pressure, continuously throughout the study. Incremental changes in MAP were produced by injection (over 10 to 15 seconds) into the inferior vena cava of small doses of phenylephrine (50 to 150 μ g) and nitroprusside (25 to 50 μ g). Each drug was diluted in 0.9% sodium chloride and the total volume of each injection never exceeded 4 ml. In each subject, 3 or 4 doses each of phenylephrine and nitroprusside were administered to produce small (10%) to moderate (25%) increases and decreases in MAP. Three- to 5-minute intervals were taken between each drug injection to allow MAP and R-R interval to return to baseline levels. After each drug injection, MAP and R-R interval increased or decreased within

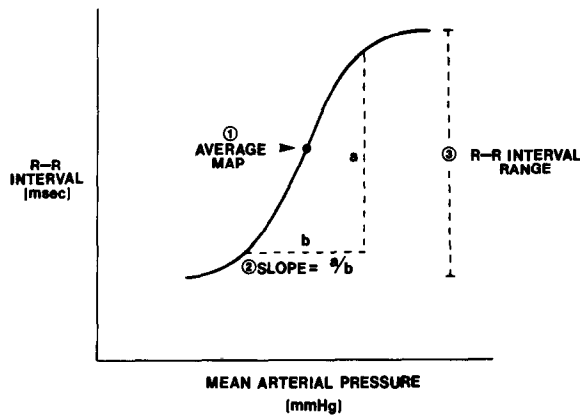


FIGURE 1. Idealized baroreceptor function curve. The curve is characterized by 3 parameters: (1) average baseline mean arterial pressure (MAP); (2) slope (a/b); and (3) R-R interval range.

15 seconds to a new plateau level which was sustained for 10 to 20 seconds. MAP was measured at baseline before, and at the plateau after each injection. The R-R interval was also measured at each baseline and plateau and the values reported represent the average of 3 consecutive R-R interval measurements. The range of MAP over which baroreceptor function was studied was comparable in the C of A repair and control group averages of 42.2 ± 2.7 mm Hg and 44.1 ± 2.9 mm Hg, respectively ($p = 0.61$).

Steady-state baroreceptor function curves were then constructed for each child by relating each R-R interval to its corresponding MAP value. Sigmoidal curves were constructed using the cumulative standard normal distribution function (ϕ)*. This sigmoidal function was utilized since previous studies have shown that the baroreceptor function curve is sigmoidal in nature.¹¹ Each patient's baroreceptor function curve was defined by the following equation: $RR = A + B \phi((MAP - \overline{MAP})/SD)$, where i represents phenylephrine or nitroprusside, RR represents each R-R interval, and MAP each corresponding MAP value. \overline{MAP} is the average baseline MAP for the patient. SD represents the standard deviation for each patient of the MAP values measured after drug injections either with phenylephrine or nitroprusside. Since the relationship between R-R interval and mean arterial pressure has been shown to be characterized by an asymmetric sigmoid curve,¹¹ 2 different standard deviations were used in constructing the upper and lower portions of the curve, respectively—one for the MAP values greater than \overline{MAP} (that is, after phenylephrine) and another for the MAP values less than MAP (such as after nitroprusside). This procedure permitted each curve to be asymmetrical if warranted by the data. A separate A and B were determined for each subject using least-squares linear regression, since data within subjects were correlated.

Each patient's baroreceptor function curve was characterized by 3 parameters: (1) average baseline MAP, (2) slope, and (3) R-R interval range. Figure 1 depicts an idealized baroreceptor curve with these 3 parameters indicated. Average baseline MAP (\overline{MAP}) is the average of all MAP values at baseline before each drug injection, and represents the set-point pressure about which the baroreflex operates. The slope of a baroreceptor function curve is an index of baroreflex

sensitivity to changes in arterial pressure. In this study the reported slope is the average slope of the linear portion of the curve over a pressure range defined as $\overline{MAP} \pm 1$ SD (standard deviation for each patient of all the MAP values measured after drug injections). For example, in Figure 1 the slope is a/b , where b represents $\overline{MAP} \pm 1$ SD. The R-R interval range is the difference between the R-R interval at the upper and lower plateaus of the baroreceptor function curves. This represents the R-R range over which the baroreflex operates.

Descending aortic angiograms were obtained on all 6 C of A patients. The descending thoracic aorta at the C of A repair site and at the level of the diaphragm were measured using calibrated grids. In the 7 control patients, the descending thoracic aorta was measured from either the levophase of a right ventricular angiogram or from a left ventricular angiogram.

Statistical significance was assessed using Student's t test. Group values are reported as mean \pm standard deviation.

Results

Arm systolic pressure at rest averaged 143.8 ± 2.9 mm Hg for the 6 children with repaired C of A (Table I). This ranks in the 90th percentile for systolic pressure according to the National Heart, Lung, and Blood Institute's blood pressure charts for children.¹³ Resting systolic pressure was significantly lower in the control group, averaging 118.3 ± 9.9 mm Hg ($p < 0.05$). Resting diastolic pressure was normal in both groups, averaging 74.5 ± 9.4 mm Hg and 73.7 ± 6.2 mm Hg in the C of A repair and control groups, respectively. The C of A pressure gradient at rest averaged 7.8 ± 10.6 mm Hg in the 6 children with repaired C of A. There was no significant difference between study and control patients with regard to resting heart rate. All children with repaired C of A had an abnormal blood pressure elevation in response to treadmill exercise (Table I). Upper extremity systolic pressure, measured immediately after exercise, averaged 223.3 ± 13.6 mm Hg in the coarctation repair group, compared with 146.6 ± 12.1 mm Hg for the control group ($p < 0.001$). There was no overlap of postexercise systolic pressure between the 2 groups. The coarctation pressure gradient increased to 75.2 ± 33.2 mm Hg immediately after exercise. Arm diastolic pressure immediately after exercise was not significantly different in the coarctation repair and control groups, averaging 73.5 ± 15.2 mm Hg and 66.6 ± 17.8 mm Hg, respectively. Maximal heart rate attained at peak exercise was also similar in the 2 groups. At cardiac catheterization, all children appeared to have a good hemodynamic repair of C of A, with peak systolic gradients from ascending to descending aorta ranging from 0 to 20 mm Hg (mean 8.5 ± 7.3 mm Hg) (Table II). In all 6 patients, aortograms demonstrated a mild indentation of the descending aorta at the site of coarctation repair. However, in no patient was this residual narrowing $< 70\%$ of the size of the descending aorta at the level of the diaphragm (Table II).

Baroreceptor function, as defined by the steady-state relationship between mean arterial pressure and R-R interval, was significantly different in the children with repaired C of A compared with control children. Figure 2 illustrates representative baroreceptor function curves of a child with repaired C of A and a control child. The

* $\phi(y) = \int_{-\infty}^y \frac{1}{\sqrt{2\pi}} e^{-x^2/2} dx$, where $y = (MAP - \overline{MAP})/SD$

TABLE II Pertinent Cardiac Catheterization in 6 Children With Repaired C of A and 7 Control Children

| Patient | HR (beats/min) | AsAo Pressure (mm Hg) | | | DescAo Pressure (mm Hg) | | | Size of Residual Narrowing (mm) | Size Descending Ao at Diaphragm (mm) |
|--------------------|-------------------|------------------------|--------------------|------------------------|-------------------------|----------------|--------------------|---------------------------------------|---|
| | | Syst | Dias | Mean | Syst | Dias | Mean | | |
| Coarctation Repair | | | | | | | | | |
| 1 | 96 | 153 | 101 | 117 | 150 | 100 | 117 | 14.4 | 15.7 |
| 2 | 84 | 128 | 83 | 97 | 123 | 80 | 97 | 15.3 | 19.4 |
| 3 | 76 | 148 | 82 | 100 | 138 | 80 | 99 | 11.8 | 15.9 |
| 4 | 80 | 145 | 80 | 100 | 145 | 80 | 100 | 16.6 | 21.4 |
| 5 | 78 | 138 | 78 | 97 | 118 | 76 | 89 | 10.0 | 14.0 |
| 6 | 72 | 142 | 78 | 100 | 129 | 76 | 96 | 12.5 | 17.8 |
| | 81 ±8.4 NS | 141.1 ±9.3 <0.05 | 83.6 ±8.7 NS | 101 ±7.5 p <0.01 | 132.6 ±14 NS | 82 ±9 NS | 99.6 ±9.3 NS | 13.4 ±2.4 ... | 17.4 ±2.7 NS |
| Control | | | | | | | | | |
| 7 | 84 | 112 | 70 | 83 | 120 | 70 | 82 | ... | 13.7 |
| 8 | 72 | 110 | 71 | 82 | 108 | 71 | 81 | ... | 17.3 |
| 9 | 93 | 125 | 78 | 93 | 130 | 77 | 94 | ... | 19.6 |
| 10 | 89 | 112 | 72 | 85 | 118 | 72 | 85 | ... | 16.7 |
| 11 | 82 | 118 | 74 | 88 | 120 | 73 | 88 | ... | 13.4 |
| 12 | 69 | 98 | 62 | 84 | 100 | 62 | 84 | ... | 17.4 |
| 13 | 88 | 123 | 88 | 99 | 123 | 88 | 99 | ... | 13.2 |
| | 82.4 ±8.9 | 114 ±9.1 | 73.5 ±8.0 | 87.7 ±6.2 | 117 ±9.9 | 73.3 ±7.9 | 87.5 ±6.6 | | 15.9 ±2.5 |

Grad = gradient; other abbreviations as in Table I.

data describe sigmoidal curves with well-defined slopes in the mid-portion of the curves and clear upper and lower plateaus. Table III lists the baroreceptor function curve characteristics for the 2 groups of children. The average baseline mean arterial pressure (MAP) was 108.8 ± 6.6 mm Hg for the repaired C of A group compared with 90.4 ± 8.6 mm Hg for the control group (p <0.01). Thus, the baroreflex has been reset to operate at high arterial pressure levels in the children with repaired C of A. The baroreceptor function curves of the children with repaired C of A have diminished slopes compared with control curves. In these children, the

slope averages 7.9 ± 3.7 ms/mm Hg, compared with 17.4 ± 5.1 ms/mm Hg in the control group (p <0.001), a 55% difference. This indicates diminished baroreflex sensitivity to changes in arterial pressure in the children with repaired C of A. Finally, the R-R interval range averages 246.7 ± 81.5 ms in the C of A repair group compared with 535.7 ± 97.2 ms in the control group (p <0.001). Figure 3 illustrates the composite baroreceptor

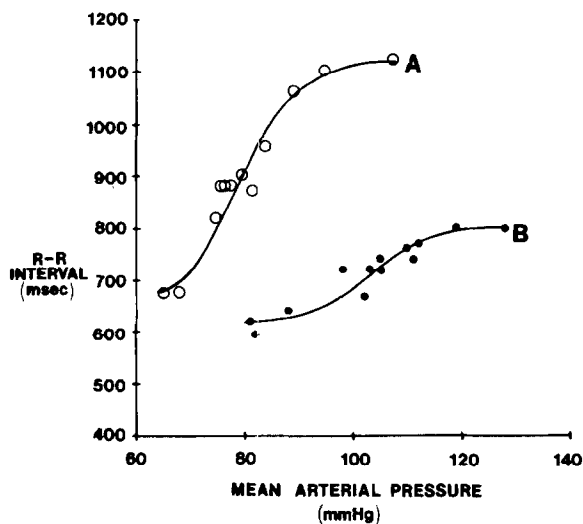


FIGURE 2. Representative individual baroreceptor function curves. A = curve of control, Patient 12. B = curve of coarctation repair, Patient 2.

TABLE III Baroreceptor Function Curve Characteristics in Children With Repaired Coarctation and in Control Children

| Patient | MAP (mm Hg) | Slope (ms/mm Hg) | R-R Interval Range (ms) |
|--------------------|-------------|------------------|-------------------------|
| Coarctation Repair | | | |
| 1 | 121 | 5.2 | 230 |
| 2 | 104 | 7.0 | 240 |
| 3 | 110 | 6.5 | 170 |
| 4 | 110 | 8.7 | 320 |
| 5 | 104 | 5.1 | 160 |
| 6 | 104 | 14.8 | 360 |
| Mean | 109 | 7.9 | 246.7 |
| ±SD | 7 | ±3.7 | ±81.5 |
| p value* | <0.01 | <0.001 | <0.001 |
| Controls | | | |
| 7 | 90 | 17.4 | 590 |
| 8 | 86 | 10.1 | 390 |
| 9 | 95 | 20.9 | 550 |
| 10 | 88 | 17.0 | 700 |
| 11 | 96 | 19.7 | 530 |
| 12 | 80 | 16.4 | 450 |
| 13 | 98 | 20.3 | 540 |
| Mean | 90 | 17.4 | 536 |
| ±SD | 9 | 3.6 | 97 |

* Compared with control values. MAP = average baseline mean arterial pressure; other abbreviations as in Table I.

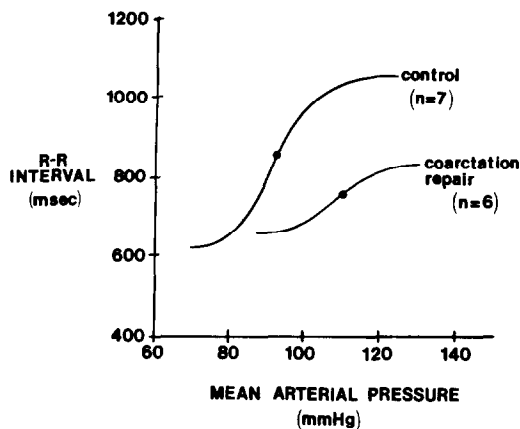


FIGURE 3. Composite baroreceptor curves of children with repaired coarctation and control children. The curves clearly differ in regard to average baseline MAP, slope, and R-R interval range.

function curves of the 6 children with repaired C of A and the 7 control children. These curves were constructed with the standard formula using averaged MAP, A, B, and SD (square root of averaged individual variances) values for the 2 groups. The two composite baroreceptor curves are clearly different. The C of A repair curve has been reset to a higher average baseline MAP and has a diminished slope compared with the control curve. The decrease in R-R interval range of the C of A repair curve is shown to be due to a lowering of the upper R-R interval plateau, indicating a diminished bradycardiac response to arterial pressure elevation.

Baseline arterial pulse pressure was higher in the children with repaired C of A than in the control children, averaging 58.6 ± 8.4 mm Hg and 40.4 ± 4.2 mm Hg, respectively ($p < 0.05$). After injection of phenylephrine or nitroprusside, however, the changes in pulse pressure were not significantly different between groups. The baroreceptor characteristics discussed above are not corrected for baseline pulse pressure differences. An adjustment for differences in pulse pressure would further increase the discrepancy in baroreceptor function between the 2 groups, since baroreflex sensitivity is known to be enhanced by increased pulse pressure.¹⁴

Discussion

The present study has documented altered baroreceptor function in a group of children with repaired C of A who have mild arm systolic hypertension. In these children the baroreflex is reset to operate at higher arterial pressure levels and has a reduced sensitivity to changes in arterial pressure compared with control children. The diminished baroreceptor sensitivity (slope) was marked, averaging only 45% of the control value. In addition, the baroreceptor curve of the C of A repair group had a reduced R-R interval plateau, due almost entirely to a decrease in the upper R-R interval plateau. The level of the upper R-R interval plateau is dependent upon the degree of vagal efferent excitation

and cardiac sympathetic inhibition, while the lower R-R interval plateau is determined by the degree of sympathetic excitation and vagal inhibition. We do not know which cardiac effector mechanism is altered in the children with repaired C of A but, based on studies of hypertensive adults,¹¹ the reduced upper R-R interval plateau seems more likely due to impaired vagal excitation than to impaired sympathetic inhibition.

The findings of the present study are similar to those of previous studies reporting altered baroreceptor function in adults with chronic essential hypertension.⁹⁻¹¹ Korner et al,¹¹ using a technique similar to ours to characterize steady-state baroreceptor function, also showed resetting, reduced baroreflex sensitivity, and a diminished R-R interval range in adults with chronic hypertension. In Korner's study the baroreceptor curve slopes of adults older than 30 years with severe resting hypertension (average MAP 151 mm Hg) averaged 11 ms/mm Hg compared with 21 ms/mm Hg for age-matched control subjects. These findings are quantitatively very similar to those of the present study. It is, however, important to note that although the technique of measuring heart rate response to injections of pressor and depressor drugs free of cardiac action is the most widely used test of baroreflex function in human subjects, it has the limitation of being unable to directly measure the baroreflex control of the peripheral circulation. Despite this, with the exception of denervation of either the carotid sinus or aortic arch baroreceptors,¹⁴ most studies in both man¹⁵ and animals^{14,16,17} have demonstrated that with intact afferents there is a positive correlation between the heart rate and the arterial pressure responses to a baroreceptor manipulation. Thus, although we have only documented an abnormal heart rate response to baroreceptor stimulation in our children after C of A repair, we feel that it is likely that an abnormality of baroreceptor regulation of vascular resistance is also present.

Baroreceptor function has not previously been evaluated in children or adults with C of A. Two previous studies have, however, demonstrated altered baroreceptor function in dogs with experimental coarctation. Bonchek et al⁷ reported resetting of the baroreflex to a higher pressure level, but normal reflex sensitivity, in 10 Labrador puppies with surgically created C of A. More recently, Iglar et al⁸ described both resetting and diminished baroreflex sensitivity in dogs with C of A studied under nonpulsatile flow conditions. Baroreceptor function was found to be normal after surgical repair of the experimental C of A.

The cause of altered baroreceptor function in hypertension has not been clearly elucidated. Although either the afferent or efferent limb of the reflex may be involved, most investigators have suspected that the changes reside in the afferent limb. Support for this view is found in animal studies, using single or multifiber preparations from the aortic and carotid sinus nerves, which have demonstrated an elevated threshold pressure (the pressure at which distinct nerve discharge first begins), an elevated saturation pressure (the pressure at which nerve firing no longer increases with increased pressure), and a decreased sensitivity (slope

of the stimulus-response curve over its linear range).^{8,18,19} Such diminished baroreceptor responsiveness to elevated arterial pressure may involve 1 or more mechanisms. First, diminished baroreceptor function in hypertension may involve dysfunction of the afferent neuronal baroreceptor fibers themselves. Generalized destruction or inactivation of these receptor fibers could, in theory, depress baroreflex sensitivity. Angell-James¹⁹ has presented histologic evidence of baroreceptor degeneration in the aortic arch of hypertensive rabbits. Similar findings in hypertensive persons have also been reported.²⁰ Second (and a more likely mechanism), with chronic hypertension, medial hypertrophy, elastic fiber degeneration, atherosclerosis, and changes in vessel sodium and water content may develop. These changes in the arterial wall may all act to diminish arterial wall compliance and thereby depress the baroreflex.²¹ In addition to the effects of chronic hypertension, morphologic arterial wall abnormalities may occur in patients with coarctation which may further decrease vessel compliance.²²⁻²⁴ Specifically, Sehested et al²⁴ have demonstrated, both by in vitro stimulation of aortic tissue from patients with C of A and by morphologic analysis, that the pre-coarctation aortic wall is more rigid and contains significantly more collagen and less smooth muscle than the postcoarctation wall. They speculated²⁴ that this reduced distensibility may influence baroreceptors in the upper vascular bed in such a way as to tolerate a higher pressure.

The findings of the present study suggest but do not prove that there may be a relationship between the observed abnormality in baroreceptor function and the presence of both resting and exercise hypertension in children with repaired C of A. There is no unanimity in the literature with regard to either the relationship between diminished baroreflex gain and hypertension^{25,26} or the ability of the baroreflexes to modify the cardiovascular response to exercise.²⁷⁻³⁰ Floras et al²⁷ demonstrated that adults with abnormal baroreflexes respond to a standard exercise test with a greater increase in blood pressure than that observed in adults with normal baroreflexes. Although altered baroreceptor function may contribute to both the maintenance of resting hypertension and the development of exercise hypertension after successful repair of C of A, we feel that it is not the major cause of the hypertension. Probably more important other causes include the magnitude²⁴ and duration¹ of preoperative hypertension, the mild residual narrowing of the descending thoracic aorta that is usually present after C of A repair,⁵ and the possible decreased distensibility of the ascending aortic wall.²⁴ Studies of the baroreceptor function in children with repaired C of A who have normal resting and exercise blood pressure would help to clarify these relationships.

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