Influence of Aortic Coarctation on Pulsatile Hemodynamics in the Proximal Aorta

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SUMMARY
Pressure and flow were recorded in the ascending aorta of three dogs with aortic coarctation, induced surgically 3 months previously, and in three control litter mates. From these data were derived input impedance of the systemic circulation, pulsatile and steady components of external heart work, aortic systolic pressure-time index, and mean systolic and mean diastolic pressures. Results were correlated with intraoperative measurements of aortic pressure in three young patients with aortic coarctation and with records taken in another 24 patients during diagnostic catheterization. Distinctive changes in aortic impedance with coarctation were explained in terms of altered peripheral reflection. Such changes were responsible for characteristic alteration in central aortic pressure pulse contour and for change in other parameters, indicating impaired arterial function in accepting pulsatile flow from the heart. Such changes in the cushioning function appear responsible for many clinical features and complications of aortic coarctation. Surgery is probably justified for restoration of the normal cushioning function of the arterial system as well as for reduction of resistance and mean arterial pressure.

Additional Indexing Words:
Vascular impedance Heart work
Medionecrosis Aortic rupture
Harmonic analysis Windkessel
Ruptured aneurysm Wave contour

The traditional reason given for surgical correction of aortic coarctation used to be that correction leads to relief of hypertension in the upper part of the body. It is now appreciated that this explanation is probably incomplete, since untreated postductal coarctation is associated with a different spectrum of complications and appears to carry a worse prognosis than simple hypertension of equal severity.1-6

In aortic coarctation and hypertension, peripheral resistance and mean arterial pressure are high, but in aortic coarctation there is in addition marked reduction in the dimensions of that part of the arterial system that is effectively pulsating. It is possible that many of the clinical features and complications of aortic coarctation may be due not to increased resistance to flow from the ventricle but to decrease in size of the arterial compression chamber.

The purpose of this paper is to demonstrate the changes in pulsatile hemodynamics in the proximal aorta that result from aortic coarctation and that are attributable to impairment of the normal cushioning function of the arterial system. We will describe the mechanism by which these changes occur and the ill effects they may have; further, we will discuss the relevance of these findings to the natural course of aortic coarctation and the implications with respect to management.

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Methods

Data were obtained from six dogs and from three patients studied at operation immediately prior to correction of aortic coarctation. Additional information was obtained from review of diagnostic catheterization records of 24 patients with aortic coarctation.

Animals

Dogs were mongrel litter mates aged 37–39 weeks at the time of study. We induced coarctation in three dogs at 26 weeks of age by tying a heavy silk ligature around the aorta at the level of the fifth thoracic vertebra. The ligature was tightened until the pressure pulse in the femoral artery became impalpable. At this stage the aorta was found to have been narrowed to approximately one-third of its original diameter; there was a prominent thrill in the vessel downstream. Postoperative convalescence was uneventful, and the dogs grew normally over the following 3 months. The remaining three animals did not undergo surgery and served as controls.

Hemodynamic studies were performed in the tenth month of life. Animals were anesthetized with pentobarbital (20–40 mg/kg iv) and ventilated with a Harvard pump. Small supplemental doses of pentobarbital were given as required. The ascending aorta was exposed through a right thoracotomy, and an electromagnetic flow transducer of appropriate size was placed about it. Pressure was measured through a needle inserted into the ascending aorta immediately distal to the flow transducer; the needle bevel was orientated to face the direction of flow. In dogs with coarctation, pressure was also measured in a side branch of the femoral artery. A bipolar catheter was advanced to the right atrium from the external jugular vein, and was attached to an electric stimulator through an isolating transformer.

The sine wave electromagnetic flowmeter used was a modification of that described by Kolin and Kado. Transducers were constructed according to the method of Kolin and Wisshaupt. Zero flow baseline was taken as the integrated level during diastole. Steady flow calibration was determined by passing saline through an excised artery enclosed within the probe. Dynamic characteristics of the flowmeter have been described; allowance was made for flowmeter frequency response in calculations. Pressure was measured with a Statham P267B manometer. This was calibrated against a column of mercury, and dynamic characteristics were determined by the pressure step or "pop" method; damped natural frequency averaged 66 Hz and damping coefficient, 0.47. As with flow, allowance was made for dynamic response in calculations.

Data were recorded on photographic paper and magnetic tape. Records were taken with the heart beating regularly and also during irregular cardiac contraction which was induced by random electrical stimulation of the right atrium.

The relationship between pressure and flow in the ascending aorta was expressed as vascular impedance. This was determined by relating corresponding frequency components of simultaneously recorded pressure and flow waves by Fourier or frequency spectrum analysis, as described previously. External heart work was calculated from records obtained with the heart beating regularly, and was divided into steady and pulsatile components. From the same records systolic pressure-time index and mean systolic and mean diastolic pressure were calculated.

Patients

The three patients were aged 9, 10, and 19 years, and had uncomplicated postductal aortic coarctation. Pressures were recorded in the aortic arch and in the descending thoracic aorta below the coarctation with Statham P267B manometers through needles inserted into the vessel wall. Data were recorded on photographic paper. Damped natural frequency of the manometer assembly in all cases exceeded 25 Hz. When recording details of the aortic pressure wave, it is desirable to have as high a natural frequency as possible. For practical purposes, a natural frequency of at least 12 Hz is necessary to record the arterial pressure wave with any degree of accuracy. In the human catheterization laboratory the limit is set by the fluid-filled tubing and connections rather than by the pressure transducer itself.

Results

Dogs

Data from all dogs are given in table 1. In the ascending aorta mean arterial pressure was greater in the dogs with coarctation (119 mm Hg) than in the controls (109 mm Hg). In the dogs with coarctation there was a difference in mean pressure of 3–15 mm Hg (average, 9 mm Hg) between the ascending aorta and femoral artery. There was no apparent difference in contour of the ascending aortic flow wave between the two groups, although cardiac output was greater in controls (21.1 compared to 15.0 ml/sec).

The most obvious difference was in the contour of the ascending aortic pressure wave
Table 1

<table>
<thead>
<tr>
<th>Dog</th>
<th>Heart rate (beats/sec)</th>
<th>Mean cycle arterial pressure (mm Hg)</th>
<th>Pulse pressure (mm Hg)</th>
<th>Cardiac output (ml/sec)</th>
<th>Pulsatile external LV work (watts)</th>
<th>Steady external LV work (watts)</th>
<th>Pressure-time index/cardiac output (mm Hg-sec/ml)</th>
<th>Pressure-time index (mm Hg-sec/sec)</th>
<th>Mean systolic minus mean diastolic aortic pressure (mm Hg)</th>
<th>Pulsatile external LV work / total external LV work (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>2.44</td>
<td>100</td>
<td>16</td>
<td>16.0</td>
<td>0.0064</td>
<td>0.213</td>
<td>37.2</td>
<td>2.33</td>
<td>5.8</td>
<td>2.9</td>
</tr>
<tr>
<td>2</td>
<td>3.57</td>
<td>125</td>
<td>14</td>
<td>22.5</td>
<td>0.0096</td>
<td>0.375</td>
<td>52.8</td>
<td>2.34</td>
<td>9.8</td>
<td>2.5</td>
</tr>
<tr>
<td>3</td>
<td>3.08</td>
<td>103</td>
<td>25</td>
<td>24.7</td>
<td>0.0257</td>
<td>0.940</td>
<td>41.1</td>
<td>1.66</td>
<td>7.5</td>
<td>7.0</td>
</tr>
<tr>
<td>Mean</td>
<td>3.03</td>
<td>109</td>
<td>18</td>
<td>21.1</td>
<td>0.0139</td>
<td>0.300</td>
<td>43.7</td>
<td>2.11</td>
<td>7.7</td>
<td>4.1</td>
</tr>
<tr>
<td>Coarctation</td>
<td>2.50</td>
<td>104</td>
<td>23</td>
<td>12.4</td>
<td>0.0092</td>
<td>0.171</td>
<td>44.4</td>
<td>3.61</td>
<td>12.1</td>
<td>5.1</td>
</tr>
<tr>
<td>2</td>
<td>2.56</td>
<td>118</td>
<td>33</td>
<td>19.7</td>
<td>0.0182</td>
<td>0.310</td>
<td>49.8</td>
<td>2.53</td>
<td>15.4</td>
<td>5.6</td>
</tr>
<tr>
<td>3</td>
<td>3.70</td>
<td>134</td>
<td>41</td>
<td>12.8</td>
<td>0.0290</td>
<td>0.229</td>
<td>56.5</td>
<td>4.41</td>
<td>18.4</td>
<td>11.2</td>
</tr>
<tr>
<td>Mean</td>
<td>2.92</td>
<td>119</td>
<td>32</td>
<td>15.0</td>
<td>0.0188</td>
<td>0.237</td>
<td>50.2</td>
<td>3.52</td>
<td>15.3</td>
<td>7.3</td>
</tr>
</tbody>
</table>

Abbreviation: LV = left ventricular.

Data from Dogs
with two fluctuations in impedance phase. In the dog with coarctation there was only one minimum of impedance modulus at 8.5 Hz, corresponding to the second minimum in the control dog, and there was little fluctuation in impedance phase. The features described were seen in all control animals and in all with coarctation (fig. 3). In two animals with coarctation, impedance was determined again after the aortic obstruction was successfully relieved and the normal pattern was reestablished; at the same time amplitude of the ascending aortic pressure pulse decreased, and a diastolic wave reappeared.

Coarctation of the aorta led to loss of the first minimum of impedance modulus at 3–4 Hz and so, to increase in values of impedance modulus between 1 and 8 Hz (figs. 2 and 3). Associated with this was relative increase in the first and second harmonics of the ascending aortic pressure wave. The pulsatile component of external heart work (heart work lost in arterial pulsations) \(^{14,15}\) is largely determined by impedance over this frequency band, since most of the energy of the ventricular ejection wave is in the first and second harmonics \(^{13}\) (fig. 2). Magnitude of pulsatile work is given by the formula:

\[
\text{Work} = \int P \cdot \frac{dV}{dP}
\]
Figure 3

Input impedance to the systemic circulation in three normal dogs (left) and in three litter mates with aortic coarctation (right).

\[ W_p = \frac{1}{2} \sum_{n=1}^{N} (Q_n)^2 \cdot Z_n \cos \phi_n \]

where \( Q_n \) is the amplitude of the \( n \)th flow harmonic and \( Z_n \) and \( \phi_n \) are the modulus and phase, respectively, of impedance at the frequency of the \( n \)th harmonic.\(^{15}\) On the average, the ratio of pulsatile to total (pulsatile + steady) external heart work was greater in the dogs with coarctation (7.3%) than in controls (4.1%) (table 1). The ratio was not, however, as high as one might have expected from impedance modulus alone, and this was due to the increase in phase angle \( \phi \) (and so, reduction in cosine \( \phi \)) which accompanied the elevated impedance modulus \( Z \) between 1 and 8 Hz.

The major determinant of myocardial oxygen consumption is the left ventricular tension-time index.\(^{17}\) This is approximated by the aortic pressure-time index, which was greater in the dogs with coarctation than in controls (average, 50.2 compared to 43.7 mm Hg·sec/sec; table 1). When expressed in relation to cardiac output, the difference was greater (average, 3.52 compared to 2.11 mm Hg·sec/ml), indicating increased energy expenditure by the heart for any given cardiac output.

By decreasing dimensions of the arterial "compression chamber," aortic coarctation alters the relationship between mean systolic and mean diastolic pressures. It will be argued...
later that the difference between mean systolic and mean diastolic pressures in the ascending aorta is an (inverse) measure of arterial efficiency in relation to cardiac performance. In the three dogs with coarctation, mean systolic pressure minus mean diastolic pressure in the ascending aorta averaged 15.3 mm Hg against 7.7 mm Hg in controls (table 1).

Patients
Pressure waves similar to those seen with acute or chronic aortic coarctation in dogs were observed in three young patients with coarctation who were studied during surgery. A typical record is shown in figure 4, taken from a 9-year-old boy. Pulse pressure (70 mm Hg) is much greater than normal. The peak of the wave is sharp and occurs late in systole, and there is no diastolic wave. In normal children the peak of the proximal aortic pressure wave is broad, and a diastolic wave is almost invariable; wave contour in figure 4 is more like that seen in elderly patients with arterial degeneration than in a normal 9-year-old child.

The findings in animals and children suggested that the proximal aortic pressure wave is deformed in characteristic fashion by aortic coarctation. To test this thesis, we reviewed catheterization records of all patients with uncomplicated postductal aortic coarctation studied during a 5-year period at two major Sydney hospitals. There were 24 patients aged 7–45 years (average, 25 years). Interpretation was not always easy because of the low natural frequency of the manometer system in many cases. Ascending aortic pulse pressure was high (average, 75 mm Hg), and the peak of the wave was sharp and occurred late in systole; what appeared to be a well-developed diastolic wave was seen in only one of 24 patients (and even in this patient it was not possible to be sure the wave was not due to manometer resonance). By way of contrast, a definite diastolic wave was seen in the proximal aortic pulse in 22 of 42 adults without coarctation studied during diagnostic catheterization at one of these hospitals.18

Discussion
There have been many reports on altered hemodynamics in aortic coarctation.19–28 The main interest has been the mechanism of hypertension and the question of whether there is or is not a general increase in arteriolar resistance in addition to increased resistance at the constricted aorta and the collateral vessels.20–22, 24, 26 Preoccupation with the conduit function of the arterial system and its disturbance in coarctation appears to have shifted emphasis away from where, from a pragmatic point of view, it may more properly be directed—that is, to the disturbance in the "Windkessel" or cushioning function of the arterial system. In aortic coarctation the physical dimensions of the effectively pulsating part of the arterial system are considerably reduced, and there are in consequence marked alterations in pulsatile hemodynamics in the proximal aorta and arteries in the upper part of the body. Though not entirely neglected,20, 24, 27, 28 it is surprising that such little attention has been devoted to this subject when the major causes of death in aortic coarctation are attributable to stresses and strains on the heart, proximal aorta, and cerebral arteries.1, 4–6 The lack of data on altered hemodynamics in the proximal aorta in aortic coarctation, the deficiency of emphasis

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on pulsatile phenomena in this condition, and the confusion on mechanisms that exists in the literature prompted this report.

Impedance

The concept of vascular impedance has proved useful in describing the relationship between pressure and flow in a vessel and in explaining the properties of the vascular bed downstream. The concept appears singularly appropriate for describing and explaining altered hemodynamics that result from large artery disease. Studies of impedance in the ascending aorta and large arteries of dogs have suggested that there are in the systemic circulation two functionally discrete reflecting sites—one close to the heart and representing the resultant of all individual reflecting sites in the upper part of the body and the other further from the heart and representing the resultant of all individual reflecting sites in the lower part of the body. The position of these sites is illustrated by the asymmetric T-shaped model of the normal arterial system shown at the left in figure 5. In a normal dog, the first minimum of impedance modulus and corresponding fluctuation in phase (at 3.75 Hz in fig. 2) are attributable to wave reflection in the trunk and lower limbs (represented in the model by the end of the longer tube). In dogs with coarctation, the first minimum of impedance modulus and the corresponding fluctuation in phase are absent (figs. 2 and 3); this is attributable to loss of the echo from the lower part of the body. The distal reflecting site in the lower body is replaced by another site (the aortic constriction itself) very much closer to the heart. The relevant model of the arterial system is that at the right in figure 5. The second minimum of impedance modulus (at approximately 8 Hz in figs. 2 and 3) is largely due to wave reflection in the upper part of the body. As expected, this is preserved in aortic coarctation. In dogs with coarctation, reflection from the narrowed aorta might be expected to result in a further minimum of impedance modulus above 8 Hz. This was not definitely

Figure 5

Model of the normal arterial system (left) and the arterial system in aortic coarctation (right), together with pressure waves recorded in a normal dog (left) and in a dog with aortic coarctation (right) at points corresponding to their position in the respective model. Superimposed on the waves are lines with arrows, which show the impulse generated by ventricular ejection passing back and forth over the arterial system at the velocity of the pulse (approximately 6 m/sec). It is shown that contour of the waves is attributable to summation of incident and reflected components. Models as previously suggested.
seen; its absence is attributable to interaction with reflections from the upper body site together with low signal/noise ratio at frequencies above 8 Hz.

Changes in impedance patterns in dogs with chronic aortic coarctation are readily explained from theoretic principles. These changes are little different from those seen in acute coarctation, with the possible exception of a lower (i.e., less negative) impedance phase. The shape of ascending aortic impedance curves in aortic coarctation is superficially similar to that seen in experimental hypertension of severe degree. In both, the minimum of impedance modulus occurs at a higher frequency than normal. In both, reflected waves return to the ascending aorta sooner than usual; but in coarctation this is due to a new reflection site which is anatomically close to the heart, whereas in hypertension it is due to more rapid travel of the pressure wave between the heart and normally placed peripheral reflecting sites.

Contour of the Pressure Wave in the Proximal Aorta

Alteration in peripheral reflection explains the characteristic contour of the aortic pressure wave. This is illustrated in figure 5, which shows how replacement of the distant reflection site in the lower body by one much closer to the heart accounts for all features noted—high pulse pressure, presence of a late systolic peak, and absence of a diastolic wave. As would be expected, similar pressure waves are seen in hypertension and arteriosclerosis.

Typical features of the aortic pressure wave in coarctation are obvious in the records of Gupta and Wiggers, who rejected the possibility of reflected waves being responsible for any of the features of the pressure pulse and who attributed increased pulse pressure to increased stroke volume and displacement of peak pressure into late systole to increased peripheral resistance. Gupta and Wiggers made no comment on the decrease in amplitude and eventual loss of the diastolic wave in the proximal aorta with increasing constriction of the descending thoracic aorta. Fuller and his associates recorded pressure waves similar to those in figure 4 in patients with aortic coarctation, but they did not draw attention to any abnormality in contour; neither did Wright and Wood in a later survey. We would regard a pressure wave like that in figure 4 as being definitely abnormal in a child and probably abnormal in a young adult. A definite diastolic wave in the proximal aortic pulse is usually seen in children, often in young adults, but rarely in the elderly. Absence of a diastolic wave in patients without coarctation is attributable to hypertension, arterial degeneration, or prolongation of ventricular systole.

There are in the literature some unusual interpretations of pressure wave contour in aortic coarctation. These should be challenged because they are still widely quoted. On the basis of elevated diastolic pressure in the femoral artery, Steele inferred increased resistance in arterioles in the lower part of the body; Bing et al. correctly pointed out that this was due simply to decreased oscillation around a lowered mean pressure. Woodbury et al. showed great increase in brachial pulse pressure and in pressure gradient across the coarctation after intravenous infusion of nor-epinephrine; the authors attributed this to constriction of collateral vessels and decreased distensibility of the proximal aorta rather than to increase in stroke volume. Gupta and Wiggers attributed increase in proximal aortic pulse pressure to a combination of increased stroke volume, increased peripheral resistance, and decreased aortic distensibility, and rapid fall of pressure during diastole to increased coronary flow. This explanation appears unnecessarily complex when decreased aortic dimensions with decreased distensibility can account for all findings.

Left Ventricular Function

Impedance derived from measurements of pressure and flow in the ascending aorta may be used not only to characterize the properties of the peripheral vascular bed but also to describe the load presented to the heart for intermittent ejection. In dogs most of the energy of the left ventricular ejection wave is contained in the frequency band 1–8 Hz.
experimental coarctation, impedance modulus is increased over this range; as a consequence pressure moduli generated by flow moduli over this range are increased. In more conventional terms, the heart must generate greater swings of pressure in the aorta in delivering the same amount of blood, quite apart from any greater mean pressure it may need to develop in order to maintain flow through the increased resistance.

The conventional division of external heart work into kinetic and potential components is from a functional point of view less useful than separation into pulsatile and steady components. Steady external work represents energy lost in maintaining forward flow and is expended mainly in the arterioles and capillaries. Pulsatile external work represents energy lost in pulsations and is dissipated almost exclusively in arteries. One would expect both to be increased in aortic coarctation (when cardiac output is normal), but from consideration of the functional defect and the changes in aortic impedance modulus one might expect pulsatile work to be disproportionately increased. In this series pulsatile work was greater relative to steady work in the dogs with coarctation compared to controls; that it was not greater still has been explained on the basis of the accompanying change in impedance phase. Impedance phase is decreased during peripheral vasodilatation, so that one would expect the most striking changes in pulsatile work with coarctation of the aorta to be seen during activity and exercise rather than under resting control conditions.

Porjé measured the pulsatile and steady components of external left ventricular work in patients with aortic coarctation and in a control group with normal arteries. He showed that the ratio of pulsatile to total external work was considerably higher in the former (24 and 37%) than in the latter (mean 14%; range 12–17%).

In this series of experiments the ratio of pulsatile to total external left ventricular work was less (even in dogs with coarctation) than in a previous series of normal dogs studied in the same laboratory. However dogs in the present series were only 9 months old at the time of study, whereas animals in the previous series were much older. Degeneration of arteries with age may explain the difference in results. This subject is being pursued further.

External heart work is only a fraction of total heart work and bears no constant relationship to myocardial oxygen needs. It has been shown that the major determinants of myocardial oxygen requirements are the tension developed by the ventricle during systole and the time this tension is held. The ventricular tension-time index is reflected by the ascending aortic pressure-time index. This was greater in the dogs with coarctation and greater still when expressed as a fraction of cardiac output. Such an alteration in coarctation indicates greater energy cost by the left ventricle per unit output, and this is a result both of the increased peripheral resistance and decreased size of the arterial compression chamber.

While the pressure maintained in the ascending aorta during systole is a major determinant of myocardial oxygen and blood requirements, the pressure maintained in the ascending aorta during cardiac diastole is the major determinant of coronary blood flow. The difference between mean systolic and mean diastolic pressures in the ascending aorta may be taken as an inverse index of the efficiency with which the arterial system couples with the heart—efficiency with which the system accepts pulsatile flow from the heart and delivers the heart's blood requirements through the coronary arteries. For optimal function there would be no pressure fluctuation in the ascending aorta, and mean systolic, mean diastolic, and mean cycle pressures would be the same. This is not possible to achieve, so that the arterial system has to be inefficient to some extent. The normal design of the arterial system is such as to maintain the difference between mean systolic and mean diastolic pressures at a very low value. Stiffening of arteries or decrease in their physical dimensions leads to increase in the difference between mean systolic and
mean diastolic pressures in the ascending aorta\textsuperscript{14, 16} and so to impairment in arterial efficiency and compromise in cardiac function. Counterpulsation\textsuperscript{33, 34} elevates mean diastolic pressure and reduces mean systolic pressure and so artificially improves the efficiency with which the arterial system couples with the heart. It is surprising that while the hemodynamic advantages of counterpulsation are well known,\textsuperscript{33, 34} the deleterious effects of arterial disease on cardiac performance and coronary flow have received little prominence. In the dog experiments reported here the difference between mean systolic and mean diastolic pressures was doubled in the presence of aortic coarctation, indicating that in aortic coarctation the left ventricle performs at considerable hemodynamic disadvantage. This disadvantage is due to decreased volume distensibility of the arterial system and not to increased peripheral resistance. As with pulsatile heart work the disadvantage is likely to be more marked during activity when cardiac output is higher and peripheral resistance lower.

**Effects of Altered Arterial Hemodynamics in Coarctation**

The hemodynamic changes described affect the heart and arteries. The heart is made less efficient in pumping intermittently because it faces a higher pressure during systole, while its muscle is perfused by a relatively lower pressure during diastole. This hemodynamic disadvantage is additive to the elevation in mean pressure that results from increased vascular resistance. All factors impair cardiac performance and must contribute to development of cardiac failure.

Arteries are affected by the large and rapid fluctuations in pressure that occur within them and that impose great stresses on the arterial wall. Accelerated arterial degeneration including atheroma,\textsuperscript{35} medionecrosis,\textsuperscript{36, 37} and development of cerebral aneurysms\textsuperscript{37} are attributable to these increased stresses, as are their further complications including myocardial infarction, dissection of the aorta, and cerebral hemorrhage. All told, the most common complications of aortic coarctation are almost certainly due to large and sudden pressure fluctuations secondary to impairment of the normal cushioning function of the arterial system rather than to sustained elevation of mean pressure secondary to interference with its conduit function. Such a suggestion is not new; effects of impaired cushioning on the heart and arteries were discussed by Mackenzie in 1902\textsuperscript{28} and amplified by others\textsuperscript{39} in subsequent years.

These considerations are relevant to the problems of medical management of patients with aortic coarctation and surgical correction of the lesion. With regard to medical management it would seem desirable to avoid the situations where great strains are placed on the heart and arteries in the upper part of the body\textsuperscript{27} and so to limit strenuous exertion and vigorous competitive sport. With regard to surgical correction of aortic coarctation one's aim should be to restore the normal anatomic structure of the aorta in order to forestall the development of arterial disease and to improve cardiac performance. One should not aim just to relieve hypertension, nor should one wait for the development of symptoms or complications. In older children and adolescents without associated defects, surgical correction of aortic coarctation under optimal conditions carries a mortality of 1-2\%. This risk of operation is far lower than the continuing danger of lethal complications in untreated patients.\textsuperscript{6}

These arguments support the current practice in most centers of advising surgery in all patients with correctable uncomplicated aortic coarctation of significant degree (mean pressure gradient $> 10$ mm Hg across the coarctation) irrespective of the presence or absence of complications and of the absolute systolic and diastolic blood pressure levels.

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