Distalward Modification of the Arterial Pulse Wave in Children with Congenital Aortic Stenosis

By P. CHARD E. HAWKER, M.B., M.R.A.C.P., CARLOS A. SEARA, M.D., and L. JEROME KROVETZ, M.D., PH.D.

SUMMARY

In 38 children with congenital aortic stenosis (32 valvar, three subvalvar, and three supravalvar: 28 preoperative, ten postoperative), the left ventricular pressure, left ventricular-to-aortic pressure difference and aortic valve area were compared with several components of the central and peripheral arterial pulse waves. In 23 patients, a catheter-tip manometer was used, and in 15, pressure was measured with a regular manometer system and corrected for artifact by resynthesis of pulse waves after harmonic analysis. The severity of the anomaly was not reflected in the central or peripheral pulse pressure, the first derivative of these pressures, mean aortic pressure, pulse wave velocity, or peripheral amplification of pulse waves or of their individual harmonics.

While in adults with aortic stenosis, the various pulse wave characteristics correlate with severity, the change in the pulse wave as it travels down the aorta in children is so modified by vascular factors as to be worthless in assessing the severity of the obstruction.

THE SLOWLY RISING, narrowed pulse characteristic of aortic stenosis was first recognized in the nineteenth century. Since then, many components of the pulse wave have been thought to be related to the severity of the stenosis. Pulse wave parameters such as peak dp/dt, the slope of the first two-thirds of the systolic upstroke and ejection time have correlated sufficiently well to be used to assess the severity of aortic stenosis in adult patients.

However, we have been impressed by the considerable modification the pulse wave undergoes in travelling peripherally in the healthy compliant arteries of children, and we wondered if these correlations would apply to pediatric patients with congenital aortic stenosis.

We, therefore, planned a study of the behavior of intravascularly recorded central and peripheral pulse waves in congenital aortic stenosis. This would serve as a background for the later assessment of the value of systolic time intervals and other parameters measured by noninvasive techniques in a similar group of patients.

Materials and Methods

Thirty-eight studies were performed in patients with aortic stenosis, three discrete, membranous subvalvar, three supravalvar, and 32 of the valvar type. The patients, whose ages ranged from three to 16 years, were studied during elective cardiac catheterization. Twenty-eight studies were preoperative and ten were performed electively about one year following surgical repair. Data from nine patients without cardiac disease were available for control values. Aortic root angiograms were available in all patients and patients with aortic regurgitation of any significant degree were excluded from the study. Patients were catheterized in a supine position after premedication with meperidine, promethazine, and chlorpromazine, or with fentanyl and droperidol (Innovar).

In 33 patients, a catheter was inserted percutaneously into the right femoral artery, and in five, a cutdown on the right brachial artery was made. In 23 patients a catheter-tip manometer was used and in the remainder, standard Goodale-Lubin or Gensini catheters were used. Frequency response of the standard catheters was determined by the "pop" method, or by the flush method in which the manometer system is opened momentarily to a pressure of 900 mm Hg and the time period of the resultant oscillations

From the Departments of Pediatrics and Biomedical Engineering, The Johns Hopkins University School of Medicine, The Johns Hopkins Hospital, Baltimore, Maryland.

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Dr. Hawker's present address is Royal Alexandra Hospital for Children, Camperdown 2050, Sydney, New South Wales, Australia. Dr. Seara's address is Hospital Durand, Diaz Velez 5044, Buenos Aires, Argentina.

Address for reprints: Dr. L. Jerome Krovetz, Department of Pediatrics, University of Virginia Hospital, Charlottesville, Virginia 22901.

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measured. Frequency response ranged from 17 to 69 Hz (mean, 41 Hz) and damping factor \( \alpha \) from 0.057 to 0.274 of critical.*

Pressure recordings were made in root of aorta, arch of aorta, and iliac artery at the level of the sacro-iliac joint within a five minute time span and recorded together with the ECG on a Honeywell 7600 multichannel analog tape recorder. This was later played back on a Tektronix R564B storage-type oscilloscope with the sweep triggered by the QRS complex of the ECG. The time from the QRS complex to the start of the pressure pulse was determined at each recording site. Arterial transmission time between root and arch and between arch and iliac artery was determined by the average difference in these intervals for ten consecutive beats. The distance (cm) between two recording sites was obtained by measuring the length of catheter withdrawn, and division by transmission time gave the pulse wave velocity.

The first derivative of both arch of aorta and peripheral arterial pulses was obtained by digital computer in 23 patients in whom a catheter-tip manometer was used. Pulse waves were digitized at 5 msec intervals, and second order equations derived for triplet points, differentiated, and peak \( dp/dt \) measured. Pulse wave amplification ratios were obtained by dividing the peripheral (iliae or brachial) pulse pressure by the aortic arch pulse pressure.

In this study, harmonic analysis was employed both for correction of artifact* (unpublished data) and for a more detailed assessment of pulse wave characteristics. Analysis was obtained by way of the discrete Fourier transform, using a digital computer. A pressure wave, like any periodic function, consists of many sinusoidal harmonics, each of which can be characterized by an amplitude (modulus) and a phase angle. In the 15 patients in whom a fluid-filled catheter-manometer system was used, harmonic analysis of the pressure waves allowed corrections to be made for manometer frequency characteristics which were applied to moduli and phase angles. Resynthesis of the first 20 harmonics then resulted in a wave with significantly less artifact (unpublished data).

A convenient method of comparing the harmonic profiles in different patients is to normalize these by making the amplitude of the first harmonic equal to one, and that of subsequent harmonics a fraction thereof (fig. 1). In aortic stenosis, the first few harmonics have a higher amplitude than those in patients with a normal value, while the degree of peripheral amplification of an individual harmonic depends on both its position in the harmonic profile and on the artery through which it travels.**

Cardiac outputs were determined by dye dilution technique as soon as practicable after measurement of the left ventricle-aortic pressure difference. Aortic valve areas were calculated from the Gorlin formula. All measurements were obtained prior to injection of contrast media to avoid possible changes in circulatory dynamics.

Left ventricular peak systolic pressure, left ventricle-aortic pressure difference and aortic valve area, the conventional indices of severity of aortic stenosis, were compared to

\[
\alpha = \frac{\sqrt{\lambda^2}}{4\pi^2 + \lambda^2}
\]

where \( \lambda \), the logarithmic decrement, is the natural logarithm of the ratio of the first and second oscillations in response to the step function.**

(a) central and peripheral aortic pulse pressures, (b) the first derivative of these pressures, (c) the first derivative of the central aortic pressure corrected for arterial compliance by using the method of Arani et al.* in which \( dp/dt \) is multiplied by the stroke volume divided by pulse pressure, (d) pulse wave velocity, and (e) peripheral amplification of pulse waves.

**Results**

The cumulated data for 38 patients is shown in table 1. Multiple comparisons were made between the three indices of severity and the variables under investigation both for the whole series and for the subgroups of aortic stenosis. We were unable to demonstrate significant differences either in arterial pressure or pulse wave transmission of these subgroups. The results for peak left ventricular systolic pressure and peak left ventricle-aortic systolic pressure difference were similar to those for aortic valve area. Because of severe catheter whip artifact on pullback across the aortic valve, valve areas were not able to be calculated in ten patients.

Table 2 shows the correlations obtained when pulse wave parameters are compared with aortic valve area. Neither the patients with preop ASV nor the entire group showed significant correlation of any pulse wave parameter with severity. The wide variation in results is illustrated in figure 2a and b. The subvalvar and supravalvar aortic stenosis groups were too small for separate statistical analysis. Even after exclusion of the patients with a milder degree of stenosis (valve area > 0.75 cm²/m²), no significant correlation of aortic valve area and peripheral amplification was found. Values for amplification between arch and brachial artery were available in only three patients, but these did not differ from amplification between arch and iliac arteries. In the eight postoperative valvar stenosis

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*HAWKER, SEARA, KROVETZ

Figure 1

Normalized harmonic moduli of aortic root pressure waves in four normal patients.

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patients, a significant correlation was found between aortic valve area and peripheral amplification, \( r = 0.81 \). However, the mean amplification ratio of 1.22 in this group did not differ significantly from the ratios of 1.39 for the preoperative aortic valve stenosis group and 1.35 for nine patients without cardiac disease. Reference to table 1 shows that the calculated aortic valve areas in the postoperative group were generally small.

Examination of the behavior of the individual harmonics also produced disappointing results. Figure 3 shows that the normalized pressure modulus of the first 12 harmonics of aortic root pressure waves in five patients with mild aortic valve stenosis did not differ from those with severe stenosis. Moreover, the harmonic profiles do not differ significantly from the four normal subjects shown in figure 1. Amplification of individual harmonics between aortic arch and iliac artery are shown in representative cases of mild and severe valvar aortic stenosis in figure 4. It is apparent that the pattern of amplification is not related to the severity of the aortic stenosis. On the other hand, it would seem likely that something more than the static properties of the vessel wall are determining the degree of amplification, since the patients were similar in age and studied under similar conditions.

**Discussion**

It is well known that clinical findings, chest X-ray, and ECG fail to reflect accurately the severity of congenital aortic valve stenosis.\(^9\)\(^,\)\(^10\) The success which Gamboa et al.\(^21\) had in predicting left ventricular pressure from the vectorcardiogram has not been experienced by others\(^22\)\(^,\)\(^23\) while the telemetered exercise electrocardiograph\(^24\) has not won wide acceptance.

Although some of the arterial pressure wave parameters which have been investiga\(^24\)\(^\text{--}^\text{10}\) show a significant correlation in large series of adult patients, the scatter in many has been too wide to allow an accurate indication of severity of aortic stenosis in an individual patient. Lyle\(^8\) concluded that although the form of the pressure pulse was affected by the size of the left ventricular outflow tract, other factors such as left ventricular performance and the properties of the vessels were also operating.

A previous study from this institution showed considerable modification of central pulse waves as they were transmitted down the aorta.\(^11\) We predicted, therefore, that distal pulses in childhood would not
faithfully reflect central ones, and that the correlation of severity with, for example, peak dp/dt of peripheral pulses would be less than that seen in adults.\textsuperscript{6, 6} However, we had hoped that the central pulse pressure and peak dp/dt would correlate better with the degree of aortic stenosis. It is possible that by the time an aortic root pulse wave reaches the aortic arch, it has been already modified by reflection from the brachiocephalics as suggested by O'Bourke.\textsuperscript{24} Unfortunately, we could not use aortic root pulse waves

\begin{table}
\centering
\caption{Accumulated Data in 38 Patients with Congenital Aortic Stenosis*}

\begin{tabular}{l|lllll}
\hline
Pt. & Diag.\dagger & Age & LV syst. & LV-Ao syst. & Ao. valve & Manometer system \\
no. & & & (mm Hg) & diff. (mm Hg) & index & Damped nat. freq. & Damping ratio \\
& & & & & (CM\(^2\)/M\(^2\)) & (Hz) & (s) \\
\hline
1. & ASB & 7.3 & 150 & 48 & 0.61 & 1007 & -- \\
2. & ASB & 10.5 & 190 & 87 & -- & SF1 & -- \\
3. & ASS & 6.8 & 142 & 30 & 0.90 & SF1 & -- \\
4. & ASV & 3.5 & 203 & 105 & 0.49 & (56) & 0.162 \\
5. & ASV & 3.8 & 220 & 125 & -- & SF1 & -- \\
6. & ASV & 5.8 & 143 & 54 & 0.62 & (69) & 0.143 \\
7. & ASV & 7.4 & 128 & 16 & -- & SF1 & -- \\
8. & ASV & 9.1 & 118 & 28 & -- & (25) & 0.142 \\
9. & ASV & 9.3 & 240 & 144 & -- & (28) & 0.139 \\
10. & ASV & 10.0 & 120 & 24 & 2.36 & SF1 & -- \\
11. & ASV & 11.2 & 172 & 72 & -- & SF1 & -- \\
12. & ASV & 13.2 & 128 & 36 & 0.66 & (20) & 0.148 \\
13. & ASV & 13.1 & 165 & 70 & -- & SF1 & -- \\
14. & ASV & 13.6 & 190 & 90 & -- & (17) & 0.265 \\
15. & ASV & 13.7 & 125 & 35 & -- & SF1 & -- \\
16. & ASV & 14.2 & 172 & 80 & -- & (30) & 0.135 \\
17. & ASV & 14.2 & 130 & 25 & -- & SF1 & -- \\
18. & ASV & 14.9 & 153 & 57 & 0.50 & SF1 & -- \\
19. & ASV & 15.2 & 155 & 45 & -- & SF1 & -- \\
20. & ASV & 15.5 & 140 & 46 & -- & (40) & 0.104 \\
21. & ASV & 15.8 & 160 & 45 & 0.81 & SF1 & -- \\
22. & ASV & 15.8 & 140 & 42 & 1.04 & SF1 & -- \\
23. & ASV & 16.1 & 138 & 36 & -- & (57) & 0.180 \\
24. & ASV & 16.3 & 151 & 45 & -- & (67) & 0.178 \\
25. & ASV & 12.8 & 137 & 40 & 0.84 & SF1 & -- \\
26. & ASBP & 9.0 & 189 & 92 & 0.34 & (38) & 0.057 \\
27. & ASSP & 10.3 & 130 & 20 & -- & SF1 & -- \\
28. & AVVP & 12.3 & 145 & 30 & 0.45 & SF1 & -- \\
29. & AVVP & 6.5 & 140 & 30 & -- & SF1 & -- \\
30. & \textbf{\Lslash} \textbf{\Lslash} & 5.5 & 120 & 34 & -- & SF1 & -- \\
31. & A\text{\Lslash} \text{\Lslash} & 10.3 & 145 & 17 & 0.48 & SF1 & -- \\
32. & A\text{\Lslash} \text{\Lslash} & 8.4 & 142 & 61 & 0.53 & SF1 & -- \\
33. & A\text{\Lslash} \text{\Lslash} & 4.6 & 170 & 52 & 0.48 & SF1 & -- \\
34. & ASS & 10.1 & 142 & 116 & -- & SF1 & -- \\
35. & ASV & 11.9 & 175 & 55 & -- & (54) & 0.085 \\
36. & ASV & 15.4 & 162 & 54 & -- & (48) & 0.154 \\
37. & ASVP & 4.7 & 190 & 73 & 0.73 & (22) & 0.274 \\
38. & ASVP & 11.0 & 215 & 127 & -- & (29) & 0.131 \\
\hline
*Patients 1 through 33 studied from the femoral artery. Patients 34 through 38 studied from R brachial artery; peripheral pulse pressure, peripheral dp/dt and its index, pulse pressure amplification and pulse wave velocity are therefore those in the upper limbs in these five patients.
\dagger ASB = subvalvar aortic stenosis.
ASS = supravalvar aortic stenosis.
ASV = valvar aortic stenosis.
Suffix P = postoperative.
Indexed dp/dt = aortic arch dp/dt \times stroke volume (reference 7)

Abbreviations: Ao = aortic; Amplif. = amplification; Art. = artery; Diag. = diagnosis; Diff. = difference; Freq. = frequency; Nat. = natural; Periph. = peripheral; Press. = pressure; Veloc. = velocity; Syst. = systolic.

\end{table}
because even with catheter-tip manometers, the waveforms here were often very distorted. The pulse pressures were abnormally low because of low lateral pressure in the jet stream and the waveforms distorted due to turbulence (fig. 5).

The effect of aortic stenosis on peripheral amplification is unclear. Though we found a positive correlation between aortic valve area and amplification in our aortic valve stenosis group after surgery, the preoperative group showed no such relationship. Over-all, the values for peripheral amplification did not differ from our normal group, whereas Wright and Wood found lower than normal amplification ratios in aortic stenosis. We could find no consistent pattern of amplification of individual harmonics between arch and iliac artery, although Farrar and Gray described increased amplification of the first and second harmonics in the descending aorta in children with aortic stenosis. On the other hand, O’Rourke found that in adults, amplification between root of aorta and brachial artery was less in patients with all types of aortic valve disease than in controls, whereas he had previously shown that amplification in the descending aorta was not related to aortic valve disease. To explain this discrepancy, he contends that the lower harmonics are most amplified in the upper limbs, while the higher harmonics are relatively more amplified in the lower limbs. Since in aortic stenosis most of the energy of the wave is in the lower harmonics, amplification in the brachial artery, but not in the

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<th>Periph. art. (mm Hg/sec)</th>
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descending aorta, might be expected. Obviously, simultaneous recording of arch, brachial, and iliac pressures in children with aortic stenosis and in normal controls would be the only way to settle this question.

It is apparent from this study that the pulse wave is so modified in transmission along the healthy compliant arteries of children that it becomes a totally unreliable basis for assessing the severity of aortic stenosis. We believe that although clinical, radiological, and various electrocardiographic parameters are sometimes of use in detecting the severity of congenital aortic stenosis, cardiac catheterization is advisable for children who exhibit any manifestation suggesting that the lesion is more than trivial.

References

14. O'Rourke MF: Influence of ventricular ejection on the

Table 2

Correlation Coefficients for the Comparison of Various Pulse Wave Parameters with Indexed Aortic Valve Area in Congenital Aortic Stenosis

<table>
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<th>Ao arch</th>
<th>Periph. art.</th>
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<th>Pulse wave velocity</th>
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<td>r</td>
<td>27</td>
<td>- .05</td>
<td>- .12</td>
</tr>
</tbody>
</table>

See table 1 for explanation of abbreviations.

Figure 5

Polaroid photograph of ECG, aortic arch and root pressure waves displayed on the storage oscilloscope. Time lines 1 cm apart; 50 msec/div. Pressure traces taken with a catheter-tip manometer. Because of turbulence in the jet stream in aortic stenosis, aortic root pressure waves are often bizarre, even when measured with high fidelity apparatus.
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