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Abnormal Biophysical Properties of the Aorta in Post Surgical Patients with Congenital heart diseases: a Non-Invasive Study

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BACKGROUND

Children with congenital heart disease (CHD) represent a growing population: The incidence of congenital heart defects is almost 1 in 100 live births and due to major improvements in medical and surgical care, most of them will reach adulthood.

Some specific diagnoses might be associated with an increased risk for adult cardiovascular disease compared with the general population. To date, relatively few data exist regarding which CHD is associated with a significant increased risk and their role in early development of atherosclerosis.

Tetralogy of Fallot (TET), coarctation of the aorta (COA) and transposition of the great arteries (TGA) are three of the most common life-threatening forms of CHD. However, the overall perioperative mortality in the recent era is less than 5%. Most of the children with this condition will survive until adulthood.

These CHDs are associated with congenital and/or post-surgical vascular anomalies. Both macroscopic (dilatation, surgical scarring) and microscopic vascular alterations (aortic cystic medial necrosis) have been described and might contribute to vascular and myocardial function impairment.

OBJECTIVE AND HYPOTHESIS

Objective

Using non invasive techniques, we sought to assess the aortic biophysical properties, total arterial compliance, hydraulic power and efficiency in post-operative children with TET, COA and TGA.

Hypothesis

We hypothesized that children born with these forms of CHD would have abnormal vascular function, increasing their risk of acquiring early-onset cardiovascular disease

PATIENTS AND METHODS

Patients

- 55 patients with TOF (n=24), COA (n=20) and TGA (n=11) were enrolled in the study during their regular follow-up
- 55 controls were recruited from volunteers
- All subjects were between the ages of 8 to 19 years, mean age was 15.2y, 13.4y, 14.3y and 14.1y respectively for TET, COA, TGA and CTRL
- CHD patients had no additional disease, especially no hypertension or renal disease
- CTRL patients had no chronic illness

Methods

- A full cardiovascular physical examination was performed. Height and weight were recorded and body mass index (BMI) calculated. Resting systolic and diastolic blood pressures (BPs and BPd) were recorded simultaneously with echocardiography via sphygmomanometry.
- A full echocardiographic assessment was performed. M-mode, and Doppler echocardiographic imaging, and carotid artery applanation tonometry were used to measure aortic flows and dimensions. Pulse-wave velocity (PWV), input (Zi) and characteristic (Zc) impedance, arterial stiffness (Ep) and β-index were calculated. Total arterial compliance (TAC), mean (Wm) and total (Wt) hydraulic power, and efficiency (HE) were calculated from carotid pulse tracings and flows using standard fluid dynamics equations.
- The calculation of these indexes is presented on the lower left panel and the technique in the upper right panel.
- *Statistical analysis:* Univariate analyses were performed on all continuous variables. Summary statistics are expressed as median (range). The statistical significance was determined using a Kruskal–Wallis ANOVA followed by Mann–Whitney U test. P values <0.05 were considered statistically significant. All statistical analyses were performed using SAS software version 9.1.3 (SAS Institute Inc., Cary, NC).

CALCULATIONS

- PWV = L / TT
- Ep = [(BPs-BPd) / (Ds-Dd)] x Dd
- β-index =ln{[(BPs/BPd)/(Ds-Dd)]xDd}
- Zi = (BPs - BPd) / AoVcsa x AoVpeak
- Zc = (PWV x ρ) / AoVcsa
  ρ = 1.06 (density of blood)
- TAC = TAC = Ad/R(P1 P2),
  P1 is end-systolic pressure, P2 is end-diastolic pressure, Ad is the area under the pressure waveform enclosed by P1 and P2, and R is the peripheral resistance, given by mean pressure divided by mean flow
- Wm = mean pressure x mean flow
- Wt = $\frac{1}{T}\int_0^T P(t)Q(t)dt$
  T is the cardiac cycle duration, and P and Q are instantaneous pressure and flow, respectively.
- HE = Wm / Wt

ECHO-DOPPLER ASSESSMENT OF VASCULAR FUNCTION

The diameter of the aortic valve annulus was measured in a standard parasternal long axis view using 2-D echocardiography. The cross-sectional area of the aortic valve annulus [AoVcsa] was calculated.

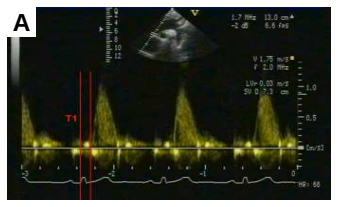
The peak aortic velocity [AoVpeak] was measured from an ascending aortic pulse wave Doppler tracing recorded in a standard suprasternal long axis view. The time from the QRS to the onset of the ascending aortic Doppler envelope (T1) was measured (Figure A).

Maintaining the same transducer position, the pulse wave Doppler sample volume was then placed as distally as possible in the descending aorta and the time from the QRS to the onset of the descending aorta Doppler envelope (T2) was measured (Figure B).

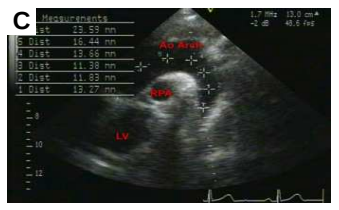
The transit time of the pulse wave [TT] was derived from the difference between T2 and T1. Using the same 2-D image, the length [L] between these two points was measured with electronic calipers (Figure C).

The diameter of the ascending aorta at end-diastole [Dd] and the peak diameter in systole [Ds] were measured using the leading edge method from an M-mode recording made at a right angle to the ascending aorta in a high right parasternal view (Figure D)

Figure E shows an example of a carotid pulse tracing and ascending aortic Doppler flow taken from the suprasternal notch. The sample volume is placed centrally in the ascending aorta just distal to the aortic valve. The short arrow points to the carotid pulse tracing. The magnification of the tracing is adjusted to fit on the screen. The longer arrow points to the onset of Doppler flow. D, Dicrotic notch; ECG, electrocardiogram; N, nadir of the pressure tracing; P, peak of the pressure tracing.

A


B


C


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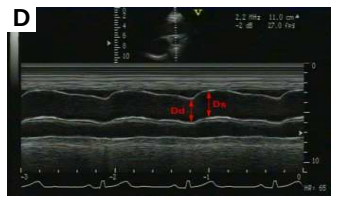

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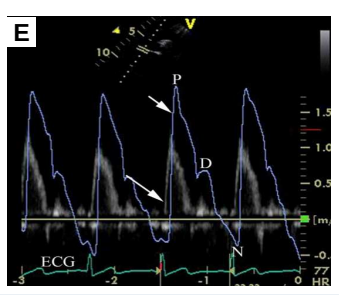

TABLE 2 - BIOPHYSICAL PROPERTIES OF THE AORTA

	CTRL	TET	COA	TGA
PWV ($cm \cdot s^{-1}$)	360 (268-600)	492 (295-930) p<0.001 vs. CTRL	458 (240-1170) p<0.001 vs. CTRL	527 (300-820) p<0.05 vs. CTRL
Zi ($dyn \cdot s \cdot cm^{-5}$)	192 (108-299)	179 (97-316)	237 (101-511)	210 (112-339)
Zc ($dyn \cdot s \cdot cm^{-5}$)	138 (85-251)	177 (69-314) p<0.001 vs. CTRL	234 (112-702) p<0.05 vs. CTRL	154 (62-277)
Ep (mm Hg)	263 (128-706)	298 (93-461)	295 (176-592)	270 (133-761)
β-index	2.27 (1.77-3.17)	2.50 (1.72-2.93)	2.38 (1.81-3.06)	2.24 (1.72-3.28)
TAC ($mL \cdot torr^{-1} \cdot m^{-2}$)	1.35 (0.94-2.13)	1.71 (0.68-4.45) p<0.05 vs. CTRL	2.32 (0.60-4.67) p<0.05 vs. CTRL	2.34 (1.66-5.88) p<0.001 vs. CTRL / p<0.05 vs. TET
Wm ($mW \cdot m^{-2}$)	680 (518-1117) p<0.05 vs. TGA	730 (523-1468) p<0.05 vs. TGA	965 (266-1510)	1010 (694-1449)
Wt ($mW \cdot m^{-2}$)	830 (602-1503) p<0.05 vs. TGA	936 (652-1811) p<0.05 vs. TGA	1175 (316-1878)	1273 (952-1852)
HE	0.86 (0.70-0.91)	0.86 (0.81-0.91)	0.83 (0.78-0.89) p<0.05 vs. TET	0.82 (0.78-0.87) p<0.01 vs. TET

Significant results are shown with colored background (blue or red).

CONCLUSION

Children with post-operative TET, COA and TGA have stiffer aortas, increased work and higher total arterial compliance than CTRL. It is unclear if this is related to intrinsic lesions of the aorta, alterations of the aorta due to surgical repair or other factors. Further studies and follow-up are needed to determine if these abnormalities predispose these patients to long-term cardiac dysfunction and cardiovascular risk.