Profiling left and right ventricular proportional output during fetal life with a novel systolic index in the aortic isthmus.

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* Presented in part at the 2012 ISUOG, 22nd World Congress, in Copenhagen.
This investigation was supported (in part) by a research grant (grant # MOP-97986) from the Canadian Institutes of Health Research.
Abstract

Objective: Left ventricular (LV) ejection causes a forward flow in the fetal aortic isthmus (AoI) while the right ventricle (RV) has a retrograde influence. This study proposes reference values for an isthmic systolic index (ISI) reflecting the changing influences of the RV and LV performances on the Doppler flow velocity waveforms of the aortic isthmus throughout normal pregnancies.

Methods: Doppler recordings of 261 normal fetuses, aged 18 to 37 weeks, were reviewed. Peak systolic velocities (Ps) and end-systolic velocities or systolic nadir (Ns) were measured on all aortic isthmus waveforms. An isthmic systolic index (ISI) was derived from the ratio Ns/Ps. Left (QLV) and right (QRV) ventricular outputs were also calculated.

Results: Up to 22-23 weeks of gestation, the ISI is stable at 0.2. At about 28 weeks, a brief end-systolic deceleration wave is observed on the aortic isthmus waveforms, progressing steadily with gestation and causing a fall of ISI towards a mean value of zero between 30 and 31 weeks. This trend continues thereafter and a mean value of -0.4 was observed at the end of pregnancy. An inverse correlation was found between QRV and Ns (r=0.334, p=0.001). Simultaneous recordings of the isthmus and the ductus arteriosus Doppler waveforms demonstrate that the essential contributor to the end-systolic deceleration and,
ultimately reversal flow at the isthmus is caused by the increasingly competitive flow from the RV.

**Conclusion:**

The transitional changes of the respective RV and LV outputs throughout pregnancy are well profiled by the ISI. This index highlights the physiological increase in fetal RV flow preponderance as pregnancy progresses. Alteration of the ISI profile could be expected in clinical conditions associated with unbalanced alteration of the fetal ventricular outputs.

**Key words:** Fetal aortic isthmus, isthmus systolic index, ventricular performances.

**INTRODUCTION**

In the fetal circulation, the right and left ventricles as well as their respective outlets, the aortic and pulmonary arches, are disposed in parallel. The aortic isthmus (AoI), localized between the origin of the left subclavian artery and the aortic extremity of the ductus arteriosus acts as a shunt between these two arches. Experimental and clinical studies have demonstrated that flow velocity patterns in the aortic isthmus are valid markers of peripheral circulatory disturbances during fetal life. During diastole, when the semi-lunar valves are closed, the direction of the isthmic flow depends on the difference between the vascular impedance of the supra versus infra-diaphragmatic vascular systems. Under normal circumstances, the isthmic flow is always antegrade due to the
low placental vascular resistances, but the flow profile has been shown to vary with physiological changes throughout gestation\textsuperscript{10}. At this date, the great majority of clinical studies have analysed the diastolic Doppler flow in the isthmus in relation to changes in peripheral resistances, essentially in fetuses with intrauterine growth restriction (IUGR). Yet, during systole, blood ejected by the left ventricle is expected to induce a forward flow in the aortic isthmus while the influence of right ventricular ejection should manifest itself as a retrograde force resulting in a reduction of the forward isthmic flow. Due to the known physiological increase in right ventricular output preponderance during the second half of pregnancy\textsuperscript{11}, this potential retrograde influence should be more evident toward the end of gestation. The objective of this study was therefore to analyse the normal pattern of aortic isthmus flow during systole in the healthy fetus and to propose an isthmic systolic index (ISI) expressing the balance between the performances of the two ventricles during the second and third trimesters of gestation.

METHODS

The data presented in this study were retrospectively measured on Doppler recordings made on normal fetuses aged between 18 and 37 weeks of gestation. Those studies were collected in 2 tertiary centers from January 2010 to March 2013. All data from both centers were verified by the principal investigator (JCF) for the proper position of the Doppler sample volume in the aortic
isthmus. Inclusion criteria were: (i) singleton pregnancies with normal medical and obstetrical histories; (ii) absence of structural cardiac malformations and chromosomal abnormalities (iii) intrauterine growth appropriate for gestational age (GA) with normal umbilical artery Doppler pattern. The normal morphology of the fetal heart was confirmed by the postnatal clinical examination and outcome. Occasional extrasystoles in an otherwise healthy heart were not considered as an exclusion criterion. All scans were performed by experienced fetal cardiac sonographers. The fetal sagittal approach used to obtain a real-time imaging of the aortic arch and the correct position of the Doppler sample volume in the aortic isthmus, just beyond the left subclavian artery has been previously described5. All reported values represent the calculated average of measurements made on 3 consecutive beats during apnea and regular sinus rhythm.

The following variables were measured on each isthmic waveform: the systolic peak velocity (Ps) and the end-systolic velocity or systolic nadir (Ns). End-systolic velocity was determined by visual assessment based on either the sudden changes of the deceleration slope caused by the closure of the aortic valve (top of figure 1) or on the clear appearance of an incisura after the 25th week of gestation. An isthmic systolic index was calculated by dividing the systolic nadir (Ns) by the systolic Peak (PS) velocity (ISI=Ns/Ps). To evaluate the left and right ventricular stroke volumes, the systolic aortic and pulmonary
arterial valve diameters (D) were measured on frames showing maximum systolic diameter (cm). Cross-sectional area was calculated assuming a circular cross section for the pulmonary and aortic valves. Time velocity integrals (TVI) of ascending aorta and main pulmonary artery were measured on high quality flow velocity spectra of their respective Doppler waveforms recorded at an angle < 20° in relation to the Doppler beam. Right or left ventricular stroke volumes (ml) were derived from the product of pulmonary or aortic arterial flow velocity integrals and pulmonary or aortic cross-sectional area respectively, according to the usual Doppler formula TVI x (π x D²/4). Right (QRV) and left (QLV) ventricular outputs (ml/min) were computed from the product of stroke volumes (ml) by the respective simultaneous heart rates (beats/min) in 93 subjects.

Statistical analysis

Intra- and interobserver reliabilities for the three target parameters were evaluated on 20 recordings using intraclass correlation coefficient (ICCs) assessment. Interobserver analysis compared parameters independently measured by two observers, each without knowledge of the results obtained by the others. Intraobserver analysis compared two measurements obtained by the same observer on the same waveform two months apart. A p-value < 0.05 was considered significant.
To assess the similarity of the data obtained from the 2 centers, regression parameters were compared by the Fisher z-test before pooling data on the final regressions. The statistical method of assessing reference intervals and Z scores described by Royston and Wright\textsuperscript{12} was used. Separate linear, quadratic and cubic regression models were applied to estimate the relationship between the aortic isthmus systolic parameters and gestational age. The best fitted model for each variable was selected based on the R-square statistic and the standard error of the estimates. The normality of the distribution of ISI in our population was validated with Z scores distribution. The 3\textsuperscript{rd} and 97\textsuperscript{th} percentiles were calculated based on the standard deviation across gestational age. Associations between Ps, Ns and ventricular output components were assessed using multiple regression analysis. All analyses were performed with SPSS version 20.0.0.

RESULTS

Doppler recordings from 269 pregnancies corresponding to the inclusion criteria were reviewed. Of these, two were excluded due to the inconsistency of the aortic isthmus waveform recording, two presented a notch on the uterine artery waveforms, and another two presented an umbilical cord anomaly. Reviewing the medical chart, one mother was found with the factor V Leiden and one foetus was presenting a tricuspid insufficiency. Finally, one recording was excluded due to the very extreme values measured on the aortic isthmus. Accordingly, a total of 261 fetuses composed the final study population.
The Ps and Ns were measured in all with excellent ICCs for the intra- and inter-
observer assessments in randomly selected samples (table 1). Examples of
normal Doppler waveforms at different gestational ages are shown on Figure 1.

The relationship between the studied variables and gestational age were best
fitted in quadratic regression models. The equations for Ps, Ns and ISI are
provided in table 2. The distribution of adjusted Z-scores \( Z = \frac{\sqrt{|\text{ISI}| - \text{mean}}}{\text{SD}} \) falls, for the most part, between -2 and +2, supportive of its normal
distribution. The gestational age related reference ranges for the aortic isthmus
systolic aortic isthmus blood velocities (peak and nadir) and the ISI are listed in
table 3.

The estimated mean and centile curves from the regression analysis for those
parameters are illustrated in Figure 3. With advancing gestation, the aortic
isthmic Ps increased up to 32\textsuperscript{nd} week of gestation and remained mostly
unchanged thereafter (figure 2-top). The Ns followed an opposite pattern,
remaining relatively stable at 13.1 ± 0.4 cm/s up to 25 weeks of gestation and
decreasing gradually thereafter to reach the zero velocity line at around 31 weeks
of gestation. From this point, an increasing retrograde wave started to appear
causing a progressive fall of Ns up to the end of gestation. As a consequence,
the ISI remained stable at 0.2, up to around 27 weeks; then, it gradually
decreased crossing the zero velocity line between 30-31 weeks and reaching a negative mean value of -0.4 by the end of pregnancy (Figure 2-bottom).

As expected, left and right ventricular outputs are highly correlated with fetal growth, both QLV and QRV increasing with advancing gestational age (Pearson’s r = 0.907 and 0.914 respectively, p<0.001). Controlling for gestational age, we studied the potential correlation between combined cardiac output components, i.e. QRV and QLV, and the ISI components (Ps and Ns). Accordingly, Ns is inversely proportional to QRV (r = -0.334, p = 0.001), with no significant correlation with QLV (r = -0.161, p = 0.126). On the other hand, Ps showed no significant correlation with QRV (r = -0.086, p = 0.413) or QLV (r = 0.147, p = 0.162). In addition, the reverse proportional relationship between Ns and QRV was predictive of the ISI (r = -3.09, p = 0.003) independently from gestational age, QLV, or Ps.

DISCUSSION

*Technical considerations:* The appearance of a brief end-systolic notching of the flow velocity waveforms at the level of the isthmus after 20 weeks of pregnancy has been previously described (10). This brief deceleration wave increases with advancing gestation and, at about 30 weeks, a brief flow reversal is observed. This flow pattern is less frequently observed when the 3 vessels and trachea approach is used for isthmic Doppler recordings, leading to the suggestion that it could be an artefact, based on the presumed notion that...
concomitant bi-directional flow waves are not possible in the same vascular segment (13). Actually, the bidirectional streams recorded in the fetal aortic isthmus occur at two different times within the same cardiac cycle. The forward waves appear first, covering more than 2/3 of the systole while the reverse waves are observed very late in the cycle. Simultaneous recordings of the aortic isthmus and ductus arteriosus as illustrated on figure 1 (bottom), clearly demonstrate the late systolic influence of right ventricular output on the isthmus flow pattern as previously reported (9,14). This controversy highlights the importance of accurate echographic identification of the aortic isthmus just beyond the origin of the left subclavian artery and before the connection of the ductus arteriosus with the descending aorta. These anatomical landmarks can be identified only on the 2-D echo imaging of the aortic arch viewed from the fetal sagittal approach.

Underlying mechanism(s): Two physiological events could be proposed as explanations for the late systolic reversal of flow in fetal aortic isthmus: either the fall in cerebrovascular resistance or the right ventricular preponderance, both events being normally observed during the third trimester of gestation. With the first hypothesis the decrease in cerebral vascular resistance would drive blood retrograde through the isthmus during systole; however, cerebral vasodilatation will primarily cause an increase in the fraction of LV stroke volume going to the brain, sufficient to eliminate any systolic retrograde effect on the downstream isthmic flow. The second hypothesis is based on known physiological specificities of the fetal circulation: in prenatal life, an increase
in cerebral flow due to a fall in cerebral vascular resistance is always associated with an isolated rise in the RV preload and stroke volume since the elevated cerebral venous return is drained by the SVC entirely into the RV; a major proportion of this elevated right ventricular output goes through the ductus arteriosus which is connected to the distal portion of the isthmus and, therefore, has a systolic retrograde influence on the isthmic flow.

Various mechanisms could be considered to explain how right ventricular preponderance influences the fetal aortic isthmic flow. First, a greater stroke volume could cause a longer right ventricular ejection time and a late closure of the pulmonary valve with an extended forward late systolic flow across the ductus arteriosus. However, previous experimental studies have shown that despite the difference in output, right and left ventricular ejection times were similar throughout gestation (15). A second explanation could be a difference in the opening time of the semi-lunar valves. Experimental studies in sheep fetuses have indeed shown that the pre-ejection period of the fetal right ventricle was longer than that of the left due to a longer right ventricular electromechanical time interval (15). If this element was significant, the end systolic reversal of isthmic flow should be observed as early as the first trimester of gestation. The most likely explanation is probably related to the variation in the compliance of the fetal pulmonary arterial network throughout gestation. A typical flow pattern has been described in the fetal branch pulmonary arteries characterized by a rapid systolic upstroke, a short
acceleration time and a late systolic flow reversal (16). This systolic retrograde flow has been attributed to the low compliance of the branch pulmonary arteries and the high peripheral pulmonary vascular resistances during the fetal period (16). This observation is in accordance with our finding of a strong inverse correlation between the right ventricular output and the nadir of the late isthmus systolic reverse flow: the greater the QRV, the more significant should be the systolic reversal of flow from the pulmonary arteries toward the ductus arteriosus and the aortic isthmus.

Potential clinical applications: The demonstration of the close relationship between ISI and respective ventricular outputs should lead to many clinical applications. Clinical conditions associated with fall in LV performance and/or increase in right ventricular output such as aortic stenosis, could potentially either reduce the forward systolic isthmic flow or cause flow reversal depending on the degree in the reduction of forward trans aortic output. The peripheral vascular resistances by their potential effect on the systemic venous return could also have an influence on ventricular performances by changing ventricular
preloads; this is especially true for the supradiaphragmatic circulation whose
venous return is drained almost exclusively into the right ventricle through the
superior vena cava; for example, a previous report has shown that cerebral
arterio-venous fistula, which causes an excessive blood return via the superior
vena cava, causes retrograde systolic flow in the aortic isthmus (9). Similarly,
situations associated with cerebral arterial vaso-dilatation such as placental
circulatory insufficiency may also benefit from the assessment of the systolic
flow pattern in the aortic isthmus. Quantifying the ISI in this pathology may
help stratifying the risk of fetal morbidity.

In conclusion, the transitional increase of the proportional QRV/QLV outputs
observed throughout pregnancy is well profiled by the ISI. This index
highlights the physiological increase in fetal right ventricular outflow
preponderance as pregnancy progresses. The ISI is easy to calculate with
excellent intra and inter-observer agreement rates. It is expected that alteration
of the ISI profile be a valuable diagnostic criterion and/or prognostic tool in
clinical conditions causing unbalanced alteration of the fetal ventricular outputs.

Acknowledgement:

The authors are very grateful for the invaluable secretarial assistance by
Nathalie Ishmael

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RÉFÉRENCES


Figure legends

Figure 1: Examples of Doppler flow waveforms in the aortic isthmus of normal fetuses throughout gestation.  **Top:** At 20 weeks of gestation, a constant forward flow is observed both in systole and diastole and the end-systolic nadir (Ns) is always above the baseline level (zero velocity line).  **Middle:** Around 28 to 30 weeks of gestation, Ns reaches the baseline level.  **Bottom:** Subsequently, Ns is below the baseline, such as in this 37 week fetus.  On this last figure, superimposed Doppler waveforms of the ductus arteriosus (DA) can be observed, illustrating the late systolic influence of the right ventricle on the flow pattern of the aortic isthmus.
Figure 2:

Top: As gestation progresses, from 18 to 37 weeks, the peak systolic velocity (Ps) continues to increase whereas end-systolic nadir velocity (Ns) diminishes in the aortic isthmus.

Bottom: This widening gap between Ps and Ns throughout gestation parallels the deflection of the aortic isthmus systolic index (ISI).

The regression lines represent the 5th, 50th and 95th percentiles.
Table 1: Intraclass Correlation coefficient (ICC) calculated from measurements on 20 fetuses.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Comparison</th>
<th>ICC</th>
<th>ICC LL</th>
<th>ICC UL</th>
<th>P value</th>
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<td>Obs-1 v.s. Obs-1</td>
<td>0.992</td>
<td>0.979</td>
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<td>ISF nadir</td>
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</table>

ISFI, isthmic systolic flow index; ISF peak, peak systolic velocity at the isthmus; ISF nadir, nadir systolic velocity at the isthmus; Obs-1 v.s. Obs-1, intra-observer comparison; Obs-1 v.s. Obs-2, inter-observer comparison; ICC, Intraclass Correlation coefficient; ICC LL and ICC UL, lower limit and upper limit of the 95% interval confidence of Intraclass Correlation coefficient.
Table 2: Regression equations for ISI components against gestational age.

<table>
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<th>Regression equations of ISI components</th>
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<tr>
<td>Ps = -67.60 + (9.49 * GA) – (0.14 * GA^2)</td>
<td>15.37</td>
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<td>Ns = -109.07 + (10.88 * GA) – (0.24 * GA^2)</td>
<td>10.69</td>
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<tr>
<td>ISI = -0.73 + (0.09 * GA) – (0.002 * GA^2)</td>
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Ps, peak systolic velocity; Ns, nadir systolic velocity; ISI, isthmic systolic index; SEE, standard error of the estimate; GA, gestational age in weeks.
Table 3: Reference ranges for the aortic isthmus systolic aortic isthmus blood velocities (peak and nadir) and the ISI according to gestational age in weeks

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<th>Mean 97th centile</th>
<th>Ps (cm/s)</th>
<th>3rd centile</th>
<th>Mean 97th centile</th>
<th>Ns (cm/s)</th>
<th>3rd centile</th>
<th>Mean 97th centile</th>
<th>ISI 3rd centile</th>
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