Chapter 7

DOPPLER OF THE FETAL DESCENDING AORTA

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OUTLINE:

I. Anatomy and Physiology

II. Image Acquisition

III. Fetal Descending Aorta Wave Form and Indices

IV. Abnormal Fetal Aorta Waveform and Indices

V. Clinical Applications

VI. Conclusion
1. Anatomy and Physiology

Anatomy

The aorta is described into different portions as ascending aorta, arch of the aorta and descending aorta. The aorta commences at the upper left ventricle and after ascending for a short distance arches at the back and to the left side and over the root of the lung. It then descends within the thorax in the left side of vertebral column and passing into the abdominal cavity through the aortic hiatus in the diaphragm and at the 4th lumbar vertebra it divides into right and left common iliac arteries. The descending aorta is further dividend into thoracic and abdominal aorta.\(^1\) (Figure 7-1)

![Diagram of the aorta](https://stepjourney.wordpress.com/2010/04/06/abdominal-aorta-anastomoses/)

Physiology

The fetal descending aorta receives majority of right ventricular output via the ductus arteriosus while majority of left ventricular output supplies fetal head ensuring the brain to be well oxygenated as well as upper extremities. The major fraction of the blood that passes the descending aorta comes from the ductus arteriosus supplying the lower body and flows via two umbilical arteries to the placenta.\(^2,3\) The combined ventricular output (right ventricle and left ventricle) of about 65% represents the blood flow in the thoracic descending aorta. The placenta receives 40% of fetal cardiac output or 65% of the flow in the thoracic descending aorta and about 75% of the flow in abdominal aorta distal to the origin of renal arteries.\(^4\) The increase in volume flow is related to the growing diameter of the aorta and this explains constant time average mean velocity in the third trimester. Likewise the volume flow that was studied in relation to fetal weight has been found to be stable in the third trimester, reported by several authors ranging from 206 to 280 ml.min\(^{-1}\)kg\(^{-1}\). (Table VII-I) The distribution of aortic blood flow at the fetal descending aorta and abdominal aorta in part of the umbilical vein has shown that the placental proportion of blood flow in the descending thoracic aorta in relation to fetal weight diminished with advancing gestational age as noted by a study as 55% at 28 weeks and 33% at term.\(^5,6\) (Figure 7-2) Flow velocity in the descending aorta represents summation of blood flow of the kidneys, intestines, lower extremities and placenta.\(^7,8\) In state of acute or chronic hypoxia leads to peripheral vasoconstriction of blood flow to the kidney, gut and lungs with diversion of blood flow to brain, heart and adrenals. This redistribution results to increase
vascular resistance to the flow in fetal aorta reflected as change in aortic FVW (flow velocity waveform). This compensatory mechanism of blood redistribution during period of hypoxia is a means of fetal survival through the process of centralization. Compromised fetus showed changes of aorta FVW as ARED (Absent-reversed end diastolic) (Figure 7-3) flow which is represented by increased placental resistance in particular in IUGR and hypoxic fetuses.\(^8,9,10\) Doppler frequency changes of fetal aortic blood flow may be affected by fetal cardiac output, fetal heart rate, blood viscosity and impedance of placental vascular system.\(^5,6,9,10,11\)

Table VII-1. Reference values of fetal aortic blood flow reported in the literature\(^5\)

<table>
<thead>
<tr>
<th>Study</th>
<th>Gestational age (weeks)</th>
<th>No.</th>
<th>Volume flow (ml·min(^{-1}·kg(^{-1}))</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Thoracic descending aorta</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Griffin et al. [27]</td>
<td>28-40</td>
<td>75</td>
<td>246 ± 30</td>
<td></td>
</tr>
<tr>
<td>van Lierde et al. [28]</td>
<td>37-40</td>
<td>20</td>
<td>216 ± 24</td>
<td></td>
</tr>
<tr>
<td>Maršál et al. [29]</td>
<td>27-40</td>
<td>64</td>
<td>238 ± 40</td>
<td></td>
</tr>
<tr>
<td>Lingman &amp; Maršál [26]</td>
<td>27-36</td>
<td>21</td>
<td>238 ± 46</td>
<td>Longitudinal study</td>
</tr>
<tr>
<td></td>
<td>37-38</td>
<td>21</td>
<td>221 ± 41</td>
<td></td>
</tr>
<tr>
<td></td>
<td>39-40</td>
<td>21</td>
<td>213 ± 37</td>
<td></td>
</tr>
<tr>
<td>Rasmussen [31]</td>
<td>29-40</td>
<td>58</td>
<td>234 (184-289)</td>
<td></td>
</tr>
<tr>
<td>Räisänen et al. [32]</td>
<td>40±2.3</td>
<td>51</td>
<td>290 ± 67</td>
<td>Low values for unclear reason</td>
</tr>
<tr>
<td>Cameron et al. [33]</td>
<td>28</td>
<td>9</td>
<td>143 ± 34</td>
<td>Longitudinal study; simultaneous automatic measurement of the velocity and diameter</td>
</tr>
<tr>
<td>Brodzski et al. [16]</td>
<td>28-31</td>
<td>20</td>
<td>213 ± 77</td>
<td></td>
</tr>
<tr>
<td></td>
<td>32-35</td>
<td>20</td>
<td>239 ± 66</td>
<td></td>
</tr>
<tr>
<td><strong>Abdominal aorta</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eldridge et al. [34]</td>
<td>18-40</td>
<td>18</td>
<td>184 ± 20</td>
<td>Longitudinal study</td>
</tr>
<tr>
<td>Lingman &amp; Maršál [26]</td>
<td>27-36</td>
<td>21</td>
<td>167 ± 43</td>
<td>Longitudinal study</td>
</tr>
<tr>
<td></td>
<td>37-38</td>
<td>21</td>
<td>133 ± 38</td>
<td></td>
</tr>
<tr>
<td></td>
<td>39-40</td>
<td>21</td>
<td>136 ± 30</td>
<td></td>
</tr>
</tbody>
</table>

Mean values±SD or range are given.

### Distribution of Fetal Aortic Blood Flow

![Distribution of Fetal Aortic Blood Flow](image)

**Figure 7-2.** (Adapted from Karel Marsal, Fetal Descending Aorta, Doppler Ultrasound, In Obstetrics and Gynecology, 11,151.)
II. Image Acquisition

Doppler flow study of fetal descending aorta is usually obtained at lower thoracic level just above the diaphragm.\(^5\,6\,8\,12\) (Figure 7-4) The color flow mapping interrogating the descending aorta will reveal many of its branches including celiac axis, superior mesenteric artery, renal arteries, adrenal arteries and the iliac bifurcation.

For methodology of acquiring waveform of the fetal descending aorta: Refer to Chapter 4, IV B2b. Technique of Fetal Aorta Doppler Assessment. In addition to the technique of acquisition of waveform of fetal descending aorta, it is recommended for patients to abstain from smoking 2 hours prior to Doppler ultrasound examination.\(^5\)

IV. Fetal Descending Aorta Waveform and Indices

Physiology of Formation of Waveform of Fetal descending aorta (FDA)

The FVW (flow velocity waveform) of aorta and umbilical artery are similar in the early part of gestation. It is highly pulsatile with minimal diastolic flow. The FVW of FDA shows a continuous forward flow during whole cardiac cycle.\(^5\,6\,9\,10\,13\)
1. The diastolic velocities is absent up until 10 weeks which is a typical end diastolic zero flow.

2. At 12-13 gestational age of gestation, the end diastolic flow velocities begin to appear due to a lowering of systemic vascular resistance with the development of the placenta.

3. Diastolic velocities are present in all aortic signals from 16 weeks onwards. S/D ratio and RI are decreasing during the first 16 weeks of gestation. PI drops from 2.5-3.0 to 1.8-2.0.

4. After 17-18 weeks there is no further decrease in aortic PI with advancing gestational age.

5. The aortic vascular impedance is stable during last trimester of pregnancy. During this time the diameter of fetal aorta becomes wider contributing to decreased peripheral resistance with increased diastolic flow. (Figure 7-5)

6. Pulsatility index (PI) and resistance index (RI) are lower in the abdominal aorta than thoracic aorta due to a greater proportion of blood flow towards the abdominal aorta. This is the same findings of study by Lingman et. al and Ballmann et. al. that the aortic vascular resistance is decreasing with increasing distance to the heart.

The waveform of aortic blood velocity changes with increasing distance to the heart. In the study of Ballmann et al. of 926 low risk pregnancies showed the aortic vascular resistance and blood flow velocities are significantly reduced with increasing distance from the heart. This finding is similar from the study of Lingman et, al. They attributed the reductions due to the abdominal vessels (celiac artery, superior and inferior mesenteric arteries and renal artery) and branching off below the diaphragm.5,13

The increased in diastolic flow of arterial vessel as in FDA and umbilical artery with advancing gestational age was explained by the study of Trudinger (1985) by these physiologic events.10

<table>
<thead>
<tr>
<th>PLACENTA</th>
<th>FETUS</th>
<th>FETAL VESSEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased placental villi</td>
<td>Increased cardiac output</td>
<td>Increased vessel compliance</td>
</tr>
<tr>
<td>Widening of placental Vessels</td>
<td>Increased blood pressure</td>
<td>Increased vessel diameter</td>
</tr>
</tbody>
</table>

Normal Pregnancy – Development of Descending Aorta

The waveform of fetal descending aorta is characterized by a steep systolic rise followed by a post-systolic notch and a low end-diastolic forward flow velocities. During systole which is the acceleration time is a reflection of cardiac contractility while the diastolic phase reflects peripheral vascular impedance. The post-systolic notch is the result of closure of aortic valve during early ventricular diastolic phase resulting in a slight decrease in flow velocity.\textsuperscript{5,6,11,13} Figure (7-6).

The S/D ratio of fetal descending aorta decreases with increasing gestational age. (Graph 7-1) The FVW of the fetal aorta shows a continuous forward stream throughout the whole cardiac cycle. (Figure 7-7) As compared to umbilical artery, the diastolic flow is less, so that the S/D ratio of fetal aorta is greater than the umbilical arteries. With advancing gestational age, the diameter of the fetal aorta increases results to decrease in peripheral resistance and contributes to increase in diastolic flow.\textsuperscript{10}
As the end-diastolic velocities decrease, all the indices rise and the changes in the values obtained correlate closely between different indices. The pulsatility index is preferred since it can still quantify the FVW in the absence of diastolic flow. Absent or reversed end diastolic flow are classified separately. Pulsatility index (PI) and resistance index (RI) of fetal descending aorta after 20 weeks AOG remains to be stable and can be plotted on a nomogram chart with gestational age. (Graphs 7-2, 7-3 and 7-4)\(^{10}\)
Points to remember:
1. Values at 90th and 95th percentile are borderline
2. Values above 95th percentile are abnormal


IV. Abnormal Fetal Aorta Waveform

In pregnancies that are associated with hypoxia most especially in growth restricted fetus with uteroplacental insufficiency; the waveform of fetal descending aorta is similar to umbilical artery described as decreased diastolic and eventual disappearance and reversed end-diastolic flow.

Lingman et. al. (1986) reported four blood flow classes (BFC) of fetal aorta waveform as:
BFC 0 (Normal) showed positive blood flow throughout the cardiac cycle
BFC I showed positive blood flow throughout the cycle with Pi>mean +2 SD of the normal or a decreasing flow during diastole
BFC II showed a nondetectable end diastolic velocity;
BFC III showed total absence of positive flow throughout diastole and reverse flow as well. BFC III is the worst fetal condition leading to increased risk of fetal death. 5(Figure 7-8)

Blood Flow Classes

Figure 7-8. Blood Flow Classes (BFC) of Fetal Aortic Waveforms; (Adapted from Karel Marsal, Fetal Descending Aorta, Doppler Ultrasound in Obstetrics and Gynecology, Dev Mulik, Chapter11:148.)
Increased in pulsatility index is a sign of an early change in the fetal circulation that has been found in several studies. The absence of end diastolic flow indicates the presence of fetal compromise and reversal of flow is correlated with increased incidence of perinatal morbidity and mortality.\textsuperscript{5, 6, 8, 10, 11}

**Pathophysiology of abnormal FVW in placental insufficiency/IUGR**

**Normal BFC**

The fetal descending aorta FVW has a similar course as in the umbilical artery with progression of pregnancy. In the early part of pregnancy the FVW of umbilical artery and fetal aorta is high resistant with absent end diastolic flow and appearance of diastolic flow after first trimester with fall in resistance index in second and third trimester. The decrease in S/D ratio, PI and RI is attributed to low impedance in the placenta.\textsuperscript{5, 9, 10}

**BFC I**

In milder form of placental insufficiency, S/D ratio increases with decreased end diastolic velocity and remains constant. Even during this time the fetal circulatory system including the aorta is usually normal. As the resistance in umbilical artery increases, this has an associated decreased in pO2 in the umbilical vein. The only compensatory mechanism observed during this time is the reopening of ductus venosus to physiologically extract a significant amount of blood from the liver and goes directly to the heart. The shunt can delay by some weeks the need of centralization of blood flow that will induce a typical FVW of a growth restricted fetus.\textsuperscript{9, 10}

**FVW of umbilical artery and FA - Increased PI**

**FVW of common carotid artery and middle cerebral artery - Decreased PI**

**Pathophysiology of increased resistance in the descending aorta\textsuperscript{9}**

1. Increased to umbilical-placental resistance
2. Arterial vasoconstriction resulting to hypoxia
3. Decreased myocardial contractility

**BFC II**

In addition to the re-opening of the ductus venosus, a redistribution of its blood flow in order to protect most important structures like the heart and brain from hypoxia. During this time is a period of centralization (selective vasodilation of brain, heart, and adrenals with vasoconstriction of other sites as lungs, intestines, kidneys, skin and skeleton)\textsuperscript{5, 8, 9, 10}

In the study by Akalin-Sel et al. postulated that redistribution in growth restricted fetuses is regulated by reflex mechanisms (the 'lower limb reflex') which result in severe vasoconstriction in the abdominal aorta, mesentery and carass, favouring the brain and myocardium. The loss of end diastolic flow (L-EDF) is a good predictor for poor neonatal outcome (sensitivity, specificity and positive predictive value, all 100\%).\textsuperscript{14}

**Three Phases of Centralization\textsuperscript{9}**

1. **Initial phase** – (BFC I) Increased PI of umbilical artery and fetal aorta and decreased PI in CCA (common carotid artery) and MCA (middle cerebral artery)
2. **Advanced phase** – (BFC II) According to Trudinger, it will need about 80% of obstruction in the villous arterial system to attain a zero or absent diastolic blood flow. During this time there is maximal dilatation of middle cerebral artery with reduced flow in the CCA.
3. **Terminal phase** – (BFC III). This showed reversal of diastolic flow in umbilical artery and FA and progression of decompensation.
BFC III

During the terminal phase, in addition to reversal of flow of umbilical artery and FA, there is an associated arterial hemodynamics in the phase of cardiac insufficiency leads to coronary vasodilatation.

Coronary vasodilation is due to:
1. Low myocardial oxygen pressure
2. Loss of myocardial contraction force
3. Decrease in the velocity of intracardiac blood flow

Decentralization
The decentralization of blood flow to irreversible hemodynamics changes the pattern of centralization of blood flow which precedes fetal death.

The persistence of hypoxia will show a phenomenon of generalized fetal vascular paralysis and during this time there is cerebral edema and tissue necrosis brought about by marked reduction of oxygen with accumulation of lactic acid as a result of sustained anaerobic metabolism.9

FVW of umbilical artery and fetal aorta- RDF (Reversed Diastolic Flow)
FVW of Middle cerebral artery- Increased PI and can appear normal or reversal of flow

It is unknown on the time interval of the appearance of these abnormal FVW to fetal death. It can be possibly occurring not more than 2-3 days or even few hours.9,10

V. Clinical Applications

A. Aortic Doppler Velocimetry Associated with IUGR and Fetal Hypoxia

The major portion of blood in the fetal descending aorta is directed to the placenta. The presence of resistance to flow in the placental vascular bed will greatly affect the umbilical artery and also the descending aorta showing either decreased or absent diastolic velocity. The initial response is to adapt to hypoxia is by increasing the blood supply with preferential delivery of nutrients and oxygen to most vital organs, as the brain, heart and adrenals and in turn decreasing the perfusion of the kidneys, gastrointestinal tract, and lower extremities. This redistribution may produce an increase in vascular resistance to the flow in the fetal aorta which is reflected in the change of aortic FVW (flow velocity waveform).5,9,10,11

1. Peripheral vasoconstriction is associated with reduction of blood flow in the viscera that may explain the increased rate of necrotizing enterocolitis reported in growth restricted fetus with associated AEDV of fetal aorta.15

2. Aortic peak systolic velocity is considered to reflect myocardial contractility. In the study by Jouppila et al., there is a significant reduction in aortic peak velocity in hypertensive pregnancies with fetal distress.5,16

3. In hypoxic fetuses, changes in aortic FVW were usually detected before the occurrence of an abnormal cardiotocographic (CTG) finding. However the time of occurrence CTG fetal distress and aortic FVW abnormalities are not consistent. Time interval varies with different studies ranging from 3 days (Jouppila et al. 1984, Lingman et al. 1986, Laurin et al 1987) to 2-3 weeks (Griffin et al., 1984).5,15,16
4. The degree of abnormality of aortic FVW correlates well with cord aterial pH suggesting that hypoxia is already present by the time FVW changes are evident. In another study has shown a decrease of end-diastolic flow in fetal aorta were significantly correlated to frequency of FHR decelerations during contraction stress tests, low pH, base excess in IUGR fetuses. A decrease in end-diastolic flow in umbilical artery paralleled with an increase in doppler indices(RI, S/D ratio) in fetal descending aorta reflect oxygen deprivation in IUGR fetuses in pre-eclamptic pregnancies with or without HELLP syndrome.\textsuperscript{17,18}

B. Fetal Cardiac Arrhythmia
Doppler recording of fetal aortic velocities provides important information of hemodynamic consequences of fetal cardiac arrhythmias.\textsuperscript{5,6}

1. Measurement of flow velocity waveform of fetal abdominal aorta reflects ventricular contractions and the inferior vena cava reflecting atrial contractions and designates cases of fetal arrhythmias.

2. Most cases of fetal arrhythmias showed normal aortic volume flow.

3. Fetuses with severe arrhythmias with concomitant heart failure showed a subnormal low aortic flow results.

4. In cases of bradyarrhythmias or tachyarrhythmias, the decrease in aortic flow was observed when the fetal heart rate exceeded its limits of 50 or 230 bpm respectively.

5. Intrauterine treatment of fetuses with digoxin diagnosed with heart failure showed an improved performance of fetal heart by measuring fetal aortic volume blood blow.

C. Fetal Anemia
Severe anemia in the fetus often resulted from red blood cell hemolysis of varied etiologies like parvovirus infection, fetal hemorrhage, blood group isoimmunization. The diagnosis of fetal anemia is possible by invasive testing by method of cordocentesis and non invasive modality has been preferred by use of doppler ultrasound examination.\textsuperscript{5,6}

1. In the descending aorta of previously untransfused isoimmunized fetuses reported an increased mean blood velocity and a negative correlation with the hematocrit of umbilical cord blood obtained by cord puncture by fetoscopy. This finding was confirmed by Nicolaides et. al., this study has showed values of mean aortic velocity to hemoglobin deficit in blood samples obtained by cordocentesis.\textsuperscript{19}

2. Fetal anemia leads to increase in cardiac output, decrease in blood viscosity and increased in peripheral blood vessel peak velocities. The findings of increased fetal aortic velocities in anemic fetuses showed increased cardiac output. It was observed that after blood transfusion the mean velocity of fetal aorta velocity became normal.\textsuperscript{5}

3. No waveform changes indices was noted in cases of fetal anemia.

Recently, the findings of hypoxia induced cerebral blood flow make the middle cerebral artery (MCA) velocity an ideal test to study fetal anemia and was found to be more sensitive and more superior to fetal ascending aorta in the determination of fetal anemia.\textsuperscript{4,5,6,7,8,9,10}
D. Postnatal Follow up of Infants with In-utero Doppler Velocimetry of Fetal Aorta

Long term follow up of infants who suffered restriction and hypoxia in utero with previous Doppler of fetal aortic velocimetry in the study by Malmo et al. and Ley et al. investigating the somatic, neurologic and psychological status of 149 children at age 7. All had Doppler velocimetry of fetal descending aorta and 50% were growth restricted at birth. Infants with abnormal intrauterine aortic blood flow had an increased frequency of minor neurologic abnormalities and lower mean intelligence quotient than infants with normal Doppler measurements. This has come to speculations that abnormal fetal aortic BFC in growth restricted fetus has a role for interventions in the early delivery to prevent fetal death. A similar study was done by Tideman et al. among young adults who were growth restricted in utero have shown impaired cognitive function with abnormal fetal blood flow in the descending aorta.

E. Fetal-Aortic-Cerebral Doppler Resistance Index Ratio

A normal (ACRI) Fetal Aortic Cerebral Doppler resistance index ratio reflects identical vascular resistance of the fetal descending aorta and cerebral vessels. In the study of Aranyosis et al. an ACRI >1.2 is a useful marker of the centralized arterial circulations indicating the earlier stage of fetal hypoxemia.

Impaired placental performance results to redistribution of the fetal arterial circulation with minor changes in the vascular resistance of fetal descending aorta and middle cerebral artery. Fetal hypoxia results in decrease in the vascular resistance in the MCA while resistance in the fetal descending aorta increases as a response to peripheral vasoconstriction in the splanchnic and skeletal region.

A study of 96 uncomplicated pregnancy at 38-40 weeks were recruited for the cross-sectional assessment of Doppler resistance index in the fetal descending aorta and middle cerebral artery. An attempt was done to establish a cut off value for the purpose of giving an estimate that this ratio will identify possible fetal compromise among small for dates fetuses. In this study the mean values of ACRI are close to 1.0 with overall mean of 1.062 (+/-0.087).

The value of cut off limit to classify normal from abnormal ACRI (overall mean +2 SD) equals to 1.236.

1. The practical application of ACRI to be considered normal is below 1.2. A normal ACRI reflects a physiologic maintenance of fetal central blood flow that identifies a normal flow in the fetal descending aorta and middle cerebral artery.

2. An abnormal ACRI of >1.2 may be used as a marker of centralized circulation that may be a sign of early fetal insult or hypoxia

VI. Conclusion

The fetal descending aorta is suitable for blood flow velocity measurements because it is an arterial vessel of interest that has good visualization appearing as straight course from thoracic to abdominal branches. It has great reliability and reproducibility based on correct sample volume and angle of insonation. The aortic thoracic blood flow velocity waveform reflects the hemodynamic state of a large portion of the fetal circulation including placental perfusion, peripheral perfusion, and cardiac output.

Doppler measurements in the fetal descending aorta provide additional diagnostic value in detecting fetuses at risk for IUGR, hypoxia and fetal acidemia. It has limited value in the detection of fetal anemia and arrhythmias. There is a possible association between abnormal fetal aortic velocity...
waveform and neurodevelopment impairment in childhood. A new ratio, the fetal aortic-cerebral resistance index is promising and further studies needs to be performed to validate its clinical significance.

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