Blood flow through the ductus venosus in singleton and multifetal pregnancies and in fetuses with intrauterine growth retardation

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OBJECTIVE: It is known from animal experiments that blood flow through the ductus venosus changes with fetal strain. Therefore the ratio of umbilical vein to ductus venosus flow rate in human intrauterine growth retardation and multifetal pregnancies was investigated and compared with that in control subjects.

STUDY DESIGN: Blood flow rates in the umbilical vein and in the ductus venosus, as well as peak velocity, minimum velocity, mean velocity, and pulsatility index (maximum velocity envelope curve) in the ductus venosus, were measured in women with normal pregnancies (n = 55), intrauterine growth retardation (n = 20), and multifetal pregnancies (10 women with 20 fetuses) with color Doppler ultrasonography.

RESULTS: Average ductus venosus blood flow rates (mean ± SD), normalized for estimated fetal body weight, were 60 ± 30, 69 ± 35, and 77 ± 28 (ml·min⁻¹·kg⁻¹) in control subjects, intrauterine growth retardation, and multifetal pregnancies, respectively. Umbilical vein blood flow rates amounted to 140 ± 59, 111 ± 54, and 141 ± 47 (ml·min⁻¹·kg⁻¹). Both absolute flow rates increased with gestational age, whereas normalized flow rates decreased. The percentage of umbilical blood flow passing through the ductus venosus in the control group was 43% ± 9%. It was significantly increased in both intrauterine growth retardation (62% ± 8%) and in multifetal pregnancies (55% ± 12%). Peak velocity, minimum velocity, mean velocity, and pulsatility index in the ductus venosus were not significantly different between groups.

CONCLUSION: The increased ratio of ductus venosus blood flow to umbilical vein blood flow may indicate fetal strain. (Am J Obstet Gynecol 1998;178:943-9.)

Key words: Ductus venosus, intrauterine growth retardation, multifetal pregnancies, blood flow, Doppler ultrasonography

The ductus venosus serves as a bypass for the hepatic microcirculation for umbilical (and portal) venous blood, which is believed to improve oxygen delivery to other fetal organs. It has been suggested (see below) that ductus venosus blood flow may change in response to fetal requirements, but little is known about the actual role and significance of the ductus venosus.1

Several investigators have measured the extent of umbilical venous blood flowing through the ductus venosus. In acute experiments in human fetuses the proportion of umbilical venous blood perfusing the ductus venosus varied considerably (8% to 92%),2 as well as in instrumented rhesus monkeys (30% to 71%)3 and fetal lambs (36% to 64%).4,7

By use of invasive techniques in animal models it has been determined that the ductus venosus flow changes significantly with variation of fetal conditions. Thus Reuss et al.7 and Rudolph et al.5 were able to show in instrumented fetal lambs that during hypoxemia the proportion of umbilical venous blood passing through the ductus venosus increased from 38% to 55% to 60% to 65%. Paulick et al.8 demonstrated in the same animal model that increases in umbilical and hepatic venous resistance can be provoked by infusions of norepinephrine and epinephrine.

Measurements of blood flow velocity with Doppler ultrasonography in fetuses seriously afflicted by intrauterine growth retardation have shown changes of the velocity profile, but no information is available in less severe cases.9

The primary goals of this study were to investigate the changes of ductus venosus blood flow during pregnancy and to determine the proportion of umbilical venous blood flow passing through the ductus venosus in patients with normal singletons, in multifetal pregnancies, and in patients with fetuses with intrauterine growth retardation. Because intrauterine growth retardation may be associated with hypoxia and chronic intrauterine...
stress,10,11 we hypothesized that a variation of ductus venosus blood flow may be found in fetuses with intrauterine growth retardation.

Material and methods

Experimental groups. The protocol of the study followed the guidelines of our institution, and oral consent to investigate the ductus venosus was given by the participants. Initially, in 147 pregnant women ductus venosus blood flow rate was measured. Of these patients, only 85 women with 95 fetuses (for reasons discussed below) entered the study; they were divided into three groups. These consisted of control subjects, a group with growth-retarded fetuses (intrauterine growth retardation), and those with multifetal pregnancies because it was noted that in fetuses of multifetal pregnancies ductus venosus blood flow rate seemed to differ from control subjects. At the time of entry gestational age was determined on the basis of the last menstrual cycle. Late in the first or early in the second trimester a level I ultrasonographic examination was performed to confirm or correct the gestational age on the basis of biparietal diameter and femur length. The basic criteria for inclusion in any group were normal fetal structure assessed by routine ultrasonographic examination and gestational age assessed before week 20. Fetal weight was estimated from the head circumference, femur length, and the abdominal circumference.12 This weight determined the assignment of singleton pregnancies to either the control or intrauterine growth retardation group regardless of weight at birth, and it was used when blood flow rates were normalized for body weight.

In the first group estimated fetal weight was between the 10th and 90th percentiles.12 Actual weight at birth was in the same range of weight percentiles.

The second intrauterine growth retardation group included women with singleton fetuses who at the time of flow measurements had estimated fetal weights <10th percentile. In three fetuses weight at birth was slightly >10th percentile.

The third group of women with multifetal pregnancies (multifetal pregnancies group) included six patients with twin and four patients with triplet gestations (only 20 fetuses entered the study).

Patients were admitted for a variety of obstetric indications. The most frequent referral diagnoses in addition to intrauterine growth retardation (23%) and multifetal pregnancies (11%) were gestational diabetes without insulin treatment (9%) and rhesus(D) isoimmunization (6%). Diabetes was well controlled; those patients who were Rh(D) negative also had good control. Complications caused by diabetes or the Rh situation did not occur in any of these pregnancies, and the rate of cesarean section or forceps delivery was not increased in these patients compared with the control group. With the exception of intrauterine growth retardation (see following text), there were no signs (Doppler velocity profiles) of abnormal aortic, umbilical artery, or uterine artery perfusion.

Doppler ultrasonographic measurements. All Doppler ultrasonographic measurements were made by the same investigator (M.T.) with an ATL Ultramark 9 (Advanced Technology Laboratories, Munich) or a Kranzbühler GE LOGIQ 500 (Kranzbühler Uedizinische Systeme GmbH, Solingen, Germany) color-coded pulsed Doppler ultrasonography system, with 5 to 7 MHz convex transducers (acoustic output <50 mW/cm² spatial peak-time average intensity).

Doppler evaluations were done with the mother in the left lateral recumbent position.

Measurements were accepted when the fetus did not breathe or move and when at least three nearly identical consecutive typical waveforms could be obtained.

The Doppler ultrasonographic systems calculated the average blood flow rate (ml · min⁻¹) from the time-averaged intensity, weighted mean envelope curve derived from the Doppler spectra of the cardiac cycles and from the vessel diameter. The inner diameter was measured in frozen B mode to the nearest tenth of a millimeter by placing the calipers at right angles to the vessel axis. Care was taken to obtain the largest diameter of the longitudinal vessel sections. In the isthmic portion of the ductus venosus color mode had to be turned off because the pixels usually covered the lumen and the vessel wall.

Switching to Doppler mode, the ultrasonographic beam (width <1 mm) was positioned at the region of interest by means of visual and audio control. Doppler angles were <60 degrees (average value 29 degrees, range 0 to 60 degrees; no significant differences between groups). Blood flow rates were normalized for the estimated fetal body weight at the time of measurement (ml · min⁻¹ · kg⁻¹).

Flow values of each patient are the mean of two to seven (average four) repeated measurements that include the repeated measurement of vessel diameters. In general, measurements were accepted only when instantaneous variation was <10%. To establish the intraobserver variability in 20 randomly selected patients (12 controls, 8 intrauterine growth retardation), SD that was based on four measurements of blood flow rate within 10 minutes was determined and found to be ≤5% of the respective mean flow values with no difference of variability between both groups.

Ductus venosus blood flow. The ductus venosus was investigated (B mode) as follows. At first the sinus venae portae and the entering umbilical vein (UV) were visualized in an oblique (or sometimes a midsagittal) section of the fetal abdomen. Then, without losing this view, the probe was manipulated until the origin of the ductus venosus from the sinus became visible. The Doppler gate...
was positioned at the vessel’s origin, which in case of the ductus venosus is identical with its isthmic portion. Care was taken in switching to Doppler mode to obtain the highest flow velocity value in the ductus venosus, which is the hallmark of isthmic flow.13

In pulsed-wave Doppler mode the B scan was frozen and not updated. It is possible therefore that during flow measurements the Doppler gate did not precisely cover the isthmic region. In most of these cases (Doppler gate shifted to the sinus region or to liver veins) the Doppler signal distinctly deviated from the expected signal, and the investigator returned to B mode for a new measurement cycle. It cannot be excluded that the gate was positioned sometimes on the ductus venosus between the isthmus and the vena cava with a Doppler signal very similar to the one typical for the isthmus. Flow measurements would be erroneous then because the site of velocity and cross-sectional area measurements were different. However, this was a rare event because mostly the ductus venosus Doppler signal was completely lost or the Doppler signal of the vena cava was picked up in addition.14

The Doppler waveform at the outlet of the ductus venosus may be distinguished from the waveform at the isthmic portion.13 In any of the above-mentioned events the measurements were discarded, and a new measurement cycle was started.

Peak velocity, minimum velocity, mean velocity, and pulsatility index (PI) in the ductus venosus derived from the maximum velocity envelope curve were recorded in addition when possible (78 patients).

UV blood flow. For UV blood flow rate measurement a straight middle segment of the intraabdominal part of the UV was selected. The Doppler sample volume was advanced to cover the vessel, and the resulting signal was maximized on the basis of audio (venous hum) and visual waveform recognition.15

Statistics. Average data are presented as mean ± SD. Number of patients or fetuses is indicated by n in the tables. Differences between groups were tested by means of the t test or the Mann-Whitney U test (percentage ductus venosus flow of UV flow) with selected casewise deletion of missing data. Differences were considered significant at p < 0.05. Standard linear regression analysis was used to correlate flow rates with gestational age. In 10 patients Doppler measurements were repeated later in the pregnancy. These results are included in Figs. 1 and 2 but not in the statistical description and analysis.

Results

Patient recruitment. In each group either patients or measurements or both had to be excluded, which indicates the technical difficulties encountered. In most of the discarded cases the diameter of the isthmic portion of the ductus venosus could not be measured accurately or variation of instantaneous consecutive flow measurements was excessive (see preceding text). Weight and gestational age at birth were not always available from pa-
tients who chose to be delivered at other hospitals. None of the fetuses was known to have died in utero or within 1 week after birth. There were, however, some pathologic conditions associated with the group of fetuses with intrauterine growth retardation. In about 64% of these patients a “notch” in the uterine artery velocity waveform was detected. Eight patients had signs of oligohydramnios or anhydramnios, and in 15 fetuses an increase of PI in the umbilical artery was noted; absence or reversal of end-diastolic flow was observed in 2 fetuses only. UV pulsations were encountered in 7 fetuses.

In the multifetal pregnancies group, three fetuses were small for gestational age at birth but not at the time of measurement.

Doppler flow recordings. Fig. 1 illustrates the increase of umbilical venous and ductus venosus blood flow rates (ml · min⁻¹) in the control group during the course of gestation. When normalized for estimated fetal weight, both flow rates decreased with gestational age (Fig. 2). The correlation coefficients and slopes of both regressions were statistically significant.

Table I summarizes results of flow measurements in control singleton pregnancies, pregnancies with intrauterine growth retardation, and multifetal pregnancies. STD, SD.

Fig. 2. Scatter plot of UV and ductus venosus blood flow rates normalized for estimated fetal weight (bw) during gestational age (control group, 55 patients). Open circles, UV flow; closed squares, ductus venosus flow. Dotted lines connect additional measurements (crosses) of individual patients. Regression: UV flow rate body weight⁻¹ (ml · min⁻¹ · kg⁻¹) = 504.2 – 0.76 · gestational age (days), r = –0.54 (open circles). Ductus venosus flow rate body weight⁻¹ (ml · min⁻¹ · kg⁻¹) = 139.5 – 0.37 · gestational age (days), r = –0.51 (closed squares).

Fig. 3. Box-and-whisker plot of percentage of ductus venosus flow on UV flow in control, intrauterine growth retardation, and multifetal pregnancies.
The proportion of umbilical venous return passing through the ductus venosus in the control group was 43% ± 9%; it increased to 62% ± 8% in the intrauterine growth retardation group and to 55% ± 12% in multifetal pregnancies, both significant (p < 0.0005) changes (Fig. 3).

Blood flow rate through the liver was estimated as the difference between umbilical venous and ductus venosus blood flow. Liver blood flow was significantly decreased in pregnancies with intrauterine growth retardation and multifetal pregnancies (Table I). The normalized liver perfusion was significantly decreased only in intrauterine growth retardation pregnancies.

Mean peak blood flow velocity in the ductus venosus (on the basis of the maximum velocity envelope curve) was in the range of 80.0 to 83.5 cm · sec−1. No significant differences were found in minimum, mean, and peak velocity or PI in the ductus venosus between groups (Table II).

### Table I. Blood flow parameters of ductus venosus and UV in control, intrauterine growth retardation, and multifetal pregnancies (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 55)</th>
<th>Intrauterine growth retardation (n = 20)</th>
<th>Multifetal pregnancies (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UV mean velocity (cm · sec−1)*</td>
<td>12.4 ± 4.4</td>
<td>9.9 ± 2.4†</td>
<td>10.4 ± 3.4</td>
</tr>
<tr>
<td>UV flow rate (ml · min−1)</td>
<td>214.9 ± 109.7</td>
<td>154.5 ± 58.7†</td>
<td>160.8 ± 108.1</td>
</tr>
<tr>
<td>UV flow rate/body weight (ml · min−1 · kg−1)</td>
<td>140.0 ± 59.4</td>
<td>110.9 ± 54.0</td>
<td>140.8 ± 46.7</td>
</tr>
<tr>
<td>Ductus venosus diameter (mm)</td>
<td>2.5 ± 0.6</td>
<td>2.5 ± 0.5</td>
<td>2.7 ± 0.6</td>
</tr>
<tr>
<td>Ductus venosus mean velocity (cm · sec−1)*</td>
<td>31.8 ± 15.0</td>
<td>32.4 ± 12.2</td>
<td>26.9 ± 13.2</td>
</tr>
<tr>
<td>Ductus venosus flow rate (ml · min−1)</td>
<td>90.9 ± 42.2</td>
<td>96.3 ± 42.2</td>
<td>93.0 ± 58.8</td>
</tr>
<tr>
<td>Ductus venosus flow rate/body weight (ml · min−1 · kg−1)</td>
<td>60.3 ± 30.0</td>
<td>68.5 ± 34.5</td>
<td>77.0 ± 28.1†</td>
</tr>
<tr>
<td>Liver flow rate (ml · min−1)</td>
<td>124.0 ± 70.8</td>
<td>18.3 ± 22.2†</td>
<td>76.9 ± 58.9†</td>
</tr>
<tr>
<td>Liver flow rate/body weight (ml · min−1 · kg−1)</td>
<td>76.3 ± 34.5</td>
<td>43.0 ± 23.2†</td>
<td>60.6 ± 31.9</td>
</tr>
</tbody>
</table>

IUGR, Intrauterine growth retardation; body weight, estimated fetal weight. In multifetal pregnancies group 20 fetuses (10 mothers) were investigated.

*Intensity-weighted mean.
†p < 0.05.
‡p < 0.0005.

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### Outcome data. As expected from the estimated fetal weight, birth weight in the intrauterine growth retardation group (2213 ± 767 gm, n = 13) was significantly less than in the control group (3272 ± 617 gm, n = 39; multifetal pregnancies: 1988 ± 638 gm, n = 18 fetuses). Gestational age at delivery (control: 272 ± 17 days, n = 39; intrauterine growth retardation: 259 ± 27 days, n = 14; multifetal pregnancies: 236 ± 19, n = 10 women) and birth weight of multifetal pregnancy fetuses were less than in the control group because triplet pregnancies were terminated routinely by cesarean section between weeks 32 and 35.

At birth arterial cord pH values were normal in controls, intrauterine growth retardation, and multifetal pregnancies, and normal Apgar scores were found in control and intrauterine growth retardation, whereas the median Apgar score of the multifetal pregnancies group was reduced (data not shown).

The averages of gestational age and estimated fetal weight at the time of investigation were 216 ± 42 days for controls (estimated weight 1775 ± 1001 gm), 221 ± 38 days for intrauterine growth retardation (estimated weight 1633 ± 808 gm), and 195 ± 29 days for multifetal pregnancy fetuses (estimated weight 1230 ± 596 gm). The differences between controls and the intrauterine growth retardation group were not significant. The multifetal pregnancy group had a small number of measurements at high gestational ages for the reasons discussed in the preceding text. Mean gestational age and estimated weight at the time of measurements were thus reduced.

### Comment

Our study (Fig. 2) confirms previous reports on blood flow rates through the UV in human fetuses. By use of Doppler ultrasonography, Gerson et al.15 demonstrated that umbilical venous blood flow per kilogram estimated fetal weight decreased with gestational age: 131 ml · min−1 · kg−1 at 30 weeks’ and 108 ml · min−1 · kg−1 at 40 weeks’ gestational age. In this study we found similar values in the control group: 135 ± 70 ml · min−1 · kg−1 at weeks 29 to 31 (n = 7), and 121 ± 28 ml · min−1 · kg−1 at weeks 39 to 41 (n = 6). Because the precision of Doppler ultrasonography flow rate measurement is debatable, this congruence suggests that the quality of our flow and weight measurements is appropriate and that our control group is comparable in this respect to others. We believe that in the hands of one skilled investigator flow measurement data are valid at least on a relative basis.

In addition, the peak velocity values (Table II) agree favorably with recent velocity measurements in the isthmic portion of the ductus venosus,13 and repeated measurements in some patients (Figs. 1 and 2) demonstrate that most of the individual sequences of data follow the general trend.
In the course of pregnancy the blood flow through the ductus venosus follows a pattern similar to that of UV flow (Figs. 1 and 2). The percentage of UV blood flow entering the ductus venosus appeared to decrease slightly (not significant) in the control group with gestational age (linear regression, \( y = 0.56 \pm 0.04 \times \)) and with estimated weight (\( y = 45.6 \pm 0.013 \times \)). At the above-mentioned gestational ages ductus venosus blood flow rates per kilogram estimated fetal weight were 64 ± 37 ml · min⁻¹ · kg⁻¹ (\( n = 7 \), weeks 29 to 31) and 45 ± 12 ml · min⁻¹ · kg⁻¹ (\( n = 6 \), weeks 39 to 41), which is equivalent to 47% ± 8% and 39% ± 8% of umbilical venous flow.

The small variation with gestational age of the ductus venosus to UV flow ratio (percentages) justifies the calculation of averages regardless of gestational age (see following text), and it makes it unlikely that the difference in flow percentages between controls and intrauterine growth retardation or multifetal pregnancies (Table I) is due to differences in gestational age or fetal weight. This was confirmed by comparing intrauterine growth retardation and multifetal pregnancy flow ratios not directly with all control values but with control values calculated from the regression at the respective average weight or gestational age of intrauterine growth retardation or multifetal pregnancy group. We again found a significant increase of the measured blood flow ratio in the intrauterine growth retardation group and in the multifetal pregnancy group compared with the calculated values. This demonstrates that the differences are not caused by differences of weight and gestational age alone.

By use of Doppler ultrasonography Emerson et al.\(^{16}\) and Weiner et al.\(^{15}\) observed similar changes of the umbilical venous return in growth-retarded human fetuses.

In fetal lambs hypoxemia and infusion of catecholamines tended to decrease liver blood flow and to increase the percentage of blood passing through the ductus venosus.\(^{5, 8}\) Because increased catecholamine concentrations in plasma (sheep) and in amniotic fluid (humans) were found in fetuses with intrauterine growth retardation,\(^{11, 18}\) we speculate that a comparable mechanism might be responsible for the blood flow redistribution in intrauterine growth retardation and possibly also in multifetal pregnancies. Most of the blood flow through the ductus venosus moves directly through the foramen ovale to the left side of the heart,\(^{19}\) and thus the relative increase of ductus venosus flow may be regarded as a compensatory mechanism to maintain oxygen supply to vital organs (e.g., brain and heart).\(^{1}\) The other aspect, however (that is, the reduction of blood supply to the liver), should not be overlooked. Because fetal proteins are predominantly synthesized in the liver,\(^{20}\) it may be speculated that the low fetal body weight in intrauterine growth retardation is partially caused by impairment of protein biosynthesis after reduction of fetal liver blood flow. In multifetal human pregnancies a similar mechanism may be effective.

We could not detect differences of peak velocity, minimum velocity, mean velocity, and PI between groups, whereas in seriously growth-retarded fetuses (of 38 fetuses 7 died in utero) reduction of minimum velocity in the ductus venosus has been observed.\(^{19}\) This suggests that Doppler velocimetry of the ductus venosus will be abnormal only when fetuses are severely compromised, whereas the ratio of ductus venosus to UV flow rates possibly is an indicator of impaired fetal condition.

The mechanism that causes the shift in blood flow ratios is still unknown. It involves a change of flow resistances of either the liver vessels or the ductus venosus. Paulick et al.\(^{8}\) demonstrated that acute fetal hypoxemia increases the vascular resistance of the hepatic venous circulation such that the proportion of umbilical venous return passing through the liver decreased by ~15%. We could not observe differences of the diameter of the ischemic portion of the ductus venosus (Table I), which presumably contributes mostly to the ductus venosus resistance. As previously described by others,\(^{21}\) in our data ductus venosus diameter increased significantly (\( r = 0.4 \)) with gestational age (data not shown). Our mean ductus venosus diameter measurements are in the upper range of values reported so far,\(^{21}\) which may be due to methodologic reasons (see above).

We suppose that the relative increase of ductus venosus blood flow in intrauterine growth retardation and multifetal pregnancies is caused by an increase in hepatic vascular resistance and not by increases of the ductus venosus.

### Table II. Blood flow velocity data in ductus venosus in control, intrauterine growth retardation, and multifetal pregnancies

<table>
<thead>
<tr>
<th>Maximum envelope curve evaluation</th>
<th>Control pregnancies (( n = 42 ))</th>
<th>Pregnancies with IUGR (( n = 18 ))</th>
<th>Multifetal pregnancies (( n = 18 ))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ductus venosus peak velocity (cm · sec⁻¹)</td>
<td>80.0 ± 29.1</td>
<td>83.5 ± 47.4</td>
<td>82.6 ± 24.8</td>
</tr>
<tr>
<td>Ductus venosus minimum velocity (cm · sec⁻¹)</td>
<td>39.8 ± 19.0</td>
<td>39.5 ± 12.9</td>
<td>39.1 ± 12.2</td>
</tr>
<tr>
<td>Ductus venosus mean velocity (cm · sec⁻¹)</td>
<td>65.5 ± 23.9</td>
<td>66.9 ± 30.0</td>
<td>66.7 ± 20.5</td>
</tr>
<tr>
<td>Ductus venosus PI</td>
<td>0.64 ± 0.22</td>
<td>0.61 ± 0.24</td>
<td>0.64 ± 0.20</td>
</tr>
</tbody>
</table>

IUGR, Intrauterine growth retardation.
sus diameter (which may be influenced by prostaglandins). Other, more passive mechanisms may be involved also.

In conclusion, we found a significant increase in the proportion of venous blood flow through the ductus venosus in pregnancies with intrauterine growth retardation and multifetal pregnancies compared with control singleton gestations. In a clinical setting the ratio of ductus venosus to UV blood flow might serve to indicate fetal strain early.

REFERENCES