Hyrtl's anastomosis is normally developed in placentas from small for gestational age infants.

Ullberg, Ulla; Lingman, Göran; Ekman-Ordeberg, Gunvor; Sandstedt, Bengt

Published in:
Acta Obstetricia et Gynecologica Scandinavica

DOI:
10.1034/j.1600-0412.2003.00161.x

Published: 2003-01-01

Citation for published version (APA):
Hyrtl’s anastomosis is normally developed in placentas from small for gestational age infants

ULLA ULLBERG1, GÖRAN LINGMAN2, GUNVOR EKMAN-ORDEBERG1 AND BENGT SANDSTEDT3

From the 1Department of Woman and Child Health, Karolinska Institute, the 2Department of Obstetrics and Gynecology, Lund University Hospital, and the 3Department of Clinical Pathology, Karolinska Institute, Danderyd Hospital, Stockholm, Sweden

Background. The aim of this study was to investigate the occurrence and appearance of the anastomosis between the two umbilical arteries in placentas from infants small for gestational age (SGA).

Methods. The arterial systems of 64 placentas from singleton pregnancies resulting in SGA infants were visualized by angiography. The method allowed study of the anastomosis between the umbilical arteries and calculation of the relative placental area supplied by each artery. The results were compared with findings in a previous study of appropriate for gestational age (AGA) infants. One-way analysis of variance (ANOVA) and χ²-analyses were used for statistics.

Results. In 56 placentas the anastomosis was represented by a true vessel, in two by a fenestration, and in another two cases by fusion of the umbilical arteries. The anastomosis was absent in one case and another three cases had a single umbilical artery (SUA). When the diameter of the anastomosis was thinner than that of the umbilical arteries, their supply areas were significantly (p ≤ 0.001) more symmetrical than in cases with a wider anastomosis.

The anatomy of the anastomosis and the relationship between its width and the symmetry between the supply areas of each umbilical artery did not differ in placentas from SGA and AGA infants, despite various types of cord insertion and placentation.

Conclusion. Static measurements of Hyrtl’s anastomosis do not indicate a contributing part for intrauterine growth retardation.

Key words: angiography; fetal growth; Hyrtl’s anastomosis; placenta; umbilical arteries

Submitted 8 October, 2002
Accepted 19 January, 2003
between the two umbilical arteries was detected, which was higher after the anastomosis. This demonstrated for the first time in vivo its important function as a pressure equalizer. The flow in the anastomosis was in all cases unidirectional towards the umbilical artery with a lower resistance index.

The occurrence and variable appearance of the Hyrtl anastomosis has been described previously in some undefined materials (2,10,11) and more recently by our group in full-term placentas from appropriate for gestational age (AGA) infants with normal umbilical artery blood flow (1). We were able to relate the width of the anastomosis to the degree of symmetry between the placental areas supplied by each umbilical artery.

The placental vascular architecture is often abnormal in intrauterine growth retardation (12–14), suggesting also that the anastomosis might differ in small for gestational age (SGA) placentas, being hypothetically more important in this condition associated with decreased placental capacity.

Hyrtl’s anastomosis has so far not been studied in placentas from SGA infants, which is why our aim was to investigate its occurrence and appearance in this setting.

Materials and methods

The placentas from 64 singleton pregnancies resulting in an SGA infant were collected and frozen at −20°C after registration of weight, size and configuration. SGA was defined as birth-weight according to gestational age <−2 SD, that is <−22% using the Scandinavian standard curve (15).

The median age of the mothers at partus was 29 years (range 15–39 years). Thirty-eight mothers were primiparas and 26 had previously given birth to 1–3 children.

Thirty-two of the 64 babies were delivered instrumentally because of threatening asphyxia, judged by cardiotocography (CTG) registration, which might reflect an acute utero-placental insufficiency. The remaining 32 babies were nonasphyctic and delivered vaginally. The pregnancy was complicated by preeclampsia in six cases in the former group and in 17 in the latter group. The main characteristics in the non-asphyctic and the asphyctic groups, including placental drained weights and median placental areas compared with AGA infants (1), are presented in Table I.

Table I. Birthweight deviation and anatomical parameters for SGA placentas compared to an AGA material (1). SGA pregnancies were divided into asphyctic (SGA<sub>asph</sub>) and nonasphyctic (SGA<sub>nonasph</sub>). For rows 1–3 one-way ANOVA and for rows 4–5 χ²-analysis was used for statistics of differences between groups

<table>
<thead>
<tr>
<th></th>
<th>SGA&lt;sub&gt;asph&lt;/sub&gt; (n=32)</th>
<th>SGA&lt;sub&gt;nonasph&lt;/sub&gt; (n=32)</th>
<th>AGA (n=67)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birthweight deviation (%)</td>
<td>−31</td>
<td>−27</td>
<td>−7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Placental weight (g)</td>
<td>268</td>
<td>298</td>
<td>428</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Placental area (cm²)</td>
<td>208</td>
<td>231</td>
<td>310</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Extrachorial placenta</td>
<td>15</td>
<td>10</td>
<td>14</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Marginal/velamentous</td>
<td>8</td>
<td>13</td>
<td>3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>cord insertion</td>
<td>(25%)</td>
<td>(41%)</td>
<td>(4.5%)</td>
<td></td>
</tr>
</tbody>
</table>
After thawing, the arterial tree of each placenta was filled with barium X-ray contrast medium and angiograms achieved in two perpendicular projections as described in earlier work (1,13). The anastomosis was measured and characterized in the angiograms according to our previous study in placentas from full-term AGA infants with normal umbilical artery blood flow (1). The width was measured in relation to that of the umbilical arteries, making the comparison between placentas of different sizes feasible.

Anastomoses that could not be clearly classified in this way were dissected. Furthermore, all placentas without anastomosis identified by X-ray were dissected to confirm its absence. After angiography the placentas were sliced to exclude major lesions. One vessel-like anastomosis and the connected umbilical artery were also cut for conventional histology and stained with an immunocytochemical myogenic marker, Dako antidesmin.

The umbilical cord insertion was considered velamentous when it was located in the membranes, marginal when the distance between the cord insertion and the placental margin was less than 3 cm and central in the remaining cases. Extrachorial placenta implies that the whole of the placental surface is not completely covered by chorion and includes both the circummarginate and the circumvallate type.

The angiograms were digitized and the placental area calculated as well as the relative area over which each umbilical artery sent its branches. The area supplied by each artery was expressed as a percentage of the total area. In perfect symmetry with each artery supplying an equal area, it is expressed as 50% and 50%, respectively. In extreme asymmetry these figures move towards 0% and 100%, respectively.

One-way analysis of variance (ANOVA) and \( \chi^2 \)-analyses were used for statistics of differences between groups.

**Results**

An anastomosis between the two umbilical arteries could be identified in 60 out of 64 placentas. In no case was there more than one anastomosis. The type of anastomosis was classified according to Fig. 3 (see also Table II). In 56 placentas the anastomosis was represented by a true vessel whereas in two it was represented by a fenestration between the two arteries. Two cases the arteries fused. The anastomosis was absent in one case, which was verified by elaborate dissection. Another three cases had a single umbilical artery (SUA) and, in these, no anastomosis was seen between allantochorial arteries. In 41 of the 58 placentas with an anastomosing vessel or fenestration the connection was between the main umbilical arteries, in 16 one of the umbilical arteries branched before the anastomosis and in one both did. The anastomosis was localized within the umbilical cord in five, at the cord insertion in 48 and on the placental surface in seven cases.

Of the five anastomoses within the umbilical cord the distance from the centre of the

| Table II. Type of anastomosis in placentas from SGA and AGA (1) infants. SGA pregnancies divided into asphyctic (SGAasph) and nonasphyctic (SGAnonasph) |
|-----------------|---------|------|-----|-----|-----|
|                 | Vessel  | Fenestration | Fusion | Absent | SUA  |
| SGAasph (n = 32)| 27 (84%) | 2 (6%) | 1 (3%) | 1 (3%) | 1 (3%) |
| SGAononasph (n = 32) | 29 (91%) | 0 (0%) | 1 (3%) | 0 (0%) | 2 (6%) |
| AGA (n = 67)    | 56 (84%) | 4 (6%) | 1 (1%) | 4 (6%) | 2 (3%) |

SUA, single umbilical artery.
cord insertion to the most distant part of the anastomosis ranged from 11 to 25 mm (median 14 mm) and to the nearest part from 4 to 14 mm (median 6 mm). Four anastomoses were vessel-shaped and one was of the fusion type. The seven anastomoses on the placental surface had their most distant part within 14–49 mm (median 17 mm) from the cord insertion. Six were vessel-shaped and one was of the fenestration type.

The length of the anastomosis varied between 0 and 20 mm (median 5.5 mm, mean 7 mm). The diameter of the anastomosis was measured in 57 of the 58 cases who had a vessel or fenestration type of anastomosis. One was not possible to measure for technical reasons. The diameter ranged between 1 and 5 mm (median 2 mm, mean 2.4 mm).

The width of the anastomosis was also classified in relation to that of the vessels (umbilical arteries or branches of them) it connected, in order to avoid bias from the differences in placental size. In one case this was not possible for technical reasons. The relative placental area supplied by each umbilical artery averaged 37% and 63% for SGA. Only in the placenta lacking anastomosis, despite two umbilical arteries, was the distribution 45% and 55%. The results are summarized in Table III. No significant difference was found between pregnancies leading to an SGA infant with or without asphyxia. However, symmetry values between placentas with wide, thin and absent anastomosis differed significantly ($p < 0.001$).

None of the placentas contained macroscopical lesions occupying more than 5% of the parenchyma. Morphologically, a transverse histological section of a vessel-like anastomosis, compared with the connected arteries, showed a considerably thinner and only a circular smooth muscle layer, but like the arteries without any elastic tissue (Figs 4 and 5a,b).

Table III. The width of Hyrtl's anastomosis related to the mean symmetry values in percent of total placental area supplied by branches of each umbilical artery. Wide Hyrtl refers to equal or larger diameter than the umbilical arteries whereas thin Hyrtl means smaller diameter. Placenta groups according to Tables I and II. Symmetry values differed significantly ($p > 0.001$) between wide, thin and absent anastomosis in all groups.

<table>
<thead>
<tr>
<th></th>
<th>Wide Hyrtl</th>
<th>Thin Hyrtl</th>
<th>No Hyrtl</th>
</tr>
</thead>
<tbody>
<tr>
<td>SGA asph</td>
<td>27/73%</td>
<td>38/62%</td>
<td>45/55%</td>
</tr>
<tr>
<td>($n = 9$)</td>
<td>($n = 19$)</td>
<td>($n = 1$)</td>
<td></td>
</tr>
<tr>
<td>SGA nonasph</td>
<td>31/69%</td>
<td>40/60%</td>
<td>–</td>
</tr>
<tr>
<td>($n = 7$)</td>
<td>($n = 22$)</td>
<td>($n = 0$)</td>
<td></td>
</tr>
<tr>
<td>AGA</td>
<td>26/74%</td>
<td>41/59%</td>
<td>45/55%</td>
</tr>
<tr>
<td>($n = 17$)</td>
<td>($n = 41$)</td>
<td>($n = 4$)</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 4. Transverse histological section of the vessel-like anastomosis in Fig. 1. Note thin circular muscle layer (Van Gieson elastic stain).

Fig. 5. (a) Detail of vessel-like Hyrtl's anastomosis seen in Figs 1 and 4. The thin single circular muscle layer brought out by immunoreactivity for desmin. Loose intimal and adventitial connective tissue. (b) Detail of connected umbilical artery wall. Note longitudinal inner and circular outer muscle layer (Van Gieson elastic stain).
Discussion

In the first month of gestation there is a meshwork of cord anastomoses (16) that is subsequently reduced to one single anastomosis (11). Functionally, Predanic et al. (7) observed a small difference in resistance to blood flow between the two umbilical arteries, which diminished during late pregnancy indicating a functional maturation or adaptation of the anastomosis. In a few cases with perfectly symmetric umbilical artery supply areas, lack of anastomosis has been observed suggesting regression of even the last connection when shunting is not needed (1).

The main function of Hyrtl’s anastomosis is probably to equalize the flow and pressure in the two arterial placental vascular territories (10). In this context it should also be mentioned that reasons to redirect the arterial flow can be present upstream due to, for example, external pressure, bleeding or vascular thrombosis in the cord or events in the fetal circulation, but we saw no signs of this in the present material. Abnormal vessel coiling in the cord has recently been found to be associated with fetal growth restriction (17). Coiling was evident in the angiograms, but we did not study the full length of the umbilical cord in this respect.

The most usual type of anastomosis was vessel shaped, which is probably able to react in the same way for stimuli as the umbilical arteries even if the muscular layer is thinner. The fenestration type would in the same way take part in contraction or dilatation of the arterial muscular wall. The various types of anastomoses in placentas from SGA infants were the same as those reported by Hyrtl (2), Bacsich and Smout (10) and Priman (16), which all were from unselected material.

Regarding the width of the anastomosis we do not know what effect the method used had on the vessels devoid of tonus. Remarkably, the reported dimensions are similar to those found in in vivo conditions as reported by Raio et al. (9), with a mean between 2 and 3 mm in both studies.

We previously found a relationship between intrauterine growth retardation and abnormal placental vascular pattern associated with marginal cord insertion and extrachorial placentalisation (13). It seems reasonable that this reflects placental growth and migration governed by the uterine vascular supply with consequences for the cotyledonary as well as the allantochorial vascular pattern. The function of the anastomosis as a safety valve may then hypothetically be more important in these conditions, especially in pregnancies with SGA infants ending with acute asphyxia or preeclampsia. However, as in the placentas from AGA infants we found the occurrence and width of the anastomosis in all settings related to the symmetry in size between the supply areas of each umbilical artery despite gross differences in type of placentation and cord insertion. The results suggest that the size of the umbilical artery supply area matters more for the anatomy of Hyrtl’s anastomosis than other factors.

We found no extensive macroscopical lesions and do not have an answer if such ones have an impact on the anastomosis. It is in this context interesting to note that Gudmundsson et al. (18), by studying placental perfusion using power Doppler, found silent areas that after selective catheterization postpartum were outlined in a normal manner in X-ray angiography and showed normal histology. Their interpretation was that the placenta is capable of a regional on–off selective perfusion, but the role of Hyrtl’s anastomosis in such dynamic flows was not commented upon.

The final size and type of Hyrtl’s anastomosis late in gestation seems to be the end result of a circulatory equilibrium, but to what extent it can redirect blood flow for acute demand is still unclear. An answer can only be given by long-term dynamic registrations.

In conclusion, we found that the Hyrtl anastomosis in placentas from SGA infants had a varied anatomy and a relationship between its width and the symmetry of the supply areas of each umbilical artery. The findings did not differ from those in placentas from AGA infants, suggesting it is a noncontributing part for intrauterine growth retardation. However, Hyrtl’s anastomosis should be borne in mind when assessing blood flow in the umbilical arteries.

References


Author for correspondence:
Ulla Ullberg
Department of Paediatric Radiology Q6:05
Astrid Lindgren Children’s Hospital at Karolinska Hospital
SE-171 76 Stockholm
Sweden