and intrapartum planning to optimize fetal salvage. These plans might include delivery at a tertiary care center and coordination with pediatric subspecialties for possible surgical intervention of the newborn infant.

We recognize that a genetic diagnosis is not always definitive. Other genetic disorders not associated with an obviously dismal prognosis may become apparent, such as mosaics, translocations, or sex chromosome abnormalities. We believe that responsible explanations and representations of expected neonatal prognoses can only be proposed when as much information as possible about a pregnancy is known. In caring for patients with pregnancies complicated by idiopathic hydramnios, important information can be obtained through knowledge of the fetal karyotype.

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REFERENCES

Circulating neurohypophyseal hormones in anencephalic infants

Herman P. Oosterbaan, M.D., Ph.D., and Dick F. Swaab, M.D., Ph.D.
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Human anencephalic infants have always been considered not to have circulating levels of oxytocin or vasopressin. However, this article shows that amniotic oxytocin levels in anencephalic infants without hydramnios fall within the control range. In addition, low levels of both oxytocin and vasopressin are present in the umbilical circulation. These peptides are probably derived from fetal sources other than the fetal brain, for example, the fetal adrenal cortex. (Am J Obstet Gynecol 1987;157:117-9.)

Key words: Oxytocin, vasopressin, anencephalic infants

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The fetal brain is hypothesized to influence labor, fetal homeostasis, adaptation of the fetus to stress, and preparation for extraterine functions by the neuro-peptides oxytocin and vasopressin which circulate in blood and amniotic fluid of normal human fetuses.
Table I. Amniotic oxytocin levels in normal human pregnancies and in some anencephalic infants with and without hydramnios

<table>
<thead>
<tr>
<th>Infants</th>
<th>n</th>
<th>Gestation length (range, wk)</th>
<th>Amniotic oxytocin (pg/ml)</th>
<th>Samples below detection level (%)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anencephalic, with hydramnios</td>
<td>7</td>
<td>31-40</td>
<td>&lt;7 ± 4*</td>
<td>&lt;0.25-33</td>
<td>71</td>
</tr>
<tr>
<td>Anencephalic, without hydramnios</td>
<td>10</td>
<td>22-44</td>
<td>22 ± 8</td>
<td>&lt;3-71</td>
<td>30</td>
</tr>
<tr>
<td>Control†</td>
<td>38</td>
<td>36-42½</td>
<td>21 ± 3</td>
<td>&lt;2.88</td>
<td>3</td>
</tr>
</tbody>
</table>

n = Number of cases determined; < = below detection level.  
*For calculations the detection levels were used if the values were below this level. Differences were calculated with the Mann-Whitney U test (two-tailed, corrected for ties).  
†Control subjects comprise a group of patients in normal labor.

Table II. Oxytocin and vasopressin levels* in human umbilical cord blood in normal human pregnancies after spontaneous labor and in some anencephalic infants

<table>
<thead>
<tr>
<th></th>
<th>Oxytocin</th>
<th>Arginine vasopressin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Umbilical artery</td>
<td>Umbilical vein</td>
</tr>
<tr>
<td>Spontaneous labor†</td>
<td>40 (65 ± 18)</td>
<td>17 (20 ± 5)</td>
</tr>
<tr>
<td>Anencephalic infant 1</td>
<td>14.7</td>
<td>&lt;10.9</td>
</tr>
<tr>
<td>Anencephalic infant 2</td>
<td>&lt;27.3</td>
<td>30.1</td>
</tr>
<tr>
<td>Anencephalic infant 3</td>
<td>&lt;15.0</td>
<td>&lt;13.6</td>
</tr>
</tbody>
</table>

< = Below detection level of the assay.  
*Values are presented in median (mean ± SEM) levels in picograms per milliliter.  
†Samples were obtained from 13 patients after normal labor.

During labor increased levels of oxytocin (umbilical circulation and amniotic fluid) and arginine vasopressin (umbilical circulation) have been reported. Anencephalic infants have no neurohypophysis that contains these peptides and have often been studied to obtain clinical support for experimental data concerning the effect of a lack of fetal oxytocin and arginine vasopressin. However, recent observations raised serious doubts as to the absence of fetal neuropeptides in anencephalic infants. We have reported on the presence of normal concentrations of oxytocin in freshly obtained amniotic fluid of eight anencephalic infants without any detectable arginine vasopressin. In this article we offer an extension of our earlier data. In addition, for the first time we make mention of detectable amounts of oxytocin and arginine vasopressin in the umbilical artery and vein of three live-born anencephalic infants, as opposed to the few anencephalic infants reported on in the literature.

The material comprises a total of 17 anencephalic infants, from whom we were able to collect amniotic fluid that had not been contaminated with blood or meconium. The material was subdivided into two groups on the basis of the presence or absence of clinically diagnosed hydramnios. The results are summarized in Table I. In the absence of hydramnios, amniotic oxytocin concentrations were similar to those found in uncomplicated pregnancies. In anencephalic infants with hydramnios, oxytocin often fell below the detection level. On no occasion was arginine vasopressin found in amniotic fluid of anencephalic infants. However, this was also the case in 75% of our samples obtained in normal pregnancies (data not shown).

In three live-born anencephalic infants after intravenous administration of sulprostone to induce labor at 33 to 35 weeks of gestation, detectable amounts of oxytocin and arginine vasopressin were demonstrated in the separate umbilical vessels. These levels were usually lower than those in the circulation of children born after normal vaginal deliveries (Table II). Oxytocin could also be demonstrated in a fourth anencephalic infant, from whom we could only obtain umbilical vein blood (11.5 pg/ml). Earlier we had reported the presence of oxytocin in mixed cord blood of three anencephalic infants. The present data show that neurohypophyseal peptides circulate in human anencephalic infants. Inevitably they must originate from alternative fetal sources outside the fetal brain, since a passage of oxytocin and arginine vasopressin from the mother to the fetus is unlikely. Because oxytocin was localized in the fetal
zone of the adrenal cortex and arginine vasopressin in the definitive zone of the adrenal cortex in both normal control subjects and anencephalic neonates,² this organ is a putative source for these circulating fetal neuropeptides. For studies concerning, for example, passage of arginine vasopressin or oxytocin and their possible role in labor and fetal homeostasis, the anencephalic infant can no longer be considered to be a child with no circulating arginine vasopressin or oxytocin.

REFERENCES

Fetal pulmonary hypoplasia with hydrothorax

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Described is a case of fetal congenital malformation with hydrothorax, for which left pulmonary hypoplasia was initially presumed by ultrasonography and for which hypoplasia was diagnosed postnatally only in the left lower pulmonary lobe by a pulmonary technetium Tc-99m scintigram. The pathogenesis of this disease is discussed. (Am J Obstet Gynecol 1987;157:119-20.)

Key words: Fetal pulmonary hypoplasia, fetal hydrothorax, ultrasound, pathogenesis

Fetal pulmonary hypoplasia, especially lobar hypoplasia, with hydrothorax is an extremely rare intrauterine complication.¹ We report here an interesting case that we experienced recently.

Case report

A 29-year-old woman, gravida 2, para 0, without any recorded medical history presented with a complaint of abdominal fullness at 28 weeks' gestation. Hydramnios and threatened premature delivery were diagnosed, and she was admitted to the hospital.

Ultrasound examination at the twenty-eighth week demonstrated moderate hydramnios, with the lungs and the heart of the fetus compressed by a large amount of fetal intrathoracic fluid (Fig. 1). The fluid was retained only in the left side of the thorax. Although the fetal heart was shifted to the right side, cardiac activity was normal and the four chambers were clearly recognized. The biparietal diameter was 7.6 cm, large but normal for the fetal age. The other organs were normal.

Fig. 1. Oblique scan through fetal thorax showing the left lung (LL), the right lung (RL), and the heart (H) compressed by a large amount of intrathoracic fluid (F).

Amniocentesis and fetal thoracentesis were performed under ultrasound guidance at the thirtieth week. Fetal monitoring (nonstress test and cardiotocogram) before and after the puncture showed no abnormal patterns. The amounts of aspirated amniotic and fetal intrathoracic fluid were 200 and 25 ml, respectively; the former was clear and watery and the latter was clear yellow. The aspirated fluids were cytologically negative for malignant cells and also for bacterial growth.