Polyhydramnios: Etiology, Diagnosis, and Treatment
John D. Yeast

*NeoReviews* 2006;7:e300-e304
DOI: 10.1542/neo.7-6-e300

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Polyhydramnios: Etiology, Diagnosis, and Treatment

John D. Yeast, MD*

Objectives  After completing this article, readers should be able to:
1. Understand the various causes of polyhydramnios.
2. Describe the pathways of amniotic fluid production and removal.
3. Appreciate the reason for preterm delivery following the diagnosis of polyhydramnios.
4. Recognize the treatment methods for polyhydramnios.
5. Explain the importance of sudden changes in fundal size during pregnancy.

Introduction
Polyhydramnios, sometimes referred to as hydramnios, is a relatively uncommon complication affecting pregnancy that refers to the presence of an excessive amount of amniotic fluid relative to gestational age. Onset may be gradual or sudden, based on the cause. Gradual onset may be largely asymptomatic. In this situation, the diagnosis is suspected when fundal height exceeds that expected for gestational age. In contrast, sudden onset of polyhydramnios often is symptomatic, characterized by contractions and significant abdominal discomfort. Polyhydramnios has potential serious consequences, primarily related to the cause, but also due to the increased risk of adverse pregnancy outcome, such as preterm labor (PTL) or preterm premature rupture of the membranes (PPROM).

Incidence and Frequency
The exact incidence of polyhydramnios is unknown because mild, asymptomatic cases may be discovered only at the time of delivery and are underreported. Several series have suggested that the incidence may range up to 1.6% in a low-risk population. Most cases are mild and often not associated with any significant sequelae. Approximately 35% of cases could be classified as moderate or severe, requiring further diagnostic or therapeutic measures.

Prior to the routine use of diagnostic ultrasonography, the incidence of polyhydramnios seemed much lower. Today, though, ultrasonography studies may reveal an abnormal increase in amniotic fluid volume earlier in pregnancy, resulting in more cases being reported. Intuitively, it seems that earlier diagnosis via ultrasonography could improve outcome, but that has not yet been proven conclusively.

Diagnosis
The clinical suspicion of abnormally large fundal size remains critical to the diagnosis of polyhydramnios. Serial measurements of uterine height at the time of prenatal visits can stimulate the clinician to consider possible causes of abnormal fundal size. Subsequently, obstetric ultrasonography can confirm the presence of abnormally increased amniotic fluid volume and allow a thorough study of the fetus for possible anatomic causes.

Over the past 25 years, a number of investigators have created models to standardize the measurement of amniotic fluid volume. In some instances, they have compared and contrasted formulas for measuring amniotic fluid volume with precise, objective invasive methods, such as dye dilution studies. Others have compared subjective assessment of amniotic fluid volume with various formula models. Subjective measurements, however, do not allow comparisons of values between different observers.

An experienced sonographer can assess the amniotic fluid volume subjectively and have
a high index of suspicion that the amount is increased. In addition, a sonographer can use one of the objective methods to diagnose polyhydramnios. However, when intraobserver results are compared, there is a poor correlation unless the fluid volume is very abnormal.

The amniotic fluid index (AFI) has been the most studied objective measure of amniotic fluid volume. The sonographer divides the uterus into four quadrants and measures the greatest vertical depth of fluid without fetal parts present in each quadrant. The sum of these measurements is the AFI. This method is not used prior to 20 weeks’ gestation. The upper limit of normal for the AFI is 20 cm, with values of 20 to 24 cm considered borderline.

Normal AFI values generally are well supported in most studies. However, diagnosing polyhydramnios by AFI compared with dye studies showed a sensitivity of only 30% and a positive predictive value of 57%. As expected, specificity was good at 98%. Other methods of objectively measuring amniotic fluid volume, such as measuring the greatest two diameters of the largest pocket of amniotic fluid noted (>50 cm being the criterion for polyhydramnios) or measuring the single deepest pocket of fluid noted (>8 cm indicating polyhydramnios) also have poor sensitivity, at 38% and 29%, respectively.

Despite the poor correlation of objective measurements of fluid volume with precise dye-dilution studies, a subjective suspicion of polyhydramnios should be followed by ultrasonography using one of the objective measures of amniotic fluid noted. Serial studies subsequently can be employed to evaluate for changes in fluid volume.

Causes
The production of amniotic fluid and the maintenance of normal amniotic fluid volume are critical to the development of a normal fetus. (See the article by Gilbert in this issue of NeoReviews.) Production of amniotic fluid varies by gestational age. Early in the pregnancy, fluid primarily results from transmembranous secretion via the amnion and the body surface of the embryo. By the early second trimester, the fetus begins to produce urine, and fluid also is secreted from the fetal lungs, egressing into the amniotic cavity. By 20 to 22 weeks’ gestation, the fetal skin surfaces keratinize and no longer are a source of amniotic fluid. Thus, most of the fluid in the latter half of gestation comes from the fetal kidneys and, to a lesser extent, fetal lungs.

Fetal urine production is estimated at 2 to 5 mL/h around 22 to 24 weeks’ gestation and almost 50 mL/h at term. These estimates are based on ultrasonography-derived measures of fetal bladder volume changes in pregnancy. Lung fluid production has been estimated to be 150 to 170 mL/d, with most of the fluid excreted into the amniotic fluid compartment. Lung fluid production appears to be critical to maintaining growth and development of the alveoli. In addition, some studies suggest that the excess amount of fluid produced creates a pressure differential in the terminal bronchioles that further expands the developing alveolar buds.

Fluid leaves the amniotic cavity in the second half of pregnancy largely via two routes. First, fetal swallowing normally accounts for the vast amount of fluid leaving the amniotic compartment. It is estimated that the late-term fetus swallows about 500 to 1,000 mL/d. The fetal gastrointestinal tract absorbs fluid and solutes and returns them to the maternal compartment via the placenta. Less well understood is the egress of fluid via the transmembranous process. It is well documented in fetal lambs and other animal models that a moderate amount of fluid can be absorbed via the placental surface. Interestingly, in fetal sheep models, occlusion of fetal swallowing results in a marked increase of intramembranous fluid flow. One estimate of normal flow via the transmembranous route is approximately 300 to 400 mL/d.

Regulation of normal amniotic fluid volume, that is, the balance of fluid production and removal, is poorly understood. Animal models that artificially increase fluid production or restrict fluid removal by one mechanism show that alternate mechanisms can readily adapt and adjust their role in homeostasis of fluid volume. However, the “sensors” that control such mechanisms are unknown. Possibly, fluid volume, osmolality, and electrolyte concentrations all contribute to amniotic fluid homeostasis in the healthy fetus.

From a clinical perspective, polyhydramnios results from an overproduction of amniotic fluid or an interruption in the removal of fluid from the amniotic cavity. Causes can be subdivided further into maternal or fetal origin (Table 1). The primary maternal cause of polyhydramnios is diabetes mellitus, which may contribute up to 25% of the cases recognized. However, such cases of polyhydramnios usually are in the mild-to-moderate range and seldom are associated with significant morbidity. The precise cause in maternal diabetes seems to be the increase in fetal urine production, probably related to increased osmotic gradients in fetal blood flow from the placenta due to hyperglycemia.

Fetal causes of polyhydramnios can be divided primarily into two general categories: neurologic inhibition of the fetal swallowing mechanism and mechanical obstruc-
tion or interruption of swallowing and gastrointestinal absorption (Table 2). Neurologic inhibition of the swallowing mechanism, and perhaps inhibition of the regulatory mechanism of amniotic fluid homeostasis, can result from congenital disorders such as some aneuploidies or neuromuscular disorders or acquired conditions such as in utero viral infections that have central nervous system manifestations. More common causes are mechanical obstruction of swallowing, such as atresia of the esophagus or bowel or obstruction of the gastrointestinal tract by intra-abdominal masses. Less common causes of polyhydramnios are severe fetal anemia with associated hydrops, usually due to isoimmunization or fetal-maternal hemorrhage. In these latter diagnoses, the increased amniotic fluid volume may result from high-output cardiac perfusion of the kidneys, with resultant increased urine production, or from cardiac failure and reduced swallowing mechanisms.

Based on some series, 40% to 60% of cases of polyhydramnios do not have an apparent cause during pregnancy. So-called “idiopathic” polyhydramnios can occur with a healthy fetus, although careful neonatal evaluation still is indicated.

### Evaluation and Treatment

Evaluation of the fetus by thorough, targeted anatomic ultrasonography is the cornerstone of determining a diagnosis and cause when polyhydramnios is suspected. The probability of diagnosing a fetal structural defect as the cause of the increased amniotic fluid volume improves as the volume of fluid increases. With severe polyhydramnios, more than 30% of fetal studies result in identification of a structural defect. Experienced perinatal consultation should be obtained to aid in overall management.

Amniocentesis for fetal karyotype is strongly recommended, especially in the presence of a structural defect. In addition, maternal screening for evidence of fetal-maternal bleeding, congenital infection, and possibly hereditary anemias may be considered. Routine prenatal laboratory results should be reviewed, especially glucose screening, isoimmunization, and maternal serum screens. If no cause can be determined prior to delivery, the pediatric staff must evaluate the newborn carefully, paying close attention to neuromuscular development and function as well as ruling out structural defects not always seen during obstetric ultrasonography (certain cardiac defects, midline cleft malformations, and tracheoesophageal fistulas).

For most cases of polyhydramnios, no intervention or aggressive therapy is indicated. However, based on the degree of excess amniotic fluid, the pregnancy may be at risk for PPROM, PTL, or maternal respiratory restriction. Also, there is an increased risk of fetal death, probably related to the underlying cause of the fluid disorder.

The pregnancy in which amniotic fluid volume is increased should be followed carefully, with screening for signs and symptoms of PTL, maternal compromise, or

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**Table 1. Causes of Polyhydramnios**

<table>
<thead>
<tr>
<th>Fetal Disorders</th>
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</thead>
<tbody>
<tr>
<td>• Structural malformations</td>
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<tr>
<td>— Central nervous system defects</td>
</tr>
<tr>
<td>— Obstruction or atresia of portions of the gastrointestinal tract</td>
</tr>
<tr>
<td>— Abdominal wall defects</td>
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<tr>
<td>• Aneuploidies</td>
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<tr>
<td>• Neuromuscular disorders</td>
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<tr>
<td>Maternal Disorders</td>
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<tr>
<td>• Diabetes mellitus</td>
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<tr>
<td>Combined Disorders</td>
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<tr>
<td>• Isoimmunization</td>
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<tr>
<td>• Congenital infections</td>
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<tr>
<td>• Congenital anemias</td>
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<tr>
<td>Idiopathic</td>
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</tbody>
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**Table 2. Findings in Polyhydramnios**

<table>
<thead>
<tr>
<th>Increase in Fetal Urine Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Hydrops</td>
</tr>
<tr>
<td>• Some central nervous system lesions associated with decreased antidiuretic hormone output</td>
</tr>
<tr>
<td>• Maternal diabetes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Decreased Fetal Swallowing</th>
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</thead>
<tbody>
<tr>
<td>• Some central nervous system lesions</td>
</tr>
<tr>
<td>• Aneuploidies</td>
</tr>
<tr>
<td>• Neuromuscular disorders</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Decreased Gastrointestinal Absorption of Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Gastrointestinal obstruction or atresia</td>
</tr>
<tr>
<td>• Nongastrointestinal obstructive masses</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Fetal Structural Disorders Associated With Large-volume Transudates</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Open neural tube defects</td>
</tr>
<tr>
<td>• Abdominal wall defects</td>
</tr>
</tbody>
</table>
fetal jeopardy. Reduction in maternal activity and increased bedrest may be prudent, although neither has been shown conclusively to change outcome. In some instances, polyhydramnios resolves spontaneously.

Maternal symptoms are the most common reason for therapeutic intervention. If the patient becomes symptomatic, either with uterine irritability, respiratory compromise, or discomfort, treatment may be necessary to prolong the pregnancy. Based on gestational age, two options are available: amnioreduction or the use of prostaglandin inhibitors to attempt a medical reduction of fluid production.

Amnioreduction should be performed by personnel familiar with the procedure. Using ultrasonography guidance, a large-bore needle is placed in the amniotic cavity, and fluid is removed via a suction pump. The goal is to remove fluid slowly, reducing fluid volume to a near-normal AFI of less than 25 cm. Some patients require mild sedation, analgesics, or tocolytics for the procedure, although most tolerate the amnioreduction without incident. The amniotic fluid volume should be evaluated frequently (at least twice weekly) and the procedure repeated when symptoms recur or volumes begin to increase significantly. Some patients may require serial procedures to prolong pregnancy. Careful informed consent must be obtained because the procedure may precipitate PTL or PPROM. Also, the risk/benefit balance versus gestational age must be considered carefully.

Some data suggest that prostaglandin inhibitors, such as indomethacin or ibuprofen, may reduce fetal urine production. Although no controlled, randomized studies have compared methods of therapy, medical therapy may be considered, especially when polyhydramnios develops at early gestational ages. Due to the concern over fetal ductus arteriosus closure with prostaglandin treatment, therapy should be conducted only in perinatal centers that have the ability to follow fetal ductal blood flow. In addition, it would seem prudent not to use such therapy beyond 32 weeks gestational age.

Treatment of significant polyhydramnios prior to term may include corticosteroid therapy to enhance fetal lung maturity. Antepartum surveillance with ultrasonography and cardiotocography should be employed prior to delivery. The goal of all therapy is to assure fetal and maternal well-being and allow the fetus to reach maturity. In some cases, however, delivery prior to fetal maturity may result or be indicated. The method of delivery depends on fetal well-being and standard obstetric indications. Increased amniotic fluid volume does increase the chance of fetal malposition or funic (cord) presentation. Sudden uterine decompression with PPROM or amniotomy can result in placental abruption. The fetus should be monitored continuously at all times during labor and delivery.

Suggested Reading
NeoReviews Quiz

4. Polyhydramnios is a complication of pregnancy whose estimated incidence is up to 1.6% of pregnancies in a low-risk population. Of the following, the most accurate statement regarding polyhydramnios is that:

A. Approximately 10% of cases have no identifiable cause.
B. Cases that have a gradual onset are generally asymptomatic.
C. Early diagnosis has been proven to improve perinatal outcome.
D. Measurement of the amniotic fluid index is best performed before 20 weeks’ gestation.
E. Most cases are moderate-to-severe and associated with sequelae.

5. Polyhydramnios results from an overproduction of amniotic fluid or an interruption in its removal from the amniotic cavity. The causes of polyhydramnios can be of maternal or fetal origin. Of the following, the most common fetal cause of polyhydramnios is:

A. Decreased absorption of amniotic fluid due to gastrointestinal atresia.
B. Decreased fetal swallowing from neuromuscular disorder.
C. Excessive transudation of fluid from an abdominal wall defect.
D. Increased fetal lung fluid secretion associated with gestational diabetes.
E. Increased fetal urine output from hydrops associated with anemia.

6. The treatment of polyhydramnios during pregnancy depends on several factors, including the degree of excess amniotic fluid, maternal symptoms, and gestational age of the fetus. Of the following, the most common obstetric intervention for polyhydramnios is:

A. Amnioreduction.
B. Expectant observation.
C. Reduction in maternal activity.
D. Treatment with diuretics.
E. Treatment with prostaglandin inhibitors.
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