# Polyhydramnios

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**INTRODUCTION** — Polyhydramnios (also known as hydramnios) refers to an excessive volume of amniotic fluid. It has been associated with an increased risk of various adverse pregnancy outcomes, including preterm birth, placental abruption, and fetal anomalies [1-3]. Polyhydramnios should be suspected clinically when uterine size is large for gestational age. The diagnosis is made prenatally by ultrasound examination using a noninvasive qualitative or quantitative approach. (See "Assessment of amniotic fluid volume".)

**INCIDENCE** — The incidence of polyhydramnios in a general obstetric population generally ranges from 1 to 2 percent [4-8]. Reported rates are influenced by variations in diagnostic criteria, the population studied (low or high risk), the subjective volume of fluid where polyhydramnios is diagnosed (eg, mild, moderate, or severe), and the gestational age (preterm, term, or postterm) at examination. In one series of 93,332 singleton pregnancies delivering at a single hospital from 1991 to 1997, polyhydramnios was diagnosed during antepartum sonography in 708 pregnancies (0.7 percent of deliveries); mild, moderate, and severe disease occurred in 66, 22, and 12 percent of cases, respectively [5].

**PATHOPHYSIOLOGY** — The volume of amniotic fluid reflects the balance between fluid production and movement of fluid out of the amniotic sac; the regulation of this process is incompletely understood (see "Physiology of amniotic fluid volume regulation"). In late gestation, the primary sources of amniotic fluid production are fetal urination and secretion of lung fluid; oral and nasal secretions make minimal contributions. The main routes of amniotic fluid removal are fetal swallowing and absorption via the intramembranous pathway. Even a relatively minor increase in daily fetal urine production or decrease in fetal swallowing can result in a marked increase in amniotic fluid volume (AFV) [9-11].

**ETIOLOGY** — The most common cause of severe polyhydramnios are fetal anomalies (often associated with an underlying genetic abnormality or syndrome), while maternal diabetes, multiple gestation, and idiopathic factors are more often associated with milder cases. In one series of 272 singleton pregnancies with polyhydramnios, approximately one-third were associated with a congenital anomaly and one-quarter were associated with maternal diabetes; the remaining 40 percent were considered idiopathic [12]. After birth, an abnormality is diagnosed in up to 25 percent of cases considered idiopathic prenatally [12-15]. Fetal infection, Bartter syndrome, anemia, and neuromuscular disorders account for some of these cases and should be considered in the differential diagnosis if a structural abnormality and maternal diabetes are excluded, although Bartter syndrome and neuromuscular diseases are quite rare and infection (TORCH, parvovirus) is rarely associated with isolated polyhydramnios. In a retrospective observational study of 294 singleton pregnancies with polyhydramnios and serum screening for TORCH and parvovirus B19 infections, only two patients tested positive for parvovirus infection and only one for toxoplasmosis infection [16]. Among these patients, 72 percent were diagnosed with idiopathic polyhydramnios, 13 percent with diabetes, 5 percent with obstructive gastrointestinal lesions, 0.3 percent with Rhesus isoimmunization, and 1 percent with chromosomal abnormalities or genetic syndromes.

Polyhydramnios has been associated with fetal anomalies in most organ systems. The most common structural anomalies associated with polyhydramnios are those that interfere with fetal swallowing and/or absorption of fluid [17,18]. Decreased swallowing may be due to a primary gastrointestinal obstruction (eg, duodenal, esophageal, or intestinal atresia), neuromuscular disorders (eg, anencephaly), or to secondary obstruction of the gastrointestinal tract (eg, massive unilateral dysplastic kidneys).

The combination of intrauterine growth restriction and polyhydramnios is suggestive of trisomy 18; other sonographic markers of trisomy 18 are typically present (see "Sonographic findings associated with fetal aneuploidy", section on 'Trisomy 18'). Excess amniotic fluid in this syndrome may be related to difficulty swallowing or to intestinal abnormalities. Other aneuploidies have also been associated with polyhydramnios, most commonly trisomy 21. (See "Sonographic findings associated with fetal aneuploidy", section on 'Trisomy 21 (Down syndrome)'.)

Increased urine production may occur in high fetal cardiac output states (eg, fetal anemia due to alloimmunization, parvovirus infection, fetomaternal hemorrhage, alpha-thalassemia, glucose-6-phosphatase deficiency) or, rarely, from entities such as fetal Bartter syndrome [19]. The most common antenatal type of Bartter syndrome is a rare autosomal recessive tubular disorder associated with intrauterine presentation of renal tubular hypokalemic alkalosis. As a result, the fetus develops polyuria and subsequent polyhydramnios between 24 and 30 weeks of gestation. Infants exhibit postnatal polyuria and persistent renal salt wasting, requiring life-long treatment. A severe but transient form of antenatal Bartter's syndrome has been attributed

to mutations in *MAGED2,* which maps to the X-chromosome and appears to be essential for fetal renal salt reabsorption and maintenance of normal amniotic fluid homeostasis [20]. This X-linked disorder has very early onset of severe polyhydramnios (median 19 to 20 weeks of gestation), often resulting in preterm birth (median gestational age 22 to 34 weeks), but signs and symptoms of renal dysfunction resolve spontaneously postnatally. (See "Bartter and Gitelman syndromes".)

In monochorionic multiple gestation, polyhydramnios/oligohydramnios sequence is diagnostic of twin-twin transfusion syndrome (TTTS). (See "Twin-twin transfusion syndrome and twin anemia polycythemia sequence: Pathogenesis and diagnosis".)

The mechanism for polyhydramnios in pregnancies complicated by maternal diabetes is unclear. Fetal hyperglycemia leading to polyuria is one likely etiology, and is supported by the observation that polyhydramnios is often associated with high maternal glycated hemoglobin (A1C) levels and fetal macrosomia [21,22]. Decreased fetal swallowing or an imbalance in water movement between the maternal and fetal compartments in diabetic pregnancies are other possible mechanisms.

**CLINICAL MANIFESTATIONS** — On physical examination, polyhydramnios should be suspected by uterine size large for gestational age. It also may be detected as an incidental finding on a prenatal ultrasound examination. The increase in amniotic fluid volume (AFV) is usually asymptomatic; however, the gravida may experience persistent shortness of breath, uterine irritability and contractions, and abdominal discomfort when uterine distention is severe.

**CLINICAL SIGNIFICANCE** — Many idiopathic cases resolve spontaneously, especially if mild [1,23]. However, polyhydramnios has been associated with an increased risk of several adverse outcomes in addition to the poor outcomes related to the associated morphologic abnormalities [24-26]:

- Maternal respiratory compromise
- Preterm labor, premature rupture of membranes (PROM), preterm delivery
- Fetal malposition
- Macrosomia (potentially leading to shoulder dystocia)
- Umbilical cord prolapse
- Abruption upon rupture of membranes
- Longer second stage of labor
- Postpartum uterine atony

These complications increase the risk of cesarean delivery and neonatal intensive care admission.

Maternal respiratory compromise, fetal malposition, cord prolapse, and postpartum uterine atony are related to uterine over-distention from excessive amniotic fluid volume (AFV). Although preterm labor and PROM may lead to spontaneous preterm delivery, preterm birth in affected pregnancies is also caused by iatrogenic intervention for management of pregnancy complications.

The risk of fetal death in nonanomalous pregnancies affected by polyhydramnios is increased compared with normal amniotic fluid pregnancies at each gestational age, with the greatest increase at term (adjusted odds ratio 5.5, 95% CI 4.1-7.6) [27]. Overall perinatal mortality in polyhydramnios is increased two- to five-fold compared with pregnancies without polyhydramnios [28]. The risk of perinatal death appears to be increased even after adjusting for confounders, such as congenital anomalies and maternal diabetes [7,27-31]. The risk increases with increasing severity of polyhydramnios in some, but not all, studies. The combination of a small for gestational age fetus and polyhydramnios has a particularly poor prognosis (including perinatal death) due to the high prevalence of fetal chromosomal and structural abnormalities in this setting [32,33].

Neonatal outcomes associated with idiopathic polyhydramnios include an increased incidence of low five-minute Apgar score, transient tachypnea of the newborn, newborn resuscitation, admission to neonatal intensive care unit, ventilator requirement, jaundice, hypoglycemia, and structural anomalies.

Polyhydramnios may also be associated with long-term sequelae. A retrospective cohort study compared 134 children ages four to nine years who had had polyhydramnios with 268 controls who had had a normal amniotic fluid index; both groups had an otherwise normal detailed ultrasound examination [34]. Compared with the normal AFI group, the polyhydramnios group had an increased rate of malformations, genetic syndromes, neurologic disorders, and developmental delay diagnosed in childhood.

**DIAGNOSIS** — The diagnosis of polyhydramnios is based upon sonographic visualization of increased amniotic fluid volume (AFV). This may be qualitative or quantitative, but generally has a strong subjective component. A 2014 consensus panel at a fetal imaging workshop suggested the following thresholds for polyhydramnios [35]:

- Single deepest pocket ≥8 cm
- Amniotic fluid index (AFI) ≥24 cm

A two diameter pocket >50 $cm^2$ and AFI >25 cm, >24 cm, >20 cm, or >18 cm are other suggested thresholds for polyhydramnios. (See "Assessment of amniotic fluid volume".)

We feel a qualitative impression of mild, moderate, or severe polyhydramnios should be followed by a quantitative measurement, such as the AFI or single deepest pocket (table 1). Quantitative approaches are standardized and provide a measurement that can be compared over serial examinations, even if sensitivity and positive predictive value (PPV) are low.

Invasive methods for measuring AFV are not used clinically. If an invasive method such as dye dilution is performed, polyhydramnios is defined as AFV more than two standard deviations above normal for the gestational age. At 34 weeks of gestation, this would be an AFV >2200 mL [36].

There are few large studies in women with polyhydramnios comparing the various methods of amniotic fluid assessment against a gold standard (ie, dye dilution) [37,38]. One such study compared AFI 25 cm to a dye standard for diagnosis of polyhydramnios and reported sensitivity, specificity, and PPVs and negative predictive values (NPVs) of 30, 98, 57, and 93 percent, respectively [39]. A two-diameter amniotic pocket volume greater than 50 $cm^2$ performed slightly better, with sensitivity, specificity, PPV and NPV of 38, 97, 62, and 94 percent, respectively. Another study by the same group found the sensitivity, specificity, PPV, and NPV of a single deepest pocket greater than 8 cm was 29, 94, 45, and 89 percent, respectively [37]. Thus, all available noninvasive diagnostic tests for polyhydramnios perform relatively poorly.

**POST-DIAGNOSTIC EVALUATION** — A comprehensive sonographic evaluation should be performed to look for fetal anomalies or fetal hydrops. The likelihood of identifying the etiology of polyhydramnios prenatally correlates with severity of fluid accumulation. In one series, an etiology was determined in only 17 percent of pregnancies with mild polyhydramnios, but in 91 percent of those with moderate or severe amniotic fluid accumulation [4]. In another series, the frequency of an anomalous infant with mild, moderate, and severe polyhydramnios was 8, 12, and 31 percent, respectively [5]. Overall, 80 percent of anomalous infants were detected prenatally. Anomalies most often missed included tracheoesophageal fistula, cardiac septal defects, and cleft palate. Although not readily available, serial bladder volume measurements have been used to assess fetal urine production. Increased fetal urine production decreases the likelihood of an undetected fetal anomaly involving decreased swallowing [40].

Fetal genetic studies should be offered when congenital anomalies are detected and this information will affect management, given the high risk of abnormality in this setting. In one large series, the prevalence of aneuploidy in anomalous fetuses with polyhydramnios was 10 percent (95% CI 5-19) [5]. Whether genetic studies should be offered in the absence of anomalies or growth restriction is controversial, as the likelihood of aneuploidy is much lower in the setting of a normal sonogram: 1 percent (95% CI 0.4-2) in the previously cited series [5]. However, studies using microarray for genetic analysis have reported almost 10 percent of fetuses (46/464) with amniotic fluid index (AFI) >25 cm had genetic abnormalities following a normal detailed fetal ultrasound examination [41]. The abnormalities included trisomy 21 (10 cases), trisomy 18 (10 cases), tetrasomy 12 p (2 cases), sex chromosome aneuploidies (8 cases), microdeletions/microduplications (15 cases), and uniparental disomy 14 (1 case).

We recommend offering chromosomal analysis in all cases of severe polyhydramnios, including those that appear isolated, because of the increased risk of aneuploidy noted in most series, the possibility that anomalies were not detected by sonographic examination, and the low risk of the procedure. In cases of severe polyhydramnios with a normal karyotype and ultrasound findings of anomalies, microarray or gene sequencing may detect a genetic abnormality of clinical significance [42,43]. For example, 22q11.2 microdeletion syndrome is associated with polyhydramnios and hypoplastic thymus as the only sonographic findings [42]. Noonan syndrome is often associated with polyhydramnios, as well as other abnormalities, and can be diagnosed by gene sequencing. (See "Use of chromosomal microarray in obstetrics", section on 'Prenatal ultrasound showing fetal structural abnormalities' and "Causes of short stature", section on 'Noonan syndrome'.)

In pregnancies with a male fetus and unexplained severe polyhydramnios in the second trimester, especially with previous history of severe polyhydramnios, genetic studies for identification of mutations in *MAGED2* should be offered [20]. Accurate diagnosis can avoid potentially harmful treatment of preterm infants.

A monochorionic multiple gestation with polyhydramnios/oligohydramnios sequence is suggestive of twin-twin transfusion syndrome (TTTS). Clinical issues related to TTTS are reviewed separately. (See "Twin-twin transfusion syndrome and twin anemia polycythemia sequence: Pathogenesis and diagnosis" and "Twin-twin transfusion syndrome: Management and outcome".)

Screening for diabetes is appropriate if not previously performed. (See "Diabetes mellitus in pregnancy: Screening and diagnosis".)

If fetal hydrops is identified, the next step is evaluation for an immune or nonimmune etiology (see "Nonimmune hydrops fetalis", section on 'Postdiagnostic evaluation'). In cases of fetal hydrops associated with polyhydramnios, evaluation of potential fetal anemia includes assessment of peak systolic velocity of the middle cerebral artery with a value >1.5 multiples of the median, suggesting moderate or severe anemia, irrespective of the cause (see "Management of pregnancy complicated by Rhesus (D) alloimmunization", section on 'Assess for severe anemia in fetuses at risk'). Work-up for fetal anemia also includes testing for fetomaternal hemorrhage, acute parvovirus infection, and hemoglobinopathy. (See "Massive fetomaternal hemorrhage" and "Parvovirus B19 infection during pregnancy" and "Prenatal screening and testing for hemoglobinopathy".).

Congenital infection may be associated with maternal signs or symptoms of infection and/or fetal abnormalities (eg, hydrops, growth restriction, hepatosplenomegaly, cerebral ventriculomegaly, intracranial and intraabdominal calcifications, hyperechogenic fetal bowel, ascites). In the absence of maternal signs and symptoms or fetal findings (other than polyhydramnios), congenital infection (rubella, cytomegalovirus, toxoplasmosis, syphilis) is an unlikely cause of isolated polyhydramnios [16,44].

A detailed medical history should be obtained to evaluate for heritable diseases associated

with polyhydramnios. These conditions often present with multiple congenital contractures (arthrogryposis), which should be identified by ultrasound. Most cases are recessive inheritance or new mutations. If suspected, the patient should be referred to a genetics specialist for three generation pedigree and parental examination.

In one study of 464 cases of polyhydramnios (AFI ≥25 cm) that appeared to be isolated, amniotic fluid biochemical analyses (total protein, alpha-fetoprotein [AFP], digestive enzyme gamma-glutamyl transpeptidase [GGTP]) was helpful for diagnosis of Bartter syndrome (28 cases; sensitivity 85.7 percent, specificity 84.2 percent) and esophageal atresia (15 cases; sensitivity 66.6 percent, specificity 100 percent) [41]. The amniotic fluid Bartter index (AF-BI) was calculated by multiplying total protein (expressed in g/L) by AFP (expressed in multiples of the median [MoM]); a level ≤1.2 was defined as abnormal. The amniotic fluid esophageal atresia index (AF-EAI) was calculated by multiplying AFP by GGTP (expressed in MoM); a level ≥3 was defined as abnormal. A major limitation of this study was that the biochemical measurements and AFI for each affected fetus were not provided. We believe that more data on the clinical significance of amniotic fluid biochemical analyses are needed before these tests are utilized in the routine evaluation of polyhydramnios. Others have reported analysis of the amniotic fluid chloride concentration can be helpful for prenatal diagnosis of Bartter syndrome [45,46]. Amniotic fluid aldosterone concentration is not helpful [47]. (See "Bartter and Gitelman syndromes", section on 'Prenatal diagnosis'.)

**MANAGEMENT OF POLYHYDRAMNIOS** — Management of idiopathic polyhydramnios depends on the gestational age, severity, presence of symptoms, and cause.

**Antepartum fetal monitoring** — No randomized trials have evaluated whether pregnancies complicated by idiopathic polyhydramnios benefit from any method of antenatal surveillance. Given the increase in risk of adverse pregnancy outcomes and the two- to five-fold increase in risk of perinatal mortality (including fetal mortality [27]) in these cases, we perform antenatal fetal monitoring.

In mild to moderate polyhydramnios, we perform a nonstress test (NST) and biophysical profile (BPP) upon diagnosis and then every 1 to 2 weeks until 37 weeks, and then weekly from 37 weeks to delivery. The BPP includes assessments of amniotic fluid index (AFI) so that the trajectory of amniotic volume change can be tracked. In severe polyhydramnios, we perform a NST and BPP every week until delivery. In interpreting the BPP score, clinicians should be cautious about conclusions of fetal well-being with a borderline score (6/8) since the two points for amniotic fluid volume (AFV) in these cases is not reassuring.

**Indications for intervention** — No randomized trials have directly compared expectant management and amnioreduction for management of polyhydramnios. In women with preterm singleton pregnancies, we offer amnioreduction only if polyhydramnios is both severe and symptomatic (associated with significant maternal discomfort or preterm labor). The goal of amnioreduction is to relieve maternal discomfort and prolong pregnancy [48,49]. Two methods are available: amnioreduction (ie, removal of a large volume of amniotic fluid via amniocentesis, also called ‘decompression amniocentesis’) and maternal administration of a prostaglandin synthetase inhibitor [50-52].

**Gestational age based approach**

**Pregnancies under 32 weeks** — For severe symptomatic polyhydramnios at less than 32 weeks of gestation, we suggest amnioreduction (to normalize fluid volume) followed by treatment with indomethacin to maintain normal AFV without exposing the fetus to the risks of serial invasive procedures (see 'Amnioreduction' below and 'Indomethacin' below). A course of prophylactic maternal steroids is appropriate prior to the amnioreduction in those pregnancies because of the increased risk of preterm birth.

During indomethacin therapy, we monitor AFV at least weekly while titrating the indomethacin dose to AFV changes. If there is no reduction in AFV, then the dose is gradually increased to a maximum of 2 to 3 mg/kg per day [50]. We taper the drug when a reduction in AFV is observed. It may be possible to discontinue indomethacin if severe polyhydramnios does not recur as the indomethacin is tapered [53]. Indomethacin is discontinued no later than 32 to 34 weeks because of the risk of premature fetal ductal constriction.

During indomethacin therapy, we obtain serial fetal echocardiographic evaluations if the duration of therapy exceeds 48 hours and the pregnancy is over 24 weeks of gestation. Doppler velocimetry is performed at intervals of two days to one week (see 'Indomethacin' below). Following the discontinuation of indomethacin, we assess the AFV weekly, and continue NSTs/BPPs even if the AFV returns to normal, since polyhydramnios may recur.

Amniocentesis and/or indomethacin treatment can be reinitiated if severe polyhydramnios recurs.

**Pregnancies over 32 weeks** — For severe symptomatic polyhydramnios between 32 and 34 weeks of gestation, we suggest amnioreduction. After 34 weeks, we offer amniocentesis for fetal lung maturity and deliver if maturity is confirmed.

Indomethacin is rarely utilized at ≥32 weeks, and only if the potential benefits (avoidance of serial amnioreduction procedures) are likely to exceed the risks of premature ductal closure and maternal side effects. As an example, we may use indomethacin in select cases when the gestational age is between 32 and 34 weeks and severe symptomatic polyhydramnios persists despite at least one attempt at amnioreduction. In these pregnancies, serial fetal echocardiographic evaluation at two- to seven-day intervals is especially important if the duration of therapy exceeds 48 hours.

**Interventions**

**Amnioreduction** — There is no consensus about how much fluid to remove, how rapidly to remove the fluid, use of tocolytic medications, or use of antibiotics during amnioreduction. A common technique is to cleanse the site with a surgical scrub and then administer local anesthesia into the skin and subcuticular tissues [48]. Under continuous ultrasound guidance to minimize fetal contact, an 18-gauge needle is inserted with the tip directed towards the fluid. It is often useful to insert the needle in a caudal direction with the hub of the needle positioned cranially so the needle can be advanced as the uterus becomes smaller with decompression. Hard-walled arterial line tubing with a three-way stopcock is attached to the needle and connected to a vacuum suction bottle. The fluid may be suctioned directly into the container or, alternatively, removed with a 50 mL syringe under controlled suction, but this can be tedious when there is a lot of fluid. A reasonable guideline is to remove no more than 2 to 2.5 liters at one time and to remove the fluid no faster than 1000 mL over 20 minutes, although rates of 100 to 125 mL/min have been reported [49]. The procedure is terminated when the AFI is normalized (generally 15 to 20 cm) or when intraamniotic pressure is less than 20 mmHg [54]. Sometimes the procedure will need to be terminated early because of patient discomfort, a clogged needle, or membrane separation. We do not use antibiotic prophylaxis, and initiate tocolytic drugs only if increased contractions occur.

On average, two procedures are needed to reduce AFV chronically, although some patients require many more procedures [48,55]. One case report described 12 amnioreductions which removed a total of 21,600 mL of fluid; the infant was diagnosed with West syndrome at follow-up 16 months after delivery [14].

Following amnioreduction, we monitor AFV every one to three weeks, as indicated by the progression and severity of the process. Amnioreduction is repeated if severe polyhydramnios recurs and the patient again becomes symptomatic. Repeat procedures can be performed at any interval.

Complications occur in 1 to 3 percent of procedures and include preterm labor, premature rupture of membranes (PROM), abruptio placentae, intraamniotic infection, and hypoproteinemia [48,55]. In cases presenting with uterine contractions, decompression has reduced symptoms within a few hours of the procedure [56]; however, there is no evidence that it prolongs gestation in these cases.

**Prostaglandin synthetase inhibitors** — Maternal administration of prostaglandin synthetase inhibitors reduces AFV in pregnancies with normal or abnormal AFV at baseline. These drugs may stimulate fetal secretion of arginine vasopressin and facilitate vasopressin-induced renal antidiuretic responses and reduced renal blood flow, thereby reducing fetal urine flow. They also may impair production or enhance reabsorption of lung liquid [57].

**Indomethacin** — Six series have reported the use of indomethacin to treat polyhydramnios [58]. Various regimens have been used, but a common approach is an initial dose of 25 mg orally four times daily. If there is no reduction in AFV after two or three days, then the dose is gradually increased up to 2 to 3 mg/kg per day [50]. We taper the drug when there is a reduction in AFV, and discontinue it when polyhydramnios is no longer severe. A literature review found this therapy to be effective in 48 of 52 patients studied [59].

Maternal side effects, such as nausea, esophageal reflux, gastritis, and emesis, have been studied primarily in women receiving the drug for inhibition of preterm labor; the prevalence of side effects in this population is 4 percent. Platelet dysfunction may occur. Alterations in maternal cardiovascular physiology are minimal. (See "Nonselective NSAIDs: Overview of adverse effects".)

The primary fetal concern with use of indomethacin is constriction of the ductus arteriosus. If the duration of therapy exceeds 48 hours and the pregnancy is over 24 weeks of gestation, we suggest serial fetal echocardiographic evaluation with Doppler velocimetry at intervals of two days to one week. Sonographic signs of ductal narrowing include tricuspid regurgitation and right ventricular dysfunction. The risk of ductal constriction increases with advancing gestational age and is almost 50 percent at 32 weeks of gestation. Constriction generally resolves within 24 hours after discontinuing the drug.

Other possible fetal adverse effects include neonatal necrotizing enterocolitis and intraventricular hemorrhage, but these associations are more controversial. Effects of indomethacin on the fetus/neonate are discussed in more detail separately. (See "Inhibition of acute preterm labor", section on 'Fetal side effects'.)

**Sulindac** — Sulindac is a nonsteroidal antiinflammatory drug that also results in reduction of AFV, but appears to have less of a constrictive effect on the fetal ductus than indomethacin [59-62]. However, there are no clinical studies evaluating this agent for treatment of polyhydramnios. We do not use it for this indication.

**Investigational approaches** — Investigational therapeutic approaches for polyhydramnios include the use of intraamniotic pharmacologic agents to reduce fetal fluid production. In ovine pregnancy, intraamniotic administration of either arginine vasopressin (AVP) or desmopressin (DDAVP) results in rapid fetal plasma absorption and a marked decrease in fetal urine flow [63], although there is no effect on fetal swallowing [64].

Modulation of the amniotic membrane aquaporin water channels, which have been reported to be altered in the amnion and chorion of patients with unexplained polyhydramnios, may represent a potential future therapeutic opportunity to normalize AFV [65,66].

**Management of labor** — During labor, we check fetal position frequently to confirm maintenance of vertex presentation. The excess amniotic fluid allows greater fetal mobility so conversion to a breech, compound, or transverse presentation may occur.

Spontaneous rupture of membranes can cause sudden severe uterine decompression with risk of cord prolapse or abruption. Prophylactic gradual abdominal or transcervical amnioreduction with a needle may prevent these complications during labor. Controlled amniotomy is performed by using a small gauge needle to puncture the fetal membranes in one or more places in the operating room.

The fetal heart rate is monitored continuously as these pregnancies are at increased risk of abnormalities [67,68].

**Induction** — Although there is no absolute contraindication to use of oxytocin or prostaglandins in cases of polyhydramnios, these agents should be used with caution. There is a marked increase in the incidence of postpartum hemorrhage related to atony in patients with polyhydramnios; use of uterine stimulants may increase this risk and that of amniotic fluid embolism [58,69]. However, we use prostaglandins for cervical ripening and oxytocin for induction, as clinically indicated.

**Timing of delivery** — In mild to moderate polyhydramnios with normal NST and BPP, we induce labor at 39 to 40 weeks of gestation as the risk of fetal death appears to increase significantly at term [27]. In severe cases, we induce labor at 37 weeks to minimize the risk of umbilical cord prolapse and/or abruption upon rupture of membranes. In severe polyhydramnios with intolerant maternal symptoms before 37 weeks, we perform amniocentesis as early as 34 weeks and deliver if the fetal lungs are mature.

## SUMMARY AND RECOMMENDATIONS

- Polyhydramnios is typically caused by decreased fetal swallowing or increased fetal urination. The most common etiologies are: fetal malformations and/or genetic disorders, maternal diabetes mellitus, multiple gestation, and fetal anemia. Rare causes include congenital viral infection or Bartter syndrome. (See 'Etiology' above.)

- If there is a subjective impression of polyhydramnios, we suggest that an objective measure such as an amniotic fluid index (AFI) be performed. We use single deepest pocket ≥8 cm or AFI ≥24 cm. Objective indices are standardized and provide a measurement that can be followed on serial examinations, even though sensitivity and positive predictive value (PPV) are suboptimal. (See 'Diagnosis' above.)

- A comprehensive sonographic evaluation is recommended to determine whether fetal anomalies or fetal hydrops is present. Suggested laboratory evaluations depend upon sonographic findings and may include screening for gestational diabetes, testing for fetomaternal hemorrhage if fetal anemia is suspected, maternal serology to determine exposure to infectious agents (eg, syphilis, parvovirus, cytomegalovirus, toxoplasmosis, rubella), and appropriate tests for hereditary anemias (eg, alpha thalassemia) or metabolic abnormalities. (See 'Post-diagnostic evaluation' above.)

- We recommend offering karyotype analysis in cases of severe polyhydramnios or if there are associated anatomic anomalies, if knowledge of the karyotype will affect management. Amniotic fluid biochemical analyses can help in the diagnosis of esophageal atresia and Bartter syndrome. (See 'Post-diagnostic evaluation' above.)

- The outcome of pregnancies complicated by polyhydramnios varies according to the severity and underlying etiology of the excessive fluid accumulation. Possible complications include maternal respiratory compromise, preterm labor, premature rupture of membranes (PROM), fetal malposition, and umbilical cord prolapse and/or postpartum uterine atony. (See 'Clinical significance' above.)

- We suggest treatment for polyhydramnios in singleton pregnancy only if there is preterm labor or significant maternal discomfort (**Grade 2C**). (See 'Management of polyhydramnios' above.)

  The therapeutic option is determined by the gestational age, degree of discomfort, and sensitivity or contraindication to prostaglandin synthetase inhibitors.

- For severe symptomatic polyhydramnios at less than 32 weeks of gestation, we suggest amnioreduction (to normalize fluid volume) and treatment with indomethacin to maintain normal amniotic fluid volume (AFV) (**Grade 2C**). A course of corticosteroids is given prior to amnioreduction because of the increased risk of preterm birth. (See 'Pregnancies under 32 weeks' above.)

  During indomethacin therapy, we monitor AFV at least weekly and titrate the indomethacin dose to AFV changes. We also monitor ductal Doppler flow at two- to seven-day intervals, with increased surveillance after 28 weeks of gestation, to look for early evidence of constriction. It may be possible to discontinue indomethacin treatment if polyhydramnios does not recur as the indomethacin is tapered. Indomethacin is discontinued no later than 32 weeks because of the risk of premature ductal constriction. (See 'Prostaglandin synthetase inhibitors' above.)

- For severe symptomatic polyhydramnios between 32 and 34 weeks of gestation, we suggest amnioreduction (**Grade 2C**). After 34 weeks, we offer amniocentesis for fetal lung maturity and deliver if maturity is confirmed. We suggest not administering indomethacin because of the high risk of premature closure of the ductus arteriosus at this gestational age (**Grade 2C**). However, in select cases, this risk may be outweighed by the risk associated with multiple amniocenteses or prematurity. In these cases, ductal Doppler flow should be followed at least weekly to detect early evidence of constriction. (See 'Pregnancies over 32 weeks' above.)



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Topic 6781 Version 28.0

## GRAPHICS

**Criteria for mild moderate and severe polyhydramnios**

| | Mild | Moderate | Severe |
|---|---|---|---|
| **Single deepest pocket** | 8 to 11.9 cm | 12 to 15.9 cm | ≥16 cm |
| **Amniotic fluid index** | 25 to 30 cm | 30.1 to 35 cm | >35 cm |

Graphic 89351 Version 1.0

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**Ron Beloosesky, MD** Nothing to disclose **Michael G Ross, MD, MPH** Nothing to disclose **Charles J Lockwood, MD, MHCM** Consultant/Advisory Boards: Celula [Aneuploidy screening (No current products or drugs in the US)]. **Deborah Levine, MD** Nothing to disclose **Vanessa A Barss, MD, FACOG** Nothing to disclose

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