Oligohydramnios

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INTRODUCTION — Oligohydramnios refers to amniotic fluid volume that is less than expected for gestational age. It is typically diagnosed by ultrasound examination and may be described qualitatively (eg, normal, reduced) or quantitatively (eg, amniotic fluid index [AFI] ≤5). Methods of amniotic fluid volume assessment are reviewed separately. (See "Assessment of amniotic fluid volume").

An adequate volume of amniotic fluid is critical to allow normal fetal movement and growth, and to cushion the fetus and umbilical cord. Oligohydramnios may inhibit these processes and can lead to fetal deformation, umbilical cord compression, and death.

INCIDENCE — Reported rates of oligohydramnios are influenced by variations in diagnostic criteria, the population studied (low or high risk, screening or indicated ultrasound examination), the threshold used, and the gestational age at the time of the ultrasound examination (preterm, term, or postterm).

A study of 3050 uncomplicated pregnancies with singleton non-anomalous fetuses between 40 and 41.6 weeks of gestation noted oligohydramnios (defined as AFI ≤5 cm) in 11 percent [1]. The incidence is high in laboring women, largely due to rupture of fetal membranes during or just before labor [2-4].

PATHOPHYSIOLOGY — The volume of amniotic fluid is ultimately determined by the volume of fluid flowing into and out of the amniotic sac. Fetal urination, lung fluid, and swallowing all make important contributions to fluid movement in late gestation, with minimal contributions from other sources. Fetal disorders that affect any of these processes will affect the amniotic fluid volume. As an example, growth restricted fetuses may redistribute blood flow away from their kidneys, which decreases fetal urine production, resulting in oligohydramnios [5].

Homeostatic mechanisms, such as intramembranous absorption (transfer of amniotic fluid across the amnion into the fetal circulation), also exist and work to maintain amniotic fluid volume. These mechanisms appear to be more successful in limiting excess fluid volume than in preventing reduced fluid volume. As an example, only half of fetuses with esophageal atresia, and two-thirds of fetuses with duodenal or proximal jejunal atresia develop polyhydramnios [6], whereas renal agenesis invariably results in oligohydramnios.

The physiology of normal amniotic fluid production and volume regulation are discussed separately. (See "Physiology of amniotic fluid volume regulation").

ETIOLOGY — Conditions commonly associated with oligohydramnios are listed in the table (table 1). The most likely etiologies of oligohydramnios vary according to severity and the trimester in which they are diagnosed. The majority of women with oligohydramnios or borderline/low normal amniotic fluid volume have no identifiable cause.
**First trimester** — The etiology of first trimester oligohydramnios is often unclear. Reduced amniotic fluid prior to 10 weeks of gestation is rare because gestational sac fluid is primarily derived from the fetal surface of the placenta, transamniotic flow from the maternal compartment, and secretions from the surface of the body of the embryo.

Criteria suggested for determining reduced amniotic fluid at this gestational age have included a difference between mean gestational sac size (MGSS) and crown-rump length of less than 5 mm or a mean gestational sac diameter/crown-rump length ratio outside the normal range for gestational age \([7-11]\). This finding has been associated with poor outcome in selected populations \([7]\). However, the prognostic value of these findings, when applied to large unselected populations, has not been adequately studied \([11]\).

**Second trimester** — By the beginning of the second trimester, fetal urine begins to enter the amniotic sac and the fetus begins to swallow amniotic fluid. Therefore, disorders related to the fetal renal/urinary system begin to play a prominent role in the etiology of oligohydramnios (table 2). Maternal and placental factors, as well as rupture of the fetal membranes, are also common causes of oligohydramnios in the second trimester.

The etiologies and relative frequencies of midtrimester oligohydramnios were illustrated in a series of 128 fetuses first noted to have severe oligohydramnios/anhydramnios at 13 to 24 weeks of gestation \([12]\). The following etiologies were observed: fetal anomaly (51 percent), preterm premature rupture of membranes (PPROM) (34 percent), placental abruption (7 percent), fetal growth restriction (FGR) (5 percent), and unknown (4 percent). Six of the 65 anomalous fetuses were aneuploid. The pregnancy outcome was generally poor due to fetal or neonatal death or pregnancy termination (see 'Prognosis and management' below).

An elevated maternal serum alpha fetoprotein (MSAFP) concentration has also been linked to second trimester oligohydramnios, with or without an anomalous fetus. This combination (elevated MSAFP, decreased amniotic fluid volume) carries an extremely poor prognosis: fetal growth restriction, fetal death, preterm delivery, neonatal death \([13-16]\). In one review of these cases, only 8 of 57 (14 percent) children survived past the neonatal period \([17]\). Oligohydramnios associated with an elevated MSAFP level may be caused by fetal membrane or placental damage, with leakage of amniotic fluid or fetal blood into the maternal circulation \([18]\).

Second trimester oligohydramnios related to amniocentesis appears to have a better prognosis. The membranes often "reseal" with reaccumulation of amniotic fluid and normal pregnancy outcome. (See "Diagnostic amniocentesis", section on 'Leakage of amniotic fluid'.)

There are a few reports of the occurrence and outcome of oligohydramnios after chorionic villus sampling \([19,20]\). (See "Chorionic villus sampling".)

**Third trimester** — Oligohydramnios first diagnosed in the third trimester is often associated with PPROM or with uteroplacental insufficiency due to conditions such as preeclampsia or other maternal vascular diseases. Oligohydramnios frequently accompanies fetal growth restriction related to uteroplacental insufficiency. Fetal anomalies and abruptio placentae also play a role at this gestational age. Amniotic fluid volume normally decreases postterm and oligohydramnios can develop in these pregnancies. In addition, many cases of third trimester oligohydramnios are idiopathic. (Refer to individual topic reviews on these subjects)

There may also be an association between pregnancy during the summer season and oligohydramnios, likely related to suboptimal maternal hydration in hot weather \([21]\). Mechanisms of isolated oligohydramnios also may include alterations in the expression of water pores (aquaporin 1, aquaporin 3) in fetal membranes and placenta \([22]\).

**CLINICAL MANIFESTATIONS AND DIAGNOSIS** — Oligohydramnios may be first suspected because the uterine size is less than expected for gestational age. Clinical diagnosis is based on the finding of decreased amniotic fluid on ultrasound examination. Methods for assessment of amniotic fluid volume are discussed in detail separately. (See "Assessment of amniotic fluid volume".)
There are both objective and subjective ultrasound criteria for oligohydramnios. Although use of an objective criterion is generally preferable (amniotic fluid index ≤5; single deepest pocket <2 cm), subjective suspicion of amniotic fluid volume by experienced examiners has similar sensitivity for diagnosing reduced amniotic fluid volume confirmed by the dye-dilution method, the gold standard for quantifying volume [23]. There are no large studies in women with oligohydramnios comparing the various sonographic methods of amniotic fluid volume assessment against the dye dilution method, and the latter is not useful clinically because it is invasive and time-consuming. Small series have found that objective sonographic measurements often grossly under- or over-estimated oligohydramnios [24-28]. In the only study comparing sonographic and magnetic resonance imaging assessment of amniotic fluid volume, both modalities had similar, and relatively poor, efficacy for the detection of oligohydramnios [29].

We use the amniotic fluid index (AFI) for diagnosing oligohydramnios. The AFI provides a means of quantifying normal and abnormal fluid volumes and comparing assessments of amniotic fluid volumes across gestation. As the 5th percentile for AFI averages approximately 7 cm throughout gestation, an AFI ≤5 cm is greater than two standard deviations below the mean value [30]. Most studies that found significant morbidity associated with oligohydramnios used an AFI ≤5 cm as the cutoff, rather than the percentile for gestational age [31]. Since the AFI remains relatively stable between 22 to 39 weeks of gestation [32,33], we feel that using the AFI cutoff ≤5 cm rather than using the 5th percentile is more clinically relevant for the diagnosis of oligohydramnios, and represents a volume that may require further assessment and/or clinical intervention.

We use the term borderline/low normal amniotic fluid volume to describe pregnancies with AFI >5.0 and ≤8.0 cm. (See "Assessment of amniotic fluid volume", section on 'Amniotic fluid index'.)

A single deepest pocket (SDP) <2 cm can also be used as an objective criterion for oligohydramnios. A randomized trial (SAFE) comparing AFI and SDP for prediction of adverse pregnancy outcome at term in low- and high-risk pregnancies found that use of AFI increased the frequency of diagnosis of oligohydramnios and labor induction but did not improve perinatal outcome compared with SDP [34].

Anhydramnios can be defined as the lack of a measurable AFI or SDP, although a thin echolucent rim may be imaged on the inner aspect of the uterus. (See "Assessment of amniotic fluid volume", section on 'Single deepest pocket'.)

**Multiple gestation** — Measurement of AFI for each sac of a multiple gestation is difficult so single deepest vertical pocket is used for diagnosis of oligohydramnios. In a twin gestation, a value ≤2 cm suggests the amniotic fluid volume is <2.5th percentile and is generally accepted as diagnostic of oligohydramnios [35,36]; this is the same vertical pocket threshold used in singletons.

**EVALUATION OF PREGNANCIES WITH OLIGOHYDRAMNIOS**

- A thorough maternal history and targeted physical examination is performed to look for maternal conditions that may be associated with oligohydramnios (table 1). The effect of maternal medications (eg, prostaglandin synthase inhibitors) may be limited to oligohydramnios, whereas maternal medical disorders often cause a combination of fetal growth restriction and oligohydramnios.

- A comprehensive sonographic evaluation with fetal biometry is performed, as well as a search for fetal anomalies (table 2), markers suggestive of aneuploidy (eg, increased nuchal translucency), fetal growth restriction, or placental abnormalities (eg, abruption) that may account for decreased amniotic fluid volume. (See "Sonographic findings associated with fetal aneuploidy" and "Fetal growth restriction: Diagnosis" and "Placental abruption: Clinical features and diagnosis".)

- If PROM is suspected but the diagnosis is uncertain after noninvasive assessments (eg, examination for vaginal pooling, Nitrazine and fern tests, Amnisure), indigo carmine dye (where available) can be injected into the amniotic fluid. Fetal membrane rupture is confirmed if dye is observed on a tampon placed in the vagina at the time of the procedure. If indigo carmine is not available, indocyanine green is a potential alternative. This dye has been used for newborn blood volume measurements in both preterm and term infants without reported adverse effects [37-39]. It should be given to a pregnant woman only if...
clearly indicated, as reproductive toxicology studies in animals have not been conducted and information in pregnancy and the neonatal period is limited. (See "Preterm premature (prelabor) rupture of membranes", section on 'Diagnosis'.)

- If there are fetal anomalies, amniocentesis may reveal an abnormal karyotype. Trisomy 13 and triploidy are the most common chromosomal abnormalities associated with early oligohydramnios. (See "Congenital cytogenetic abnormalities", section on 'Fetuses with congenital anomalies'.)

MSAFP, if elevated, also has prognostic significance and can be helpful in counseling [13-16].

METHODS OF INCREASING AMNIOTIC FLUID VOLUME — There is no treatment of oligohydramnios that has been proven to be effective long-term. However, short-term improvement of amniotic fluid volume is possible and may be considered under certain circumstances, such as when a fetal anatomic survey is needed.

Amnioinfusion — Amnioinfusion temporarily increases amniotic fluid volume. It has been used in the following settings (see "Amnioinfusion":)

- To improve detection of fetal anomalies — During the second trimester, oligohydramnios may limit optimal ultrasound assessment of the fetus. In such cases, transabdominal amnioinfusion of approximately 200 milliliters of saline under ultrasound guidance can provide better visualization of fetal anatomy and thus improve diagnostic precision [40-42]. It is a reasonable option when the information obtained is likely to affect pregnancy management.

A review of patients with unexplained midtrimester oligohydramnios who underwent diagnostic antenatal amnioinfusion found that the overall rate of adequate visualization of fetal structures improved from 51 to 77 percent [40]. There was also an improvement (from 12 to 31 percent) in the identification of associated anomalies in fetuses having preinfusion-identified obstructive uropathy. Other studies reported that information obtained at amnioinfusion at a median gestational age of 22 weeks led to a change of etiologic diagnosis in 13 percent of cases [41] and the most common postamnioinfusion new findings were renal anomalies, rupture of membranes, and growth restriction [42].

Fetal magnetic resonance imaging, alone or in combination with amnioinfusion, has been used as a complementary imaging method for detecting fetal anomalies in second trimester oligohydramnios.

- To facilitate cephalic version — Decreased amniotic fluid is considered a relative contraindication to version and increases the failure rate. Minimal data have been published on amnioinfusion for this indication. (See "External cephalic version", section on 'Amnioinfusion'.)

- To prevent fetal sequelae of oligohydramnios — Although investigational, serial transabdominal amnioinfusions have been used to improve fetal outcome in pregnancies with idiopathic oligohydramnios [43] or early oligohydramnios due to premature rupture of membranes [44]. A comparative study using an ovine model with complete obstructive uropathy found that serial amnioinfusions through an intraamniotic Port-A-Cath prevented pulmonary hypoplasia compared to controls who did not undergo serial amnioinfusion [45]. The treated group had lung volumes comparable to the control lambs that underwent a sham operation. Additional studies to confirm these findings are needed prior to clinical utilization of this approach for second trimester oligohydramnios.

Maternal hydration — In cases of isolated oligohydramnios in which delivery is not indicated, oral hydration with one to two liters of water can transiently increase amniotic fluid volume and may have some benefit, particularly in patients with dehydration. This approach is easier and safer than intravenous fluid administration or amnioinfusion. Hydration with water appears to reduce maternal plasma osmolality and sodium concentration, resulting in osmotically driven maternal to fetal water flux; it also improves uteroplacental perfusion.
A 2015 systematic review and meta-analysis of the efficacy of maternal hydration strategies for improving amniotic fluid volume found that maternal hydration was most effective in pregnancies with isolated oligohydramnios and that hypotonic solutions were more effective than isotonic fluids [46]. Whether maternal hydration improved clinical outcome in pregnancies with isolated oligohydramnios was unclear because of heterogeneity in patient selection, sonographic diagnostic criteria, hydration protocols, and outcome assessment.

A prospective study not included in the analysis provided an example of the potential effect of maternal hydration. In this study, 10 women with third trimester oligohydramnios (amniotic fluid index [AFI] < 5 cm) and 10 women with normal amniotic fluid volume asked to consume two liters of water over two hours [47]. In women with oligohydramnios, hydration increased mean AFI by 3.2 cm (95% CI 1.1-5.3) but had no effect on AFI in women with normal amniotic fluid volume.

**Water and DDAVP** — The combined use of oral water ingestion and desmopressin (DDAVP) markedly and transiently increases amniotic fluid volume [48,49]. This effect is attributed to both of maternal hydration and antidiuresis, and thus maternal plasma hypoosmolality. Use of DDAVP for this indication should be considered experimental, and used only under approved research protocols.

**Fetal membrane sealants** — A variety of tissue sealants (eg, fibrin glue, gelatin sponge, amniopatch) have shown some success in stopping leakage from ruptured membranes in case reports. Neither the safety nor the efficacy of these sealants has been established. (See "Midtrimester preterm premature rupture of membranes", section on 'Repair of leaks'.)

**Investigational therapies** — Investigational approaches to oligohydramnios have explored novel treatments. In a pilot trial of women with idiopathic oligohydramnios diagnosed after 30 weeks of gestation, administration of sildenafil citrate three times daily and intravenous hydration of 1 L followed by oral hydration significantly increased the AFI compared with intravenous hydration of 1 L followed by oral hydration alone [50].

**PROGNOSIS AND MANAGEMENT** — The fetal/neonatal prognosis depends on the cause, severity, gestational age at onset, and duration of oligohydramnios. In fetuses with renal anomalies, the type of extrarenal anomalies, if present, also affects prognosis. In all cases, the clinician must consider the complete clinical picture and the risks of intervention prior to clinical management decisions.

**First trimester** — Reduced amniotic fluid at this time is an ominous finding; the pregnancy usually aborts. In one series, 15 of 16 patients (94 percent) with a normal fetal heart rate and small sac noted on first trimester sonogram went on to spontaneously abort compared with only 4 of 52 control patients (8 percent) with normal sac size [7]. (See "Spontaneous abortion: Risk factors, etiology, clinical manifestations, and diagnostic evaluation", section on 'Pelvic ultrasound'.)

We counsel these patients regarding the poor prognosis and inform them of the signs of miscarriage. Serial sonographic examinations are helpful for following the natural history of the process (eg, worsening oligohydramnios, embryonic/fetal demise, or [rarely] resolution).

**Second trimester** — In the second trimester, prognosis and management depend upon the underlying etiology and the magnitude of amniotic fluid reduction.

- **Borderline/low normal amniotic fluid volume** — Pregnancies with borderline/low normal amniotic fluid volume generally have a good prognosis [4,14,51,52]. Serial sonographic examinations are helpful for following the natural history of the process, which may remain stable, resolve, or progress to development of oligohydramnios and/or fetal growth restriction.

- **Oligohydramnios** — In the second trimester, oligohydramnios often ends in fetal or neonatal death [12,53]. In the large series described above (128 fetuses first noted to have oligohydramnios at 13 to 24 weeks of gestation), survival occurred in 9/43 (21 percent) fetuses with PPROM, 2/9 (22 percent) fetuses with abruptio, 1/5 (20 percent) idiopathic cases, 1/65 (1.5 percent) fetuses with congenital anomalies,
and 0/6 fetuses with fetal growth restriction [12]. Many of these women chose pregnancy termination because of the poor prognosis.

Preterm delivery, either spontaneous or indicated by maternal or fetal complications, occurs in more than 50 percent of cases [4,13-15,41,54,55]. Infants may have anatomical and functional abnormalities, such as skeletal deformations, contractures, and pulmonary hypoplasia. (See "Midtrimester preterm premature rupture of membranes", section on 'Musculoskeletal development' and "Midtrimester preterm premature rupture of membranes", section on 'Pulmonary hypoplasia'.)

We initially perform a fetal anatomic survey to look for fetal malformation, which may influence future management. We administer oral maternal hydration or perform amnioinfusion in pregnancies in which the fetus cannot be visualized adequately. If rupture of membranes is uncertain, amnioinfusion with instillation of indigo carmine dye (where available) facilitates both diagnosis of PPROM and visualization of fetal anomalies. Serial sonographic examinations are obtained to monitor amniotic fluid volume, fetal growth, and fetal well-being. (See "Preterm premature (prelabor) rupture of membranes").

Specific pregnancy complications associated with oligohydramnios are managed as appropriate for the condition. (Refer to individual topic reviews on specific chromosomal and congenital anomalies, midtrimester PPROM, maternal medical disorders, preeclampsia, abruptio placentae, etc).

**Third trimester** — Some [2,56], but not all [57], studies have shown an inverse relationship between amniotic fluid volume in the third trimester and the incidence of adverse pregnancy outcome. Small sample size likely affected the results of negative studies. Adverse outcomes are related to umbilical cord compression, uteroplacental insufficiency, and meconium aspiration. In particular, uteroplacental insufficiency and cord compression are associated with fetal heart rate abnormalities that result in cesarean delivery and low Apgar scores [12,58-61].

A study on 28,555 pregnancies to estimate pregnancy and neonatal outcomes in women with decreased AFI between 24 and 34 weeks of gestation found that major malformations were more common in pregnancies with oligohydramnios (AFI ≤5 cm) and borderline AFI (5 to 8 cm) than in those with normal fluid; major malformations were present in 25, 10, and 2 percent of fetuses, respectively (P<.001) [52]. Among nonanomalous fetuses, complications that occurred more often in pregnancies with oligohydramnios and borderline AFI included preterm birth: 62, 37, and 8 percent, respectively; cesarean delivery for nonreassuring fetal status: 9, 9, and 4 percent, respectively; and birth weight below the third percentile: 37, 21, and 4 percent, respectively.

Idiopathic isolated oligohydramnios at term appears to have a better prognosis. In a retrospective cohort study of term pregnancies with sonographic finding of isolated oligohydramnios (n = 987) and a control group of pregnancies with normal AFI (n = 22,280), isolated oligohydramnios was not independently associated with increased risk for a composite adverse outcome when pregnancies complicated by thrombophilia, hypertension, diabetes, deviant fetal growth, or chromosomal/structural abnormalities were excluded [62].

The duration of oligohydramnios is also a prognostic factor. Patients who present with idiopathic oligohydramnios at an earlier gestational age are at risk for adverse perinatal outcomes compared with those presenting later in gestation [12,63]. As an example, in one series of 122 cases of oligohydramnios initially diagnosed in the third trimester and 128 cases initially diagnosed in the second trimester, perinatal survival was 85 and 10 percent, respectively [12]. Two large meta-analyses reviewed cases of isolated oligohydramnios. In an analysis of 12 studies with 35,999 women, 2414 (6.7 percent) women demonstrated isolated oligohydramnios at term, which was associated with significantly higher rates of labor induction, cesarean sections, and short-term neonatal morbidity [64]. When patients with oligohydramnios are stratified into high risk (patients with comorbid conditions such as hypertension) and low risk (women with normal AFI), those with isolated oligohydramnios had significantly higher rates of an infant with meconium aspiration syndrome, cesarean delivery for fetal distress, and admission to the neonatal intensive care unit. Patients with oligohydramnios and comorbidities were more likely to have an infant with low birth weight, though rates
of meconium aspiration syndrome, cesarean delivery, and neonatal intensive care unit admission were similar to those for women with normal AFI. Although the authors concluded that, in high-risk pregnancy, management should be dictated by the comorbid condition and not the presence of oligohydramnios, we believe the oligohydramnios management should be similar in both low- and high-risk pregnancies [61].

Given the potential high risk of adverse outcome, we deliver patients with oligohydramnios who are at term (see 'Labor' below). Patients with oligohydramnios early in the third trimester may be hospitalized to undergo evaluation of possible causes (see 'Evaluation of pregnancies with oligohydramnios' above), daily nonstress tests, and maternal hydration in an attempt to increase amniotic fluid volume (see 'Maternal hydration' above). In ambulatory patients, we perform a nonstress test (NST) and AFI (or biophysical profile) once or twice weekly until delivery, depending upon the maternal and fetal condition. Combined use of the NST and AFI is associated with a low rate of unexpected fetal death [65-67].

Doppler velocimetry may have a role in identifying patients with idiopathic oligohydramnios who are at higher risk of adverse outcome [68]. In one retrospective series of 76 patients with oligohydramnios unrelated to PROM or congenital anomalies, the 46 patients with normal systolic/diastolic ratios demonstrated markedly reduced perinatal morbidity compared with the 30 patients with abnormal systolic/diastolic ratios (11 versus 80 percent) [68]. These findings, and observations from other studies [69], suggest that avoiding intervention in pregnancies with oligohydramnios and normal Doppler velocimetry may decrease iatrogenic morbidity related to prematurity. Although the role of Doppler velocimetry in managing fetal growth restriction is well established, its use in other circumstances remains to be validated.

**Timing of delivery** — The indications for delivery in women with oligohydramnios attributable to a specific condition (eg, preeclampsia, premature rupture of membranes, fetal growth restriction, congenital anomaly, postterm pregnancy, etc) are discussed separately in topic reviews on these disorders.

Timing of delivery in pregnancies complicated by idiopathic oligohydramnios is controversial [70]. Only one small, randomized trial has evaluated outcomes with intervention versus expectant management. In this trial, 54 pregnancies beyond 40 weeks of gestation with isolated oligohydramnios were randomly assigned to either induction of labor or expectant management [71]. No differences were found for any important maternal or neonatal outcome.

Observational studies have reported conflicting results. In one retrospective series, the corrected perinatal mortality rate in structurally normal fetuses with oligohydramnios was significantly lower when delivery was initiated upon diagnosis (in gestations of at least 28 weeks) than with conservative management (18 versus 90 per 1000 births) [72]; this suggests that intervention is indicated. However, the groups came from different institutions and time periods, so they may not have been comparable.

Most studies have reported no increased risk of fetal acidosis and generally good outcomes in pregnancies with isolated oligohydramnios (AFI <5 cm) in the third trimester (ie, appropriately grown nonanomalous fetus, reassuring fetal heart rate pattern, no maternal disease) when compared to controls with normal amniotic fluid volumes [60,73-83]. Importantly, most studies examining the benefits of AFI assessments instituted scheduled antenatal testing for these patients, suggesting that oligohydramnios may precede fetal compromise [75]. Alternatively, this may be related, in part, to the low sensitivity and specificity of AFI for uteroplacental insufficiency, especially in the absence of other indicators of impaired placental perfusion, such as fetal growth restriction, preeclampsia, fetal abnormalities, postterm pregnancy, or abortion [56,84].

Our indications for delivery in patients with idiopathic oligohydramnios include, but are not limited to, nonreassuring fetal testing or reaching 37 to 38 completed weeks of gestation, regardless of the cervical Bishop score. Delivery as early as 36 to 37\(\frac{6}{7}\)th weeks has been suggested [85,86]. Although induction increases the risk of cesarean delivery [74,83], there is insufficient evidence to assure us that perinatal outcome with continued conservative management of oligohydramnios at term is comparable to that with delivery, even in the presence of an appropriately grown, uncompromised fetus and absence of maternal disease.
We do not typically confirm pulmonary maturity prior to delivery in well-dated pregnancies beyond 36 weeks. Amniocentesis can be challenging in the presence of oligohydramnios due to the lack of a fetus- or placenta-free site for needle insertion.

Alternatively, the patient can be followed with serial nonstress testing and biophysical profiles until term gestation is reached; the risks and benefits of various management plans should be discussed with the patient.

**Labor** — Neither AFI nor single deepest pocket performed on admission to the labor unit accurately predicts the risk of peripartum complications [87-89]. We recommend obtaining a short electronic fetal heart rate recording upon admission of patients in labor to help determine whether the fetal heart rate should be monitored continuously or whether intermittent monitoring is likely to be sufficient. If the fetal heart rate is not reactive or fetal heart rate decelerations are present, then we recommend continuous electronic fetal heart rate monitoring during labor. We also consider transcervical amnioinfusion for patients with oligohydramnios and variable fetal heart rate decelerations in labor. (See "Amnioinfusion".)

**SUMMARY AND RECOMMENDATIONS**

- **Oligohydramnios** refers to amniotic fluid volume that is less than expected for gestational age. It is typically diagnosed by ultrasound examination and may be described qualitatively or quantitatively (eg, amniotic fluid index [AFI] ≤5; single deepest pocket <2 cm). (See 'Clinical manifestations and diagnosis' above.)

- Conditions commonly associated with oligohydramnios are listed in the table ([table 1](#)). The most likely etiologies of oligohydramnios vary according to the trimester in which it is diagnosed. The majority of women with mild oligohydramnios have no identifiable cause. (See 'Etiology' above.)

- Maternal history and physical examination and a comprehensive sonographic evaluation are recommended for all pregnancies with oligohydramnios. Use of additional tests (eg, karyotype, instillation of dye) depends upon individual clinical circumstances. (See 'Evaluation of pregnancies with oligohydramnios' above.)

- There is no effective long-term treatment of oligohydramnios. In idiopathic oligohydramnios, maternal treatment with intravenous isotonic solution, oral hydration, or amnioinfusion can lead to short-term improvement. These procedures may be useful under certain circumstances, such as to facilitate diagnostic ultrasound evaluation when the fetal anatomic survey is suboptimal. (See 'Methods of increasing amniotic fluid volume' above.)

- Reduced amniotic fluid in the first trimester appears to be an ominous finding. We counsel these patients regarding the poor prognosis, discuss the signs of miscarriage, and follow the pregnancy with serial ultrasound examinations to determine its course. (See 'First trimester' above.)

- The prognosis and management of second trimester oligohydramnios depend upon the underlying etiology and severity of oligohydramnios. Pregnancies with borderline/low normal amniotic fluid volume generally have a good prognosis. Anhydramnios diagnosed at this time may induce anatomical and functional fetal abnormalities and often results in fetal or neonatal death.

  We initially perform a fetal structural survey to rule out a fetal malformation since serious abnormalities may influence future management. Serial sonographic examinations to monitor amniotic fluid volume, fetal growth, and fetal well-being are performed until delivery. (See 'Second trimester' above.)

- Some studies have shown an inverse relationship between amniotic fluid volume in the third trimester and the incidence of adverse pregnancy outcome. Adverse outcomes are related to umbilical cord compression, uteroplacental insufficiency, and meconium aspiration. Given the potential high risk of adverse outcome, we perform a nonstress test (NST) and AFI (or biophysical profile) once or twice weekly until delivery. (See 'Third trimester' above.)
For women with idiopathic oligohydramnios, we suggest delivery at 37 to 38 completed weeks of gestation rather than expectant management (Grade 2C). Although induction of an unfavorable cervix may increase the risk of cesarean delivery, there is insufficient evidence to assure us that perinatal outcome with continued conservative management of oligohydramnios at term is comparable to that with delivery. (See ‘Timing of delivery’ above.)

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REFERENCES


36. Reddy UM, Abuhamad AZ, Levine D, et al. Fetal imaging: executive summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, American Institute of Ultrasound in Medicine, American College of Obstetricians and Gynecologists,


75. Driggers RW, Holcroft CJ, Blakemore KJ, Graham EM. An amniotic fluid index < or =5 cm within 7 days of delivery in the third trimester is not associated with decreasing umbilical arterial pH and base excess. J Perinatol 2004; 24:72.


Causes of oligohydramnios

### Maternal
- Medical or obstetrical conditions associated with uteroplacental insufficiency (e.g., preeclampsia, chronic hypertension, collagen vascular disease, nephropathy, thrombophilia)
- Medications (e.g., angiotensin converting enzyme inhibitors, prostaglandin synthetase inhibitors, trastuzumab)

### Placental
- Abruptio
- Twin to twin transfusion (i.e., twin polyhydramnios-oligohydramnios sequence)
- Placental thrombosis or infarction

### Fetal
- Chromosomal abnormalities
- Congenital abnormalities, especially those associated with impaired urine production
- Growth restriction
- Demise
- Postterm pregnancy
- Ruptured fetal membranes

### Idiopathic

Graphic 79447 Version 3.0
### Type and frequency of congenital anomalies associated with oligohydramnios in a literature review

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<th>Type</th>
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Graphic 78227 Version 2.0
Contributor Disclosures

Ron Beloosesky, MD Nothing to disclose Michael G Ross, MD, MPH Nothing to disclose Lynn L Simpson, MD Nothing to disclose Deborah Levine, MD Nothing to disclose Vanessa A Barss, MD, FACOG Nothing to disclose

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