Oligohydramnios: Etiology, diagnosis, and management in singleton gestations

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INTRODUCTION

Oligohydramnios refers to amniotic fluid volume (AFV) that is less than the minimum expected for gestational age. It is diagnosed by ultrasound examination, preferably based on an objective measurement such as amniotic fluid index (AFI) ≤5 cm or single deepest pocket (SDP) <2 cm, but a subjective assessment of reduced AFV is also acceptable. Some cases have an identifiable maternal, fetal, or placental cause (table 1); the remainder are considered idiopathic.

The fetal prognosis depends on several factors, particularly the underlying cause, severity (reduced versus no amniotic fluid), and gestational age at occurrence. Because an adequate AFV is critical to normal fetal movement and second-trimester lung development and for cushioning the fetus and umbilical cord from uterine compression, pregnancies complicated by oligohydramnios from any cause are at risk for pulmonary hypoplasia (if second-trimester oligohydramnios), fetal deformation (if prolonged oligohydramnios), and umbilical cord compression. Oligohydramnios is associated with an increased risk for fetal or neonatal death, which may be related to the underlying cause of the reduced AFV, the sequalae of reduced AFV, or both.

This topic will discuss issues related to oligohydramnios in singleton gestation. Methods of AFV assessment in singleton and multiple gestations and the significance of oligohydramnios in multiple gestations, particularly monochorionic twins, are reviewed separately.

- (See "Assessment of amniotic fluid volume".)
- (See "Twin-twin transfusion syndrome: Screening, prevalence, pathophysiology, and diagnosis", section on 'Diagnostic criteria'.)

INCIDENCE

In the overall obstetric population, oligohydramnios occurs in <1 percent of preterm pregnancies and in 2 to 10 percent of pregnancies at 40 to 42 weeks [1-4]. Rates of oligohydramnios reported in individual studies vary because they are highly influenced by the gestational age at the time of the ultrasound examination, the population studied (low or high risk, screening or indicated ultrasound examination, antepartum or intrapartum), and variations in diagnostic criteria.

PATHOGENESIS

AFV reflects the balance between AF flow pathways that increase AFV (urination, outflow of lung secretions) and those that decrease AFV (swallowing, intramembranous absorption [the transport of amniotic water and solutes across the amniotic membrane into the fetal circulation], leakage because of membrane rupture). The two most common disturbances leading to oligohydramnios are (1) reduced urination due to fetal kidney disease or lower urinary tract obstruction and (2) AF loss due to rupture of membranes.

Reductions in the outflow of lung fluid and increased swallowing do not play major roles in pathogenesis of oligohydramnios. Intramembranous absorption is an important pathway for maintaining normal AFV; however, it appears to be more successful in preventing hydramnios than in preventing oligohydramnios. Nevertheless, idiopathic cases may be due to alterations in the expression of specific aquaporins in the membranes and placenta or other alterations in activity of vesicular transport pathways across the amnion [5-7]. (See "Physiology of amniotic fluid volume regulation".)

ETIOLOGY

Many of the conditions associated with oligohydramnios are listed in the table (table 1). The most likely etiologies vary according to oligohydramnios severity and the trimester of diagnosis. The majority of cases present in the third trimester and have no identifiable cause.

Second trimester — By the beginning of the second trimester, fetal urine is being produced and entering the amniotic sac. Therefore, congenital anomalies related to the fetal kidney and urinary tract (CAKUT) begin to play a prominent role in the etiology of oligohydramnios

(table 2). These anomalies include intrinsic renal disorders (eg, cystic renal disease) and obstructive lesions of the lower urinary tract (eg, posterior urethral valves, urethral atresia). Maternal and placental factors, as well as rupture of the membranes (traumatic or spontaneous), are also common causes of oligohydramnios in the second trimester (table 1).

The etiologies and relative frequencies of second-trimester oligohydramnios were illustrated in a series of 128 fetuses first noted to have severe oligohydramnios/anhydramnios at 13 to 24 weeks of gestation [8]. The following etiologies were observed:

- Fetal anomaly (51 percent). Six of the 65 anomalous fetuses were aneuploid.
- Preterm prelabor rupture of membranes (PPROM, 34 percent)
- Placental abruption (7 percent)
- Fetal growth restriction (FGR, 5 percent)
- Idiopathic (4 percent)

The pregnancy outcome was generally poor due to fetal or neonatal death or pregnancy termination. (See 'Prognosis and counseling by etiology' below.)

Third trimester — Oligohydramnios first diagnosed in the third trimester is often due to PPROM. Other common causes are uteroplacental insufficiency (manifested by FGR, preeclampsia, and/or chronic abruption) and fetal anomalies (table 2). In addition, many cases are idiopathic. Fetal TORCH (toxoplasma gondii, rubella virus, cytomegalovirus, herpes simplex virus) and parvovirus B19 infections are uncommon but may be associated with second- or third-trimester oligohydramnios; other evidence of fetal infection is often present [9,10].

Oligohydramnios is more prevalent in postterm pregnancies since AFV normally begins to decline late in the third trimester (figure 1). Rapid development of oligohydramnios has been described postterm [11].

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Presentation — Oligohydramnios may be suspected because the uterine size is less than expected for gestational age or because the patient presents with prelabor rupture of membranes.

It may be initially detected on an ultrasound examination performed to assess AFV in a patient at risk for an AF abnormality (eg, ruptured membranes, preeclampsia) or as an incidental finding on an ultrasound examination performed for another reason (eg, fetal anatomy scan at 18 to 20 weeks).

Diagnosis — The diagnosis is based on any of the following:

- Amniotic fluid index (AFI) ≤5 cm
- Single deepest pocket (SDP) <2 cm
- Subjective assessment of oligohydramnios by an experienced ultrasound examiner

Some clinicians classify oligohydramnios as mild (AFI 4.1 to 5.0 cm), moderate (AFI 2.1 to 4.0 cm) and severe (AFI 0 to 2.0 cm) [12]. Anhydramnios is the extreme end of the oligohydramnios spectrum, defined by lack of a measurable AFI or SDP, although a thin echolucent rim may be imaged on the inner aspect of the uterus.

Use of objective criteria is generally preferable for diagnosis; however, subjective assessment of oligohydramnios by an experienced ultrasound examiner has similar diagnostic sensitivity as objective criteria when compared with the dye-dilution method, the gold standard for quantifying AFV [13].

It appears that the AFI overly diagnoses oligohydramnios, while the SDP overly diagnoses polyhydramnios [14]. Accordingly, one may consider using the SDP in patients with low AFV and the AFI for patients with high AFV, though this approach may not be practical. A meta-analysis comparing SDP versus AFI for predicting adverse perinatal outcomes in pregnancies with oligohydramnios reported that use of the SDP was associated with a lower rate of pregnancy intervention without any increase in adverse pregnancy outcome [15].

The diagnostic performance of ultrasound assessment of amniotic fluid is reviewed in detail separately. (See "Assessment of amniotic fluid volume".)

Ultrasound technique — The transducer can be held perpendicular to the floor or perpendicular to the uterine contour when obtaining the measurements [16,17]. The fluid pocket should not contain fetal extremities or umbilical cord (on gray-scale examination) and the depth is only measured when a transverse diameter of AF ≥1 cm is present. The technique for ultrasound assessment of amniotic fluid is reviewed in detail separately. (See "Assessment of amniotic fluid volume".)

MANAGEMENT

Postdiagnostic evaluation — Our approach to evaluation of pregnancies with oligohydramnios follows. Patients with newly diagnosed oligohydramnios early in the third trimester undergo evaluation of possible causes and daily nonstress tests (NSTs) until fetal status is clarified. Patients with anhydramnios are typically hospitalized for both diagnostic evaluation and fetal surveillances.

- **History and physical**

- Perform a thorough maternal and family history and a targeted physical examination to look for maternal and familial conditions that may be associated with oligohydramnios (table 1). Maternal medications that cause oligohydramnios typically affect fetal urination and do not cause growth restriction, whereas maternal medical disorders often result in uteroplacental insufficiency, leading to both fetal growth restriction (FGR) and oligohydramnios.

- Rule out prelabor rupture of membranes (PROM). (See "Preterm prelabor rupture of membranes: Clinical manifestations and diagnosis", section on 'Diagnostic evaluation and diagnosis'.)

- **Ultrasound –** Perform a detailed sonographic evaluation for findings that may account for reduced AFV. The evaluation should include:

  - Assessment for fetal anomalies (table 2), particularly assessment of the:

    - Kidneys (presence, size, location, appearance [echogenicity, cysts, urinary tract dilation])
    - Bladder (size and shape)
    - Umbilical cord fetal insertion site and vessel number

  - Fetal sex (males are prone to renal dysplasia and agenesis, posterior urethral valves)

  - Second-trimester markers suggestive of aneuploidy (see "Sonographic findings associated with fetal aneuploidy")

  - FGR (see "Fetal growth restriction: Screening and diagnosis")

  - Placental abnormalities (eg, chronic abruption) (see "Acute placental abruption: Pathophysiology, clinical features, diagnosis, and consequences")

- **Genetic testing –** If fetal anomalies are identified, offering fetal diagnostic genetic testing (amniocentesis for microarray on amniocytes) is routine. For patients who decline amniocentesis, we offer a noninvasive screening test (cell-free DNA) and discuss its limitations. Trisomy 13 and triploidy are the most common chromosomal abnormalities associated with early oligohydramnios.

  If no fetal anomalies are detected (ie, isolated oligohydramnios), the risk of genetic abnormalities does not appear to be increased above the baseline risk [18]. Genetic testing may be offered to these patients as part of standard obstetric care or because of the possibility of undetected anomalies that would increase the risk for an underlying chromosomal disorder. (See "Prenatal genetic evaluation of the fetus with anomalies or soft markers" and "Sonographic findings associated with fetal aneuploidy".)

Exome sequencing was diagnostic in two families with recurrent oligohydramnios [19]. The decision to use exome sequencing should be made in consultation with a clinician specializing in genetic testing.

- **Ancillary imaging and interventions**

  - **Diagnostic amnioinfusion –** If oligohydramnios prevents adequate ultrasound assessment of the fetus, we typically recommend transabdominal amnioinfusion of approximately 200 mL of saline under ultrasound guidance to provide better visualization of fetal anatomy and thus improve diagnostic precision [20-23]. Amnioinfusion temporarily increases AFV; the duration of increase remains unclear, but appears to be for a few days. It is a reasonable option when the information obtained is likely to affect pregnancy management. During the amnioinfusion, fluid may be collected for genetic studies. In addition, in patients with second-trimester oligohydramnios, intraamniotic dye injection can be of value for diagnosis of prelabor rupture of membranes (PROM) if physical and laboratory examinations do not support the diagnosis.

    A review of patients with unexplained second-trimester oligohydramnios who underwent diagnostic antenatal amnioinfusion found that the overall rate of adequate visualization of fetal structures improved from 51 percent before amnioinfusion to 77 percent after amnioinfusion [20]. In fetuses having preinfusion-identified obstructive uropathy, the identification of associated anomalies increased from 12 percent before amnioinfusion to 31 percent after amnioinfusion. Other studies have 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 [21] and the most common new postamnioinfusion findings were renal anomalies, rupture of membranes, and growth restriction [22].

    The procedure is described separately. (See "Amnioinfusion".)

  - **MRI –** Fetal magnetic resonance imaging (MRI) can be helpful to better define complex fetal anomalies when this information will alter patient care, because it is less limited by lack of amniotic fluid than ultrasound. It may also detect anomalies missed on ultrasound. T1 and T2 imaging is preferable to T3 imaging because the latter can increase the temperature of amniotic fluid [24,25]. Whether MRI findings in oligohydramnios cases will alter patient care, counseling, or outcomes, as compared with ultrasound with or without amnioinfusion, is controversial.

  - **Diagnostic maternal hydration –** Oral hydration with one to two liters of water may be an alternative to amnioinfusion to transiently increase AFV for up to 48 hours, particularly in hypovolemic patients. This approach is easier and safer than

intravenous (IV) 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. The combined use of oral water ingestion and desmopressin (DDAVP) markedly and transiently increases AFV [26,27]; however, use of DDAVP for this indication should be considered experimental, and used only under approved research protocols.

A meta-analysis of the efficacy of maternal hydration strategies for improving AFV found that maternal hydration was most effective in pregnancies with isolated oligohydramnios and that hypotonic solutions were more effective than isotonic fluids [28]. A prospective study not included in the analysis provided an example of the potential effect of maternal hydration on AFV. In this study, 10 patients with third-trimester oligohydramnios (amniotic fluid index [AFI] <5 cm) and 10 patients with normal AFV were asked to consume two liters of water over two hours [29]. In patients with oligohydramnios, hydration increased mean AFI by 3.2 cm (95% CI 1.1-5.3) but had no effect on AFI in those with normal AFV.

- **Doppler –** There is no clear role for Doppler imaging in the evaluation of fetuses with kidneys and isolated oligohydramnios. Most studies have not observed changes in arterial blood flow in these cases [30-32]. In contrast, color Doppler is used to assess for the presence of renal arteries when the kidneys are not well visualized since failure to image renal vessels using appropriately low gain settings is suggestive, although not definitive evidence, of renal agenesis, which is a cause of oligohydramnios.

- **Laboratory testing –** No routine testing is required.

  - Testing maternal serum or amniotic fluid for an infection-related etiology (maternal TORCH [toxoplasma gondii, rubella virus, cytomegalovirus, herpes simplex virus] and parvovirus 19 infection) depends on the degree of suspicion (eg, suggestive maternal history and symptoms, other fetal abnormalities on ultrasound) in individual cases. See individual topic reviews on TORCH and parvovirus B19.

  - Maternal serum alpha-fetoprotein (MSAFP) may have been checked as part of biochemical marker screening performed between 15 and 20 weeks of gestation. If performed and elevated, it has prognostic significance and can be helpful in counseling [33-36]. Oligohydramnios associated with an elevated second-trimester MSAFP level may be caused by damage to the fetal membranes or placenta, leading to leakage of amniotic fluid from the vagina or transplacental passage of fetal blood into the maternal circulation [37]. The fetus may or may not be anomalous. The combination (elevated MSAFP, decreased AFV) carries an extremely poor prognosis,

including FGR, fetal death, preterm birth, and neonatal death [33-36]. In one review of these cases, only 8 of 57 children (14 percent) survived past the neonatal period [38].

Prognosis and counseling by etiology — The fetal/neonatal prognosis depends on the etiology, severity, gestational age at onset, and duration of oligohydramnios [8,12,39,40]. In a series of 128 fetuses first noted to have oligohydramnios at 13 to 24 weeks of gestation, survival was reported in 9 out of 43 (21 percent) fetuses with preterm prelabor rupture of membranes (PPROM), 2 out of 9 (22 percent) fetuses with abruption, 1 out of 5 (20 percent) idiopathic cases, 1 out of 65 (1.5 percent) fetuses with congenital anomalies, and 0 out of 6 fetuses with FGR [8]. Many of the deaths were the result of pregnancy termination because of the poor prognosis.

On the other hand, pregnancies with borderline/low normal AFV rather than oligohydramnios generally have a good prognosis [34,41-43]. Serial sonographic examinations every one to two weeks are helpful for following the natural history of the process, which may remain stable, resolve, or progress to development of oligohydramnios and/or FGR.

The prognosis for ongoing pregnancies with specific etiologies of oligohydramnios is discussed in detail below:

- **Renal oligohydramnios –** Patients with oligohydramnios due to a renal disorder (called "renal oligohydramnios") should receive multidisciplinary counseling, when appropriate, regarding the suspected etiology of oligohydramnios, further evaluation, short- and long-term prognosis, and their options (eg, termination, continuation of pregnancy, palliative neonatal care, aggressive neonatal care). The type of extra-renal anomalies, if present, also affects prognosis. Consultation with a pediatric nephrologist is very important in these cases, as the prognosis (mortality and morbidity) of children treated with dialysis and future renal transplant is improving and these may be options in some cases [44,45]. In a series of 103 consecutive pregnancies from 2008 to 2015 with renal oligohydramnios, eight underwent termination because of early onset, underlying renal disease, and extrarenal manifestations [45]. Eight infants died in utero. Of the 49 liveborn children, 11 received palliative care and 38 underwent active care; overall survival of the latter group was 84 percent. One-third of the surviving infants needed renal replacement therapy during the first six weeks of life. Eight pregnancies were lost to follow-up.

- **Idiopathic oligohydramnios –** Idiopathic oligohydramnios has a better prognosis than renal oligohydramnios, but the risk for adverse outcome is still increased, in part because some of these cases represent placental pathology, such as maternal vascular malperfusion [43,46-49].

- In a meta-analysis evaluating adverse pregnancy outcomes in singleton pregnancies diagnosed with oligohydramnios, compared with normal AFV, low-risk pregnancies with isolated oligohydramnios (ie, no pregnancy comorbidity that could explain the oligohydramnios) were at increased risk for [47]:

  - Meconium aspiration syndrome (1.4 versus 0.2 percent; risk ratio [RR] 2.83, 95% CI 1.38-5.77)
  - Cesarean birth for an abnormal fetal heart rate pattern (4.2 versus 1.4 percent; RR 2.16, 95% CI 1.64-2.85)
  - Admission to the neonatal intensive care unit (NICU) (8.7 versus 3.7 percent; RR 1.71, 95% CI 1.20-2.42)

  Patients with oligohydramnios and comorbidities that may have led to oligohydramnios (excluding fetal anomalies known to cause oligohydramnios and those with PROM) were at increased risk for low birth weight (19.1 versus 8.8 percent; RR 2.35; 95% CI 1.27-4.34) compared with those with normal AFV, whereas rates of five-minute Apgar score <7, NICU admission, meconium-stained amniotic fluid, and cesarean birth for an abnormal fetal heart rate pattern were similar for both groups.

  Stillbirth rates were too low to analyze.

Therefore, in ongoing pregnancies with idiopathic oligohydramnios, the risks for and potential sequelae of cord compression, preterm birth, and neonatal abnormalities should be discussed [8,47,50-53]. Cord compression can lead to fetal demise, postnatal asphyxia, or meconium aspiration syndrome. Preterm birth, either spontaneous or indicated by maternal or fetal complications, occurs in more than 50 percent of cases of oligohydramnios and is associated with increased morbidity and mortality [21,33-35,41,54,55]. Neonatal abnormalities as a result of prolonged development in a severely low amniotic fluid environment may be structural or functional and include skeletal deformations, contractures, and pulmonary hypoplasia. (See "Prelabor rupture of membranes before and at the limit of viability", section on 'Musculoskeletal development' and "Prelabor rupture of membranes before and at the limit of viability", section on 'Pulmonary hypoplasia'.)

- **Oligohydramnios after PPROM** – The gestational age at the time of membrane rupture is the critical factor in the risk of subsequent neonatal pulmonary hypoplasia [56-61]. Several series have reported a low incidence (less than 1.4 percent) of pulmonary hypoplasia when PPROM occurred after 26 weeks of gestation [58-60,62,63]. This gestational age corresponds to the end of the canalicular stage of lung development, after which the developing acinar structure becomes less sensitive to external perturbations (table 3) [64]. The degree of oligohydramnios is an additional

risk factor for pulmonary hypoplasia; lower volumes of residual fluid confer the highest risk [57,65,66]. In one study, the incidence of pulmonary hypoplasia with severe, moderate, and no to mild oligohydramnios was 43, 19, and 7 percent, respectively [65]. It is less clear whether there is an association between the length of the latency period and risk of pulmonary hypoplasia. However, oligohydramnios due to PPROM is associated with reduced latency [67]. (See "Prelabor rupture of membranes before and at the limit of viability".)

- **Oligohydramnios associated with uteroplacental disorders –** The prognosis of oligohydramnios associated with uteroplacental disorders (FGR, abruption, preeclampsia) largely depends upon the prognosis of the underlying disorder. (See "Fetal growth restriction: Evaluation" and "Acute placental abruption: Pathophysiology, clinical features, diagnosis, and consequences" and "Acute placental abruption: Management and long-term prognosis" and "Preeclampsia: Clinical features and diagnosis" and "Preeclampsia: Antepartum management and timing of delivery".)

- **Oligohydramnios after amniocentesis –** An exception to the poor prognosis described above is oligohydramnios related to second-trimester amniocentesis. In these cases, the membranes often "reseal," amniotic fluid reaccumulates, and pregnancy outcome is normal (see "Diagnostic amniocentesis", section on 'Leakage of amniotic fluid'). There are few reports of the occurrence of oligohydramnios after chorionic villus sampling; outcomes have been mixed [68,69]. (See "Chorionic villus sampling".)

Investigational therapies to achieve a long-term increase in AFV — Severe oligohydramnios in the second trimester is almost uniformly lethal due to pulmonary hypoplasia. The poor prognosis has led to investigation of interventions to restore AF that may allow pulmonary development, prevent death from postnatal pulmonary hypoplasia, and reduce the risk of orthopedic sequelae. Such interventions are most likely to be successful in pregnancies with idiopathic oligohydramnios or ruptured membranes. Fetuses with oliguria/anuria as the cause of oligohydramnios typically have a primary kidney disorder that would require postnatal dialysis and renal transplantation for survival or they have a lower urinary tract obstruction that compromises normal kidney function and thus might require similar postnatal intervention for survival.

- **Serial amnioinfusion or amnioport –** Transabdominal amnioinfusion performed serially percutaneously or serially or continuously through a port has been used with some success in research studies to improve fetal outcome in pregnancies with idiopathic oligohydramnios, early oligohydramnios due to PPROM, and oligohydramnios due to lower urinary tract obstruction or fetal renal disease [70-72]; however, significant postnatal medical, emotional, and financial implications exist [73].

Normal saline is infused to achieve a single deepest pocket (SDP) >2 cm. The procedure is discussed in more detail separately. (See "Renal agenesis: Prenatal diagnosis", section on 'Pregnancy management and parental counseling'.)

- **Vesicoamniotic shunt** – In fetuses with oligohydramnios due to lower urinary tract obstruction, fetal cystoscopy for diagnostic confirmation of posterior urethral valves followed by vesicoamniotic shunting has been used with some success to increase AFV and prevent adverse pulmonary, orthopedic, and renal sequelae. This is discussed in more detail separately. (See "Management of posterior urethral valves", section on 'Prenatal intervention'.)

- **Tissue 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. This is discussed in more detail separately. (See "Prelabor rupture of membranes before and at the limit of viability", section on 'Repair of leaks'.)

- **Therapeutic maternal hydration** – A meta-analysis (16 studies, 1121 pregnant participants) concluded that maternal hydration may be a useful long-term strategy to improve AFV in cases of idiopathic oligohydramnios and noted the absence of reported harms; however, the effect on perinatal outcomes could not be assessed [28]. The authors suggested oral intake of about 1500 mLs of hypotonic solutions daily, ideally for two weeks. There were many limitations to the available data, including inability to evaluate pregnancy, delivery, and neonatal outcome after the interventions; large differences in gestational age at diagnosis of oligohydramnios; different cut-offs for diagnosis; different hydration protocols (IV, oral, combination of IV and oral; different hydration volumes, hydration solutions, and durations of hydration); and different outcome measures and time intervals for their assessment.

- **Hydration and** sildenafil **citrate** – In a pilot trial of patients with idiopathic oligohydramnios diagnosed after 30 weeks of gestation, administration of sildenafil citrate 25 mg thrice daily and one liter of IV hydration followed by oral hydration significantly increased the AFI compared with IV and oral hydration alone [74]. The sildenafil group also delivered at a more advanced gestational age (38.3 versus 36.0 weeks of gestation), had a lower cesarean birth rate (28 versus 73 percent), and a lower rate of NICU admission (11 versus 41 percent). However, a multicenter Dutch trial (STRIDER) of sildenafil for treatment of poor prognosis early-onset growth restriction using the same dose [75] was halted early because of higher than expected rates of lung disease and death of newborns in the intervention group [76]. At the time the trial was closed, use of sildenafil did not result in any benefits.

The populations differed between the two trials (idiopathic oligohydramnios versus poor prognosis growth restriction), which might account for some of the differences in outcome. Although the idiopathic oligohydramnios trial demonstrated neonatal benefits in the sildenafil group versus the hydration-only group, we believe the STRIDER trial results are striking. Until more data are available about the postnatal effects of antenatal administration of sildenafil and the mechanism for the adverse postnatal effects is understood, the use of sildenafil in pregnancy should be restricted to carefully designed clinical trials monitored by data and safety monitoring boards.

Prenatal care

- Specific disorders associated with oligohydramnios are managed as appropriate for the disorder. (Refer to individual topic reviews on specific chromosomal and congenital anomalies, second-trimester PPROM, maternal medical disorders, preeclampsia, placental abruption, congenital anomalies related to the fetal kidney and urinary tract [CAKUT], renal agenesis, etc).

- We perform a nonstress test (NST) and evaluate the SDP (or AFI) once or twice weekly until birth, depending upon the maternal and fetal condition. Combined use of the NST and SDP (or AFI) is associated with a low rate of unexpected fetal death [77-79]. Performing a biophysical profile is a reasonable alternative.

- We obtain serial sonographic examinations every three to four weeks to monitor fetal growth. Doppler velocimetry is only used to monitor pregnancies with FGR. (See "Fetal growth restriction: Evaluation".)

Timing of delivery

- **Oligohydramnios of known etiology –** The indications for delivery in pregnancies with oligohydramnios attributable to a specific condition (eg, preeclampsia, PROM, FGR, congenital anomaly, postterm pregnancy, etc) are discussed separately in topic reviews on these disorders.

- **Idiopathic oligohydramnios –** In pregnancies with idiopathic oligohydramnios and reassuring fetal testing we suggest delivery at 36+0 to 37+6 weeks of gestation or at diagnosis if diagnosed later, in accord with the American College of Obstetricians and Gynecologists' guidelines [80]. Within this time window, we consider the clinical scenario (eg, diabetes status, past obstetric history, and patient preference) in timing the day of delivery, regardless of the Bishop score [80]. Although induction may increase the chances for mild morbidity from preterm birth, there is insufficient evidence to assure us that perinatal outcome with continuing conservative management to 39 weeks is comparable to that with earlier delivery, even in the presence of an appropriately grown, noncompromised fetus and absence of maternal

disease. Use of cervical ripening agents is an option for patients with an unfavorable cervix [81].

Alternatively, the patient can be followed with serial NSTs and biophysical profiles; however, we suggest delivery when the pregnancy is full term (39+0 weeks) in all cases, given minimal neonatal morbidity at this gestational age and the increased potential for maternal and perinatal morbidity with expectant management even in low-risk pregnancies [82].

The risks and benefits of these options should be discussed with the patient so they can make an informed decision. Nonreassuring fetal testing is a standard indication for delivery.

- **Evidence –** Timing of delivery in pregnancies complicated by idiopathic oligohydramnios is controversial [83]. 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 or expectant management [84]. Although no differences were found for any important maternal or neonatal outcome, the trial was underpowered to produce meaningful results.

  Observational studies have reported conflicting results. In one retrospective series, the perinatal mortality rate in structurally normal fetuses with oligohydramnios was significantly lower when delivery was initiated within 48 hours of diagnosis (in gestations of at least 28 weeks) compared with expectant management (18 versus 90 per 1000 births) [85]; this suggests that intervention is beneficial. 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, SDP <2 cm) in the third trimester (ie, appropriately grown nonanomalous fetus, reassuring fetal heart rate pattern, no maternal disease) when compared with controls with normal AFV [52,86-96]. Importantly, most studies initiated scheduled antenatal testing for these patients because of concerns that oligohydramnios may be an early sign of uteroplacental insufficiency; this testing with appropriate intervention when indicated may have prevented an adverse outcome [88]. It is also possible that the generally good outcomes were 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 FGR, preeclampsia, fetal abnormalities, postterm pregnancy, or abruption [3,97].

Route and site of birth — The route of birth should be determined by usual obstetric considerations. Although the chances of cord compression and cesarean birth are increased, vaginal birth is often successful [98].

The neonatal care level at the planned site of birth should be appropriate for the potential respiratory and renal needs of the newborn.

Intrapartum fetal monitoring — In patients with oligohydramnios, we recommend using continuous electronic fetal monitoring, given the increased risk for cord compression. However, most patients will have a normal tracing [99].

We consider transcervical amnioinfusion for patients with oligohydramnios and variable fetal heart rate decelerations in labor. (See "Intrapartum category I, II, and III fetal heart rate tracings: Management", section on 'Variable decelerations without loss of variability or accelerations' and "Amnioinfusion".)

SPECIAL POPULATIONS

First-trimester "oligohydramnios"

- **Etiology –** The etiology of first-trimester oligohydramnios is usually unclear. Reduced amniotic (coelomic) fluid prior to 10 weeks of gestation is rare because fluid in the gestational sac 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.

- **Diagnosis –** First-trimester diagnosis of oligohydramnios is rare. Criteria suggested for determining reduced AFV at this gestational age have included a difference between mean gestational sac size and crown-rump length that is less than 5 mm or a mean gestational sac diameter/crown-rump length ratio outside the normal range for gestational age [100-104].

- **Prognosis and counseling –** Reduced AFV in the first trimester is an ominous finding; pregnancy loss is the usual outcome. In one series, 15 of 16 patients (94 percent) with a normal fetal heart rate and small sac noted on first-trimester sonogram had a spontaneous pregnancy loss compared with only 4 of 52 control patients (8 percent) with normal sac size [100]. (See "Pregnancy loss (miscarriage): Ultrasound diagnosis".)

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

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Oligohydramnios and polyhydramnios".)

SUMMARY AND RECOMMENDATIONS

- **Terminology, pathogenesis, and diagnosis** (See 'Pathogenesis' above and 'Clinical manifestations and diagnosis' above.)
  - The term oligohydramnios refers to amniotic fluid volume (AFV) that is less than expected for gestational age.
  - The two most common mechanisms leading to oligohydramnios are (1) reduced urination due to fetal kidney disease or lower urinary tract obstruction and (2) AF loss due to rupture of membranes.
  - The diagnosis is based on ultrasound examination and may be described qualitatively or preferably quantitatively as amniotic fluid index (AFI) ≤5 cm or single deepest pocket (SDP) <2 cm.
- **Etiology –** Conditions commonly associated with oligohydramnios are listed in the table (table 1). The most likely etiologies vary according to the trimester in which it is diagnosed. The majority of cases present in the third trimester and have no identifiable cause. (See 'Etiology' above.)
- **Postdiagnostic evaluation –** Maternal history and physical examination (including evaluation for membrane rupture) and a detailed fetal sonographic evaluation are performed in all pregnancies with oligohydramnios. Use of additional tests (eg, fetal genetic testing) depends upon individual clinical circumstances. If oligohydramnios prevents adequate fetal assessment, transabdominal diagnostic amnioinfusion of approximately 200 mL of saline under ultrasound guidance can provide better visualization of fetal anatomy and thus improve diagnosis of the etiology. In selected patients, oral hydration or magnetic resonance imaging may be helpful to improve visualization of fetal anatomy. (See 'Postdiagnostic evaluation' above.)
- **Prognosis –** The fetal/neonatal prognosis is affected by the etiology, severity, gestational age at onset, and duration of oligohydramnios. Pregnancies with borderline/low normal AFV generally have a good prognosis and those with anhydramnios have the poorest prognosis, especially when it occurs in the second-

trimester. Adverse fetal/newborn outcomes in pregnancies with oligohydramnios may be related to umbilical cord compression, uteroplacental insufficiency, meconium aspiration, fetal deformation from prolonged crowding, or, in the second trimester, pulmonary hypoplasia. There is no effective long-term treatment of oligohydramnios. (See 'Prognosis and counseling by etiology' above and 'Investigational therapies to achieve a long-term increase in AFV' above.)

- **Prenatal care** – Specific pregnancy complications associated with oligohydramnios are managed as appropriate for the condition. In idiopathic cases, we perform a nonstress test (NST) and SDP (or AFI) once or twice weekly until birth, depending upon the maternal and fetal condition. Performing a biophysical profile is a reasonable alternative. We also obtain serial sonographic examinations every three to four weeks to monitor fetal growth. Doppler velocimetry is only used to monitor pregnancies with fetal growth restriction (FGR). (See 'Prenatal care' above.)

- **Delivery timing** – For patients with idiopathic oligohydramnios, we suggest delivery at 36+0 to 37+6 weeks of gestation (or upon diagnosis if diagnosed later) rather than expectant management (**Grade 2C**). Available evidence is insufficient to assure us that perinatal outcome of continuing conservative management until 39 weeks is comparable to that with earlier delivery. Timing and indications for delivery in pregnancies with oligohydramnios attributable to a specific condition depend on the specific condition. (See 'Timing of delivery' above.)

REFERENCES

1. Locatelli A, Zagarella A, Toso L, et al. Serial assessment of amniotic fluid index in uncomplicated term pregnancies: prognostic value of amniotic fluid reduction. J Matern Fetal Neonatal Med 2004; 15:233.
2. Hou L, Wang X, Hellerstein S, et al. Delivery mode and perinatal outcomes after diagnosis of oligohydramnios at term in China. J Matern Fetal Neonatal Med 2020; 33:2408.
3. Morris JM, Thompson K, Smithey J, et al. The usefulness of ultrasound assessment of amniotic fluid in predicting adverse outcome in prolonged pregnancy: a prospective blinded observational study. BJOG 2003; 110:989.
4. Murzakanova G, Räisänen S, Jacobsen AF, et al. Adverse perinatal outcomes in 665,244 term and post-term deliveries-a Norwegian population-based study. Eur J Obstet Gynecol Reprod Biol 2020; 247:212.

5. Zhu XQ, Jiang SS, Zhu XJ, et al. Expression of aquaporin 1 and aquaporin 3 in fetal membranes and placenta in human term pregnancies with oligohydramnios. Placenta 2009; 30:670.
6. Shao H, Gao S, Ying X, et al. Expression and Regulation of Aquaporins in Pregnancy Complications and Reproductive Dysfunctions. DNA Cell Biol 2021; 40:116.
7. Cheung CY, Brace RA. Altered proteomics profile in the amnion of patients with oligohydramnios. Physiol Rep 2020; 8:e14381.
8. Shipp TD, Bromley B, Pauker S, et al. Outcome of singleton pregnancies with severe oligohydramnios in the second and third trimesters. Ultrasound Obstet Gynecol 1996; 7:108.
9. Kishore J, Misra R, Paisal A, Pradeep Y. Adverse reproductive outcome induced by Parvovirus B19 and TORCH infections in women with high-risk pregnancy. J Infect Dev Ctries 2011; 5:868.
10. Abdel-Fattah SA, Bhat A, Illanes S, et al. TORCH test for fetal medicine indications: only CMV is necessary in the United Kingdom. Prenat Diagn 2005; 25:1028.
11. Clement D, Schifrin BS, Kates RB. Acute oligohydramnios in postdate pregnancy. Am J Obstet Gynecol 1987; 157:884.
12. Zilberman Sharon N, Pekar-Zlotin M, Kugler N, et al. Oligohydramnios: how severe is severe? J Matern Fetal Neonatal Med 2022; 35:5754.
13. Magann EF, Perry KG Jr, Chauhan SP, et al. The accuracy of ultrasound evaluation of amniotic fluid volume in singleton pregnancies: the effect of operator experience and ultrasound interpretative technique. J Clin Ultrasound 1997; 25:249.
14. Hughes DS, Magann EF, Whittington JR, et al. Accuracy of the Ultrasound Estimate of the Amniotic Fluid Volume (Amniotic Fluid Index and Single Deepest Pocket) to Identify Actual Low, Normal, and High Amniotic Fluid Volumes as Determined by Quantile Regression. J Ultrasound Med 2020; 39:373.
15. Sekhon S, Rosenbloom JI, Doering M, et al. Diagnostic utility of maximum vertical pocket versus amniotic fluid index in assessing amniotic fluid volume for the prediction of adverse maternal and fetal outcomes: a systematic review and meta-analysis. J Matern Fetal Neonatal Med 2021; 34:3730.
16. Hughes D, Simmons B, Magann E, et al. Amniotic Fluid Volume Estimation from 20 Weeks to 28 Weeks. Do You Measure Perpendicular to the Floor or Perpendicular to the Uterine Contour? Int J Womens Health 2021; 13:1139.
17. Hughes DS, Whittington JR, Kim H, et al. Is There a Difference in Sonographic Estimation of Amniotic Fluid Volume When Measuring With the Probe Perpendicular to the Floor Compared With Perpendicular to the Uterine Contour? J Obstet Gynaecol Can 2019; 41:1295.

18. Singer A, Maya I, Sukenik-Halevy R, et al. Microarray findings in pregnancies with oligohydramnios - a retrospective cohort study and literature review. J Perinat Med 2019; 48:53.
19. Lin SY, Chuang GT, Hung CH, et al. Rapid Trio Exome Sequencing for Autosomal Recessive Renal Tubular Dysgenesis in Recurrent Oligohydramnios. Front Genet 2021; 12:606970.
20. Pryde PG, Hallak M, Lauria MR, et al. Severe oligohydramnios with intact membranes: an indication for diagnostic amnioinfusion. Fetal Diagn Ther 2000; 15:46.
21. Fisk NM, Ronderos-Dumit D, Soliani A, et al. Diagnostic and therapeutic transabdominal amnioinfusion in oligohydramnios. Obstet Gynecol 1991; 78:270.
22. Vikraman SK, Chandra V, Balakrishnan B, et al. Impact of antepartum diagnostic amnioinfusion on targeted ultrasound imaging of pregnancies presenting with severe oligo- and anhydramnios: An analysis of 61 cases. Eur J Obstet Gynecol Reprod Biol 2017; 212:96.
23. Ahmed B. Amnioinfusion in severe oligohydramnios with intact membrane: an observational study. J Matern Fetal Neonatal Med 2022; 35:6518.
24. Tocchio S, Kline-Fath B, Kanal E, et al. MRI evaluation and safety in the developing brain. Semin Perinatol 2015; 39:73.
25. Cannie MM, De Keyzer F, Van Laere S, et al. Potential Heating Effect in the Gravid Uterus by Using 3-T MR Imaging Protocols: Experimental Study in Miniature Pigs. Radiology 2016; 279:754.
26. Ross MG, Nijland MJ, Kullama LK. 1-Deamino-[8-D-arginine] vasopressin-induced maternal plasma hypoosmolality increases ovine amniotic fluid volume. Am J Obstet Gynecol 1996; 174:1118.
27. Nijland MJ, Ross MG, Kullama LK, et al. DDAVP-induced maternal hyposmolality increases ovine fetal urine flow. Am J Physiol 1995; 268:R358.
28. Gizzo S, Noventa M, Vitagliano A, et al. An Update on Maternal Hydration Strategies for Amniotic Fluid Improvement in Isolated Oligohydramnios and Normohydramnios: Evidence from a Systematic Review of Literature and Meta-Analysis. PLoS One 2015; 10:e0144334.
29. Flack NJ, Sepulveda W, Bower S, Fisk NM. Acute maternal hydration in third-trimester oligohydramnios: effects on amniotic fluid volume, uteroplacental perfusion, and fetal blood flow and urine output. Am J Obstet Gynecol 1995; 173:1186.
30. Selam B, Koksal R, Ozcan T. Fetal arterial and venous Doppler parameters in the interpretation of oligohydramnios in postterm pregnancies. Ultrasound Obstet Gynecol 2000; 15:403.

31. Bar-Hava I, Divon MY, Sardo M, Barnhard Y. Is oligohydramnios in postterm pregnancy associated with redistribution of fetal blood flow? Am J Obstet Gynecol 1995; 173:519.
32. Budunoglu MD, Yapca OE, Yldildiz GA, Atakan Al R. Fetal renal blood flow velocimetry and cerebro-placental ratio in patients with isolated oligohydramnios. J Gynecol Obstet Hum Reprod 2019; 48:495.
33. Dyer SN, Burton BK, Nelson LH. Elevated maternal serum alpha-fetoprotein levels and oligohydramnios: poor prognosis for pregnancy outcome. Am J Obstet Gynecol 1987; 157:336.
34. Richards DS, Seeds JW, Katz VL, et al. Elevated maternal serum alpha-fetoprotein with oligohydramnios: ultrasound evaluation and outcome. Obstet Gynecol 1988; 72:337.
35. Koontz WL, Seeds JW, Adams NJ, et al. Elevated maternal serum alpha-fetoprotein, second-trimester oligohydramnios, and pregnancy outcome. Obstet Gynecol 1983; 62:301.
36. Los FJ, Hagenaars AM, Cohen-Overbeek TE, Quartero HW. Maternal serum markers in second-trimester oligohydramnios. Prenat Diagn 1994; 14:565.
37. Los FJ, Beekhuis JR, Marrink J, et al. Origin of raised maternal serum alpha-fetoprotein levels in second-trimester oligohydramnios. Prenat Diagn 1992; 12:39.
38. Peipert JF, Donnenfeld AE. Oligohydramnios: a review. Obstet Gynecol Surv 1991; 46:325.
39. Vink J, Hickey K, Ghidini A, et al. Earlier gestational age at ultrasound evaluation predicts adverse neonatal outcomes in the preterm appropriate-for-gestational-age fetus with idiopathic oligohydramnios. Am J Perinatol 2009; 26:21.
40. Ulkumen BA, Pala HG, Baytur YB, Koyuncu FM. Outcomes and management strategies in pregnancies with early onset oligohydramnios. Clin Exp Obstet Gynecol 2015; 42:355.
41. Mercer LJ, Brown LG, Petres RE, Messer RH. A survey of pregnancies complicated by decreased amniotic fluid. Am J Obstet Gynecol 1984; 149:355.
42. Chamberlain PF, Manning FA, Morrison I, et al. Ultrasound evaluation of amniotic fluid volume. I. The relationship of marginal and decreased amniotic fluid volumes to perinatal outcome. Am J Obstet Gynecol 1984; 150:245.
43. Petrozella LN, Dashe JS, McIntire DD, Leveno KJ. Clinical significance of borderline amniotic fluid index and oligohydramnios in preterm pregnancy. Obstet Gynecol 2011; 117:338.
44. Loos S, Kemper MJ. Causes of renal oligohydramnios: impact on prenatal counseling and postnatal outcome. Pediatr Nephrol 2018; 33:541.
45. Mehler K, Gottschalk I, Burgmaier K, et al. Prenatal parental decision-making and postnatal outcome in renal oligohydramnios. Pediatr Nephrol 2018; 33:651.

46. Miremberg H, Grinstein E, Herman HG, et al. The association between isolated oligohydramnios at term and placental pathology in correlation with pregnancy outcomes. Placenta 2020; 90:37.
47. Rabie N, Magann E, Steelman S, Ounpraseuth S. Oligohydramnios in complicated and uncomplicated pregnancy: a systematic review and meta-analysis. Ultrasound Obstet Gynecol 2017; 49:442.
48. Figueroa L, McClure EM, Swanson J, et al. Oligohydramnios: a prospective study of fetal, neonatal and maternal outcomes in low-middle income countries. Reprod Health 2020; 17:19.
49. Dorot A, Wainstock T, Sheiner E, et al. Isolated oligohydramnios and long-term neurological morbidity of the offspring. J Dev Orig Health Dis 2020; 11:648.
50. Sarno AP Jr, Ahn MO, Phelan JP. Intrapartum amniotic fluid volume at term. Association of ruptured membranes, oligohydramnios and increased fetal risk. J Reprod Med 1990; 35:719.
51. Chauhan SP, Sanderson M, Hendrix NW, et al. Perinatal outcome and amniotic fluid index in the antepartum and intrapartum periods: A meta-analysis. Am J Obstet Gynecol 1999; 181:1473.
52. Alchalabi HA, Obeidat BR, Jallad MF, Khader YS. Induction of labor and perinatal outcome: the impact of the amniotic fluid index. Eur J Obstet Gynecol Reprod Biol 2006; 129:124.
53. Shrem G, Nagawkar SS, Hallak M, Walfisch A. Isolated Oligohydramnios at Term as an Indication for Labor Induction: A Systematic Review and Meta-Analysis. Fetal Diagn Ther 2016; 40:161.
54. Moore TR. Superiority of the four-quadrant sum over the single-deepest-pocket technique in ultrasonographic identification of abnormal amniotic fluid volumes. Am J Obstet Gynecol 1990; 163:762.
55. Mercer LJ, Brown LG. Fetal outcome with oligohydramnios in the second trimester. Obstet Gynecol 1986; 67:840.
56. Moretti M, Sibai BM. Maternal and perinatal outcome of expectant management of premature rupture of membranes in the midtrimester. Am J Obstet Gynecol 1988; 159:390.
57. Shumway JB, Al-Malt A, Amon E, et al. Impact of oligohydramnios on maternal and perinatal outcomes of spontaneous premature rupture of the membranes at 18-28 weeks. J Matern Fetal Med 1999; 8:20.
58. Falk SJ, Campbell LJ, Lee-Parritz A, et al. Expectant management in spontaneous preterm premature rupture of membranes between 14 and 24 weeks' gestation. J Perinatol 2004; 24:611.

59. Nimrod C, Varela-Gittings F, Machin G, et al. The effect of very prolonged membrane rupture on fetal development. Am J Obstet Gynecol 1984; 148:540.

60. Blott M, Greenough A. Neonatal outcome after prolonged rupture of the membranes starting in the second trimester. Arch Dis Child 1988; 63:1146.

61. Lauria MR, Gonik B, Romero R. Pulmonary hypoplasia: pathogenesis, diagnosis, and antenatal prediction. Obstet Gynecol 1995; 86:466.

62. Farooqi A, Holmgren PA, Engberg S, Serenius F. Survival and 2-year outcome with expectant management of second-trimester rupture of membranes. Obstet Gynecol 1998; 92:895.

63. Rotschild A, Ling EW, Puterman ML, Farquharson D. Neonatal outcome after prolonged preterm rupture of the membranes. Am J Obstet Gynecol 1990; 162:46.

64. Glasser SW, Korfhagen TR, Wert SE, Whitsett JA. Transgenic models for study of pulmonary development and disease. Am J Physiol 1994; 267:L489.

65. Kilbride HW, Yeast J, Thibeault DW. Defining limits of survival: lethal pulmonary hypoplasia after midtrimester premature rupture of membranes. Am J Obstet Gynecol 1996; 175:675.

66. Vergani P, Ghidini A, Locatelli A, et al. Risk factors for pulmonary hypoplasia in second-trimester premature rupture of membranes. Am J Obstet Gynecol 1994; 170:1359.

67. Point F, Ghesquiere L, Drumez E, et al. Risk factors associated with shortened latency before delivery in outpatients managed for preterm prelabor rupture of membranes. Acta Obstet Gynecol Scand 2022; 101:119.

68. Bronshtein M, Blumenfeld Z. First- and early second-trimester oligohydramnios-a predictor of poor fetal outcome except in iatrogenic oligohydramnios post chorionic villus biopsy. Ultrasound Obstet Gynecol 1991; 1:245.

69. Cheng EY, Luthy DA, Hickok DE, et al. Transcervical chorionic villus sampling and midtrimester oligohydramnios. Am J Obstet Gynecol 1991; 165:1063.

70. Turhan NO, Atacan N. Antepartum prophylactic transabdominal amnioinfusion in preterm pregnancies complicated by oligohydramnios. Int J Gynaecol Obstet 2002; 76:15.

71. Locatelli A, Ghidini A, Verderio M, et al. Predictors of perinatal survival in a cohort of pregnancies with severe oligohydramnios due to premature rupture of membranes at <26 weeks managed with serial amnioinfusions. Eur J Obstet Gynecol Reprod Biol 2006; 128:97.

72. Polzin WJ, Lim FY, Habli M, et al. Use of an Amnioport to Maintain Amniotic Fluid Volume in Fetuses with Oligohydramnios Secondary to Lower Urinary Tract Obstruction or Fetal Renal Anomalies. Fetal Diagn Ther 2017; 41:51.

73. Soffer OD, Mauch TJ, Muff-Luett MA. Ethical Concerns for Amnioinfusions to Treat Early-Onset Anhydramnios. JAMA 2023; 329:1913.
74. Maher MA, Sayyed TM, Elkhouly N. Sildenafil Citrate Therapy for Oligohydramnios: A Randomized Controlled Trial. Obstet Gynecol 2017; 129:615.
75. https://clinicaltrials.gov/ct2/show/NCT02277132 (Accessed on July 24, 2018).
76. Hawkes N. Trial of Viagra for fetal growth restriction is halted after baby deaths. BMJ 2018; 362:k3247.
77. Clark SL, Sabey P, Jolley K. Nonstress testing with acoustic stimulation and amniotic fluid volume assessment: 5973 tests without unexpected fetal death. Am J Obstet Gynecol 1989; 160:694.
78. Nageotte MP, Towers CV, Asrat T, Freeman RK. Perinatal outcome with the modified biophysical profile. Am J Obstet Gynecol 1994; 170:1672.
79. Miller DA, Rabello YA, Paul RH. The modified biophysical profile: antepartum testing in the 1990s. Am J Obstet Gynecol 1996; 174:812.
80. American College of Obstetricians and Gynecologists' Committee on Obstetric Practice, Society for Maternal-Fetal Medicine. Medically Indicated Late-Preterm and Early-Term Deliveries: ACOG Committee Opinion, Number 831. Obstet Gynecol 2021; 138:e35.
81. Krispin E, Netser T, Wertheimer A, et al. Induction of labor methods in isolated term oligohydramnios. Arch Gynecol Obstet 2019; 299:765.
82. Grobman WA, Rice MM, Reddy UM, et al. Labor Induction versus Expectant Management in Low-Risk Nulliparous Women. N Engl J Med 2018; 379:513.
83. Bannerman CG, Chauhan SP. Oligohydramnios at 34-36 weeks: observe or deliver. Am J Obstet Gynecol 2011; 205:163.
84. Ek S, Andersson A, Johansson A, Kublicas M. Oligohydramnios in uncomplicated pregnancies beyond 40 completed weeks. A prospective, randomised, pilot study on maternal and neonatal outcomes. Fetal Diagn Ther 2005; 20:182.
85. Bastide A, Manning F, Harman C, et al. Ultrasound evaluation of amniotic fluid: outcome of pregnancies with severe oligohydramnios. Am J Obstet Gynecol 1986; 154:895.
86. Zhang J, Troendle J, Meikle S, et al. Isolated oligohydramnios is not associated with adverse perinatal outcomes. BJOG 2004; 111:220.
87. Danon D, Ben-Haroush A, Yogev Y, et al. Prostaglandin E2 induction of labor for isolated oligohydramnios in women with unfavorable cervix at term. Fetal Diagn Ther 2007; 22:75.
88. 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.

89. Sherer DM. A review of amniotic fluid dynamics and the enigma of isolated oligohydramnios. Am J Perinatol 2002; 19:253.
90. Conway DL, Adkins WB, Schroeder B, Langer O. Isolated oligohydramnios in the term pregnancy: is it a clinical entity? J Matern Fetal Med 1998; 7:197.
91. Rainford M, Adair R, Scialli AR, et al. Amniotic fluid index in the uncomplicated term pregnancy. Prediction of outcome. J Reprod Med 2001; 46:589.
92. Magann EF, Kinsella MJ, Chauhan SP, et al. Does an amniotic fluid index of </=5 cm necessitate delivery in high-risk pregnancies? A case-control study. Am J Obstet Gynecol 1999; 180:1354.
93. Magann EF, Doherty DA, Field K, et al. Biophysical profile with amniotic fluid volume assessments. Obstet Gynecol 2004; 104:5.
94. Kreiser D, el-Sayed YY, Sorem KA, et al. Decreased amniotic fluid index in low-risk pregnancy. J Reprod Med 2001; 46:743.
95. Elsandabesee D, Majumdar S, Sinha S. Obstetricians' attitudes towards 'isolated' oligohydramnios at term. J Obstet Gynaecol 2007; 27:574.
96. Manzanares S, Carrillo MP, González-Perán E, et al. Isolated oligohydramnios in term pregnancy as an indication for induction of labor. J Matern Fetal Neonatal Med 2007; 20:221.
97. Magann EF, Chauhan SP, Kinsella MJ, et al. Antenatal testing among 1001 patients at high risk: the role of ultrasonographic estimate of amniotic fluid volume. Am J Obstet Gynecol 1999; 180:1330.
98. Tahmina S, Prakash S, Daniel M. Maternal and perinatal outcomes of induction of labor in oligohydramnios at term-a retrospective cohort study. J Matern Fetal Neonatal Med 2020; 33:2190.
99. Rhoades JS, Stout MJ, Macones GA, Cahill AG. Effect of Oligohydramnios on Fetal Heart Rate Patterns during Term Labor Induction. Am J Perinatol 2019; 36:715.
100. Bromley B, Harlow BL, Laboda LA, Benacerraf BR. Small sac size in the first trimester: a predictor of poor fetal outcome. Radiology 1991; 178:375.
101. Tadmor OP, Achiron R, Rabinowiz R, et al. Predicting first-trimester spontaneous abortion. Ratio of mean sac diameter to crown-rump length compared to embryonic heart rate. J Reprod Med 1994; 39:459.
102. Nazari A, Check JH, Epstein RH, et al. Relationship of small-for-dates sac size to crown-rump length and spontaneous abortion in patients with a known date of ovulation. Obstet Gynecol 1991; 78:369.
103. Dickey RP, Olar TT, Taylor SN, et al. Relationship of small gestational sac-crown-rump length differences to abortion and abortus karyotypes. Obstet Gynecol 1992; 79:554.

104. Rowling SE, Coleman BG, Langer JE, et al. First-trimester US parameters of failed pregnancy. Radiology 1997; 203:211.

Topic 6777 Version 45.0

GRAPHICS

Causes of oligohydramnios

Maternal
Medical or obstetric conditions associated with uteroplacental insufficiency (eg, preeclampsia, chronic hypertension, collagen vascular disease, nephropathy, thrombophilia)
Medications (eg, angiotensin converting enzyme inhibitors, prostaglandin synthetase inhibitors [eg, NSAIDs], trastuzumab)
Diabetes insipidus (rare cases have presented with oligohydramnios that improve upon maternal treatment with desmopressin)
Placental
Abruption
Twin to twin transfusion (ie, twin polyhydramnios-oligohydramnios sequence)
Placental thrombosis or infarction
Fetal
Chromosomal abnormalities
Congenital abnormalities, especially those associated with impaired urine production or excretion
Growth restriction
Demise
Postterm pregnancy
Ruptured fetal membranes
Infection
Idiopathic

NSAIDs: nonsteroidal anti-inflammatory drugs.

Graphic 79447 Version 7.0

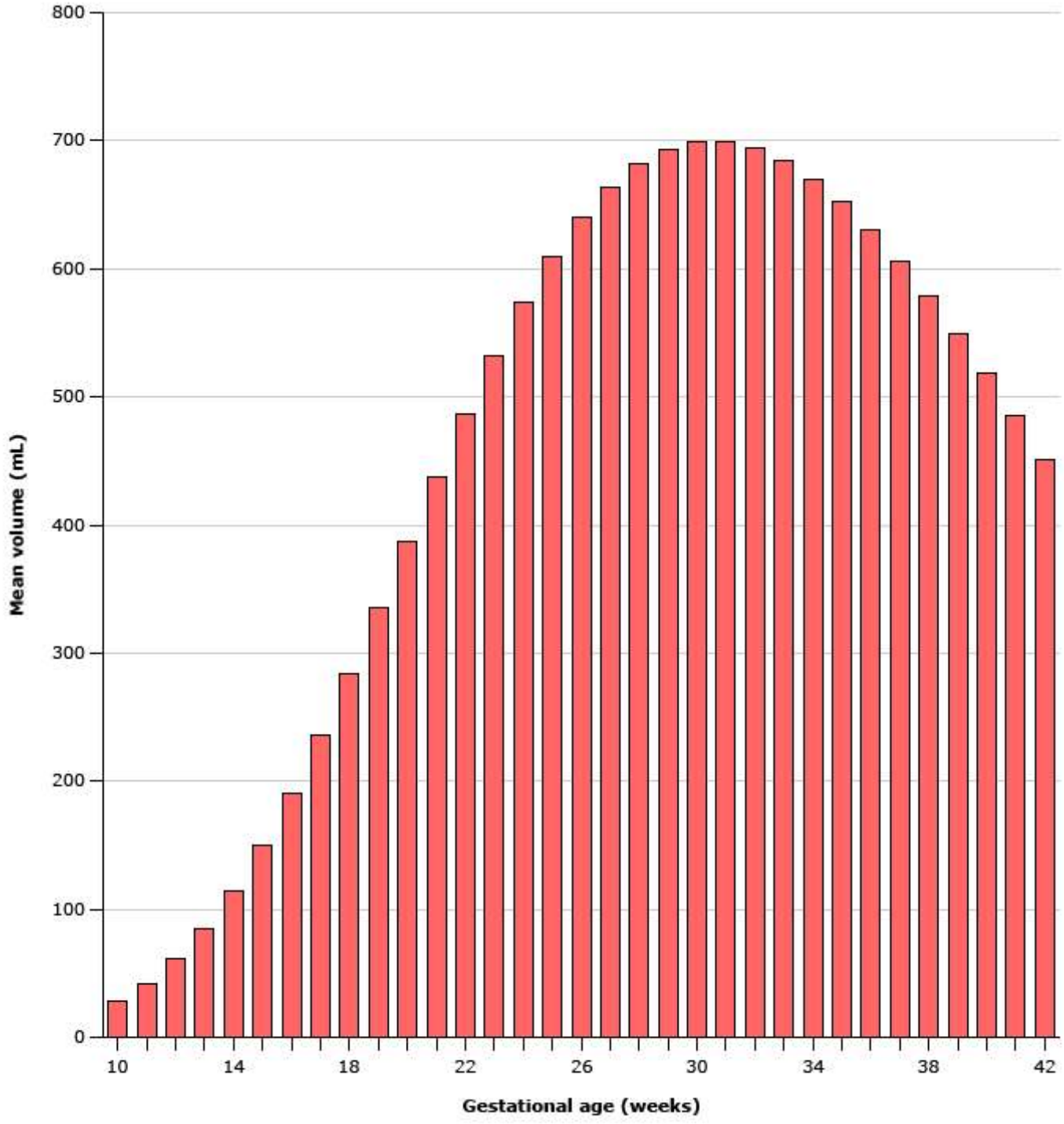
Type and frequency of congenital anomalies associated with oligohydramnios in a literature review

Type	Number of cases (percent)
Renal	94 (65)
Multiple	17 (12)
Aneuploidy	12 (8)
Central nervous system	7 (5)
Skeletal system	5 (4)
Cardiovascular system	4 (3)
Other	6 (4)
Total	145

Adapted from: Hill LM. Clin Obstet Gynecol 1997; 40:314.

Graphic 78227 Version 3.0

Mean amniotic fluid volume across normal pregnancy



These values represent the 50th percentile. There is considerable variability around the mean. The 5th, 50th, and 95th percentiles at 30 weeks of gestation are approximately 260, 700, and 1900 mL, respectively.

Data from: Ounpraseuth ST, Magann EF, Spencer HJ, Rabie NZ, Sandlin AT Normal amniotic fluid volume across gestation: Comparison of statistical approaches in 1190 normal amniotic fluid volumes. J Obstet Gynaecol Res; 2017: 43:1122.

Graphic 59704 Version 9.0

Phases of lung development

Gestational age, weeks	Stage of lung development	Description
0 to 5	Embryonic	Lung bud arises
6 to 17	Pseudoglandular	Nonrespiratory bronchi and bronchioles develop
17 to 24	Canalicular	First gas exchanging acini and pulmonary capillaries are forming
25 to 37	Terminal sac	Subsaccules and alveoli develop with extensive capillary invasion and expansion of the alveolar-blood barrier surface area
38 to age 3 years	Alveolar	Subsaccules become alveoli

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Conflict of interest policy

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