

CHAPTER 11: Amnionic Fluid

It is generally agreed that amniotic fluid represents in great part a transudation from the maternal vessels, but many authorities consider that a portion of it is derived from urinary secretion of the foetus.

—J. Whitridge Williams (1903)

INTRODUCTION

At the time Williams wrote this, the fetal kidney was thought by many to be nonfunctional. Since that time, however, much has been learned of this complex multifunctional *liquor amnii*. Amnionic fluid serves several roles during pregnancy. Fetal breathing of amnionic fluid is essential for normal lung growth, and fetal swallowing permits gastrointestinal (GI) tract development. Amnionic fluid also creates a physical space for fetal movement, which is necessary for neuromusculoskeletal maturation. It further guards against umbilical cord compression and protects the fetus from trauma. Amnionic fluid even has bacteriostatic properties. Abnormalities of volume may result from fetal or placental pathology—indicating a problem with fluid production or its circulation. These volume extremes may be associated with increased risks for adverse pregnancy outcome.

NORMAL AMNIONIC FLUID VOLUME

Amnionic fluid volume increases from approximately 30 mL at 10 weeks to 200 mL by 16 weeks and reaches 800 mL by the mid-third trimester (Brace, 1989; Magann, 1997). The fluid is approximately 98-percent water. A full-term fetus contains roughly 2800 mL of water and the placenta another 400 mL, such that the term uterus holds nearly 4 liters of water (Modena, 2004). Abnormally decreased fluid volume is termed *oligohydramnios*, whereas abnormally increased fluid volume is termed *hydramnios* or *polyhydramnios*.

Physiology

Early in pregnancy, the amnionic cavity is filled with fluid that is similar in composition to extracellular fluid. During the first half of pregnancy, transfer of water and other small molecules takes place across the amnion—*transmembranous flow*; across the fetal vessels on placental surface—*intramembranous flow*; and *transcutaneous flow*—across fetal skin. Fetal urine production begins between 8 and 11 weeks' gestation, but it does not become a major component of amnionic fluid until the second trimester, which explains why fetuses with lethal renal abnormalities may not manifest severe oligohydramnios until after 18 weeks. Water transport across the fetal skin continues until keratinization occurs at 22 to 25 weeks. This explains why extremely preterm neonates can experience significant fluid loss across their skin.

With advancing gestation, four pathways play a major role in amnionic fluid volume regulation (Table 11-1). First, fetal urination is the primary source of amnionic fluid in the second half of pregnancy. By term, fetal urine production may exceed 1 liter per day, and the entire amnionic fluid volume is recirculated on a daily basis. Fetal urine osmolality is similar to that of amnionic fluid and significantly hypotonic to that of maternal and fetal plasma. Specifically, the osmolality of maternal and fetal plasma approximates 280 mOsm/mL, whereas that of amnionic fluid is about 260 mOsm/L. The hypotonicity of amnionic fluid accounts for significant *intramembranous* fluid transfer across and into fetal vessels on the placental surface. This transfer reaches 400 mL per day and is a second regulator of fluid volume (Mann, 1996). In the setting of maternal dehydration, the resultant increase in maternal osmolality favors fluid transfer from the fetus to the mother, and then from the amnionic fluid compartment into the fetus (Moore, 2010).

TABLE 11-1

Amniotic Fluid Volume Regulation in Late Pregnancy

Pathway	Effect on Volume	Approximate Daily Volume (mL)
Fetal urination	Production	1000
Fetal swallowing	Resorption	750
Fetal lung fluid secretion	Production	350
Intramembranous flow across fetal vessels on the placental surface	Resorption	400
Transmembranous flow across amniotic membrane	Resorption	Minimal

Data from [Magann, 2011](#); [Modena, 2004](#); [Moore, 2010](#).

An important third source of amniotic fluid regulation is the respiratory tract. Approximately 350 mL of lung fluid is produced daily late in gestation, and half of this is immediately swallowed. Last, fetal swallowing is the primary mechanism for amniotic fluid resorption and averages 500 to 1000 mL per day ([Mann, 1996](#)). Impaired swallowing, secondary to either a central nervous system abnormality or GI tract obstruction, can result in an impressive degree of hydramnios. The remaining pathways are transmembranous and transcutaneous flow, which together account for a far smaller proportion of fluid transport in the second half of pregnancy.

Measurement

From a practical standpoint, the actual volume of amniotic fluid is rarely measured outside of the research setting. That said, direct measurement and dye-dilution methods of fluid quantification have contributed to our understanding of normal physiology. These measurements have further been used to validate sonographic fluid assessment techniques. Dye dilution involves injecting a small quantity of a dye such as aminohippurate into the amniotic cavity under sonographic guidance and then sampling the amniotic fluid to determine the dye concentration and hence to calculate the volume.

[Brace and Wolf \(1989\)](#) reviewed 12 studies done through the 1960s in which amniotic fluid volume was assessed using these measurement techniques. Although fluid volume increased across gestation, they found that the mean value did not change significantly between 22 and 39 weeks—it was approximately 750 mL. There was considerable variation at each week of gestation, especially in the mid-third trimester, when the 5th percentile was 300 mL and the 95th percentile nearly 2000 mL. In contrast, [Magann and colleagues \(1997\)](#), using dye-dilution measurements, found that amniotic fluid volume rose with advancing gestation. Specifically, the average fluid volume was approximately 400 mL between 22 and 30 weeks, doubling thereafter to a mean of 800 mL. The volume remained at this level until 40 weeks and then declined by approximately 8 percent per week. The two reports differed in the regression methodology employed, and despite different conclusions, both identified a wide normal range, particularly in the third trimester. This normal variation is similarly observed sonographically.

Sonographic Assessment

Amniotic fluid volume evaluation is a component of every standard sonogram performed in the second or third trimester ([Chap. 10, Amniotic Fluid Volume](#)). It may be measured using either of two semi-quantitative techniques, the single deepest pocket of fluid or the amniotic fluid index (AFI), which was described by [Phelan and associates \(1987\)](#). Both measurements are reproducible and, in the setting of a fluid abnormality, can be followed serially over time to assess trends and to aid communication among providers. For these reasons, semiquantitative assessment of amniotic fluid is preferred to qualitative or subjective estimation ([American College of Obstetricians and Gynecologists, 2016](#)). Using either technique, a fluid pocket must be at least 1 cm in width to be considered adequate. Fetal parts or loops of umbilical cord may be visible in the pocket, but they are not included in the measurement. Color Doppler is generally used to verify that umbilical cord is not within the measurement.

Single Deepest Pocket

This is also called the largest or *maximal vertical pocket* of amniotic fluid. The ultrasound transducer is held perpendicular to the floor and parallel to the long axis of the woman. Then, while scanning in the sagittal plane, the largest vertical pocket of fluid is identified and measured. *The single deepest pocket measurement is considered normal if above 2 cm and less than 8 cm, with values below and above this range indicating oligohydramnios and hydramnios, respectively.* These thresholds are based on data from Chamberlain and associates (1984) and correspond to the 3rd and 97th percentiles. When evaluating twin pregnancies and other multifetal gestations, a single deepest pocket of amniotic fluid is assessed in each gestational sac, again using a normal range of more than 2 cm to less than 8 cm (Hernandez, 2012; Society for Maternal-Fetal Medicine, 2013). The fetal biophysical profile similarly uses a single deepest vertical pocket threshold of more than 2 cm to indicate normal amniotic fluid volume. This is discussed further in Chapter 17 (Acoustic Stimulation Tests).

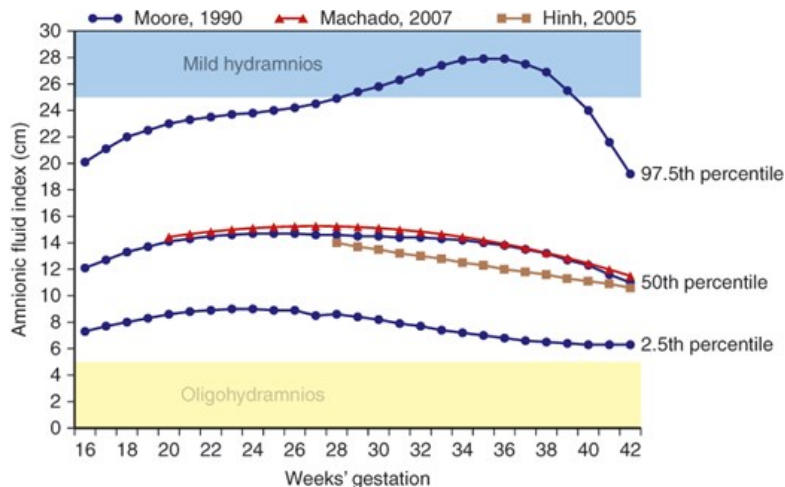
Amniotic Fluid Index

As with the single deepest fluid pocket measurement, the ultrasound transducer is held perpendicular to the floor and parallel to the long axis of the woman. The uterus is divided into four equal quadrants—the right and left upper and lower quadrants, respectively. The AFI is the sum of the single deepest pocket from each quadrant. The intraobserver variability of the AFI approximates 1 cm, and the interobserver variability is about 2 cm. Variations are larger when fluid volumes are above the normal range (Moore, 1990; Rutherford, 1987). A useful guideline is that the AFI approximates three times the single deepest pocket of fluid (Hill, 2003).

Determination of whether the AFI is normal may be based on either a static numerical threshold or a gestational age-specific percentile reference range. The AFI is generally considered normal if greater than 5 cm and below 24 or 25 cm. Values outside these ranges indicate oligohydramnios and hydramnios, respectively. The upper threshold of 24 cm is used in consensus documents (American College of Obstetricians and Gynecologists, 2016; Reddy, 2014). The 25-cm threshold is often applied in research studies (Khan, 2017; Luo, 2017; Pri-Paz, 2012). Moore and Cayle (1990) have provided normal curves for AFI values based on a cross-sectional evaluation of nearly 800 uncomplicated pregnancies. The mean AFI was found to be between 12 and 15 cm from 16 weeks until 40 weeks' gestation. Other investigators have published nomograms with similar mean values (Hinh, 2005; Machado, 2007). Figure 11-1 depicts these AFI nomogram reference values in relation to commonly used thresholds for hydramnios and oligohydramnios.

FIGURE 11-1

Amniotic fluid index (AFI) according to gestational-age-specific and threshold values. The blue curves represent the 2.5th, 50th, and 97.5th AFI percentile values, based on the nomogram by Moore (1990). Red and tan curves represent 50th percentile values for AFI from Machado (2007) and from Hinh and Ladinsky (2005), respectively. The light blue and yellow shaded bars indicate threshold values used to define hydramnios and oligohydramnios, respectively.



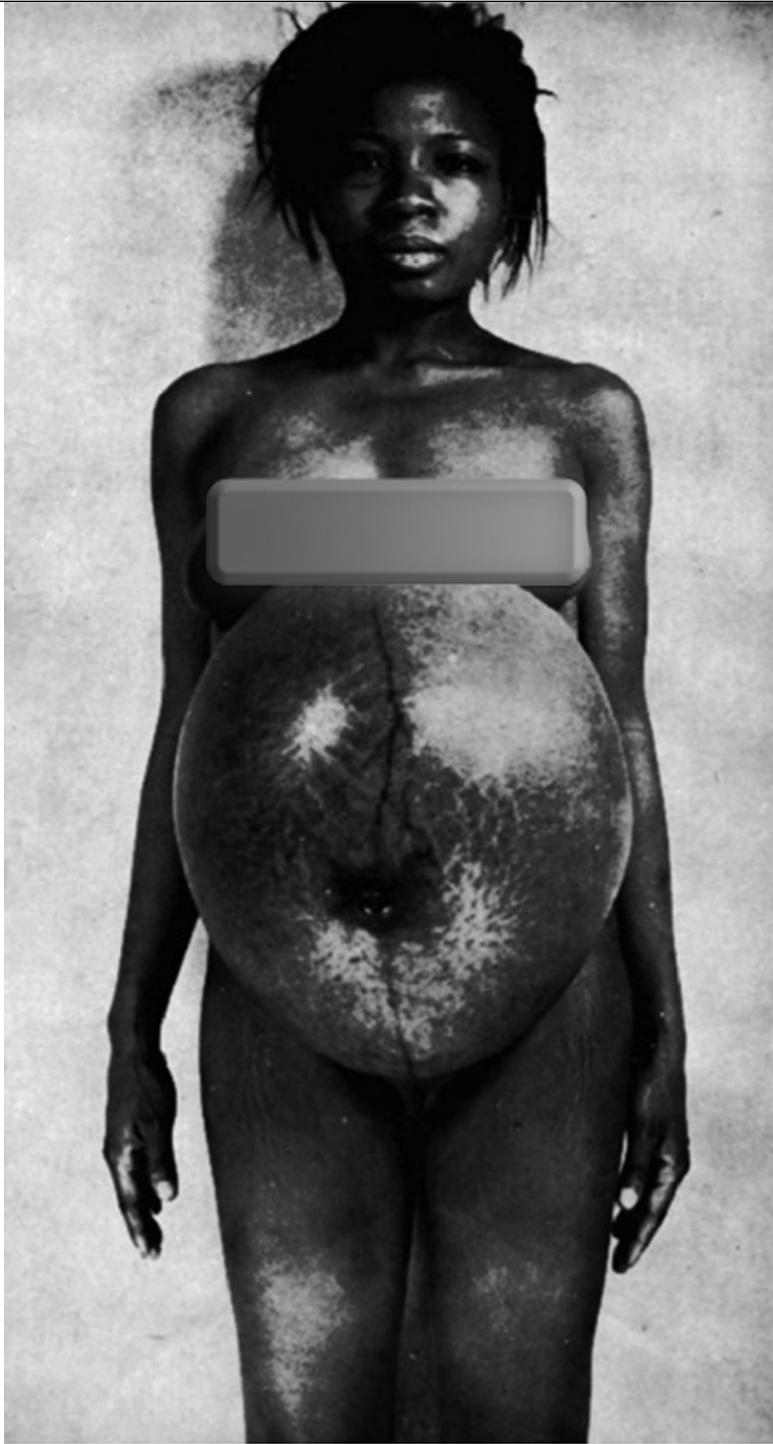
Source: F. Gary Cunningham, Kenneth J. Leveno, Steven L. Bloom, Catherine Y. Spong, Jodi S. Dashe, Barbara L. Hoffman, Brian M. Casey, Jeanne S. Sheffield. Williams Obstetrics, 25th Edition. Copyright © McGraw-Hill Education. All rights reserved.

HYDRAMNIOS

This is an abnormally increased amniotic fluid volume, and it complicates 1 to 2 percent of singleton pregnancies (Dashe, 2002; Khan, 2017; Pri-Paz, 2012). It is more frequently noted in multifetal gestations (Hernandez, 2012). Hydramnios may be suspected if the uterine size exceeds that expected for gestational age. The uterus may feel tense, and palpating fetal small parts or auscultating fetal heart tones may be difficult. An extreme example is shown in Figure 11-2.

FIGURE 11-2

Severe hydramnios—5500 mL of amniotic fluid was measured at delivery.



Source: F. Gary Cunningham, Kenneth J. Leveno, Steven L. Bloom, Catherine Y. Spong, Jodi S. Dashe, Barbara L. Hoffman, Brian M. Casay, Jeanne S. Sheffield. *Williams Obstetrics*, 25th Edition
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Hydramnios may be further categorized according to degree. Such categorization is primarily used in research studies to stratify risks. Several groups have termed hydramnios as *mild* if the AFI is 25 to 29.9 cm; *moderate*, if 30 to 34.9 cm; and *severe*, if 35 cm or more (Lazebnik, 1999; Luo, 2016; Odibo, 2016; Pri-Paz, 2012). Mild hydramnios is the most common, comprising approximately two thirds of cases; moderate hydramnios accounts for about 20 percent; and severe hydramnios for approximately 15 percent. Using the single deepest pocket of amniotic fluid, mild hydramnios is defined as 8 to 9.9 cm, moderate as 10 to 11.9 cm, and severe hydramnios as 12 cm or more (Fig. 11-3). In general, severe hydramnios is far more likely to have an underlying etiology and to have consequences for the pregnancy than mild hydramnios, which is frequently idiopathic and benign.

FIGURE 11-3

Sonogram of severe hydramnios at 35 weeks in a pregnancy complicated by fetal aqueductal stenosis. This pocket of amniotic fluid measures >15 cm, and the amniotic fluid index measured nearly 50 cm.



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Etiology

Underlying causes of hydramnios include fetal anomalies—either structural abnormalities or genetic syndromes—in approximately 15 percent, and diabetes in 15 to 20 percent (Table 11-2). Congenital infection, red blood cell alloimmunization, and placental chorioangioma are less frequent etiologies. Infections that may present with hydramnios include cytomegalovirus, toxoplasmosis, syphilis, and parvovirus. Hydramnios is often a component of *hydrops fetalis*, and several of the above causes—selected anomalies, infections, and alloimmunization—may result in a hydropic fetus and placenta. The underlying pathophysiology in such cases is complex but is frequently related to a high cardiac-output state. Severe fetal anemia is a classic example. Because the etiologies of hydramnios are so varied, hydramnios treatment also differs and is tailored in most cases to the underlying cause.

TABLE 11-2

Hydramnios: Prevalence and Associated Etiologies—Values in Percent

	Golan (1993) n = 149	Many (1995) n = 275	Biggio (1999) n = 370	Dashe (2002) n = 672	Pri-Paz (2012) n = 655
Prevalence	1	1	1	1	2
Amniotic fluid index	—	72	—	66	64
Mild 25–29.9 cm		20		22	21
Moderate 30–34.9 cm		8		12	15
Severe >35 cm					
Etiology					
Idiopathic	65	69	72	82	52
Fetal anomaly ^a	19	15 ^a	8	11 ^a	38 ^a
Diabetes	15	18	20	7	18

^aA significant correlation was identified between severity of hydramnios and likelihood of an anomalous infant.

Congenital Anomalies

Selected anomalies and the likely mechanism by which they cause hydramnios are shown in [Table 11-3](#). Many of these abnormalities are depicted and discussed in [Chapter 10](#). Because of this association, targeted sonography is indicated whenever hydramnios is identified. If a fetal abnormality is encountered concurrent with hydramnios, amniocentesis with chromosomal microarray analysis should be offered, because the aneuploidy risk is significantly elevated ([Dashe, 2002](#); [Pri-Paz, 2012](#)).

TABLE 11-3

Selected Anomalies and Mechanism for Hydramnios

Mechanism	Anomaly Examples
Impaired swallowing (CNS)	Anencephaly
	Hydranencephaly
	Holoprosencephaly
Impaired swallowing (craniofacial)	Cleft lip/palate
	Micrognathia
Tracheal compression or obstruction	Neck venolymphatic abnormality
	CHAOS ^a

Thoracic etiology (mediastinal shift)	Diaphragmatic hernia ^a
	Cystic adenomatoid malformation ^a
	Pulmonary sequestration ^a
High-output cardiac state	Ebstein anomaly ^a
	Tetralogy of Fallot with absent pulmonary valve ^a
	Thyrotoxicosis ^a
Functional cardiac etiology	Cardiomyopathy, myocarditis ^a
Cardiac arrhythmia	Tachyarrhythmia ^a : atrial flutter, atrial fibrillation, supraventricular tachycardia
	Bradyarrhythmia ^a : heart block
GI obstruction	Esophageal atresia
	Duodenal atresia
Renal-Urinary	Ureteropelvic junction obstruction (“paradoxical hydramnios”)
	Baarter syndrome
Neurological or muscular etiology	Arthrogryposis, akinesia sequence
	Myotonic dystrophy
Neoplastic etiology	Sacroccocygeal teratoma ^a
	Mesoblastic nephroma ^a
	Placental chorioangioma ^a

^aPoses risk for hydrops.

CHAOS = congenital high-airway obstruction sequence; GI = gastrointestinal.

Importantly, the degree of hydramnios correlates with the likelihood of an anomalous infant (Lazebnik, 1999; Pri-Paz, 2012). At Parkland Hospital, the prevalence of an anomalous neonate was approximately 8 percent with mild hydramnios, 12 percent with moderate hydramnios, and more than 30 percent with severe hydramnios (Dashe, 2002). Even if no abnormality was detected with targeted sonography, the likelihood of a major anomaly identified at birth was 1 to 2 percent if hydramnios was mild or moderate and 10 percent if hydramnios was severe. The overall reported risk that an underlying anomaly will be discovered after delivery has ranged from 9 percent in the neonatal period to 28 percent among infants followed to 1 year of age (Abele, 2012; Dorleijn, 2009). The anomaly risk is particularly high with hydramnios coexistent with fetal-growth restriction (Lazebnik, 1999).

Although amniotic fluid volume abnormalities are associated with fetal malformations, the converse is not usually the case. In the Spanish Collaborative Study of Congenital Malformations that included more than 27,000 anomalous infants, only 4 percent of pregnancies were complicated

by hydramnios, and another 3 percent with oligohydramnios (Martinez-Frias, 1999).

Diabetes Mellitus

The amniotic fluid glucose concentration is higher in diabetic women than in those without diabetes, and the AFI may correlate with the amniotic fluid glucose concentration (Dashe, 2000; Spellacy, 1973; Weiss, 1985). Such findings support the hypothesis that maternal hyperglycemia causes fetal hyperglycemia, with resulting fetal osmotic diuresis into the amniotic fluid compartment. That said, rescreening for gestational diabetes in pregnancies with hydramnios does not appear to be beneficial, provided that the second-trimester glucose tolerance test result was normal (Frank Wolf, 2017).

Multifetal Gestation

Hydramnios is generally defined in multifetal gestations as a single deepest amniotic fluid pocket measuring 8 cm or more. It may be further characterized as moderate if the single deepest pocket is at least 10 cm and severe if this pocket is at least 12 cm. In a review of nearly 2000 twin gestations, Hernandez and colleagues (2012) identified hydramnios in 18 percent of both monochorionic and dichorionic pregnancies. As in singletons, severe hydramnios was more strongly associated with fetal abnormalities. In monochorionic gestations, hydramnios of one sac and oligohydramnios of the other are diagnostic criteria for twin-twin transfusion syndrome (TTTS), discussed in Chapter 45 (Twin-Twin Transfusion Syndrome). Isolated hydramnios of one sac also may precede the development of this syndrome (Chon, 2014). In the absence of TTTS, hydramnios does not generally raise pregnancy risks in nonanomalous twins (Hernandez, 2012).

Idiopathic Hydramnios

This accounts for up to 70 percent of cases of hydramnios and is thus identified in as many as 1 percent of pregnancies (Panting-Kemp, 1999; Pri-Paz, 2012; Wiegand, 2016). Idiopathic hydramnios is rarely identified during midtrimester sonography and is often an incidental finding later in gestation. The gestational age at sonographic detection usually lies between 32 and 35 weeks (Abele, 2012; Odibo, 2016; Wiegand, 2016). Although it is a diagnosis of exclusion, an underlying fetal abnormality may subsequently become apparent with advancing gestation, particularly if the degree of hydramnios becomes severe. In the absence of an etiology, idiopathic hydramnios is mild in approximately 80 percent of cases, and resolution is reported in more than a third of affected pregnancies (Odibo, 2016; Wiegand, 2016). Mild, idiopathic hydramnios is most commonly a benign finding, and associated pregnancy outcomes are usually good.

Complications

Unless hydramnios is severe or develops rapidly, maternal symptoms are infrequent. With chronic hydramnios, fluid accumulates gradually, and a woman may tolerate excessive abdominal distention with relatively little discomfort. Acute hydramnios, however, tends to develop earlier in pregnancy. It may result in preterm labor before 28 weeks or in symptoms that become so debilitating as to necessitate intervention.

Symptoms may arise from pressure exerted within the overdistended uterus and upon adjacent organs. When distention is excessive, such as that shown in Figure 11-2, the mother may suffer dyspnea and orthopnea to such a degree that she may be able to breathe comfortably only when upright. Edema may develop as a consequence of major venous system compression by the enlarged uterus, and it tends to be most pronounced in the lower extremities, vulva, and abdominal wall. Rarely, oliguria may result from ureteral obstruction by the enlarged uterus (Chap. 53, Lower Genital Tract Lesions). Maternal complications such as these are typically associated with severe hydramnios from an underlying etiology.

Maternal complications associated with hydramnios include placental abruption, uterine dysfunction during labor, and postpartum hemorrhage. Placental abruption is fortunately infrequent. It may result from the rapid decompression of an overdistended uterus that follows fetal-membrane rupture or therapeutic amnioreduction. With prematurely ruptured membranes, a placental abruption occasionally occurs days or weeks after amniorrhexis. Uterine dysfunction consequent to overdistention may lead to postpartum atony and, in turn, postpartum hemorrhage.

Pregnancy Outcomes

Some outcomes more common with hydramnios include birthweight >4000 g, cesarean delivery, and importantly, perinatal mortality. Pregnancies with idiopathic hydramnios are associated with birthweights exceeding 4000 g in nearly 25 percent of cases, and the likelihood appears to be greater if the hydramnios is moderate or severe (Luo, 2016; Odibo, 2016; Wiegand, 2016). A rationale for this association is that larger fetuses have higher urine

output, by virtue of their increased volume of distribution, and fetal urine is the largest contributor to amniotic fluid volume. Cesarean delivery rates are also higher in pregnancies with idiopathic hydramnios, with reported rates of 35 to 55 percent (Dorleijn, 2009; Khan, 2017; Odibo, 2016).

An unresolved question is whether hydramnios alone raises the risk for perinatal mortality. Some studies have found no increase in stillbirth or neonatal death rates with idiopathic hydramnios, whereas others show a greater risk (Khan, 2017; Pilliod, 2015; Wiegand, 2016). Using birth certificate data from the state of California, Pilliod and coworkers (2015) identified hydramnios in 0.4 percent of singleton, nonanomalous pregnancies, and affected pregnancies had significantly greater stillbirth rates. At 37 weeks, the stillbirth risk was sevenfold higher in pregnancies with hydramnios. By 40 weeks, this risk was more than tenfold higher—66 per 10,000 births compared with 6 per 10,000 without hydramnios.

Risks appear to be compounded when a growth-restricted fetus is identified with hydramnios (Erez, 2005). The combination also has a recognized association with trisomy 18. When an underlying cause is identified, degree of hydramnios has been associated with likelihood of preterm delivery, small-for-gestational age newborn, and perinatal mortality (Pri-Paz, 2012). However, *idiopathic* hydramnios is generally not associated with preterm birth (Magann, 2010; Many, 1995; Panting-Kemp, 1999).

Management

As previously noted, treatment is directed to the underlying cause. Occasionally, severe hydramnios may result in early preterm labor or the development of maternal respiratory compromise. In such cases, large-volume amniocentesis—termed *amnioreduction*—may be needed. The technique is similar to that for genetic amniocentesis, described in Chapter 14 (Technique). One difference is that it is generally done with a larger needle, 18- or 20-gauge, and uses either an evacuated container bottle or a larger syringe. Approximately 1000 to 2000 mL of fluid is slowly withdrawn over 20 to 30 minutes, depending on the severity of hydramnios and gestational age. The goal is to restore amniotic fluid volume to the upper normal range. Hydramnios severe enough to necessitate amnioreduction almost invariably has an underlying cause, and subsequent amnioreduction procedures may be required as often as weekly or even semiweekly.

In a review of 138 singleton pregnancies requiring amnioreduction for hydramnios, a fetal GI malformation was identified in 20 percent, a chromosomal abnormality or genetic condition in almost 30 percent, and a neurological abnormality in 8 percent (Dickinson, 2014). In only 20 percent of cases was the hydramnios idiopathic. The initial amnioreduction procedure in this series was performed at 31 weeks' gestation, and the median gestational age at delivery was 36 weeks. Complications within 48 hours of amnioreduction included delivery in 4 percent and ruptured membranes in 1 percent. There was no instance of chorioamnionitis, placental abruption, or bradycardia requiring delivery (Dickinson, 2014).

OLIGOHYDRAMNIOS

This is an abnormally decreased amount of amniotic fluid. Oligohydramnios complicates approximately 1 to 2 percent of pregnancies (Casey, 2000; Petrozella, 2011). When no measurable pocket of amniotic fluid is identified, the term *anhydramnios* may be used. Unlike hydramnios, which is often mild and often confers a benign prognosis in the absence of an underlying etiology, oligohydramnios is always a cause for concern, as discussed in [Pregnancy Outcomes](#).

The sonographic diagnosis of oligohydramnios is usually based on an AFI less than 5 cm or a single deepest pocket of amniotic fluid below 2 cm (American College of Obstetricians and Gynecologists, 2016). Using the Moore nomogram, an AFI threshold of 5 cm is below the 2.5th percentile throughout the second and third trimesters (see [Fig. 11-1](#)). Either criterion is considered acceptable. However, use of AFI rather than single deepest pocket will identify more pregnancies as having oligohydramnios, without evidence of improvement in pregnancy outcomes (Kehl, 2016; Nabhan, 2010). When evaluating multifetal pregnancies for TTTS, a single deepest pocket below 2 cm is used to define oligohydramnios (Society for Maternal-Fetal Medicine, 2013).

Etiology

Pregnancies complicated by oligohydramnios include those in which the amniotic fluid volume has been severely diminished since the early second trimester and those in which the fluid volume was normal until near-term or even full-term. The prognosis depends heavily on the underlying cause and thus varies. Whenever oligohydramnios is diagnosed, it becomes an important consideration in clinical management.

Early-Onset Oligohydramnios

When amniotic fluid volume is abnormally decreased from the early second trimester, it may reflect a fetal abnormality that precludes normal urination, or it may represent a placental abnormality sufficiently severe to impair perfusion. In either circumstance, the prognosis is poor. Ruptured membranes should be excluded, and targeted sonography is performed to assess for fetal and placental abnormalities.

Oligohydramnios after Midpregnancy

When amniotic fluid volume becomes abnormally decreased in the late second or in the third trimester, it is very often associated with fetal-growth restriction, with a placental abnormality, or with a maternal complication such as preeclampsia or vascular disease (Table 11-4). The underlying cause in such cases is frequently uteroplacental insufficiency, which can impair fetal growth and reduce fetal urine output. Exposure to selected medications has also been linked with oligohydramnios as discussed subsequently. Investigation of third-trimester oligohydramnios generally includes evaluation for ruptured membranes and sonography to assess fetal growth. Umbilical artery Doppler studies are recommended if growth restriction is identified (Chap. 10, Doppler). Oligohydramnios is commonly encountered in late-term and *postterm pregnancies* (Chap. 43, Placental Dysfunction). Magann and coworkers (1997) found that amniotic fluid volume decreased by approximately 8 percent per week beyond 40 weeks.

TABLE 11-4

Pregnancy Outcomes in Women Diagnosed with Oligohydramnios between 24 and 34 Weeks' Gestation

Factor	AFI ≤5 cm (n = 166)	AFI 8 to 24 cm (n = 28,185)	p Value
Major malformation	42 (25)	634 (2)	<.001
Stillbirth	8 (5)	133 (<1)	<.001
Gestational age at delivery ^a	35.1 ± 3.3	39.2 ± 2.0	<.001
Preterm birth, spontaneous ^a	49 (42)	1698 (6)	<.001
Preterm birth, indicated ^a	23 (20)	405 (2)	<.001
Cesarean delivery for nonreassuring fetal status ^a	10 (9)	1083 (4)	<.001
Birthweight <10th percentile ^a	61 (53)	3388 (12)	<.001
<3rd percentile ^a	43 (37)	1130 (4)	<.001
Neonatal death ^a	1 (1)	24 (<1)	<.001 ^b

Data expressed as No. (%) and mean ± standard deviation.

^aAnomalous infants excluded.

^bThis difference was no longer significant after adjustment for gestational age at delivery.

Data from Petrozella, 2011.

Congenital Anomalies

By approximately 18 weeks, the fetal kidneys are the main contributor to amniotic fluid volume. Selected renal abnormalities that lead to absent fetal urine production include bilateral *renal agenesis*, bilateral *multicystic dysplastic kidney*, unilateral renal agenesis with contralateral multicystic

dysplastic kidney, and the infantile form of *autosomal recessive polycystic kidney disease*. Urinary abnormalities may also result in oligohydramnios because of fetal *bladder outlet obstruction*. Examples of this are *posterior urethral valves*, *urethral atresia* or *stenosis*, or the *megacystis microcolon intestinal hypoperistalsis syndrome*. Complex fetal genitourinary abnormalities such as *persistent cloaca* and *sirenomelia* similarly may result in a lack of amniotic fluid. Many of these renal and urinary abnormalities are discussed and depicted in [Chapter 10 \(Renal Pelvis Dilatation\)](#). If no amniotic fluid is visible beyond the mid-second trimester due to a genitourinary etiology, the prognosis is extremely poor unless fetal therapy is an option. Fetuses with bladder-outlet obstruction may be candidates for vesicoamniotic shunt placement ([Chap. 16, Urinary Shunts](#)).

Medication

Oligohydramnios has been associated with exposure to drugs that block the renin-angiotensin system. These include angiotensin-converting enzyme (ACE) inhibitors, angiotensin-receptor blockers, and nonsteroidal antiinflammatory drugs (NSAIDs). When taken in the second or third trimester, ACE inhibitors and angiotensin-receptor blockers may create fetal hypotension, renal hypoperfusion, and renal ischemia, with subsequent anuric renal failure ([Bullo, 2012](#); [Guron, 2000](#)). Fetal skull bone hypoplasia and limb contractures have also been described ([Schaefer, 2003](#)). NSAIDs can be associated with fetal ductus arteriosus constriction and with lower fetal urine production. In neonates, their use may result in acute and chronic renal insufficiency ([Fanos, 2011](#)). These agents are discussed in [Chapter 12 \(Angiotensin-Converting Enzyme Inhibitors and Angiotensin-Receptor Blocking Drugs\)](#).

Pregnancy Outcomes

Oligohydramnios is associated with adverse pregnancy outcomes. [Casey and colleagues \(2000\)](#) found that an AFI ≤ 5 cm complicated 2 percent of pregnancies undergoing sonography at Parkland Hospital after 34 weeks' gestation. Fetal malformation rates were elevated in those with oligohydramnios. Even in their absence, rates of stillbirth, growth restriction, nonreassuring heart rate pattern, and meconium aspiration syndrome were higher than in nonaffected pregnancies. [Petrozella and associates \(2011\)](#) similarly reported that an AFI ≤ 5 cm identified between 24 and 34 weeks was associated with increased risks for stillbirth, spontaneous or medically indicated preterm birth, heart rate pattern abnormalities, and growth restriction (see [Table 11-4](#)). In one metaanalysis comprising more than 10,000 pregnancies, women with oligohydramnios had a twofold greater risk for cesarean delivery for fetal distress and a fivefold higher risk for an Apgar score < 7 at 5 minutes compared with pregnancies with a normal AFI ([Chauhan, 1999](#)).

As discussed, evidence suggests that if oligohydramnios is defined as an AFI ≤ 5 cm rather than a single deepest pocket ≤ 2 cm, more pregnancies will be classified as such. One review of trials encompassing more than 3200 high-risk and low-risk pregnancies compared outcomes according to which definition was used ([Nabhan, 2008](#)). Rates of cesarean delivery, neonatal intensive care unit admission, umbilical artery pH < 7.1 , or Apgar score < 7 at 5 minutes did not differ between groups. Using AFI criteria, however, twice as many pregnancies were diagnosed with oligohydramnios. In this group, there was a doubling of the labor induction rate and a 50-percent increase in the cesarean delivery rate for fetal distress. [Kehl and colleagues \(2016\)](#) performed a prospective trial with more than 1000 term pregnancies in which women with oligohydramnios, defined either by an AFI < 5 cm or a single deepest pocket < 2 cm, were randomized to labor induction or expectant care. Significantly more pregnancies were diagnosed with oligohydramnios using the AFI criterion—10 percent compared with just 2 percent—when single deepest pocket was used. This led to a higher rate of labor induction in the AFI group, but no difference in neonatal outcomes.

Pulmonary Hypoplasia

When diminished amniotic fluid is first identified before the mid-second trimester, particularly before 20 to 22 weeks, pulmonary hypoplasia is a significant concern. The underlying etiology is a major factor in the prognosis for such pregnancies. Severe oligohydramnios secondary to a renal abnormality generally has a lethal prognosis. If a placental hematoma or chronic abruption is severe enough to result in oligohydramnios—the *chronic abruption-oligohydramnios sequence (CAOS)*—it commonly also causes growth restriction ([Chap. 41, Frequency](#)). The prognosis for this constellation is similarly poor. Oligohydramnios that results from membrane rupture in the second trimester is reviewed in [Chapter 42 \(Considerations with Expectant Management\)](#).

Management

Initially, an evaluation for fetal anomalies and growth is essential. In a pregnancy complicated by oligohydramnios and fetal-growth restriction, close fetal surveillance is important because of associated morbidity and mortality ([Chap. 44, Management](#)). Oligohydramnios detected before 36 weeks'

gestation in the presence of normal fetal anatomy and growth is generally managed expectantly in conjunction with enhanced fetal surveillance. However, evidence of fetal or maternal compromise will override potential complications from preterm delivery. Antepartum management of oligohydramnios may include maternal hydration. In a recent review of 16 trials of pregnancies with apparent isolated oligohydramnios, oral or intravenous hydration was associated with significant improvement in the AFI. However, it was not clear whether this translated into better pregnancy outcomes (Gizzo, 2015).

Amnioinfusion, discussed in [Chapter 24 \(Management Options\)](#), may be used intrapartum to help resolve variable fetal heart rate decelerations. It is not considered treatment for oligohydramnios per se, although the decelerations are presumed secondary to umbilical cord compression resulting from lack of amniotic fluid. Amnioinfusion is not the standard of care for other etiologies of oligohydramnios and is not generally recommended.

“Borderline” Oligohydramnios

The term *borderline AFI* or *borderline oligohydramnios* is somewhat controversial. It usually refers to an AFI between 5 and 8 cm (Magann, 2011; Petrozella, 2011). Through the mid-third trimester, an AFI value of 8 cm is below the 5th percentile on the Moore nomogram (see [Fig. 11-1](#)). Petrozella and colleagues (2011) found that pregnancies between 24 and 34 weeks with an AFI between 5 and 8 cm were not more likely than those with an AFI above 8 cm to be complicated by maternal hypertension, stillbirth, or neonatal death. That said, higher rates of preterm delivery, cesarean delivery for a nonreassuring fetal heart rate pattern, and fetal-growth restriction were found. Wood and colleagues (2014) similarly reported a higher rate of fetal-growth restriction in pregnancies with borderline AFI. Thus, results evaluating pregnancy outcomes with borderline AFI have been mixed. Magann and associates (2011) concluded that evidence is insufficient to support fetal testing or delivery in this setting.

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