Society for Maternal-Fetal Medicine
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Society for Maternal-Fetal Medicine (SMFM) Consult Series | #46
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SMFM Consult Series #46: Evaluation and management of polyhydramnios

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Polyhydramnios, or hydramnios, is an abnormal increase in the volume of amniotic fluid. Identification of polyhydramnios should prompt a search for an underlying etiology. Although most cases of mild polyhydramnios are idiopathic, the 2 most common pathologic causes are maternal diabetes mellitus and fetal anomalies, some of which are associated with genetic syndromes. Other causes of polyhydramnios include congenital infection and alloimmunization. The purpose of this document is to provide guidance on the evaluation and management of polyhydramnios. The following are Society for Maternal-Fetal Medicine recommendations: (1) we suggest that polyhydramnios in singleton pregnancies be defined as either a deepest vertical pocket of ≥8 cm or an amniotic fluid index of ≥24 cm (GRADE 2C); (2) we recommend that amnioreduction be considered only for the indication of severe maternal discomfort, dyspnea, or both in the setting of severe polyhydramnios (GRADE 1C); (3) we recommend that indomethacin should not be used for the sole purpose of decreasing amniotic fluid in the setting of polyhydramnios (GRADE 1B); (4) we suggest that antenatal fetal surveillance is not required for the sole indication of mild idiopathic polyhydramnios (GRADE 2C); (5) we recommend that labor should be allowed to occur spontaneously at term for women with mild idiopathic polyhydramnios; that induction, if planned, should not occur at <39 weeks of gestation in the absence of other indications; and that mode of delivery should be determined based on usual obstetric indications (GRADE 1C); and (6) we recommend that women with severe polyhydramnios deliver at a tertiary center due to the significant possibility that fetal anomalies may be present (GRADE 1C).

Key words: hydramnios, idiopathic polyhydramnios, isolated polyhydramnios

Introduction

Polyhydramnios, or hydramnios, is an abnormal increase in amniotic fluid volume and is typically diagnosed in the second or third trimester. Amniotic fluid can be assessed with ultrasonography using 1 of 2 semiquantitative methods: (1) the single deepest vertical pocket (DVP) of amniotic fluid, with polyhydramnios defined as ≥8 cm, or (2) the amniotic fluid index (AFI) with polyhydramnios defined as ≥24 cm.[1–8] When these thresholds are applied, polyhydramnios is reported to complicate 1–2% of singleton pregnancies.[1–3,9–11] In twin gestations, polyhydramnios is more common and occurs primarily due to complications of monochorionic placentation.[12,13]

Identification of polyhydramnios should prompt a search for an underlying etiology. Although mild polyhydramnios is most commonly idiopathic, the 2 most common pathologic causes of polyhydramnios are maternal diabetes mellitus and fetal anomalies. Other causes of polyhydramnios include congenital infection and alloimmunization. Physiologically, the fluid increase in many of these cases can be attributed to 1 of 2 mechanisms: (1) impaired fetal swallowing, or (2) overproduction of fetal urine due to a high-output cardiac state, renal abnormality, or osmotic fetal diuresis (Table 1).

When no etiology for the excess amniotic fluid is identified, polyhydramnios is termed "idiopathic." Idiopathic polyhydramnios is a diagnosis of exclusion, and a

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Received July 17, 2018; accepted July 17, 2018.

TABLE 1
Fetal/neonatal etiologies of polyhydramnios

Impaired Swallowing			Excess Urine Production		
GI Obstruction	Neuro-muscular	Craniofacial	Renal/Urinary	Cardiac	Osmotic diuresis/Other
Duodenal atresia	Myotonic dystrophy	Cleft lip/palate	UPJ obstruction	Cardiac structural anomaly	Diabetes
TE Fistula	Arthrogryposis	Micrognathia	Mesoblastic nephroma	Tachyarrhythmia	Hydrops
Thoracic mass	Intracranial anomaly	Neck mass	Bartter syndrome	Sacrococcygeal teratoma	Idiopathic
Diaphragmatic hernia				Chorioangioma	

GI, gastrointestinal; *TE*, tracheoesophageal; *UPJ*, ureteropelvic junction.
Society for Maternal-Fetal Medicine. Polyhydramnios. Am J Obstet Gynecol 2018.

predisposing condition may become evident with advancing gestation or after delivery. Therefore, polyhydramnios with no identified cause in the prenatal period may also be referred to as unexplained. Idiopathic polyhydramnios accounts for approximately 60–70% of cases of polyhydramnios in published series and is identified in nearly 1% of pregnancies.[1,3,10,14,15] Affected women are more likely to undergo cesarean delivery and to have infants weighing >4000 g.[2–4,15] There is also a risk of identification of an underlying fetal abnormality or genetic syndrome after delivery; this risk varies with the degree of polyhydramnios.[5,9,16]

How is amniotic fluid volume assessed?

After 20 weeks of gestation, amniotic fluid volume is assessed by using either the DVP or AFI.[17] In multiple gestations, the DVP is used. These semiquantitative measures are preferred over subjective assessment because of their reproducibility, and in the setting of amniotic fluid abnormalities, a numerical estimate permits serial assessment over time and facilitates communication among providers.[18] To measure the DVP, the ultrasound transducer is held perpendicular to the floor while scanning in the sagittal plane (parallel to the long axis of the patient's body), and the largest vertical pocket of amniotic fluid is measured. To calculate the AFI, the uterus is divided into 4 equally sized quadrants and the depth of the single deepest fluid pocket in each quadrant is measured; the sum of these measurements is the AFI.[17,18] To be included in either the DVP or AFI, each measured amniotic fluid pocket must be at least 1 cm wide.[18] The measured pocket(s) should not contain fetal parts or loops of umbilical cord. Color Doppler is useful to avoid overmeasurement of a pocket that contains loops of umbilical cord. A recent study comparing fluid assessment with and without color Doppler found that compared with grayscale assessment, use of color Doppler decreased the frequency of diagnosis of polyhydramnios without a change in pregnancy outcomes.[19]

When the single DVP is used, polyhydramnios is defined as a measurement of ≥8.0 cm.[1–7] With AFI, the threshold is generally considered to be 24 cm or 25 cm, depending on whether a 95th or 97th percentile is selected.[17,20] Using a recent nomogram, 24 cm exceeds the 97.5th percentile for all gestational ages between 20–42 weeks.[21] However, research studies have generally selected ≥25 cm as the threshold for polyhydramnios.[1,2,9,22–24] In the previously cited study by Odibo et al,[19] the researchers noted that the DVP technique identified more cases of polyhydramnios than the AFI, which was normal (<24 cm) in all cases in their cohort diagnosed with polyhydramnios based on a DVP of ≥8 cm. The authors suggest that just as the use of the AFI has led to the overdiagnosis of oligohydramnios, it is possible that use of the DVP may lead to the overdiagnosis of polyhydramnios.[19] However, further research into optimal methods for diagnosis of polyhydramnios is needed.[8] **We suggest that polyhydramnios in singleton pregnancies be defined as either a DVP of ≥8 cm or an AFI of ≥24 cm (GRADE 2C).**

Polyhydramnios is most often identified in the third trimester and is less commonly identified during ultrasound examinations conducted between 18–22 weeks of gestation. Polyhydramnios may be suspected when there is a discrepancy between fundal height and gestational age (a size-date discrepancy), but it is more commonly an incidental finding detected during an ultrasound examination performed for another indication. Idiopathic polyhydramnios is usually detected in the third trimester, at a mean gestational age of 31–36 weeks in various series.[4,5,15,16] In term pregnancies, the prevalence of idiopathic polyhydramnios is reported to be 0.3%.[24]

The degree of polyhydramnios is frequently categorized as mild, moderate, or severe, based on an AFI of 24.0–29.9 cm, 30.0–34.9 cm, and ≥35 cm, or a DVP of 8–11 cm, 12–15 cm, or ≥16 cm, respectively.[3,4,9,23–25] Using these definitions, mild polyhydramnios accounts for approximately 65–70% of cases, moderate polyhydramnios for 20%, and severe polyhydramnios for <15%.[3,9,14] The likelihood of an underlying fetal abnormality is significantly higher with greater degrees of polyhydramnios.[3,9,14,23] In pregnancies with an identified underlying etiology, the degree of polyhydramnios is associated with an increased likelihood of preterm delivery, a small-for-gestational age infant, macrosomia, and perinatal mortality.[3] The

association with preterm birth has not been reported with idiopathic polyhydramnios, likely in part because a larger proportion of idiopathic cases are mild (approximately 80% overall and 90% at term).[15,24]

What are the underlying causes of polyhydramnios?

Although most cases of polyhydramnios are mild and idiopathic, when an etiology is identified, it is most commonly due to a fetal anomaly or maternal diabetes. Many of the fetal abnormalities associated with polyhydramnios impair swallowing, such as central nervous system abnormalities, cleft palate, micrognathia, abnormalities that compress the trachea (neck, mediastinal, or lung masses, congenital high airway obstruction sequence, diaphragmatic hernia), gastrointestinal tract obstruction, and neurologic or muscular disorders such as myotonic dystrophy.

Fetal abnormalities that cause a high-output cardiac state or heart failure may also lead to polyhydramnios, often associated with nonimmune hydrops fetalis (NIHF). Examples include sacrococcygeal teratoma, placental chorioangiomas, and other vascular lesions; severe cardiac abnormalities, such as Ebstein anomaly or tetralogy of Fallot with absent pulmonary valve, cardiomyopathy, supraventricular tachycardia, and complete heart block; or fetal thyrotoxicosis. In addition, polyhydramnios may be caused by anomalies that cause fetal urine overproduction, such as ureteropelvic junction obstruction (termed "paradoxical" polyhydramnios).

Small placental chorioangiomas are relatively common and rarely cause pregnancy complications, but large ($\geq$5 cm) chorioangiomas have been associated with NIHF and with polyhydramnios.[26,27] In general, any anomaly severe enough to cause hydrops may result in polyhydramnios, as these entities are often associated with each other.

In addition to maternal diabetes, other potential causes of apparently isolated polyhydramnios in a structurally normal fetus include alloimmunization and congenital infection. With diabetes, it is hypothesized that maternal hyperglycemia leads to fetal hyperglycemia, with subsequent osmotic diuresis into the amniotic fluid compartment. This hypothesis is supported by the observation that amniotic fluid glucose concentration often correlates with the amniotic fluid volume.[28–30] Alloimmunization can lead to fetal anemia with resultant NIHF and polyhydramnios. Congenital infections, such as parvovirus, cytomegalovirus, or syphilis, can lead to polyhydramnios by a variety of mechanisms, including anemia or cardiac dysfunction.

What evaluation should be performed when polyhydramnios is detected?

Importantly, idiopathic polyhydramnios is a diagnosis of exclusion, and while the cause may be unexplained during pregnancy, an etiology may become evident after birth. Evaluation will vary depending on the degree of polyhydramnios, the presence of structural anomalies, and the gestational age. Initial evaluation for polyhydramnios involves targeted ultrasonography to assess for fetal abnormalities (Table 1). It is important to assess fetal growth because idiopathic polyhydramnios may be associated with macrosomia, and fetal growth restriction associated with polyhydramnios presents a high risk for an underlying fetal abnormality, including trisomy 13 or 18.[31,32] In most cases of idiopathic mild polyhydramnios with normal diabetes screening, this evaluation is adequate.

In a structurally normal fetus with mild polyhydramnios, consideration should be given to causes such as diabetes, alloimmunization, and potentially congenital infection. Routine prenatal care includes screening for diabetes and alloimmunization, as well as testing for syphilis. Although there are no data to support a benefit of rescreening for gestational diabetes, it may be considered when polyhydramnios is identified in the third trimester and/or >1 month has elapsed since diabetes screening was completed. Congenital infection usually presents with additional sonographic findings, such as NIHF, hepatomegaly, splenomegaly, or placentomegaly. In cases of polyhydramnios associated with NIHF or additional sonographic features, evaluation for fetal anemia and congenital infection is recommended.[27]

Severe polyhydramnios presenting earlier in gestation should raise a greater concern for an underlying etiology. In cases of severe polyhydramnios, especially early in gestation, it is important to review the medical and family history, in addition to obtaining a detailed ultrasound examination (Box).

It should be noted that not all of the above abnormalities are detectable by ultrasound, and certainly not in every case. Specifically, fetal esophageal atresia and

BOX

Detailed ultrasound examination for severe polyhydramnios

The following parameters should be assessed:

- Fetal growth
- Fetal cardiac anatomy
- Placenta for presence of large chorioangiomas
- Fetal movement to assess neurologic function
- Positioning of fetal hands and feet to rule out arthrogryposis syndromes
- Presence and size of fetal stomach to rule out tracheoesophageal fistula or esophageal atresia
- Anatomy of the fetal face and palate
- Positioning and appearance of the fetal neck to rule out an obstructing mass
- Fetal kidneys to assess for ureteropelvic junction obstruction
- Lower spine and pelvis for evidence of sacrococcygeal teratoma

tracheoesophageal fistula are among the most common abnormalities associated with polyhydramnios. These abnormalities may be difficult to diagnose by ultrasound, but should be suspected in cases in which the fetal stomach is visualized but small. Disorders associated with apparently isolated polyhydramnios include genetic syndromes for which there may not be sonographic findings or a screening or diagnostic test available.

Currently, there are no data to support diagnostic amniocentesis for apparently isolated polyhydramnios, although amniocentesis with chromosomal microarray analysis should be made available to all pregnant women.[33] With severe polyhydramnios, especially with decreased fetal movement, genetic counseling and consideration of testing for neurologic disorders such as congenital myotonic dystrophy should be considered.[34]

The underlying risk that a structural or genetic abnormality will be discovered postnatally in a pregnancy associated with apparently idiopathic polyhydramnios ranges from 9% in the neonatal period to as high as 28% when infants were followed up to 1 year of age.[5,16] In one study, the residual risk that an abnormality would be detected in the immediate neonatal period ranged from 1% with mild polyhydramnios to >10% with severe polyhydramnios[9] (Table 2).

How is a pregnancy with polyhydramnios managed?

Should polyhydramnios be treated?

Polyhydramnios severe enough to cause maternal respiratory compromise, significant discomfort, or preterm labor often has an underlying etiology, whereas idiopathic polyhydramnios, because it is usually mild and does not present until the mid-third trimester, does not typically require treatment.[7] In selected cases, however, amnioreduction may be considered in an effort to relieve maternal dyspnea or discomfort.

In a recent review of 138 singleton pregnancies with polyhydramnios in which amnioreduction was performed for maternal abdominal pain or dyspnea, only 16% of cases were considered idiopathic.[7] The mean gestational age at first procedure was 31.4 weeks; the median duration from procedure until delivery was 26 days; 4% of women delivered within 48 hours of the procedure; the mean volume removed was 1750 mL; 46% required >1 procedure; and the mean gestational age at delivery was 36.4 weeks. In a smaller series, Abele et al[5] reported that of 118 pregnancies with polyhydramnios thought to be idiopathic, amnioreduction was performed in 4 cases, and in 2 of these cases an abnormality was identified postnatally. Overall, in cases with severe polyhydramnios that results in maternal respiratory compromise such that amnioreduction is considered, an underlying fetal abnormality is usually present.[5,7] In addition, polyhydramnios usually recurs after amnioreduction, making its efficacy somewhat limited. **We recommend that amnioreduction be considered only for the indication of severe maternal discomfort, dyspnea, or both in the setting of severe polyhydramnios (GRADE 1C).** Almost all such cases will be associated with fetal abnormalities, and if an etiology has not been identified, further evaluation should be considered.

Indomethacin, which is often used for tocolysis, decreases fetal urine production. Kirshon et al[35] described a series of women with recurrent, symptomatic polyhydramnios following amnioreduction who were treated with indomethacin therapy and who did not subsequently require additional amnioreduction procedures. However, preterm infants exposed to indomethacin in utero have been shown to have decreased neonatal urine output and elevated serum creatinine concentrations, which have in some cases persisted for weeks.[36,37] In addition, meta-analyses of indomethacin therapy for preterm labor have associated this treatment with an increased risk for other neonatal morbidities, including intraventricular hemorrhage, periventricular leukomalacia, and necrotizing enterocolitis.[38,39] Due to reported neonatal complications, and in the absence of data on improved maternal or neonatal outcomes, **we recommend that indomethacin should not be used for the sole purpose of decreasing amniotic fluid in the setting of polyhydramnios (GRADE 1B).**

Antepartum management

Multiple groups of investigators have reported that idiopathic polyhydramnios is associated with infant

TABLE 2
Outcomes of polyhydramnios based on severity

	Amniotic fluid index	Deepest vertical pocket	Incidence	Risk of fetal abnormality[a]	Risk of neonatal abnormality[b]
Polyhydramnios, overall, cm	≥24.0	≥8.0	0.3–1.0%		
Mild, cm	24.0–29.9	8–11	65–70% of total	6–10%	1%
Moderate, cm	30.0–34.9	12–15	20% of total	10–15%	2%
Severe, cm	≥35.0	≥16	<15% of total	20–40%	10%

[a] Chance that fetal anomaly will be identified prenatally;[9,23,25] [b] Chance that neonatal abnormality will be identified in cases without a detected prenatal anomaly.
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birthweight >4000 g in approximately 15–30% of cases.[4,15,16,24] This is in contrast to the overall prevalence of birthweight >4000 g across the United States of 8%.[40,41] Because larger fetuses have higher urine output, and fetal urine is the largest contributor to amniotic fluid volume, mild polyhydramnios in these cases is likely to be physiologic. The appropriate role and frequency of follow-up ultrasonography is also unclear but may be warranted in cases where there is concern for progression of polyhydramnios or to evaluate fetal growth.[11,42] Progression of polyhydramnios is suggestive of an underlying etiology, and resolution is not uncommon. In one recent report, polyhydramnios resolved in more than one third of cases and was more likely to resolve when the initial AFI was lower.[4]

Preterm delivery rates are not generally increased with idiopathic polyhydramnios (which is usually mild) but is associated with more severe polyhydramnios.[2,14,15]

Reported data on whether perinatal mortality is increased with idiopathic polyhydramnios have been inconsistent. Several groups of investigators, 2 in case-control studies and 1 in a retrospective cohort investigation, have identified no increase in rates of stillbirth or neonatal death associated with idiopathic polyhydramnios.[2,15,42] In contrast, Pri-Paz et al[3] reported a fetal death rate of 1.2% and Biggio et al[1] reported a perinatal mortality rate of 3.7% with apparently idiopathic polyhydramnios, which was more than twice the rate among controls. In a study of >100,000 term pregnancies in China, in which the prevalence of idiopathic polyhydramnios was 0.3%, Luo et al[24] reported 2 (0.7%) fetal deaths with mild idiopathic polyhydramnios, which was 17 times higher than the background rate. They also observed a substantial (85 times) increase in fetal deaths with moderate or severe polyhydramnios, although there were only 2 fetal deaths in this group as well.

In 2015, Pilliod et al[43] used birth certificate data to review >1.8 million singleton nonanomalous births in California, of which 0.4% were identified to have polyhydramnios. Acknowledging the limitations of birth certificate data, including a lack of information on the degree of polyhydramnios, the authors found that the ongoing risk of fetal demise was greater in otherwise low-risk pregnancies affected by polyhydramnios at all gestational ages, with the greatest increase in risk seen at term.[43]

Antepartum fetal surveillance is performed in pregnancies at risk for uteroplacental insufficiency, and there is no evidence to indicate that isolated polyhydramnios is associated with poor placental function. The most recent guidance from the American Congress of Obstetricians and Gynecologists on antepartum fetal surveillance does not specifically address isolated polyhydramnios or list it as an indication for surveillance.[44] Although antepartum surveillance is often performed in this setting, there are no data to suggest that such assessment decreases perinatal mortality. **We suggest that antenatal fetal surveillance is not required for the sole indication of mild idiopathic polyhydramnios (GRADE 2C).** Likewise, there are no data to suggest that induction of labor or preterm delivery are associated with an improved outcome in the setting of mild idiopathic polyhydramnios. **We recommend that labor should be allowed to occur spontaneously at term for women with mild idiopathic polyhydramnios; that induction, if planned, should not occur at <39 weeks of gestation in the absence of other indications; and that mode of delivery should be determined based on usual obstetric indications (GRADE 1C).**

Intrapartum management

There are limited data regarding intrapartum management of women with polyhydramnios. Labor in the presence of polyhydramnios has been reported to be complicated by fetal nonvertex presentation, with rates increasing as the severity of polyhydramnios increases.[24,42,45] Clinical or sonographic determination of the fetal presenting part should be performed upon presentation in labor. External version for nonvertex fetal presentation may be considered if there are no contraindications to this procedure.

There is a higher rate of dysfunctional labor in the presence of polyhydramnios. Panting-Kemp et al[42] reported a higher rate of cesarean delivery for failure to progress in patients with idiopathic polyhydramnios. Other studies have also demonstrated that women with pregnancies complicated by idiopathic polyhydramnios are significantly more likely to undergo a cesarean delivery, with rates ranging from 35–55%.[2–4,16] An increased risk of operative vaginal delivery in the presence of polyhydramnios has also been reported.[46] In a retrospective case-controlled study, Zeino et al[45] demonstrated that the first stage of labor was prolonged in the presence of polyhydramnios and that the rate of amniotomy was significantly increased. If amniotomy is to be performed, and the polyhydramnios is moderate to severe, performing a "controlled" amniotomy in the operating room using a spinal or pudendal block needle has been suggested; however, a clear advantage of this approach has not been demonstrated.

Nonreassuring fetal heart rate tracings have been reported to be more frequent with polyhydramnios by some,[3] but not all, investigators.[4,41] Likewise, an increased risk for postpartum hemorrhage has been inconsistently reported; Wiegand et al[15] reported a greater risk for postpartum hemorrhage even in the presence of mild polyhydramnios, while Luo et al[24] did not. Nevertheless, having uterotonic agents readily available in the delivery room is reasonable when delivering a patient with polyhydramnios.

As previously noted, there is an increased rate of structural abnormalities or genetic syndromes in the neonate following a gestation complicated by apparently idiopathic polyhydramnios.[2,3,15,43] Idiopathic polyhydramnios is also associated with an increased risk of neonatal intensive care unit admission in some series[2,15] but not others[24,42]; this is most commonly due to transient tachypnea of the newborn.[4] Pediatric support should be available at delivery for women with mild, idiopathic polyhydramnios. **We recommend that women with severe polyhydramnios deliver**

Summary of recommendations

	Recommendation	GRADE
1	We suggest that polyhydramnios in singleton pregnancies be defined as either a DVP of ≥8 cm or an amniotic fluid index of ≥24 cm.	2C Weak recommendation, low-quality evidence
2	We recommend that amnioreduction be considered only for the indication of severe maternal discomfort, dyspnea, or both in the setting of severe polyhydramnios.	1C Strong recommendation, low-quality evidence
3	We recommend that indomethacin should not be used for the sole purpose of decreasing amniotic fluid in the setting of polyhydramnios.	1B Strong recommendation, moderate-quality evidence
4	We suggest that antenatal fetal surveillance is not required for the sole indication of mild idiopathic polyhydramnios.	2C Weak recommendation, low-quality evidence
5	We recommend that labor should be allowed to occur spontaneously at term for women with mild idiopathic polyhydramnios; that induction, if planned, should not occur at <39 weeks of gestation in the absence of other indications; and that mode of delivery should be determined based on usual obstetric indications.	1C Strong recommendation, low-quality evidence
6	We recommend that women with severe polyhydramnios deliver at a tertiary center due to the significant possibility that fetal anomalies may be present.	1C Strong recommendation, low-quality evidence

at a tertiary center due to the significant possibility that fetal anomalies may be present (GRADE 1C).

Summary

Idiopathic polyhydramnios is usually mild and detected in the third trimester. Either a DVP of ≥8 cm or AFI ≥24 cm can be used to diagnose polyhydramnios, although some data suggest that use of the DVP is associated with more cases detected and no change in outcome. The identification of polyhydramnios should prompt a search for an underlying etiology. The 2 most common causes of polyhydramnios are maternal diabetes mellitus and fetal anomalies. Even in the absence of diabetes, idiopathic polyhydramnios is associated with macrosomia in approximately 15–30% of cases, and affected patients are significantly more likely to undergo cesarean delivery. Progression of polyhydramnios is suggestive of an underlying structural or genetic etiology. Reports associating idiopathic polyhydramnios with perinatal mortality have been inconsistent and there are no data to suggest that antenatal fetal surveillance improves perinatal outcomes.

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All authors and committee members have filed a conflict of interest disclosure delineating personal, professional, and/or business interests that might be perceived as a real or potential conflict of interest in relation to this publication. Any conflicts have been resolved through a process approved by the executive board. The Society for Maternal-Fetal Medicine has neither solicited nor accepted any commercial involvement in the development of the content of this publication.

This document has undergone an internal peer review through a multilevel committee process within the Society for Maternal-Fetal Medicine (SMFM). This review involves critique and feedback from the SMFM Publications and Document Review Committees and final approval by the SMFM Executive Committee. SMFM accepts sole responsibility for document content. SMFM publications do not undergo editorial and peer review by the American Journal of Obstetrics & Gynecology. The SMFM Publications Committee reviews publications every 18-24 months and issues updates as needed. Further details regarding SMFM Publications can be found at www.smfm.org/publications.

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  https://doi.org/10.1016/j.ajog.2018.07.016