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Fetal Growth Biometry as Predictors of Shoulder Dystocia in a Low-Risk Obstetrical Population

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Abstract

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Conflict of Interest

D.A.W. has been a consultant for Parsogen, for which she received no compensation. The other authors did not report any potential conflicts of interest.

The study is registered with the ClinicalTrials.gov, identifier: NCT00912132.

Objective—This study aimed to evaluate fetal biometrics as predictors of shoulder dystocia (SD) in a low-risk obstetrical population.

Study Design—Participants were enrolled as part of a U.S.-based prospective cohort study of fetal growth in low-risk singleton gestations (n = 2,802). Eligible women had liveborn singletons ≥2,500 g delivered vaginally. Sociodemographic, anthropometric, and pregnancy outcome data were abstracted by research staff. The diagnosis of SD was based on the recorded clinical impression of the delivering physician. Simple logistic regression models were used to examine associations between fetal biometrics and SD. Fetal biometric cut points, selected by Youden's J and clinical determination, were identified to optimize predictive capability. A final model for SD prediction was constructed using backward selection. Our dataset was randomly divided into training (60%) and test (40%) datasets for model building and internal validation.

Results—A total of 1,691 women (98.7%) had an uncomplicated vaginal delivery, while 23 (1.3%) experienced SD. There were no differences in sociodemographic or maternal anthropometrics between groups. Epidural anesthesia use was significantly more common (100 vs. 82.4%; p = 0.03) among women who experienced SD compared with those who did not. Amniotic fluid maximal vertical pocket was also significantly greater among SD cases (5.8 ± 1.7 vs. 5.1 ± 1.5 cm; odds ratio = 1.32 [95% confidence interval: 1.03,1.69]). Several fetal biometric measures were significantly associated with SD when dichotomized based on clinically selected cut-off points. A final prediction model was internally valid with an area under the curve of 0.90 (95% confidence interval: 0.81, 0.99). At a model probability of 1%, sensitivity (71.4%), specificity (77.5%), positive (3.5%), and negative predictive values (99.6%) did not indicate the ability of the model to predict SD in a clinically meaningful way.

Conclusion—Other than epidural anesthesia use, neither sociodemographic nor maternal anthropometrics were significantly associated with SD in this low-risk population. Both individually and in combination, fetal biometrics had limited ability to predict SD and lack clinical usefulness.

Keywords

fetal biometrics; fetal growth ultrasound; prediction of shoulder dystocia; asymmetric fetal growth

Shoulder dystocia (SD) is an obstetrical emergency with an incidence between 0.2 and 3.0%. Between 20 and 25% of neonates experiencing a SD will sustain a neonatal injury.[1,2] Multiple maternal and fetal characteristics have been associated with shoulder dystocia.[1,2] Unfortunately, despite statistical association, no single factor or combination of factors have been able to reliably predict the occurrence of SD, especially in a low-risk obstetrical population.[2,3]

Fetal macrosomia (birth weight > 4,000 g) has the strongest association with SD.[4–7] Yet, macrosomia is only known after delivery and fetal weight estimation is plagued by inaccuracy.[8,9] Moreover, most SD cases occur in nonmacrosomic infants.[2,4,7] Authors of a decision analysis concluded that elective cesarean delivery based on ultrasound estimated fetal weight was not a clinically reasonable or cost-effective policy.[10,11]

Another factor associated with SD is asymmetric fetal growth. Well-recognized SD risk factors, maternal diabetes,[12] and fetal macrosomia[13] are both associated with asymmetric fetal growth. Prior studies characterizing fetal growth asymmetry as the difference between the abdominal diameter (AD) and biparietal diameter (BPD) revealed a significantly higher mean AD-BPD difference among fetuses experiencing SD.[14–16] Other measures of fetal asymmetry associated with SD include differences in the abdominal-to-head circumferences,[17,18] abdominal-to-head circumference ratio,[17] and the femur length-to-abdominal circumference ratio.[19] The retrospective design, lack of a standardized, research-quality data collection, and restriction to higher prevalence cohorts with maternal diabetes or larger estimated fetal weights represent data gaps in the existing literature regarding the ability of fetal measurements to effectively predict SD.

Our study objective was to examine associations between sociodemographic variables, maternal and paternal anthropometrics, and fetal biometric measures with subsequent shoulder dystocia in a prospective observational study performed in an obstetrical population without medical or obstetrical risk factors for excessive or asymmetrical fetal growth. We further sought to identify the predictive capability of a comprehensive panel of fetal biometric measures for the clinical occurrence of SD and determine whether their simultaneous assessment would allow for the development of a parsimonious regression model for the clinically meaningful prediction of SD in a low-risk obstetrical population.

Materials and Methods

This was a planned secondary analysis of the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) Fetal Growth Studies-Singletons, a prospective study conducted in twelve U.S. health care centers between July 2009 and January 2013.[20–22] Nonobese (body mass index [BMI] = 19.0 to <30.0 kg/m^2) pregnant women with spontaneous singleton conceptions without medical comorbidities or recognized obstetrical risk factors for abnormal fetal growth were eligible for enrollment. Women with a BMI of 30.0 to 45.0 kg/m^2, meeting all other eligibility criteria, were also recruited as a separate obese cohort and are included in the present analysis to improve generalizability. Recalled pregravid weight and self-reported maternal height were used to calculate BMI (kg/m^2) at enrollment. Paternal height and weight were also reported by study patients. Human subjects' approval was obtained from all participating sites, the NICHD, and the data coordinating center. All women provided informed consent prior to enrollment.

Women underwent ultrasound screening between $8^{0/7}$ and $13^{6/7}$ weeks to ensure consistency with recalled last menstrual period dating within a prescribed range. Following a standardized study ultrasound between $10^{0/7}$ and $13^{6/7}$ weeks, women were randomized to one of four sonography schedules with five additional planned visits (16–22, 24–29, 30–33, 34–37, and 38–41 gestational weeks). Study sonographers underwent training and credentialing prior to enrollment and followed rigorous scanning protocols. Enrollment was stratified to achieve relatively equal numbers of four self-identified racial/ethnic groups as follows: (1) non-Hispanic white, (2) non-Hispanic black, (3) Hispanic, and (4) Asian or Pacific Islander.[20–22]

At each ultrasound examination, biometric measurements were obtained of biparietal diameter (BPD), occipital-frontal diameter (OFD), humerus length (HL), anterior–posterior abdominal diameter (AD), femur length (FL), and amniotic fluid maximum vertical pocket (AFMVP) using linear function and the head circumference (HC) and abdominal circumference (AC) using ellipse function. All measurements were performed using identical equipment (Voluson E8 GE Healthcare; Milwaukee, WI) with a transabdominal multifrequency volume transducer (General Electric Real Time Abdominal (RAB): 4–8 MHz). A transvaginal multifrequency volume transducer (General Electric Real Time Intra-cavitary (RIC): 6–12 MHz) was used in the first trimester. All measurements and images were captured in ViewPoint (GE Healthcare; Milwaukee, WI) and electronically transferred to the imaging data coordination center. Estimated fetal weight (EFW) was computed from HC, AC, and FL using the Hadlock formula.[23] Quality assurance was performed on 5% of the scans and demonstrated correlations between the study sonographers and blinded experts exceeding 0.99 for all biometric parameters and coefficients of variation <3%.[24]

In-person interviews were conducted at each visit by study personnel to ascertain and update demographic data, family history, lifestyle, reproductive, and intercurrent medical/obstetric history. At each scheduled ultrasound examination, research staff measured maternal weight and weight gain, natural waist, waist over iliac crest, hip circumference, upper arm length, mid-upper arm circumference, triceps, and subscapular skinfold according to standard protocol. After birth, antenatal history, labor, delivery, neonatal course, and neonatal outcomes were abstracted from medical records by the same study personnel. The primary outcome was the occurrence of shoulder dystocia which was a specific delivery outcome inquiry. The diagnosis of SD was based on the recorded subjective clinical impression of the delivery attendant at each site as recorded in the birth records. The diagnostic criteria for SD were not standardized across participating centers. The occurrence of brachial plexus palsy, clavicular, or humeral fracture was based on neonatal diagnostic codes extracted from the neonatal discharge summary by assigned study staff.[22] No Zavenelli's maneuvers were performed. As some cases of SD may not be ascertained fully in the medical records, a sensitivity analysis was performed which included those cases of neonatal injury potentially associated with SD but without physician documentation of SD.

Fig. 1 illuminates our study population, inclusions, and exclusions. Of 2,802 mother–infant dyads enrolled in the original NICHD Fetal Growth Study-Singletons, 1,714 mother–infant dyads were included in final analyses.

Students *t*-tests, Chi-square, and Fisher's exact were used to assess significant ($p \leq 0.05$) differences in sociodemographic, anthropometric, and ultrasound measured fetal biometrics between pregnancies with or without the primary outcome. Missing data were uncommon but when it occurred, that woman or fetus was excluded. Fetal biometric measures were compared using data from the most recent ultrasound research visit preceding delivery (average: 17.5 days before delivery). The average of three individual fetal anthropometric measures of BPD, OFD, HC, AC, FL, and HL were obtained. Derived measurements used to describe fetal growth asymmetry included the absolute difference between the AD and BPD (AD–BPD), between the AC and HC (AC–HC), and ratios between the HC and AC (HC/AC) and FL and AC (FL/AC).

Unadjusted logistic regression models were run to assess the association between continuous fetal biometrics and shoulder dystocia using odds ratios (ORs) and 95% confidence intervals (95% CIs). From these models, we evaluated the predictive capability (i.e., area under the curve [AUC]) from receiver operating curves (ROCs) for continuous fetal biometrics. The AUC may be interpreted as the probability that a randomly chosen subject with SD is more likely to have experienced such an event than a randomly chosen subject who did not.

Next, we identified two dichotomous cut-off points for fetal biometric measurements. First was the Youden's J which is a statistical cut-off point that optimizes the discriminating ability of a variable if equal weight is given to both sensitivity and specificity.[25] Second was a clinical cut-off point selected to optimize specificity while maintaining a sensitivity of approximately 10%. This approach was used to select a cut-off point allowing detection of as many at-risk women as possible (sensitivity) while also minimizing the number of unnecessary cesareans which might be performed to avoid a shoulder dystocia (false positive rate = 1-specificity). Unadjusted logistic regression models were run for each of these dichotomous cut-off points to examine ORs and 95% CIs for their potential association with SD comparing individuals with an ultrasound measurement equal to or greater versus less than the suggested cut-off point. We then examined the performance characteristics (i.e., AUC, sensitivity, specificity, and positive and negative predictive values) from these regression models.

Finally, we built a prediction model for SD using backward model selection procedures with *p*-value for entry of 0.2 and *p*-value for remaining in the model of 0.05. Variables assessed for inclusion were sociodemographic and clinical characteristics, continuous and dichotomized fetal biometrics, and all two-way interactions. Other than measures of height and weight, maternal and paternal anthropometrics were not included in this final model, as they are not routinely measured in clinical practice. Our dataset was randomly divided into a training (60%) cohort for model building and a test (40%) cohort for internal validation. The sensitivity, specificity, and positive and negative predictive values describing internal validity are from this model applied to the test dataset. Supplementary Table S1 (available in the online version) provides more information on model building procedures. All analyses were performed using SAS (version 9.4; SAS Institute Inc, Cary, NC) and R (version 3.6.1).

Results

Our analysis included 1,714 women delivered vaginally of whom 23 (1.3%) had a SD. Of the 23 SD cases, 3 experienced brachial plexus injuries; none experienced a fractured clavicle. There were seven clavicular fractures and three brachial plexus injuries in the absence of a documented SD. No fractured humeri occurred. A sensitivity analysis which included those neonatal birth injuries reported in the absence of a SD diagnosis along with the SD cases did not reveal any appreciable improvement in the predictive value of the sociodemographic, anthropometric, or fetal biometric variables for the combination of SD and birth injury (data not shown).

No maternal sociodemographic or clinical variables were significantly associated with SD (Table 1) except for epidural use in labor. Every woman experiencing an SD received

epidural anesthesia during labor as opposed to 82.4% ($p = 0.03$) of the women who did not experience an SD. Maternal gestational diabetes was diagnosed in 13% (3 of 23) of the SD cases as opposed to 4.5% in the unaffected deliveries. The corresponding p-value was 0.48, but the statistical power may be insufficient to confirm this difference as meaningful. No maternal anthropometric measures were significantly associated with SD (Table 2), although reported paternal height was greater among those pregnancies experiencing SD (177.5 ± 8.4 vs. 181.5 ± 9.2 cm; $p = 0.03$). Previously reported associations with prepregnancy maternal BMI ≥ 30.0 kg/m^2 (17.4 vs. 14.7%; $p = 0.72$) and gestational weight gain (11.1 vs. 11.4 lbs; $p = 0.78$) were not observed.

Differences in mean fetal biometric measures at the final study ultrasound between those pregnancies complicated by SD and those without SD are presented in Table 3. The mean AFMVP was significantly greater (OR: 1.32 [95% CI: 1.03, 1.69]) in the SD group (5.8 ± 1.7 cm) compared with uncomplicated deliveries (5.1 ± 1.5 cm). The difference between the AC and HC had a mean of 14.8 ± 22.8 mm for the 23 SD cases compared with 7.7 ± 19.7 mm for the uncomplicated deliveries. However, the unadjusted OR (95% CI) was only 1.02 (1.00, 1.04). Fig. 2 presents the ROC and associated AUCs for the measured and derived fetal biometrics as continuous variables.

Table 4 presents selected dichotomous cut-off points for fetal biometric measurements and their performance characteristics as predictors of SD within the simple logistic regression models. When using the Youden's J to dichotomize, sensitivities associated with various fetal biometric cut-off points ranged from 8.7% for AC ≥ 243.1 mm to 65.2% for the HC-to-AC ratio of <0.9. Specificity was overall higher than sensitivity, ranging from 50.4% for an FL/AC ratio of ≥0.2 mm to 98.5% for FL ≥ 50.7 mm. Using a fetal biometric clinical cut-off point selected to maintain high specificity (range 89.4% for FL-to-AC ratio of ≥0.2 to 99.1% for HL ≥ 69.2 mm) was associated with sensitivities that ranged from 13.0% (all biometrics except for HL) to 17.4% (HL ≥ 69.2 mm).

A final, best prediction model for SD was constructed using fetal biometric measures, maternal clinical factors, and their interactions. The model was internally valid with an AUC of 0.90 (95% CI: 0.81–0.99) in our test dataset (Fig. 3). At a model probability of 1%, sensitivity was 71.4%, specificity was 77.5%, positive predictive value was 3.5%, negative predictive value was 99.6%, and the false positive rate (1-specificity) was 22.5%.

Discussion

Principal Findings

While several fetal biometric measures are associated with SD, when measured over a continuum, fetal biometrics had little association with SD risk and a poor ability to reliably predict SD occurrence either individually or in combination with sociodemographic and clinical characteristics in a low-risk obstetrical population. When fetal biometric measures were dichotomized using Youden's J or clinically significant cut-off points selected to optimize specificity, greater differences in the odds of SD could be identified. However, even when these more significant biometric cut-off points are used, we were unable to create a final predictive model with test characteristics that could be clinically reliable. At an

anticipated outcome probability of 1%, which is consistent with the SD prevalence in this cohort, the sensitivity and positive predictive values were poor, and the false positive rate was unacceptably high.

Surprisingly, no maternal sociodemographic or clinical factors, other than epidural use in labor, were associated with an increased risk of SD in this population by univariate analysis. Factors previously reported to be associated with SD including maternal age, race, parity, operative vaginal delivery, fetal sex, and family history of diabetes were not significantly associated with SD in this prospective cohort. The occurrence of maternal gestational diabetes was also statistically unassociated with SD. However, the difference in SD occurrence (13% in women diagnosed with gestational diabetes vs. 4.5% in women without gestational diabetes) raises the possibility of insufficient statistical power to identify a truly meaningful difference. Also contrary to the findings of others, maternal prepregnancy weight, BMI, and gestational weight gain were not associated with SD.

Results in the Context of What Is Known

While multiple maternal, fetal, and obstetrical factors have been statistically associated with SD, their ability to predict this outcome has not been established.[3,26,27] The most consistently identified clinical risk factors for SD are fetal macrosomia and maternal gestational or pregestational diabetes.[2,4,28,29] Using birth weight or diabetes as predictors of SD suffers two major limitations. First, such prediction requires an accurate EFW which is problematic, especially at the extremes of fetal weights.[2,30,31] Second, only a minority of SD cases occur among macrosomic newborns or infants of diabetic mothers. Predicting SD in lower prevalence populations is a far greater obstetrical challenge and one which has not been well studied.

Interest in asymmetric fetal growth comes from fetal and neonatal anthropometric measurements indicating that infants of diabetic mothers experiencing SD had greater shoulder circumferences, larger chest-to-head and shoulder-to-head ratios and longer bisacromial lengths compared with unaffected infants.[12,13,32] Several late pregnancy ultrasound studies have suggested the AD–BPD difference to be a promising predictor of SD.[14–16,32,33] However, small sample sizes, retrospective designs, and inclusion criteria limited to diabetic mothers or EFWs >3,400 or >3,800 g restrict the usefulness or generalizability of these studies. In a broader-based retrospective study of 12,794 term singletons, Burkhardt et al[17] reported multiple fetal biometric parameters associated with SD, most notably the AD–BPD difference. Using an AD–BPD cut-off point of >2.6 cm, the number of cesareans needed to prevent one shoulder dystocia was 14.2 and 9.5 when combined with an EFW ≥ 3,500 g. In this investigation, only the AC-to-HC difference was of borderline significance (unadjusted OR: 1.02 [95% CI: 1.00,1.04]; $p = 0.09$) in terms of its association with SD. In all these studies, positive predictive values were poor and false positive rates were high due to the uncommon occurrence of SD even in higher risk cohorts. All these investigators cautioned against overinterpretation of their data until larger prospective studies could be performed.

The current study contributes to our understanding of SD prediction in that it represents a reassessment of the associations between fetal biometric, anthropometric, and maternal

clinical variables and SD in a prospective, multicentered, racial/ethnically diverse cohort study of women at low risk for excessive or asymmetric fetal growth. It confirms that these biometric measures cannot be reliably used to predict SD in a low prevalence cohort. This is important given the multiple methodologically inferior, retrospective studies previously described which have suggested potentially predictive biometric measures. Our null findings add strong support to the understanding that SD is not a predictable event in a low-risk obstetrical population despite consideration of a large number and combinations of available, potentially useful, maternal-fetal predictors.

Strengths and Limitations

The major strength of this study was the quality of the ultrasound data and prospective ascertainment of maternal and fetal anthropometrics as predictors of SD using a standardized protocol across 12 U.S. centers. The study was performed by specifically trained and study certified sonographers. Quality-control mechanisms insured that the ultrasound data were reliable and reproducible.[24] Other strengths are the use of EFW rather than birth weight to model prediction and the depth of the data achieved through the simultaneous evaluation of multiple fetal biometric parameters. Our study takes advantage of a demographically, racially, and ethnically diverse obstetrical population without recognizable medical or obstetrical conditions likely to increase the prevalence of SD. As a result, the SD prevalence in our study population was an anticipated 1.3%. Since the majority of SD occurs among women without identifiable risk factors for accelerated or asymmetric fetal growth, there is a clear need for studies performed in an unselected population such as ours.

We also acknowledge several limitations. Since fetal macrosomia and maternal diabetes are two significant risk factors, the exclusion of women with such histories will result in a decreased outcome prevalence and will diminish the performance parameters of any predictive model. The infrequent occurrence of SD makes predictive model building problematic, given the imbalance between the primary outcome and the multiple variables considered. Among women with gestational or pregestational diabetes, various degrees of suspected fetal macrosomia, or a prior pregnancy complicated by SD, the empiric SD risk could be as high as 10 to 25%. [2,4,5,10,34,35] It is impossible to know if the associations found in the current lower prevalence study population are proportional to cohorts with higher risk profiles.

We are unable to exclude the possibility of elective cesarean for suspected macrosomia in our cohort. As a competing risk bias, elective cesarean for suspected macrosomia could reduce the positive predictive value of any identified measures. “Suspected fetal macrosomia” was identified as the indication for cesarean delivery in only 15 of the 2,802 women included in this analysis, and it is unknown if these were elective antepartum or intrapartum indications. The lack of a standardized diagnostic outcome criteria for SD is also a limitation. Although objective criteria for SD diagnosis have been proposed, these were not being used at the participating centers where the clinical diagnosis was left to subjective impression of the delivering physician.[36] It is well documented that brachial plexus injury and clavicular fractures can occur in the absence of a subjectively diagnosed

SD.[2,10,37,38] It is possible that the diagnosis of SD was missed in these deliveries with birth-associated injury. A sensitivity analysis which added those cases of documented birth injury without reported SD did not reveal any significant differences in the predictive capability of fetal biometric measures.

Use of recalled pregravid weight and self-reported maternal height and paternal height and weight may also be considered a limitation, as it is inherently subjective. However, recalled pregravid weight is the current clinical standard in the United States, and the evidence suggests that maternal BMI is accurately classified based on maternal recall in 85% of pregnancies.[39] In a large study including more than 30,000 parenteral couples where paternal BMI was based on maternal report, agreement between maternal and paternal reported obesity was quite high (kappa = 0.91). The mean difference between paternal weight and maternal report was −0.3 ± 2.7 kg and the mean height difference was negligible (0.0 ± 1.5 cm).[40]

As previously mentioned, the infrequent occurrence of SD could have missed some significant associations as a result of type-II errors. This study was performed over 4 years at 12 different clinical sites. It is unlikely that a prospective study could be performed with such detailed fetal biometry and a large enough sample size to make these uncommon outcomes plentiful. Our final study ultrasound being 17.5 days prior to delivery may also be considered a limitation. However, this interval is consistent with the American Congress of Obstetricians and Gynecologists recommendation to repeat ultrasounds every 2 to 4 weeks when abnormalities of fetal growth are suspected.[41] Still, ultrasounds performed closer to delivery might have demonstrated stronger associations.

Clinical and Research Implications

While the association between maternal and fetal anthropometrics and SD may have etiologic importance, no combination of fetal biometric measures has been shown to reliably predict the clinical occurrence of SD. While excessive somatic growth (EFW and AC > HC and AD–BPD difference) has been reported to increase SD risk, we did not find these associations to stand out in this prospective, multicenter study of unselected relatively low-risk pregnancies. The association between SD and maternal epidural use could potentially represent a type-II error, however, it has been repetitively reported.[42,43] It is conceivable that the effect of epidural anesthesia on maternal pelvic floor muscle relaxation may impact the cardinal movements associated with normal fetal descent in the second stage of labor. That effect, along with the shape and type of the maternal pelvis, likely contributed to the elusiveness of predicting SD using ultrasound measures of fetal biometry.

In this low prevalence population, both individual and combined fetal biometric measures failed to provide sufficient positive predictive value for clinical use. False positive rates were unacceptably high. It remains to be determined what our findings might have been in a population of women with an a priori higher risk for SD such as those with poorly controlled diabetes, prior SD or macrosomia. Previous studies involving such women have also failed to identify any combinations of clinically reliable sociodemographic or biometric predictors capable of efficient SD prediction.[9–11]

Conclusion

In this low-risk obstetrical cohort followed prospectively, SD remains an essentially unpredictable clinical event. Maternal sociodemographics and anthropometrics had no association with SD in this cohort. While some significant associations were identified between selected fetal biometrics and SD, they could not be modeled, either individually or in combination, to predict SD in a clinically meaningful or reliable fashion. Interestingly, neither the univariable associations nor the best predictive continuous or dichotomous models identified differences in previously touted measurements of fetal asymmetry such as the HC/AC ratio, absolute differences between the AC and HC, or differences between the AD and BPD as determining factors in SD occurrence. Our final multivariable model, including both fetal biometry and maternal factors, performed poorly in this prospective cohort with a 1.3% risk of SD. The inability of detailed and multiple fetal and parenteral variables to predict SD risk in this prospective cohort is disappointing and confirms the unpredictability of SD, especially in low-risk populations. The methodological superiority of the current study, the simultaneous consideration of multiple fetal ultrasound-derived and maternal variables, and the focus on a low-risk obstetrical cohort strengthens and solidifies the existing literature informing clinicians that they must anticipate the possibility of SD with every delivery and be prepared to effectively manage the unexpected SD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Key Points

- SD unpredictable in low-risk women.
- Fetal biometry does not reliably predict SD.
- Epidural use associated with increased SD risk.
- SD prediction models clinically inefficient.

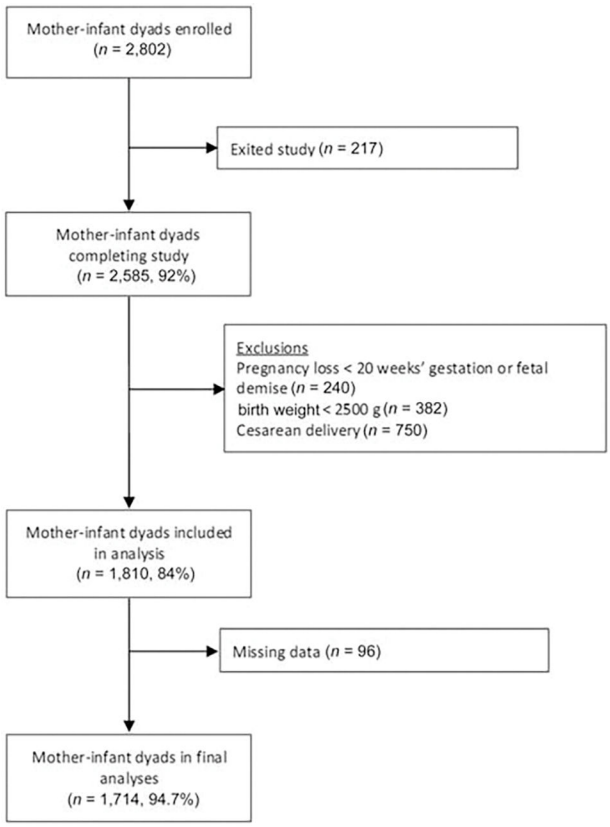

Fig. 1.
Flow diagram of study population and exclusions.

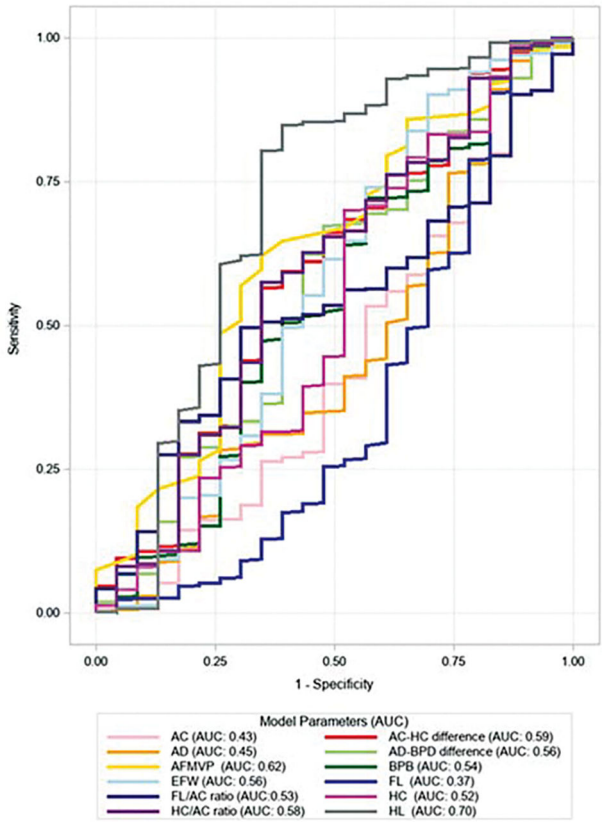

Fig. 2.

Receiver operating characteristic (ROC) curves for prediction of shoulder dystocia using univariate continuous fetal biometric measures. AC-HC, abdominal circumference-head circumference difference; AD, abdominal diameter; AD-BPD, abdominal diameter–biparietal diameter difference; AFMVP, amniotic fluid maximum vertical pocket; AUC, area under curve; BPD, biparietal diameter; EFW, estimated fetal weight; FL/AC, femur length/abdominal circumference ratio; HC, head circumference; HL, humerus length; HC/AC, head circumference/abdominal circumference ratio; SD, shoulder dystocia.

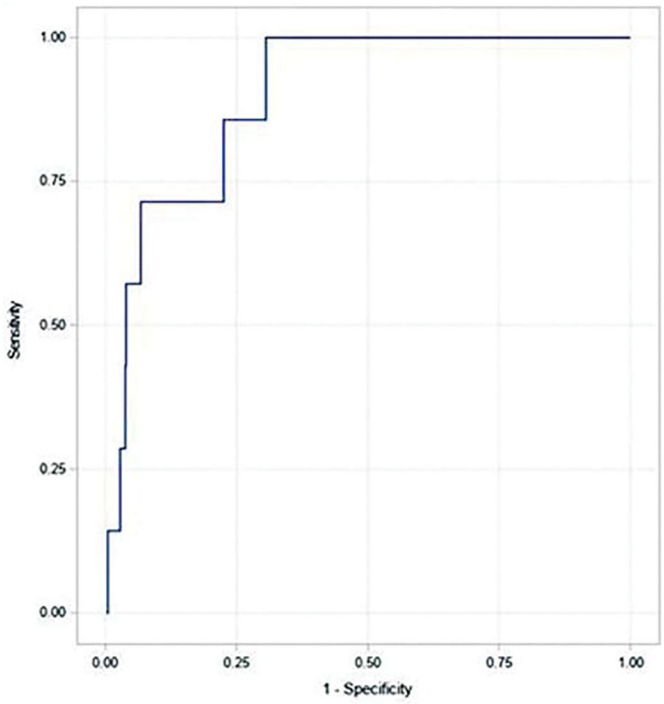

Fig. 3.

Receiver operating characteristic (ROC) curve for prediction of shoulder dystocia using multivariable model including fetal biometric measures and maternal factors. Represents internal validation study (n = 685; 9 cases) AUC = 0.98. AUC, area under curve; CI, confidence interval; SD, shoulder dystocia.

Am J Perinatol. Author manuscript; available in PMC 2023 December 30.

Table 1

Sample sociodemographics and clinical characteristics by shoulder dystocia

Sociodemographics and clinical characteristics[a]	**No shoulder dystocia** ***n*** **= 1,691**	**Shoulder dystocia** ***n*** **= 23**	**Total** ***n*** **= 1,714**	***p*-Value**[b]
Maternal age (y)	27.8 (5.3)	27.0 (6.9)	27.8 (5.4)	0.35
Gestational age at delivery (wk)	39.4 (1.2)	39.6 (1.0)	39.4 (1.2)	0.24
Gestational age at final ultrasound (wk)	37.0 (3.1)	36.7 (4.9)	37.0 (3.2)	0.24
Offspring sex				0.81
Male	840 (49.7%)	12 (52.2%)	852 (49.7%)	
Female	851 (50.3%)	11 (47.8%)	862 (50.3%)	
Maternal race/ethnicity				0.49
Non-Hispanic White	456 (27.0%)	8 (34.8%)	464 (27.1%)	
Non-Hispanic Black	448 (26.5%)	8 (34.8%)	456 (26.6%)	
Hispanic	499 (29.5%)	4 (17.4%)	503 (29.3%)	
Asian and Pacific Islander	288 (17.0%)	3 (13.0%)	291 (17.0%)	
Parity				0.37
0	771 (45.6%)	8 (34.8%)	779 (45.4%)	
1	572 (33.8%)	11 (47.8%)	583 (34.0%)	
2	348 (20.6%)	4 (17.4%)	352 (20.5%)	
Maternal highest level of education				0.23
Less than high school	200 (1 1.8%)	4 (17.4%)	204 (11.9%)	
High school diploma or GED or equivalent	317 (18.7%)	8 (34.8%)	325 (19.0%)	
Some college or associate degree	515 (30.5%)	6 (26.1%)	521 (30.4%)	
Bachelor’s degree	375 (22.2%)	3 (13.0%)	378 (22.1%)	
Master’s degree or advanced degree	284 (16.8%)	2 (8.7%)	286 (16.7%)	
Maternal marital status				0.35
Not married	442 (26.2%)	8 (34.8%)	450 (26.3%)	
Married or living with partner	1,247 (73.8%)	15 (65.2%)	1,262 (73.7%)	
Insurance				0.38
Managed	93 (5.5%)	3 (13.0%)	96 (5.6%)	
Medicaid	654 (38.7%)	10 (43.5%)	664 (38.7%)	
Other	98 (5.8%)	1 (4.3%)	99 (5.8%)	

Sociodemographics and clinical characteristics[a]	No shoulder dystocia *n* = 1,691	Shoulder dystocia *n* = 23	Total *n* = 1,714	*p*-Value[b]
Private	846 (50.0%)	9 (39.1%)	855 (49.9%)	
Maternal family history of GDM or diabetes				0.74
No	1,299 (76.8%)	17 (73.9%)	1,316 (76.8%)	
Yes	392 (23.2%)	6 (26.1%)	398 (23.2%)	
Maternal gestational diabetes				0.48
No	1,614 (95.5%)	20 (87.0%)	1,634 (95.3%)	
Yes	77 (4.5%)	3 (13.0%)	80 (4.7%)	
Mode of delivery				0.83
Spontaneous vaginal delivery	1,578 (93.3%)	21 (91.3%)	1,599 (93.3%)	
Outlet/low forceps or outlet vacuum	104 (6.2%)	2 (8.7%)	106 (6.2%)	
Mid-forceps or vacuum, forceps rotation	9 (0.5%)	0 (0.0%)	9 (0.5%)	
Mode of onset of labor				0.64
No labor	12 (0.7%)	0 (0.0%)	12 (0.7%)	
Spontaneous	1,193 (70.5%)	19 (82.6%)	1,212 (70.7%)	
Induced	483 (28.6%)	4 (17.4%)	487 (28.4%)	
Unknown	3 (0.2%)	0 (0.0%)	3 (0.2%)	
Oxytocin				0.38
No	1,103 (65.2%)	13 (56.5%)	1,116 (65.1%)	
Yes	588 (34.8%)	10 (43.5%)	598 (34.9%)	
Epidural				0.03
No	1,393 (82.4%)	23 (100.0%)	1,416 (82.6%)	
Yes	298 (17.6%)	0 (0.0%)	298 (17.4%)	
1 hour glucose at final visit (mg/dl)	111.6 (27.5)	118.5 (42.3)	111.7 (27.8)	0.45
Hematocrit at final visit (%)	34.9 (3.5)	34.5 (3.6)	34.9 (3.5)	0.67
Hemoglobin at final visit (g/dL)	11.7 (1.4)	11.5(1.1)	11.7 (1.4)	0.43

Abbreviations: GED, General Education Diploma; GDM, Gestational Diabetes.

[a]Continuous variables presented as mean (standard deviation) and categorical variables presented as N (%).

[b]*p*-Value for the comparison between shoulder dystocia and no shoulder dystocia groups calculated using Chi-square or Fisher's exact (categorical variables).

Am J Perinatol. Author manuscript; available in PMC 2023 December 30.

Table 2

Maternal and paternal anthropometrics by pregnancy outcome

Paternal and maternal anthropometries[a]	**No shoulder dystocia** ***n*** **= 1,691**	**Shoulder dystocia** ***n*** **= 23**	**Total** ***n*** **= 1,714**	***p*-Value**[b]
Paternal height (cm)	177.5 (8.4)	181.5 (9.2)	177.6 (8.4)	0.03
Paternal weight (kg)	84.2 (15.8)	81.8 (17.3)	84.1 (15.8)	0.53
Maternal Prepregnancy BMI				0.72
BMI <30 kg/m^2	1442 (85.3%)	19 (82.6%)	1461 (85.2%)	
BMI ≥30 kg/m^2	249 (14.7%)	4 (17.4%)	253 (14.8%)	
Maternal natural waist (cm)	81.6 (11.0)	84.9 (13.1)	81.6 (11.0)	0.15
Maternal waist over iliac crest (cm)	92.6 (12.3)	94.3 (14.8)	92.7 (12.3)	0.53
Maternal hip circumference (cm)	102.5 (11.1)	104.4 (11.6)	102.5 (11.1)	0.41
Maternal upper arm length (cm)	35.3 (2.9)	36.4 (3.2)	35.3 (2.9)	0.06
Maternal mid-upper arm circumference (cm)	28.9 (4.3)	29.4 (4.1)	28.9 (4.3)	0.57
Maternal triceps skinfold (mm)	25.9 (8.2)	25.9 (7.8)	25.9 (8.2)	0.98
Maternal subscapular skinfold (mm)	21.9 (8.4)	22.9 (7.8)	21.9 (8.4)	0.62
Gestational weight gain (kg)	11.4 (6.6)	11.1 (6.1)	11.4 (6.6)	0.78

Abbreviations: BMI, body mass index.

[a]Continuous variables presented as mean (standard deviation) and categorical variables presented as N (%).

Table 3

Unadjusted association of fetal biometrics as continuous measures at final visit by pregnancy outcome

Fetal biometrics[a]	No. of measurements	Shoulder dystocia Mean (SD)	No shoulder dystocia Mean (SD)	Unadjusted OR (95% CI)
AC	1,712	327.7 (58.7)	328.7 (38.2)	1.00 (0.99, 1.01)
AC-HC difference	1,712	14.8 (22.8)	7.7 (19.7)	1.02 (1.00, 1.04)[b]
AFMVP	1,700	5.8 (1.7)	5.1 (1.5)	1.32 (1.03, 1.69)[c]
AD	1,712	104.3 (19.4)	103.9 (14.1)	1.00 (0.97, 1.03)
AD-BPD difference	1,712	18.3 (9.9)	16.1 (8.1)	1.03 (0.98, 1.09)
BPD	1,712	86.2 (12.5)	88.1 (8.5)	0.98 (0.95, 1.02)
EFW	1,712	2,944 (1018)	2,921 (675)	1.00 (1.00, 1.00)
FL	1,712	68.5 (12.2)	68.6 (7.4)	1.00 (0.95, 1.05)
FL/AC ratio	1,712	0.2 (0.0)	0.2 (0.0)	-
HC	1,712	312.0 (44.0)	319.5 (29.8)	0.99 (0.99, 1.00)
HC/AC ratio	1,712	1.0 (0.1)	1.0 (0.1)	0.02 (0.00, 15.92)
HL	1,712	61.6 (10.5)	60.3 (6.4)	1.05 (0.95, 1.17)

Abbreviations: AC, abdominal circumference; AD, abdominal diameter; AFMVP, amniotic fluid maximum vertical pocket; BPD, biparietal diameter; CI, confidence interval; EFW, estimated fetal weight; FL, femur length; HC, head circumference; HL, humerus length; OR, odds ratio; SD, standard deviation.

[a] AC, AD, BPD, FL, HC, HL measured in millimeters; AFMVP measured in centimeters; and EFW measured in grams.

[b] $p = 0.09$.

[c] $p = 0.03$.

Table 4

Unadjusted associations and performance characteristics between dichotomized fetal biometric measurements and shoulder dystocia in simple logistic regression models

Youden's J[a]	**AC**	**AC-HC difference**	**AFMVP**	**AD**	**AD-BPD difference**	**BPD**	**EFW**	**FL**	**FL/AC ratio**	**HC**	**HC/AC ratio**	**HL**
Cut-off point	243.1	11.2	5.5	109.6	20.1	86.8	3,668	50.7	0.2	316.3	1.0	64.7
OR (95% Cl)[b]	0.22 (0.05, 0.96)	2.05 (0.88, 4.76)	3.06 (1.29, 7.26)	2.03 (0.89, 4.63)	1.92 (0.84, 4.39)	0.74 (0.31, 1.76)	3.22 (1.25, 8.27)	0.10 (0.03, 0.36)	0.63 (0.27, 1.47)	0.48 (0.21, 1.09)	0.39 (0.17, 0.93)	8.75 (3.75, 20.43)
AUC	0.54	0.59	0.64	0.59	0.58	0.54	0.59	0.56	0.56	0.59	0.56	0.73
Sensitivity (%)	8.7	60.9	65.2	52.2	47.8	34.8	26.1	13.0	60.9	47.8	65.2	60.9
Specificity (%)	98.0	56.8	62.0	65.1	67.7	71.7	90.1	98.5	50.4	69.5	57.6	84.9
PPV (%)	5.6	1.9	2.3	2.0	2.0	1.7	3.5	10.7	1.6	2.1	2.1	5.2
NPV (%)	98.8	99.1	99.2	99.0	99.0	98.8	98.9	98.8	99.0	99.0	99.2	99.4
Clinical criteria[c]												
Cut-off point	243.4	46.7	8.6	121.6	27.6	71.7	4002	50.7	0.2	262.2	0.9	69.2
OR (95% Cl)	0.14 (0.04, 0.50)	6.18 (1.77, 21.66)	6.47 (1.84, 22.70)	5.02 (1.44, 17.46)	1.91 (0.56, 6.52)	0.15 (0.04, 0.51)	4.82 (1.39, 16.73)	0.10 (0.03, 0.36)	0.79 (0.23, 2.68)	0.14 (0.04, 0.48)	0.13 (0.04, 0.47)	23.50 (7.14, 77.41)
AUC	0.56	0.55	0.55	0.55	0.53	0.56	0.55	0.56	0.52	0.56	0.56	0.58
Sensitivity (%)	13.0	13.0	13.0	13.0	13.0	13.0	13.0	13.0	13.0	13.0	13.0	17.4
Specificity (%)	97.9	97.6	97.7	97.1	92.7	97.9	97.0	98.5	89.4	98.0	98.1	99.1
PPV (%)	7.9	7.0	7.3	5.8	2.4	7.7	5.6	10.7	1.7	8.1	8.3	21.1
NPV (%)	98.8	98.8	98.8	98.8	98.7	98.8	98.8	98.8	98.7	98.8	98.8	98.9

Abbreviations: AC, abdominal circumference; AD, abdominal diameter; AFMVP, amniotic fluid maximum vertical pocket; AUC, area under the curve; BPD, biparietal diameter; Cl, confidence interval; EFW, estimated fetal weight; FL, femur length; HC, head circumference; HL, humerus length; NPV, negative predictive value; OR, odds ratio; PPV, positive predictive value.

Note: AC, AD, BPD, FL, HC, HL measured in millimeters; AFMVP measured in centimeters; EFW measured in grams.

[a] Youden's J optimizes discriminating power of a variable if equal weight is given to both sensitivity and specificity.

[b] OR for greater than the cut-off point compared with less than with the 95th percentile confidence intervals.

[c] Clinical criteria was defined as the cut-off point which maximized specificity while maintaining a sensitivity of at least 10%.