



May 2016 Literature Alert

1.

Am J Obstet Gynecol. 2016 Apr;214(4):509.e1-7. doi: 10.1016/j.ajog.2015.10.931. Epub 2015 Nov 4.

[INTERGROWTH-21st vs customized birthweight standards for identification of perinatal mortality and morbidity.](#)

Anderson NH, Sadler LC, McKinlay CJ, McCowan LM.

Abstract

BACKGROUND:

The recently published INTERGROWTH-21st Project international population standard for newborn size is intended for global use, but its ability to identify small infants at risk of adverse outcomes in a general obstetric population has not been reported.

OBJECTIVE:

The objective of the study was to compare adverse neonatal outcomes among small-for-gestational-age (SGA) infants between the INTERGROWTH-21st standard and a customized birthweight standard (accounting for maternal characteristics of height, weight, parity, and ethnicity). We hypothesized that in a multiethnic general obstetric population in Auckland, New Zealand, a customized birthweight standard would better identify SGA infants at-risk of neonatal morbidity/mortality and stillbirth than the INTERGROWTH-21st standard.

STUDY DESIGN:

Using prospectively gathered maternity data from a general obstetric population in Auckland, New Zealand, from 2006 to 2013 (n = 53,484 births at ≥ 33 weeks), infants were classified as SGA (birthweight < 10th centile) by INTERGROWTH-21st and customized standards. Infants were further categorized as SGA by both criteria, INTERGROWTH-21st only, customized only, or not SGA (met neither criteria). Composite adverse neonatal outcome was defined as neonatal death, neonatal intensive care admission > 48 hours, or ventilation > 4 hours or 5-minute Apgar score < 7. Relative risks for primary outcomes were estimated using modified Poisson regression, with the non-SGA group as the referent.

RESULTS:

Incidence of SGA was 4.5% by INTERGROWTH-21st and 11.6% by customized standard. Compared with those not SGA, infants identified as small for gestational age by both criteria had the highest risk of adverse neonatal outcome (relative risk [RR], 4.1, 95% confidence interval [CI], 3.7-4.6) and stillbirth (RR, 8.3, 95% CI, 5.1-13.4). Infants SGA by customized standard only (n = 4015) had an increased risk of adverse neonatal outcome (RR, 2.0, 95% CI, 1.8-2.2) and stillbirth (RR, 3.0, 95% CI, 1.7-5.3). Few infants

were identified as SGA by INTERGROWTH-21st only ($n = 172$), and risks of adverse neonatal outcome and stillbirth were not increased. Findings were unchanged when analyses were limited to term infants ($n = 50,739$). The INTERGROWTH-21st standard identified more Indian (12.8%) and Asian (5.8%) but fewer European (3.0%) and Pacific (2.9%) infants as SGA ($P < .01$). Customized criteria identified more than 3 times as many SGA infants among Maori (14.5%), Pacific (13.5%), and European (11.2%) infants and twice as many among Asian (10.3%) infants ($P < 0.01$) compared with INTERGROWTH-21st criteria. The majority of SGA infants by INTERGROWTH-21st only were born to Indian and Asian mothers (95.4%).

CONCLUSIONS:

In our general obstetric population, birthweight customization identified more SGA infants at risk of perinatal mortality and morbidity compared with the INTERGROWTH-21st standard. The INTERGROWTH-21st standard failed to detect many at-risk SGA infants, particularly among ethnic groups with larger maternal size while disproportionately identifying higher rates of SGA among those with smaller maternal size. Local validation is needed prior to implementation of the INTERGROWTH-21st standard to avoid misclassification of infant birth size.

Copyright © 2016 Elsevier Inc. All rights reserved.

KEYWORDS:

INTERGROWTH-21st Project; customized birthweight; perinatal morbidity; perinatal mortality; small for gestational age

PMID: 26546850 [PubMed - in process]

2.

Am J Obstet Gynecol. 2016 Apr;214(4):484-9. doi: 10.1016/j.ajog.2015.10.926. Epub 2015 Nov 4.

[Second-stage labor: how long is too long?](#)

Leveno KJ, Nelson DB, McIntire DD.

Abstract

The management of labor has come under increased scrutiny due to the rapid escalation of cesarean delivery in the United States. A workshop of the Society for Maternal-Fetal Medicine, the Eunice Kennedy Shriver National Institute of Child Health and Human Development, and the American Congress of Obstetricians and Gynecologists was convened to address the rising cesarean delivery rates and one of their recommendations was that the accepted upper limit of the second stage of labor should be increased to ≥ 4 hours in nulliparous women with epidural analgesia and to ≥ 3 hours in parous women with epidural. This led to the inaugural Obstetric Care Consensus series document, "Safe Prevention of the Primary Cesarean Delivery," wherein the workshop recommendations on second-stage labor were promulgated nationally. The result is that the now acceptable maximum length of the second stage of labor exceeds the obstetric precepts that have been in use for >50 years. In this Clinical Opinion, we review the evidence on infant safety, vis-à-vis length of the second stage of labor. Our examination of the evidence begins at the outset of the 20th century and culminates in the very recent (2014) recommendation to abandon the long accepted obstetric paradigm that second-stage labor >3 hours in nulliparous women with labor epidural is unsafe for the unborn infant. We conclude that the currently available evidence fails to support the Obstetric Care Consensus position that longer second-stage labor

is safe for the unborn infant. Indeed, the evidence suggests quite the opposite. We suggest that when infant safety is at stake the evidence should be robust before a new clinical road is taken. The evidence is not robust.

Copyright © 2016 Elsevier Inc. All rights reserved.

KEYWORDS:

cesarean delivery; epidural analgesia; infant outcome; prolonged; second stage of labor; second-stage labor

PMID: 26546847 [PubMed - in process]

3.

N Engl J Med. 2016 Apr 21;374(16):1506-9. doi: 10.1056/NEJMp1602708. Epub 2016 Mar 9.

Zika Virus as a Cause of Neurologic Disorders.

Broutet N, Krauer F, Riesen M, Khalakdina A, Almiron M, Aldighieri S, Espinal M, Low N, Dye C.

PMID: 26959308 [PubMed - indexed for MEDLINE]

NO ABSTRACT- below is full text

Zika virus infections have been known in Africa and Asia since the 1940s, but the virus's geographic range has expanded dramatically since 2007. Between January 1, 2007, and March 1, 2016, local transmission was reported in an additional 52 countries and territories, mainly in the Americas and the western Pacific, but also in Africa and southeast Asia. Zika virus infections acquired by travelers visiting those countries have been discovered at sites worldwide. *Aedes aegypti* mosquitoes are the principal vectors, though other mosquito species may contribute to transmission. The virus was found to be neurotropic in animals in experiments conducted in the 1950s, and recent experiments have shown how it can cause neural-cell death. A rise in the incidence of Guillain-Barré syndrome, an immune-mediated flaccid paralysis often triggered by infection, was first reported in 2013 during a Zika outbreak in French Polynesia. An increase in the incidence of microcephaly, a clinical sign that can be caused by underdevelopment of the fetal brain, was first reported in northeastern Brazil in 2015, after Zika virus transmission had been confirmed there. These reports of excess cases of Guillain-Barré syndrome and microcephaly led the World Health Organization (WHO) to declare a Public Health Emergency of International Concern on February 1, 2016, and to recommend accelerated research into possible causal links between Zika virus and neurologic disorders.¹

As researchers investigate whether and by what mechanisms Zika virus infections could affect the nervous system, there is a key question for public health: How can currently available evidence about causality guide the choice and implementation of interventions? For this purpose, the WHO is developing a framework for the systematic appraisal of evidence about these causal relationships. How does the available evidence inform current WHO recommendations, and what are the priorities for research going forward?

Besides advancing scientific understanding, the main practical purpose of investigating causality is to evaluate, as accurately as possible, what reduction in the incidence of illness (here, especially neurologic disorders) can be expected from reducing human exposure to the putative cause (Zika virus infection).

The conceptual framework is based on factors first proposed by Bradford Hill and commonly used to assess causality: temporality (cause precedes effect), biologic plausibility of causal mechanisms, consistency (same association found in different studies and populations), strength of association (as measured by risk ratio, rate ratio, or odds ratio in cohort or case–control studies), exclusion of alternative explanations, dose–response relationship, cessation (removing the supposed cause reverses the effect), and analogy to cause-and-effect relationships in other diseases.² Temporality is the single necessary condition; none of the factors on its own is sufficient.

Causal relationships cannot be proven with absolute certainty in epidemiologic studies, but these factors help analysts judge the existence and strength of possible causal links. Their assessment should be complemented by controlled experiments, the most robust approach to drawing inferences about cause and effect.

A systematic strategy for identifying relevant evidence will enable a transparent and replicable approach that can be updated to capture new information. Study methods can be assessed for risks of selection and measurement bias, confounding, and the effect of chance. To illustrate the approach, we conducted a search of PubMed and selected journal and public health websites for information posted through March 4, 2016. The table Studies of Guillain–Barré Syndrome or Microcephaly in Association with Zika Virus Infection, According to Study Design and Date of Publication, and the Supplementary Appendix (available with the full text of this article at NEJM.org) provide a preliminary summary of population- and individual-level studies on possible associations between Zika virus infection and Guillain–Barré syndrome or microcephaly.

We found three published reports on Guillain–Barré syndrome studied at the population level. During the 2013–2014 outbreak in French Polynesia, the rise and fall of Zika virus infections was followed by a similar rise and fall in the incidence of Guillain–Barré syndrome, with a delay of about 3 weeks.³ In the Americas, Guillain–Barré syndrome in the presence of Zika virus has now been reported to the WHO from Brazil, Colombia, El Salvador, Martinique, Panama, Puerto Rico, Suriname, and Venezuela, but we found no reports from these countries linking the syndrome with trends in Zika virus infections.

One study of microcephaly showed a higher-than-expected incidence in the state of Paraíba, Brazil, during the period when Zika transmission began, but data on the timing of Zika infections were not available (see table). In the state of Bahia, Brazil, an outbreak of cases of acute rash, suspected to be Zika virus disease, was followed by an increase in cases of microcephaly. Additional surveillance data describing the temporal relationship between Zika infections and neurologic disorders are likely to be published soon. Some recent epidemiologic observations invite further investigation: microcephaly has been reported in association with Zika infection in Brazil, but not yet in neighboring countries, perhaps because it is still too soon after the introduction of the virus. An outbreak of Zika virus infection in Cape Verde during 2015–2016 involving thousands of cases and possibly caused by an African strain of the virus has not been linked to any neurologic disorders.⁴

At the level of individual patients, we found 3 studies on Guillain–Barré syndrome and 14 on microcephaly. The only published case–control study showed, among other results, that 41 of 42 patients with Guillain–Barré syndrome diagnosed during the 2013–2014 outbreak in French Polynesia (98%) were carrying Zika virus antibodies, as compared with 35 of 98 hospitalized controls (odds ratio, 59.7; 95% confidence interval, 10.4 to ∞).³ Serologic tests excluded some other infectious triggers for Guillain–Barré syndrome, and there was no association between the syndrome and exposure to dengue, which has the same mosquito vector and was circulating at the same time as the Zika virus outbreak.

One prospective study has compared ultrasound findings in pregnant women with confirmed Zika virus disease and in women with rash apparently from other causes (see table). Ultrasound findings were abnormal, including indications of microcephaly, in 12 of 42 women with Zika virus disease and were normal in all 16 women with no Zika virus disease. Eleven other reports, published between November 2015 and February 2016, involved a total of 93 neonates or fetuses with microcephaly, all in, or linked to, Brazil. In 9 of the 93 cases, Zika infection was detected in fetal or neonatal brain tissue or in amniotic fluid. In 4 cases, Zika virus was found in the brain but not in other organs on postmortem examination. In most case reports or case series, other infections and toxic exposures known to cause microcephaly were not completely excluded. One additional report compiled after the Zika virus outbreak in French Polynesia identified 17 cases of fetal or neonatal brain malformations, which included a number of cases of microcephaly.

The prevailing uncertainty about Zika virus infection and its consequences is now driving a vigorous program of research. One case–control study of Guillain–Barré syndrome and one cohort study of pregnant women described above provide evidence for a causal link. However, most of the data summarized here derive from studies whose designs are typically classified as weak, and the data are not entirely consistent. The available data are mainly observations regarding temporal associations between infection and disease from routine population surveillance and clinical and pathological studies of single cases or groups of cases. Such data are essential for the discovery of new phenomena and as a source of testable hypotheses about cause and effect,⁵ but a more comprehensive approach to causality is needed. The WHO framework will set out research questions to address the various dimensions of causality as they apply to Zika virus and neurologic disorders and will ensure a systematic review of the literature to synthesize the evidence. Further case–control and cohort studies are already under way to fill critical knowledge gaps.

Even with limited evidence linking Zika virus to neurologic disorders, the severe potential risks demand decisive, immediate action to protect public health. The WHO recommends applying key interventions such as intensive mosquito control; personal protection against mosquito bites; provision of appropriate clinical care for all patients with Guillain–Barré syndrome and for women before, during, and after pregnancy; and prevention of Zika virus transmission through sexual contact or blood transfusion.⁴ Most of these are not new interventions, but they do need strengthening. Populations must be informed of the potential current and future risks of neurologic disorders, wherever the virus is being or could be locally transmitted and in other regions inhabited by the mosquito vectors. As the putative link between Zika virus and neurologic disorders is reinforced, refined, or even refuted, public health measures will be adjusted accordingly.

4.

Am J Epidemiol. 2016 Apr 6. pii: kww263. [Epub ahead of print]

[Maternal Fatty Acid Status During Pregnancy and Child Autistic Traits The Generation R Study.](#)

Steenweg-de Graaff J, Tiemeier H, Ghassabian A, Rijlaarsdam J, Jaddoe VW, Verhulst FC, Roza SJ.

Abstract

ω-3 and ω-6 polyunsaturated fatty acids are important for brain function and development. We examined whether maternal polyunsaturated fatty acid status during pregnancy affects risk of autistic traits in childhood. Within the Generation R cohort, we measured maternal plasma polyunsaturated

fatty acid concentrations and the ω -3: ω -6 ratio in midpregnancy (Rotterdam, the Netherlands, 2001-2005). Child autistic traits at 6 years were assessed by using the Social Responsiveness Scale short form in 4,624 children. A lower maternal ω -3: ω -6 ratio during pregnancy was associated with more autistic traits in the offspring ($\beta = -0.008$, 95% confidence interval: $-0.016, -0.001$). In particular, a higher total ω -6 and linoleic acid status were associated with more autistic traits (all P's < 0.05). Associations were independent of child intelligence, suggesting that the fatty acid distribution specifically affects the development of autistic traits in addition to general neurodevelopment. Maternal plasma ω -3 status was not associated with child autistic traits and, consistently, neither was prenatal dietary fish intake. Our study shows that a lower prenatal ω -3: ω -6 ratio is associated with more child autistic traits, which is largely accounted for by higher ω -6 instead of lower ω -3 status. These results suggest a biological pathway between maternal fatty acid intake during pregnancy and autistic traits in the offspring.

© The Author 2016. Published by Oxford University Press on behalf of the Johns Hopkins Bloomberg School of Public Health. All rights reserved. For permissions, please e-mail: journals.permissions@oup.com.

KEYWORDS:

autistic traits; child; polyunsaturated fatty acids; pregnancy; ω -3: ω -6 fatty acid ratio

PMID: 27052119 [PubMed - as supplied by publisher]

5.

J Matern Fetal Neonatal Med. 2016 Apr;29(8):1252-6. doi: 10.3109/14767058.2015.1043262. Epub 2015 Jun 1.

[Neonatal outcome of late preterm uncomplicated monochorionic twins: what is the optimal time for delivery?](#)

Berezowsky A, Mazkereth R, Ashwal E, Mazaki-Tovi S, Schiff E, Weisz B, Lipitz S, Yinon Y.

Abstract

OBJECTIVE:

To determine the neonatal outcome at late prematurity of uncomplicated monochorionic (MC) twin pregnancies.

METHODS:

A retrospective cohort study of 166 patients with uncomplicated MC diamniotic twins delivered between 34 and 37 weeks of gestation at a single tertiary center. The study population was classified into four groups according to the gestational age at delivery: (1) 34 weeks, (2) 35 weeks, (3) 36 weeks and (4) 37 weeks. Neonatal outcome measures were compared between the groups.

RESULTS:

Neonatal morbidity was significantly higher at 34 weeks of gestation compared to the other three groups including respiratory distress syndrome, oxygen requirement, hypothermia and hyperbilirubinemia. Moreover, the rate of admission to the special care unit and need for phototherapy were significantly higher in newborns born at 36 weeks compared to 37 weeks of gestation ($p = 0.02$ and

0.03 respectively). Multiple regression analysis revealed that the risk for adverse neonatal outcome was significantly associated with gestational age at delivery. Of note, there were no fetal or neonatal deaths in our cohort.

CONCLUSIONS:

The risk of neonatal morbidity of uncomplicated MC twins delivered at 34-37 weeks of gestation significantly decreases with advanced gestation. Therefore, under close fetal surveillance, uncomplicated MC twin pregnancies should be delivered at 37 weeks of gestation.

KEYWORDS:

Late prematurity; monochorionic twins; neonatal outcome; timing of delivery

PMID: 26030679 [PubMed - in process]

6.

Am J Perinatol. 2016 Apr;33(5):473-9. doi: 10.1055/s-0035-1566308. Epub 2015 Nov 2.

[Can Transabdominal Cervical Length Measurement Exclude Short Cervix?](#)

Rhoades JS, Park JM, Stout MJ, Macones GA, Cahill AG, Tuuli MG.

Abstract

Objective This study aims to determine if transabdominal (TA) cervical length may be used to rule out a short cervix on transvaginal (TV) ultrasound. **Study Design** We conducted a prospective cohort study of women undergoing routine anatomic survey at 17 to 23 weeks gestation. TA and TV cervical length measurements were obtained in each patient. A short cervix was defined as TV cervical length < 30 mm. Predictive characteristics were calculated for different cutoff values of TA cervical length. **Results** There were 404 patients enrolled, a TA cervical length could not be obtained in 83 women (20.6%) and 318 women had both TA and TV measurements. Of those, 14 (4.4%) had a TV cervical length < 30 mm. TA cervical length measurement \geq 35 mm excluded the possibility of TV cervical length < 30 mm (negative predictive value, 99.5%; 95% confidence interval [CI], 97.4; 100%). In our cohort, 67.6% (95% CI, 62.2; 72.7%) of TV ultrasounds could have been avoided using a TA cervical length cutoff of \geq 35 mm. **Conclusion** ATA cervical length of at least 35 mm excludes a short cervix of < 30 mm. While TA cervical length screening may not be feasible in 1 in 5 women, it may be used to decrease the burden of universal TV cervical length screening.

Thieme Medical Publishers 333 Seventh Avenue, New York, NY 10001, USA.

PMID: 26523740 [PubMed - in process]

7.

Circulation. 2016 Apr 5;133(14):1397-409. doi: 10.1161/CIRCULATIONAHA.115.020491.

[Peripartum Cardiomyopathy.](#)

Arany Z, Elkayam U.

Abstract

Peripartum cardiomyopathy is a potentially life-threatening pregnancy-associated disease that typically arises in the peripartum period and is marked by left ventricular dysfunction and heart failure. The disease is relatively uncommon, but its incidence is rising. Women often recover cardiac function, but long-lasting morbidity and mortality are not infrequent. Management of peripartum cardiomyopathy is largely limited to the same neurohormonal antagonists used in other forms of cardiomyopathy, and no proven disease-specific therapies exist yet. Research in the past decade has suggested that peripartum cardiomyopathy is caused by vascular dysfunction, triggered by late-gestational maternal hormones. Most recently, information has also indicated that many cases of peripartum cardiomyopathy have genetic underpinnings. We review here the known epidemiology, clinical presentation, and management of peripartum cardiomyopathy, as well as the current knowledge of the pathophysiology of the disease.

© 2016 American Heart Association, Inc.

KEYWORDS:

cardiomyopathies; heart failure; pre-eclampsia; pregnancy

PMID: 27045128 [PubMed - in process]

8.

Am J Obstet Gynecol. 2016 May;214(5):559-65. doi: 10.1016/j.ajog.2016.01.182. Epub 2016 Jan 29.

[Obesogens: an emerging threat to public health.](#)

Janesick AS, Blumberg B.

Abstract

Endocrine disrupting chemicals (EDCs) are defined as exogenous chemicals, or mixtures of chemicals, that can interfere with any aspect of hormone action. The field of endocrine disruption is historically rooted in wildlife biology and reproductive endocrinology where EDCs are demonstrated contributors to infertility, premature puberty, endometriosis, and other disorders. Recently, EDCs have been implicated in metabolic syndrome and obesity. Adipose tissue is a true endocrine organ and, therefore, an organ that is highly susceptible to disturbance by EDCs. A subset of EDCs, called "obesogens," promote adiposity by altering programming of fat cell development, increasing energy storage in fat tissue, and interfering with neuroendocrine control of appetite and satiety. Obesity adds more than \$200 billion to US healthcare costs and the number of obese individuals continues to increase. Hence, there is an urgent, unmet need to understand the mechanisms underlying how exposures to certain EDCs may predispose our population to be obese. In this review, we discuss the history of obesogen discovery from its origins in reproductive biology to its latest role in the transgenerational inheritance of obesity in mice. We discuss the development of adipose tissue in an embryo, maintenance of adipocyte number in adults, how EDC disruption programs stem cells to preferentially make more adipocytes, the mechanisms by which chemicals can permanently alter the germline epigenome, and whether there are barriers to EDCs in the gametes.

Copyright © 2016 Elsevier Inc. All rights reserved.

KEYWORDS:

adipogenesis; endocrine disruptors; metabolic disruptors; obesogens; transgenerational obesity

PMID: 26829510 [PubMed - in process] PMCID: PMC4851574 [Available on 2017-05-01]

9.

J Perinatol. 2016 Apr;36(4):278-83. doi: 10.1038/jp.2015.202. Epub 2016 Jan 7.

[Maternal and neonatal outcomes in obese women who lose weight during pregnancy.](#)

Cox Bauer CM, Bernhard KA, Greer DM, Merrill DC.

Abstract

OBJECTIVE:

To evaluate neonatal and maternal outcomes in obese pregnant women whose weight gain differed from the Institute of Medicine (IOM) recommendations.

STUDY DESIGN:

Maternal and neonatal outcomes associated with weight change in pregnancy were retrospectively investigated in women with obesity (body mass index (BMI) ≥ 30 kg m⁻²); N=10734) who gave birth at 12 hospitals. Using a 1:1:1:1 design (n=778 matched groups), we matched women with obesity who lost, maintained, gained appropriate (IOM recommended) and gained excessive weight during pregnancy by gestational age at delivery, maternal age, race/ethnicity, prepregnancy BMI, chronic hypertension, pregestational diabetes and smoking status. Regression techniques were used to adjust for confounders and compare outcomes across weight change categories.

RESULT:

Compared with IOM recommendations, weight loss was associated with twofold greater odds of low birth weight infants and a mean decrease in estimated blood loss of 30 ml; excessive weight gain was associated with doubled odds of gestational hypertension or preeclampsia, fourfold greater odds of macrosomia and a mean decrease in 5-min APGAR of 0.09. From lost to excessively gained weight, the odds of cesarean delivery increased 1.4 times and mean infant birth weight increased by 197 g. In contrast, the odds of small-for-gestational age were 1.8 times greater for women who lost than gained excessive weight.

CONCLUSION:

Weight loss in obese pregnant women is associated with increased risk for low birth weight neonates but significantly decreased or maintained risk for other maternal and neonatal morbidities, as compared with appropriate or excessive weight gain. This study supports re-evaluation of the current IOM guidelines for women with obesity.

PMID: 26741574 [PubMed - in process]

10.

[Relationship Between Third-Trimester Sonographic Estimate of Fetal Weight and Mode of Delivery.](#)

Yee LM, Grobman WA.

Abstract

OBJECTIVES:

Some have suggested, based on limited data, that knowledge of an estimated fetal weight from a sonogram in a low-risk population, particularly in the setting of a larger fetus, is associated with increased risk of cesarean delivery. We aimed to investigate, among women delivering neonates weighing greater than 3500 g, whether having had a sonographically estimated fetal weight in temporal proximity to delivery was associated with the risk of cesarean delivery.

METHODS:

We conducted a retrospective cohort study of term nulliparous women delivering live-born, cephalic, singleton, nonanomalous fetuses with birth weights of greater than 3500 g. The study was powered to detect a 30% change in cesarean delivery frequency with the presence of a sonographic examination after 36 weeks' gestation.

RESULTS:

Of the 2099 women meeting inclusion criteria, 419 (20%) had a sonographic examination after 36 weeks' gestation. Women were similar with respect to demographic and obstetric characteristics regardless of whether they underwent sonography. There were no differences in rates of cesarean delivery regardless of whether women had or did not undergo sonography after 36 weeks (33.2% versus 29.4%, respectively; $P = .13$). There also were no differences in rates of chorioamnionitis, postpartum hemorrhage, episiotomy, third- or fourth-degree perineal laceration, or neonatal adverse outcomes based on sonographic status. Findings were similar in a multivariable analysis, as well as when the study population was restricted to those with birth weights of greater than 4000 and 4500 g.

CONCLUSIONS:

In this population of neonates weighing greater than 3500 g, the presence of a sonographic examination was not associated with the frequency of cesarean delivery.

© 2016 by the American Institute of Ultrasound in Medicine.

KEYWORDS:

cesarean delivery; estimated fetal weight; growth sonography; mode of delivery; obstetric ultrasound

PMID: 26931788 [PubMed - in process]

11.

Santana DS, Cecatti JG, Surita FG, Silveira C, Costa ML, Souza JP, Mazhar SB, Jayaratne K, Qureshi Z, Sousa MH, Vogel JP; WHO Multicountry Survey on Maternal and Newborn Health Research Network.

Abstract

OBJECTIVE:

To evaluate maternal complications (potentially life-threatening conditions, maternal near miss, and maternal death) that are mutually exclusive and severe maternal outcomes (maternal near miss or maternal death) associated with twin pregnancies.

METHODS:

We performed a secondary analysis of a cross-sectional World Health Organization Multicountry Survey, which was implemented in 29 countries. Data from 4,756 twin deliveries were compared with 308,111 singleton deliveries. Factors associated with maternal morbidity and twin pregnancies were reported with adjusted prevalence ratio (95% confidence interval).

RESULTS:

Potentially life-threatening conditions, maternal near miss, severe maternal outcomes, and maternal deaths were 2.14 (1.99-2.30), 3.03 (2.39-3.85), 3.19 (2.58-3.94), and 3.97 (2.47-6.38) times higher, respectively, among twin pregnancies. Maternal age older than 20 years, having a partner, multiparity, and elective cesarean delivery were associated with twin pregnancies. Postpartum hemorrhage and chronic hypertension were more frequently associated with severe maternal outcomes among twin pregnancies. Conditions indicating organ dysfunction (maternal near miss) were twofold to fivefold higher for twins. Poisson multiple regression analysis identified several factors independently associated with a severe maternal outcome, but not twin pregnancies.

CONCLUSION:

Twin pregnancy is associated with greater severe maternal morbidity and a higher rate of maternal death than singleton pregnancy.

PMID: 26959199 [PubMed - in process]

12.

Pediatrics. 2016 Mar 4. pii: peds.2015-3002. [Epub ahead of print]

[Mental Health in Children Born Extremely Preterm Without Severe Neurodevelopmental Disabilities.](#)

Fevang SK, Hysing M, Markestad T, Sommerfelt K.

Abstract

OBJECTIVE:

To describe the prevalence and gender characteristics of mental health problems in extremely preterm/extremely low birth weight (EP/ELBW) children without intellectual disabilities, blindness, deafness, or severe cerebral palsy compared with a reference group at 11 years of age.

METHODS:

In a national cohort of EP/ELBW children, mental health was assessed by parental and teacher report by using the Autism Spectrum Screening Questionnaire, the Swanson, Noland, and Pelham Questionnaire IV (attention-deficit/hyperactivity disorder), the Screen for Child Anxiety Related Emotional Disorders, symptoms of obsessive-compulsive disorder (OCD), and a total difficulties score from the Strength and Difficulties Questionnaire. Pervasive rating was defined as both parent and teacher scoring the child ≥ 95 th percentile (≥ 90 th percentile for total difficulties score) of the reference group, which was the population-based Bergen Child Study.

RESULTS:

Of eligible children, 216 (64%) EP/ELBW and 1882 (61%) reference children participated. EP/ELBW children were at significantly increased risk of pervasive rated symptoms of autism (odds ratio 4.3, 95% confidence interval 2.0-9.3), inattention (8.3, 4.4-15), anxiety (2.3, 1.4-3.7), OCD (2.6, 1.4-3.7), and ≥ 90 th percentile for total difficulties score (4.9, 2.9-8.2). Reported by either parents or teachers, 54% of the EP/ELBW and 21% of the reference children had ≥ 1 mental health problem (odds ratio 4.5, 95% confidence interval 3.3-6.1). There were no significant interactions between EP/ELBW and gender in mental health outcomes.

CONCLUSIONS:

EP/ELBW children without severe disabilities had increased risk of symptoms of autism, inattention, anxiety, and OCD. Gender differences were comparable to the reference group.

Copyright © 2016 by the American Academy of Pediatrics.

PMID: 26944946 [PubMed - as supplied by publisher]