Antenatal Corticosteroids for Small for Gestational Age in Late Preterm Newborns

Keren Zloto MD[1,3], Eyal Krispin MD[2,3], Anat Shmueli MD[2,3], Eran Hadar MD[2,3], and Lina Salman MD MSc[2,3]

[1]Department of Obstetrics and Gynecology, Sheba Medical Center, Tel Hashomer, Israel
[2]Department of Obstetrics and Gynecology, Rabin Medical Center (Beilinson Campus), Petah Tikva, Israel
[3]Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

ABSTRACT **Background:** The administration of antenatal corticosteroids (ACS) is standard practice for management of threatened preterm birth. Its benefit, especially in small for gestational age (SGA) late preterm, is unclear.

Objectives: To evaluate the impact of ACS on perinatal outcome of late preterm SGA neonates.

Methods: We conducted a retrospective cohort study of all women carrying a singleton gestation who had late preterm delivery (34–36 gestational weeks) of SGA neonates at a single tertiary university-affiliated medical center (July 2012–December 2017). Exclusion criteria included termination of pregnancy, intrauterine fetal death, and birth weight ≥ 10th percentile. Outcomes were compared between ACS and non-ACS treatment prior to delivery. Neonatal composite outcome included neonatal intensive care unit (NICU) admission, respiratory distress syndrome, mechanical ventilation, and transient tachypnea.

Results: Overall, 228 women met inclusion criteria; 102 (44.7%) received ACS and 126 did not (55.3%). Median birth weight among the non-ACS group was significantly higher (1896.0 vs. 1755.5 grams $P < 0.001$). Rates of NICU and jaundice requiring phototherapy were higher among the ACS group (53.92% vs. 31.74%, $P = 0.01$; 12.74% vs. 5.55%, $P = 0.05$, respectively). Composite neonatal outcome was significantly higher among the ACS group (53.92% vs. 32.53%, odds ratio [OR] 2.42, 95% confidence interval [95%CI] 1.41–4.15, $P = 0.01$). After adjustment for potential confounders, this association remained significant (OR 2.15, 95%CI 1.23–3.78, $P = 0.007$).

Conclusions: ACS given during pregnancy did not improve respiratory outcome for SGA late preterm neonates. ACS may be associated with a worse outcome.

IMAJ 2023; 25: 683–687

KEY WORDS: antenatal corticosteroids (ACS), intrauterine growth restriction, late preterm, perinatal outcome, small for gestational age (SGA)

Antenatal corticosteroid (ACS) therapy for preterm gestations has been shown to improve neonatal outcome and reduce respiratory distress syndrome (RDS) in preterm deliveries [1]. The latest Cochrane review [2], which included the results of 30 trials, concluded that a single course of corticosteroids, given to the parturient in preterm labor, reduced the rates and severity of serious adverse outcomes related to prematurity. These outcomes included RDS, intraventricular hemorrhage (IVH), necrotizing enterocolitis (NEC), and need for respiratory support and neonatal intensive care unit (NICU) admission. In addition, ACS treatment was found to significantly reduce the risk of perinatal and neonatal death. The impact of ACS on late preterm neonates was assessed in a randomized trial [3]. The authors found that antenatal administration of betamethasone to women at risk for late preterm delivery (34 weeks 0 days to 36 weeks 6 days of gestation) decreased the need for substantial respiratory support during the first 72 hours after birth.

ACS are associated with several adverse effects, especially when repeated courses are given. Murphy and colleagues [4] demonstrated that multiple courses of ACS every 14 days did not improve preterm birth outcomes and were associated with decreased birth weight, length, and head circumference at birth. Another study that evaluated long-term outcomes after repeated doses of ACS, where children were followed for up to 2–3 years of age, found higher rate of cerebral palsy among children who had been exposed to repeated doses of corticosteroids [5].

Although ACS are widely used, there are several unanswered questions regarding such treatment. One unknown is the impact of ACS treatment on growth-restricted fetuses [6]. Newborns that are small for gestational age (SGA) have increased risk for neonatal morbidity as well as long-term adverse outcomes such as cerebral palsy, major psychiatric sequelae in later years, and adult cardiovascular diseases [7]. In one study, the neonatal outcome was compared between growth-restricted fetuses that received ACS and those who did not. The authors concluded that administration of corticosteroid to growth-restricted preterm fetuses was not beneficial with respect to short-term neonatal outcome [8]. However, this study included only early preterm (up to 34 weeks of gestation). Hence, in this study we assessed whether exposure to ACS during pregnancy impacted adverse neonatal outcome in SGA neonates born in the late preterm period.

PATIENTS AND METHODS

POPULATION

A retrospective cohort study was conducted of all women, carrying a singleton gestation, who had a preterm delivery at a single, tertiary, university-affiliated medical center between July 2012 and December 2017.

We only included late preterm deliveries (34+0 to 36+6 gestational weeks) with SGA neonates, defined as birth weight below the 10th percentile according to the Israeli national birthweight curves [9]. Exclusion criteria included termination of pregnancy (TOP), intrauterine fetal death (IUFD), and birth weight at or above the 10th percentile.

ETHICS APPROVAL

This study was approved by the institutional review board at Rabin Medical Center (RMC-19-0557). Informed consent was waived due to the retrospective design of the study.

DATA COLLECTION

Data were retrieved from the comprehensive computerized perinatal database of our center. Data from the neonatal unit and the neonatal intensive care unit (NICU) were integrated into the delivery room database using the unique admission number assigned to each woman and her offspring. Collected data included demographic and obstetric parameters, mode of delivery, and short-term maternal and neonatal outcome (up to discharge).

OUTCOME MEASURES

The study population comprised two groups: those who were treated with ACS prior to delivery and those who did not receive ACS prior to delivery. Maternal and neonatal outcomes were compared between groups. The primary outcome was composite and included at least one of the following: NICU admission, RDS, mechanical ventilation, and transient tachypnea of the newborn (TTN). Secondary outcomes included other neonatal outcomes: umbilical arterial pH, IVH, mechanical ventilation, NEC, retinopathy, sepsis, antibiotic treatment, neonate major anomaly, blood products transfusion, and hypoglycemia. Adverse maternal and labor outcome were also evaluated as secondary outcomes, including postpartum hemorrhage (PPH), blood product transfusion, mode of delivery, and onset of labor (elective, spontaneous, or induction).

By departmental protocol during the study period ACS was delivered for gestations with suspected preterm delivery before 34 + 0 weeks of gestation. The treatment course includes two 12 mg doses of betamethasone given intramuscularly 24 hours apart. In a few circumstances, where pregnancy was continued and there was an imminent threat of preterm delivery, an additional rescue course was given in the same manner. Delivery methods of induction were prostaglandin E2 (PGE2), extra-amniotic balloon, and oxytocin infusion, which were chosen at the physician's discretion and local institutional practice.

STATISTICAL ANALYSIS

Continuous variables were evaluated for normal distribution using histograms and Q-Q plots. Non-normally distributed parameters were compared using Mann–Whitney test. Correlations between continuous variables were evaluated using the Spearman correlation coefficient. The chi-square test was used to compare categorical variables. Logistic regression analysis was used to determine which factors were significantly and independently associated with ACS treatment. Odds ratios (OR) with 95% confidence interval (95%CI) were reported. All statistical tests were 2-tailed, and $P < 0.05$ was considered as statistically significant. Statistical analyses were performed using IBM Statistical Package for the Social Sciences statistics software, version 26 (SPSS, IBM Corp, Armonk, NY, USA).

RESULTS

Overall, 228 patients met the inclusion criteria; 102 (44.7%) received ACS and 126 did not (55.3%). Among the group that received ACS, 27 (26.47%) received a second (rescue) course of ACS. Maternal characteristics are summarized in Table 1.

In our cohort, there was a significantly higher mean birthweight among the non-ACS group (1891.20 ± 173.75 vs.

Table 1 Demographic characteristics of the study population

Characteristics	ACS (n=102)	Non-ACS (n=126)	*P*-value
Maternal age (years)	32 (28–35)	32 (28–36)	0.272
Pregestational BMI (kg/m^2)	22.57 (19.87–26.30)	22.58 (20.94–26.66)	0.516
Gravidity	2 (1–3)	2 (1–3)	0.207
Parity	0 (0–1)	1 (0–2)	0.177
Nulliparity	48 (47.05%)	48 (38.09%)	0.17
Gestational age at delivery (weeks)	35.5 (34.6–36.2)	36.1 (35.4–36.3)	< 0.001

ACS = antenatal corticosteroids, BMI = body mass index

Continuous variables are presented as median and interquartile range. Categorical variables are presented as n (%)

1773.76 ± 224.68 grams, $P < 0.001$). Regarding neonatal outcome, rates of NICU admission and jaundice were significantly higher among the ACS group (53.92% vs. 31.74%, $P = 0.01$ and 12.74% vs. 5.55%, $P = 0.05$, respectively). The rate of RDS was not significantly different between the groups (3.92% vs. 1.5%, $P = 0.27$) [Table 2]. In addition, the rate of composite neonatal outcome was significantly higher among the ACS group (53.92% vs. 32.53%, OR 2.42, 95%CI 1.41–4.15, $P = 0.001$). After adjustment for nulliparity, sex, and birth weight percentiles using regression analysis, this association remained significant (OR 2.15, 95%CI 1.23–3.78, $P = 0.007$).

There were no differences between groups regarding maternal age, body mass index, gravidity, and parity. There were significantly higher rates of gestational diabetes and lower rates of preeclampsia among women who received ACS (9.8% vs. 2.38%, $P = 0.01$ and 9.8% vs. 19.9%, $P = 0.03$, respectively).

Table 2: Neonatal outcomes

Characteristic	ACS (n=102)	Non-ACS (n=126)	*P*-value
Birth weight in grams	1755.5 (1611.75–1971.0)	1896.0 (1797.8–2021.2)	< 0.001
Birth weight percentiles	4 (1–6)	4.6 (1–7.4)	0.06
5-minute Apgar score < 7	1 (0.75)	1 (0.75)	0.88
Umbilical artery pH	7.34 (7.3–7.38)	7.32 (7.28–7.36)	0.02
Composite neonatal outcome	55 (53.92)	41 (32.53)	< 0.001
Respiratory distress syndrome	4 (3.92)	2 (1.5)	0.27
Transient tachypnea of the newborn	5 (4.9)	4 (3.17)	0.50
Intraventricular hemorrhage	2 (1.96)	1 (0.75)	0.44
Mechanical ventilation	1 (0.98)	4 (3.17)	0.26
Necrotizing enterocolitis	0 (0)	1 (0.75)	0.55
Neonatal intensive care unit admission	55 (53.92)	40 (31.74)	0.01
Jaundice requiring phototherapy	13 (12.74)	7 (5.55)	0.05
Neonate major anomaly	8 (7.84)	8 (6.34)	0.66
Transfusion	3 (2.94)	5 (3.96)	0.67
Hypoglycemia	6 (5.88)	9 (7.14)	0.70

Continuous variables are presented as median and interquartile range. Categorical variables are presented as n (%)

ACS = antenatal corticosteroids

Table 3 Obstetric outcomes of the study population

Variables		ACS (n=102)	Non-ACS (n=126)	*P*-value
Gestational diabetes		10 (9.8)	3 (2.38)	0.01
Oligohydramnios		8 (7.84)	5 (3.96)	0.21
Polyhydramnios		0 (0)	2 (1.5)	0.20
Episiotomy		3 (2.94)	8 (6.34)	0.23
Postpartum hemorrhage		1 (0.98)	3 (2.38)	0.42
Transfusion		1 (0.98)	2 (1.5)	0.68
Abruption		2 (1.96)	1 (0.75)	0.44
Hypertensive disease of pregnancy	Pregnancy-induced hypertension	0 (0)	2 (1.58)	0.20
	Chronic hypertension	1 (0.98)	1 (0.75)	0.88
	Pre-eclampsia	10 (9.8)	25 (19.84)	0.03
Mode of delivery	Vaginal	29 (28.43)	47 (37.3)	0.33
	Cesarean	67 (65.67)	71 (56.34)	
Mode of starting delivery	Elective	48 (47.05)	47 (37.3)	0.03
	Spontaneous	11 (10.78)	30 (23.8)	
	Induction	27 (26.47)	35 (27.77)	

Continuous variables are presented as median and interquartile range. Categorical variables are presented as n (%)

ACS = antenatal corticosteroids

Regarding obstetric outcomes, the group that received ACS delivered earlier (35.5 vs. 36.1 gestational weeks, $P = 0.00$). As for onset of labor, in the non-ACS group, there was a higher prevalence of spontaneous delivery versus elective delivery and induction of labor [Table 3].

A subgroup analysis was performed for the group that received ACS. There was a higher rate of NICU admission among the group that received a rescue course of ACS (70.37% vs. 47.14%, $P = 0.04$). In addition, composite neonatal outcome was significantly higher among individuals receiving a rescue course of ACS (70.37% vs. 47.14%, OR 2.66, 95%CI 1.03–6.88, $P = 0.04$). After adjustment for nulliparity, using multivariable logistic regression analysis, this association remained significant (OR 2.65, 95%CI 1.01–6.91, $P = 0.04$).

DISCUSSION

We found that administration of ACS for SGA neonates born in the late preterm did not result in reduced RDS and other neonatal complications and was even associated with increased odds for adverse composite neonatal outcome. This association remained significant among fetuses that received a rescue course of ACS in the subgroup analysis for the ACS group only.

ACS therapy for decreasing neonatal morbidity and mortality is recommended by guidelines around the world and is widely used. Its benefits among SGA fetuses remains largely unknown. There is insufficient evidence of the benefits of routine ACS therapy in gestations with suspected intra-uterine growth restriction (IUGR), especially in the late preterm [10].

The benefit of ACS in specific obstetric populations, such as SGA neonates, has yet to be determined. Gyamfi-Bannerman et al. [3] investigated the effects of ACS in women at risk for late preterm delivery. In their randomized trial, they recruited women with a singleton pregnancy who were at high risk for delivery during the late preterm period. The study showed that ACS caused a significant reduction in rates of neonatal respiratory complications. It should be noted though that the frequency of IUGR among the ACS group was 3.2% and among the control group was 3.4%. The impact of ACS on that subgroup was not analyzed. Haviv and colleagues [10] investigated the role of ACS on late preterm in special populations. They concluded that there was insufficient evidence regarding the benefit or harm of ACS therapy in pregnancies with IUGR, especially in the late preterm period. They recommended an individualized approach when administering ACS at later gestations in specific obstetric populations such as IUGR. Bitar and co-authors [11] investigated the effect of administering ACS in the late preterm period in pregnancies with growth restriction. They showed that ACS did not significantly decrease the need for respiratory support and increased the rate of neonatal hypoglycemia. The effectiveness of ACS administration in late preterm is still controversial and requires more research [12].

Several studies reported no effect of ACS on neonatal morbidity or mortality among IUGR fetuses in the early preterm (up to 34 weeks of gestational age) [8,13-16]. Van Stralen et al. [8] found that administration of ACS to IUGR fetuses was not beneficial with respect to short-term neonatal outcome in preterm deliveries. Another recent study also showed that ACS did not improve neonatal morbidities in SGA neonates delivered between 29 and 34 gestational weeks. Rather, ACS seemed to increase the risk of RDS. The authors concluded that ACS therapy for women who are at risk for preterm delivery with IUGR fetuses need to be further evaluated, especially after 32 weeks of gestation [15]. A recent meta-analysis on 16 observational cohort and case-control studies published from 1995 to 2018 showed that ACS reduced neonatal mortality in SGA infants delivered preterm, with no apparent effect on neonatal morbidity (RDS, NEC, IVH, periventricular leukomalacia, bronchopulmonary dysplasia, chronic lung disease of prematurity, or neonatal sepsis). The study concluded that future studies are needed on the effect of ACS administration to SGA infants in the late preterm period because data on this issue was limited [16].

One hypothesis that may explain our results is that poor intrauterine growth, by itself, actually enhances lung maturation. This assumption has been demonstrated in several studies. The physiological adaptations that growth-restricted fetuses experience in response to nutrient and oxygen restriction alter the ability to regulate endogenous glucocorticoid availability. As a result, these fetuses may be exposed to higher ACS concentrations, which may result in an exacerbation of the potentially negative side effects of antenatal glucocorticoid treatment, especially in cardiovascular development, and possibly without the full capacity to benefit from the lung maturational effects [17]. Conversely, another study demonstrated that IUGR fetuses accelerated lung maturation was not supported in comparisons of SGA and appropriate for gestational age (AGA) infants of the same gestational age, sex, and race [18].

Second, elimination of ACS via the placenta or the blood–brain barrier is impaired with IUGR, and hence, the fetus is exposed to excessive corticosteroids in the lung, brain, and heart tissues [17].

Nevertheless, some studies showed a lower risk of adverse outcomes [18-22]. Bernstein and colleagues [21] demonstrated an association of IUGR fetuses with increased morbidity and mortality. Furthermore, they showed that the benefits of ACS therapy were similar among infants with IUGR and normally grown infants for neonates from 25 to 30 weeks of gestation. A population-based study on singleton infants of 24–31 weeks of gestation concluded that ACS therapy was associated with significantly reduced mortality and reduced neonatal morbidities among preterm SGA neonates, which was generally like the effect in the AGA preterm infants [19]. A review published in 2018, showed that based on the current clinical evidence, it would be reasonable to administer a single course of glucocorticoids to pregnant women with IUGR fetuses that are at risk of preterm birth; however, there was insufficient evidence to

conclude whether repeated or rescue ACS administration was beneficial for IUGR infants [20].

We found that birth weights were significantly higher among the non-ACS group. Our results concur with previous studies that have shown that ACS is associated with reduction in birth size for infants born preterm, near term, or at term [23]. These studies even showed a reduction in head circumference among preterm newborns [23,24].

The novelty of our study is that we examined ACS therapy for SGA neonates that were born at late preterm. To the best of our knowledge, most existing studies examined ACS therapy for SGA neonates in the early preterm. Moreover, most studies refered to IUGR fetuses (defined as an estimated fetal weight < 10th percentile) and not SGA neonates. Discrepancies might exist between antenatal fetal weight estimation and the actual birthweight.

The study has limitations. The main limitation is its retrospective design, which could lead to an unknown selection bias, such as the reason for administering or withholding ACS. Our cohort was small, and the time of administering ACS was not collected; therefore, we were not able to assess whether there was any association between the duration of time from ACS to delivery and neonatal adverse outcomes. Furthermore, the group that received ACS included patients who received one dose of ACS as well as patients who received a full course of two doses. We were not able to capture the number of ACS doses administered through our medical records and therefore we combined them into one group. Another limitation is that our data were collected from pregnancies in 2012–2017; however, the recommendation to administer ACS in the late preterm only started in 2016. We also studied only short-term neonatal outcomes in this specific population. The long-term impact of ACS was not evaluated.

CONCLUSIONS

ACS did not decrease neonatal morbidity in late preterm SGA neonates. While these results need to be interpreted with caution, we found that ACS might be associated with an increased risk for adverse neonatal outcomes. These findings should be further evaluated in large prospective studies to better understand the impact of ACS on this unique cohort of SGA neonates born at the late preterm.

Correspondence

Dr. L. Salman
Dept. of Obstetrics and Gynecology, Rabin Medical Center (Beilinson Campus), Petah Tikva 49100, Israel
Phone: (972-3) 937-7680
Fax: (972-3) 937-7683
Email: linasalman@gmail.com

Reference

1. Liggins GC, Howie RN. A controlled trial of antepartum glucocorticoid treatment for prevention of the respiratory distress syndrome in premature infants. *Pediatrics* 1972; 50 (4): 515-25.
2. Roberts D, Brown J, Medley N, Dalziel SR. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. *Cochrane Database Syst Rev* 2017; 3 (3): CD004454. Update in: *Cochrane Database Syst Rev* 2020; 12: CD004454.
3. Gyamfi-Bannerman C, Thom EA, Blackwell SC, et al; NICHD Maternal–fetal medicine units network. antenatal betamethasone for women at risk for late preterm delivery. *N Engl J Med* 2016; 374 (14): 1311-20.
4. Murphy KE, Hannah ME, Willan AR, et al; MACS Collaborative Group. Multiple courses of antenatal corticosteroids for preterm birth (MACS): a randomised controlled trial. *Lancet* 2008; 372 (9656): 2143-51.
5. Wapner RJ, Sorokin Y, Mele L, et al; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Long-term outcomes after repeat doses of antenatal corticosteroids. *N Engl J Med* 2007 20; 357 (12): 1190-8.
6. Torrance HL, Derks JB, Scherjon SA, Wijnberger LD, Visser GH. Is antenatal steroid treatment effective in preterm IUGR fetuses? *Acta Obstet Gynecol Scand* 2009; 88 (10): 1068-73.
7. Tan TY, Yeo GS. Intrauterine growth restriction. *Curr Opin Obstet Gynecol* 2005; 17 (2): 135-42.
8. van Stralen G, van der Bos J, Lopriore E, et al. No short-term benefits of antenatal corticosteroid treatment in severely preterm growth restricted fetuses: a case-control study. *Early Hum Dev* 2009; 85 (4): 253-7.
9. Dollberg S, Haklai Z, Mimouni FB, Gorfein I, Gordon ES. Birth weight standards in the live-born population in Israel. *IMAJ* 2005; 7 (5): 311-4.
10. Haviv HR, Said J, Mol BW. The place of antenatal corticosteroids in late preterm and early term births. *Semin Fetal Neonatal Med* 2019; 24 (1): 37-42.
11. Bitar G, Merrill SJ, Sciscione AC, Hoffman MK. Antenatal corticosteroids in the late preterm period for growth-restricted pregnancies. *Am J Obstet Gynecol MFM* 2020; 2 (3): 100153.
12. Vidaeff AC, Belfort MA, Kemp MW, et al. Updating the balance between benefits and harms of antenatal corticosteroids. *Am J Obstet Gynecol* 2023; 228 (2): 129-32.
13. Ley D, Wide-Swensson D, Lindroth M, Svenningsen N, Marsal K. Respiratory distress syndrome in infants with impaired intrauterine growth. *Acta Paediatr* 1997; 86 (10): 1090-6.
14. Mitsiakos G, Kovacs L, Papageorgiou A. Are antenatal steroids beneficial to severely growth restricted fetuses? *J Matern Fetal Neonatal Med* 2013; 26 (15): 1496-9.
15. Kim WJ, Han YS, Ko HS, Park IY, Shin JC, Wie JH. Antenatal corticosteroids and outcomes of preterm small-for-gestational-age neonates in a single medical center. *Obstet Gynecol Sci* 2018; 61 (1): 7-13.
16. Blankenship SA, Brown KE, Simon LE, Stout MJ, Tuuli MG. Antenatal corticosteroids in preterm small-for-gestational age infants: a systematic review and meta-analysis. *Am J Obstet Gynecol MFM* 2020; 2 (4): 100215.
17. Morrison JL, Botting KJ, Soo PS, et al. Antenatal steroids and the IUGR fetus: are exposure and physiological effects on the lung and cardiovascular system the same as in normally grown fetuses? *J Pregnancy* 2012; 2012: 839656.
18. Tyson JE, Kennedy K, Broyles S, Rosenfeld CR. The small for gestational age infant: accelerated or delayed pulmonary maturation? Increased or decreased survival? *Pediatrics* 1995; 95 (4): 534-8.
19. Riskin-Mashiah S, Riskin A, Bader D, et al; Israel Neonatal Network. Antenatal corticosteroid treatment in singleton, small-for-gestational-age infants born at 24-31 weeks' gestation: a population-based study. *BJOG* 2016; 123 (11): 1779-86.
20. Ting JY, Kingdom JC, Shah PS. Antenatal glucocorticoids, magnesium sulfate, and mode of birth in preterm fetal small for gestational age. *Am J Obstet Gynecol* 2018; 218 (2S): S818-28.
21. Bernstein IM, Horbar JD, Badger GJ, Ohlsson A, Golan A. Morbidity and mortality among very-low-birth-weight neonates with intrauterine growth restriction. The Vermont Oxford Network. *Am J Obstet Gynecol* 2000; 182 (1 Pt 1): 198-206.
22. Ražem K, Tul N, Blickstein I, Trojner Bregar A. The effect of antenatal corticosteroids on small-for-gestational age preterm neonates. *J Matern Fetal Neonatal Med* 2022; 35 (2): 362-5.
23. Rodriguez A, Wang Y, Ali Khan A, Cartwright R, Gissler M, Järvelin MR. Antenatal corticosteroid therapy (ACT) and size at birth: a population-based analysis using the Finnish Medical Birth Register. *PLoS Med* 2019; 16 (2): e1002746.
24. Thorp JA, Jones PG, Knox E, Clark RH. Does antenatal corticosteroid therapy affect birth weight and head circumference? *Obstet Gynecol* 2002; 99 (1): 101-8.