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ORIGINAL ARTICLE

Group B Streptococcal Infection in Taiwan: Maternal Colonization and Neonatal Infection

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Background: There is no national data on group B streptococcus (GBS) infection in Taiwan. We investigated incidence of maternal GBS colonization and neonatal GBS infection rate and clinical pictures of neonatal GBS infection to estimate the value of intrapartum chemoprophylactic strategy in Taiwan.
Methods: From January 2004 to June 2005, a prospective study to estimate maternal colonization rate by maternal rectovaginal culture at six hospitals was conducted. Neonatal GBS infection rate based on inborn infants was calculated retrospectively from January 2001 to June 2005; clinical pictures of infants diagnosed with invasive GBS disease were reviewed.
Results: Maternal colonization rate of GBS was around 20% at hospital base, incidence of neonatal GBS infection was 1 per 1000 live births of infants born at hospitals. There were 221 infants with GBS infection: in 142, the disease occurred within 7 days of birth (early-onset disease, EOD), and in 79, it developed later (late-onset disease). Infantile EOD was more often seen in mothers with premature rupture of membrane, often accompanied by respiratory failure necessitating ventilator support. Infants with late-onset disease often

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manifested fever, leukopenia, thrombocytopenia, and meningitis. Fifteen infants died, mostly of EOD type (12 of 15). Risk factors of mortality included rescue at delivery room, leukopenia, thrombocytopenia, sepsis, respiratory distress, persistent hypertension of newborn, respiratory failure needing intensive respiratory support (intermittent mandatory ventilator and high frequency oscillatory ventilator), surfactant use, shock, and congenital heart diseases.

Conclusions: We concluded that universal maternal rectovaginal culture of GBS with intrapartum antibiotic prophylaxis is an urgent call to reduce EOD and mortality because of GBS infection in neonates in Taiwan.

1. Introduction

Group B streptococcus (GBS), or *Streptococcus agalactiae*, is the most common cause of early neonatal infection, with 5—20% mortality and serious complications.[1,2] Since implementation of intrapartum antibiotic prophylaxis in 1996, incidence of early-onset disease (EOD) of GBS has dropped by 50—85% in the United States.[3] Incidence of invasive GBS infection, which was defined as isolation of GBS from a normally sterile site [such as blood or cerebrospinal fluid (CSF) or, less commonly, joint, pleural, or pericardial fluid],[4] is around 0.2—5.4 infants per 1000 live births before intrapartum antibiotic prophylaxis.[5,6]

GBS is commonly found in the female genital tract and exists at a lower rate in male urethras.[7] An estimated of 15—28.7% women at childbearing age are colonized with GBS.[8,9] Approximately 50% of infants born from these women will become infected,[10] with 1—2% of infants with GBS colonization developing disease.[11] There were several reports of neonatal GBS infection in Taiwan,[12—16] but data were from regional single institutes that could not validly represent incidence of neonatal GBS infection in Taiwan. Although researchers believe that Taiwan needs its own policy to reduce neonatal GBS infection, no intrapartum antibiotics strategy exists in Taiwan at present because of lack of accurate data. We speculated that maternal colonization and incidence of neonatal GBS infection and mortality rate of EOD of GBS by hospital base will match those in previous reports; intrapartum chemoprophylactic strategy might be warranted for Taiwan to reduce GBS infection in neonates.

2. Materials and Methods

2.1. Maternal colonization

From July 2004 to June 2005, we performed a prospective study of maternal rectovaginal colonization rate from six hospitals (China Medical University Hospital, Taichung Veterans General Hospital, Shinyaton Hospital, Cheng Ching Hospital, Mackay Memorial Hospital, and Chang Gung Memorial Hospital). This study encouraged pregnant women to do rectovaginal culture, paid by themselves. Collection of cultures was conducted by swabbing both lower vagina and rectum (through the anal sphincter) between 35 and 37 weeks' gestation.[17]

2.2. Incidence of neonatal GBS infection

From January 2002 to June 2005, a retrospective study enrolled all infants born in the hospital in six medical centers (China Medical University Hospital, Taichung Veterans General Hospital, Changhua Christian Hospital, National Taiwan University Hospital, Chang Gung Memorial Hospital, and Mackay Memorial Hospital) and recorded inborn infants with GBS infection to calculate the neonatal GBS infection rate.

2.3. Clinical pictures of neonatal GBS infection

From January 2002 to June 2005, all infants with invasive GBS infection and with gestational age more than 24 weeks and less than 3 months of age, including inborn and transferred infants, were enrolled. Invasive GBS infection included GBS pneumonia, bacteremia, or meningitis diagnosed at six medical centers and five hospitals (Cheng Ching, Tai-An, Taipei City, Shin Kong Wu Ho-Su Memorial, and Taiwan Adventist) were reviewed retrospectively. Infants were identified from computerized medical records and reports of positive body fluid culture.

GBS was identified based on gram-stained smear and growth characteristics, production of beta-hemolysin on sheep blood agar, resistance to bacitracin disc, negative hydrolysis of bile-esculin agar, and positive CAMP test showing typical arrowhead enlargement by GBS of the zone of hemolysis caused by *Staphylococcus aureus*; this was confirmed by Streptex agglutination test (Wellcome Diagnostics, Dartford, UK). Typing of GBS isolates was not routinely performed. The following items were reviewed: gender, gestation age, body weight, age at onset, delivery method, chorioamnionitis, preterm delivery, premature rupture of membrane (PROM), Apgar score at 1 and 5 minutes, rescue at delivery room, neonatal fever, white blood cell count, platelet count, sepsis, meningitis, shock, respiratory distress, persistent pulmonary hypertension, and mode of ventilatory support.

For data collection, the following terminologies were defined: PROM, rupture of membranes was more than 18 hours; chorioamnionitis: maternal fever, uterine tenderness, foul smelling vaginal discharge/amniotic fluid, maternal leukocytosis, maternal and/or fetal tachycardia; very low birth weight, birth weight less than 1500 g. EOD means symptoms present at less than 7 days of age. Late-onset disease (LOD) means symptoms present at 7 days of

Table 1 Incidence of maternal GBS colonization (prospective study)

Hospital	All mothers	GBS(+) mothers	Colonization rate
1	1028	155	0.15
2	109	12	0.11
3	1247	162	0.13
4	168	31	0.18
5	452	101	0.22
6	1581	457	0.29
Total	4585	918	0.20

GBS = group B streptococcus; Hospital 1 = China Medical University Hospital; Hospital 2 = Taichung Veterans General Hospital; Hospital 3 = Shinyaton Hospital; Hospital 4 = Cheng Ching Hospital; Hospital 5 = Mackay Memorial Hospital; Hospital 6 = Chang Gung Memorial Hospital.

age or later. Sepsis was defined as a positive growth of GBS from blood culture with clinical symptoms. Meningitis: (1) Positive culture and/or positive latex particle agglutination test of a nontraumatic CSF; (2) positive gram-stained smear of CSF and/or significant pleocytosis in a nontraumatic CSF, in the course of GBS infection; or (3) pathologic findings consistent with acute meningeal inflammation. Pneumonia: (1) GBS infection; (2) abnormal chest roentgenogram (consistent with respiratory distress syndrome, pneumonia, transient tachypnea in newborn); (3) respiratory signs and symptoms; or (4) oxygen requirement. Shock: hypotension, corrected for birth weight, requiring dopamine and/or albumin administration. Urinary tract infection (UTI): pure culture of GBS from urine sample obtained by bladder tap or sterile catheterization. Persistent pulmonary hypertension in neonate (PPHN) was diagnosed by persistent hypoxemia despite application of 100% oxygen in conjunction with mechanical ventilation and right-to-left or bidirectional shunt from the *ductus arteriosus* and/or foramen ovale revealed by an echocardiogram. Fatality: any infant death attributed to complications of GBS infection under diagnosis of invasive GBS disease.

2.4. Statistical methods

Significance of comparison between proportions was determined by the χ^2 test with Yates correction for continuity when required. A p value less than 0.05 was considered statistically significant.

Table 3 Clinical features and laboratory findings of early and late group B streptococcus infection

Clinical findings	Early onset (≤7 d) [n = 142 (%)]	Late onset (>7 d) [n = 79 (%)]	p
Pneumonia	53 (37.3)	8 (10.1)	0.000
Sepsis	83 (58.4)	54 (68.3)	0.146
Meningitis	29 (20.4)	30 (37.9)	0.004
UTI	80 (56.3)	38 (48.1)	0.239
Shock	29 (20.4)	11 (13.9)	0.229
PPHN	4 (2.8)	0 (0)	0.132
Surfactant use	18 (12.6)	3 (3.7)	0.031
IMV	29 (20.4)	8 (10.1)	0.049
HFOV	18 (12.6)	4 (5.0)	0.070
Mortality	12 (8.4)	3 (3.8)	0.187
Fever >38°C	41 (28.8)	60 (75.9)	0.000
Leukopenia <5000	20 (14.0)	20 (25.3)	0.037
Thrombocytopenia	12 (8)	1 (1)	0.029

HFOV = high frequency oscillatory ventilator; IMV = intermittent mandatory ventilator; PPHN = persistent pulmonary hypertension in neonate; thrombocytopenia = platelet count less than 100,000/mm^3; UTI = urinary tract infection.

3. Results

Prospective maternal rectovaginal colonization of GBS was 20% in average (11–29%) (Table 1). Retrospective chart review plotted incidence of neonatal GBS infection as 1.1 per 1000 live births, based on inborn babies in medical centers (Table 2). Over 4 years at 11 hospitals, 221 infants showed GBS infection, 142 presented within 7 days of life, and 79 developed diseases later. Pneumonia developed in 61, sepsis in 136, meningitis in 59, UTI in 118, shock in 40, and PPHN in 4 (Table 3). Thirty-seven had severe respiratory failure requiring intermittent mandatory ventilator (IMV) support, and 10 required high frequency oscillatory ventilator (HFOV) (Table 3). Twenty-two received surfactant therapy. Fifteen died, mostly of the early type (12 of 15, 80.0%). Overall mortality was 15 of 221 (6.8%); mortality

Table 2 Incidence of neonatal group B streptococcus infection (retrospective study in inborn)

Infected persons/no. of newborns	2002	2003	2004	2005	Total
Hospital 1	0/2358	2/2401	3/2063	4/2321	9/9143 (0.0010)
Hospital 2	0/1146	1/1126	1/1037	1/1155	3/5856 (0.0005)
Hospital 3	0/2913	0/2548	1/2006	4/1987	5/9454 (0.0005)
Hospital 4	2/2517	2/2936	5/2254	4/2108	13/9815 (0.0013)
Hospital 5	5/5233	11/5001	6/4303	5/3763	27/16136 (0.0017)
Hospital 6	5/4403	6/4499	6/3629	0/3605	17/12963 (0.0013)
Mean					74/63367 (0.0011)

Hospital 1 = China Medical University Hospital; Hospital 2 = Taichung Veterans General Hospital; Hospital 3 = Changhua Christian Hospital; Hospital 4 = National Taiwan University Hospital; Hospital 5 = Chang Gung Memorial Hospital; Hospital 6 = Mackay Memorial Hospital.

Table 4 Differences of obstetric and neonatal risk factors between early and late onset of group B streptococcus infection

Risk factors	EOD (≤7 d) [$n = 142$ (%)]	LOD (>7 d) [$n = 79$ (%)]	p
Maternal fever	3 (2.1)	1 (1.2)	0.162
Chorioamnionitis	10 (7.0)	2 (2.5)	0.156
PROM >18 hr	28 (19.7)	5 (6.3)	0.007
Prenatal antibiotics	6 (4.2)	4 (5.0)	0.773
NSD	108 (76.0)	55 (69.6)	0.297
Rescue at DR	24 (16.9)	6 (7.5)	0.052
Male	78 (54.9)	36 (45.5)	0.182
Gestational age ≤ 34 wk	26 (18.3)	11 (13.9)	0.402
Birth weight ≤ 2500 g	40 (28.1)	17 (21.5)	0.278
Apgar score 1 min <6	11 (7.7)	5 (6.3)	0.696
Apgar score 5 min <6	3 (2.1)	3 (3.7)	0.460

Antibiotics = mother received antibiotic; DR = delivery room; EOD = early-onset disease; LOD = late-onset disease; NSD = normal spontaneous delivery; PROM = premature rupture of membrane for more than 18 hours.

of EOD was 12 of 142 (8.5%), with 3 of 79 (3.8%) in LOD (Table 3).

Pneumonia, respiratory failure with IMV support and surfactant use, thrombocytopenia (platelet count less than 100,000/mm^3) occurred more often in EOD group; and meningitis, fever more than 38°C, leukopenia (white blood cell count less than 5000/mm^3) occurred more in LOD group (Table 3).

PROM is the most important risk factor for EOD of GBS. There is no significance among maternal fever, chorioamnionitis, prenatal antibiotics use, normal spontaneous delivery, rescue at delivery room, gestational age less than 34 weeks, and birth weight less than 2500 g (Table 4). Risk factors of mortality included rescue at delivery room ($p = 0.002$), leukopenia ($p = 0.022$), thrombocytopenia ($p = 0.000$), sepsis ($p = 0.001$), respiratory distress ($p = 0.020$), PPHN ($p = 0.000$), surfactant use ($p = 0.001$), IMV ($p = 0.000$), HFOV ($p = 0.000$), shock ($p = 0.000$), and congenital heart disease ($p = 0.002$) (Table 5).

4. Discussion

This is the first prospective study of maternal rectovaginal GBS cultures with a large sample size in Taiwan; results revealed incidence of maternal GBS colonization of around 20% (918/4585), which is within the range of previous reports.[8–10] Hospitals 5 and 6 showed a higher colonization rate, which may prove more accurate than other hospitals; they performed nearly universal maternal GBS screening. Hospital 2 showed the lowest colonization rate, possibly owing to small sample size of maternal GBS cultures.

Table 5 Risk factors of mortality in GBS infection

Risk factors	Survival [$n = 206$ (%)]	Fatality [$n = 15$ (%)]	p
PROM >18 hrs	30 (14.5)	3 (20.0)	0.568
CS	52 (25.2)	3 (20.0)	0.650
Rescue at delivery room	24 (11.6)	6 (40.0)	0.002
Inborn	95 (46.1)	6 (40.0)	0.646
Male	108 (52.4)	9 (60.0)	0.570
GA ≤34 wk	34 (16.5)	3 (20.0)	0.726
BBW ≤2500 g	50 (24.2)	7 (46.6)	0.055
Neonatal fever >38°C	100 (48.5)	2 (13.3)	0.008
Leukocyte <5000	34 (16.5)	6 (40.0)	0.022
Platelet <100,000	8 (3.8)	5 (33.3)	0.000
Sepsis	122 (59.2)	15 (100)	0.001
Respiratory distress	53 (25.7)	8 (53.3)	0.020
PPHN	1 (0.4)	3 (20.0)	0.000
Meningitis	56 (27.1)	3 (20.0)	0.543
Surfactant	16 (7.7)	5 (33.3)	0.001
IMV	27 (13.1)	10 (66.6)	0.000
HFOV	4 (1.9)	6 (40.0)	0.000
Shock	25 (12.1)	15 (100)	0.000
Congenital heart disease	7 (3.3)	3 (20.0)	0.002

BBW = birth body weight; CS = cesarean section; GA = gestational age; HFOV = high frequency oscillatory ventilator; IMV = intermittent mandatory ventilator; PPHN = persistent pulmonary hypertension of newborn; PROM = premature rupture of membrane for more than 18 hours.

Colonization rate may correlate with socioeconomic status and race.[1]

Many pediatricians and obstetricians believed GBS infection not prevalent in Taiwan.[16,17] The impression of a low incidence of GBS infection in Taiwan may arise from few rectovaginal cultures done for expectant women plus antibiotics prescribed for high-risk pregnant mothers very often. That resulted in a partial or complete treatment of GBS colonization in pregnant women, masking low incidence of invasive GBS disease for infants. It is interesting that if we calculate incidence of newborn infection from the colonization rate of pregnant women, the answer is 0.1%, (0.20 maternal colonization $\times$ 0.5 newborn colonization $\times$ 0.01 newborn infection rate), and the magic number by calculation matched that of retrospective study of GBS infection in newborn infants.

Most infections in newborns occur within the first week of life defined as EOD by Baker et al.[18] It usually manifests as sepsis or pneumonia and varies from 1—4 cases per 1000 live births in the United States to 0.3—1 cases per 1000 live births in Europe[19,20] and is associated with high mortality rates.[21] Bromberger et al[22] found that 95% of neonates with GBS-positive infections and signs of sepsis exhibit them in the first 24 hours of life. Among GBS infections, sepsis was most common in both early and late onset types in our study. In the EOD group, sepsis was the most common clinical presentation, followed by UTI. Pneumonia was found in 53 (37.3%) infants and meningitis was found in 29 (20.4%) infants in the EOD group, similar to reports of Hoshina et al[23] in Japan, and Chung et al[18] from Chang Gung Memorial Hospital of Kaohsiung. EOD of GBS infection is usually from vertical transmission from mother to child; not surprisingly, PROM was the most common risk factor of infection, this further making antibiotic prophylactic strategy against vertical transmission valuable.

Thirty-eight (26.8%) infants in the EOD group developed respiratory failure necessitating ventilator support (including HFOV or IMV). Ho et al[16] reported 50% with respiratory distress in EOD group of GBS infection over an 11-year review. Chung et al[18] also reported respiratory distress in 50% of EOD babies. Because PROM was the most often seen risk factor in the EOD group, it's reasonable to speculate that infants in EOD group might aspirate the infectious amniotic fluid to get pneumonia and suffer from respiratory distress soon after they are born. McKenna and lams[24] found that LOD usually manifests as neonatal sepsis or meningitis with lower mortality. Our study showed sepsis (54, 68.3%) as the most common manifestation in LOD of GBS infection, followed by UTI (38, 48.1%), meningitis (37.9%), and pneumonia (10.1%). Fever was found in 60 (75.9%) infants and leukopenia in 20 (25.3%) of LOD (both significant); infants in LOD group seemed to have more obvious systemic reaction to GBS infection.

Although Hoshina et al[23] reported deaths significantly higher in LOD than in EOD, Liao et al[12] reported death rates higher in EOD. No significant difference in mortality was found between EOD and LOD (Table 3). There was no statistical significance between maternal fever, chorioamnionitis, and premature labor (less than 34 weeks) between the EOD and LOD group compared with the previous report,[18] maybe because of low patient numbers.

There is limited data regarding risk factors of mortality because of GBS infection in Taiwan. Chung et al[18] reported that gestational age and pneumonia were two most important factors influencing the mortality rate. Our study revealed risk factors of mortality: rescue at delivery room, leukopenia, thrombocytopenia, sepsis, respiratory distress syndrome, PPHN, surfactant use, respiratory failure with either IMV or HFOV, shock, and congenital heart disease. Owing to limited number of fatalities, we performed no further multivariate logistic regression analysis. Mortality in this study was 6.8%, within the range (5—20%) of other reports,[1,2] most fatalities (12/15, 80.0%) happened within the first 7 days, as in other reports.[25] High prevalence of EOD and case fatality in this study further underscores effectiveness of intrapartum antibiotic prophylaxis in Taiwan as in other countries.[5,26—28]

Readers might suspect selective bias because it is mainly from medical centers that might have more high-risk pregnant women and high social-economic class with a high prevalence. To overcome this bias, we did a small sample size by purposive sampling, as suggested by a statistical expert. We hired medical assistants to explain thoroughly to every pregnant woman and persuaded her to receive GBS culture from April to June 2005 in two medical centers. Colonization rate was around 18%, nearly the same as in the six hospitals.

5. Conclusions

With GBS colonization of pregnant women, incidence of EOD and mortality rate nearly the same as those in other countries, universal maternal rectovaginal culture of GBS with subsequent intrapartum antibiotics prophylaxis is mandatory for reducing EOD and mortality of neonatal GBS infection in Taiwan.

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References

1. Centers for Disease Control and Prevention. Diminishing racial disparities in early-onset neonatal group B streptococcal disease United States, 2000-2003. *MMWR* 2004;**53**:502—5.
2. Schrag SJ, Zywicki S, Farley MM, et al. Group B streptococcal disease in the era of intrapartum antibiotic prophylaxis. *N Engl J Med* 2000;**342**:15—20.
3. Schuchat A, Whitney C, Zangwill K. Prevention of perinatal group B streptococcal disease: a public health perspective. *MMWR* 1996;**45**:1—24.
4. Matsubara K, Yamamoto G. Invasive group B streptococcal infections in a tertiary care hospital between 1998 and 2007 in Japan. *Int J Infect Dis* 2009;**13**:679—84.
5. Aavitsland P, Hoiby EA, Lystad A. Systemic group B streptococcal disease in neonates and young infants in Norway 1985-94. *Acta Paediatr* 1996;**85**:104—5.

6. Baker CJ, Edwards MS. Group B streptococcal infections. In: Remington JS, Klein JO, editors. *Infectious Diseases of the Fetus and Newborn Infant*. 4th edition. Philadelphia: W.B. Saunders; 1995. p. 980—1054.
7. Girgitzova B, Minkov N, Zozikov B. Streptococcus agalactiae as a urinary tract pathogen in males and non-pregnant females. *Int Urol Nephrol* 1991;**23**:365—9.
8. Regan JA, Klebanoff MA, Nugent RP. The epidemiology of group B streptococcal colonization in pregnancy. *Obstet Gynecol* 1991;**77**:604—10.
9. Davies HD, Miller MA, Faro S, et al. Multicenter study of a rapid molecular-based assay for the diagnosis of group B streptococcus colonization in pregnant women. *Clin Infect Dis* 2004; **39**:1129—35.
10. Yancey MK, Duff P, Clark P, et al. Peripartum infection associated with vaginal group B streptococcal colonization. *Obstet Gynecol* 1994;**84**:816—9.
11. Agnoli FL. Group B streptococcal. Perinatal consideration. *Fam Pract* 1994;**39**:171—7.
12. Liao CH, Huang LM, Lu CY, et al. Group B streptococcus infection in infancy: 21-year experience. *Acta Paediatr Taiwan* 2002;**43**:326—9.
13. Wu CS, Wang SM, Ko WC, et al. Group B streptococcal infections in Children in a tertiary care hospital in southern Taiwan. *J Microbiol Immunol Infect* 2004;**37**:169—75.
14. Ho MY, Wu CT, Ku YT, et al. Group B Streptococcal infection in neonates: an 11-year review. *Acta Paediatr Taiwan* 1999;**40**: 83—6.
15. Huang FY. Neonatal group B streptococcus infection in Taiwan: an increasing trend. *Acta Paediatr Taiwan* 2002;**43**:312.
16. Chung MY, Ko DJ, Chen CC, et al. Neonatal group B streptococcal infection: a 7-year experience. *Chang Gung Med J* 2004; **27**:501—8.
17. Schrag S, Gorwitz R, Fultz-Butts K, Schuchat A. Prevention of perinatal groupB streptococcal disease. Revised guidelines from CDC. *MMWR Recomm Rep* 2002;**51**:1—22.
18. Baker CJ, Barrett FF, Gordon RC, Tow MD. Suppurative meningitis due to streptococci of Lancefield group B: a study of 33 infants. *J Pediatr* 1973;**82**:724—9.
19. Pass MA, Gray BM, Khare S, Dillon HC. Prospective studies of group B streptococcal infection in infants. *J Pediatr* 1979;**95**: 437—43.
20. Carstensen H, Henrichsen J, Jepsen OB. A national survey of severe group B streptococcal infections in neonates and young infants in Denmark. *Acta Pediatr Scand* 1985;**74**:934—41.
21. Stoll BJ, Schuchat A. Maternal carriage of group B streptococci in developing countries. *Pediatr Infect Dis J* 1998;**17**: 499—503.
22. Bromberger P, Lawrence JM, Braun D, Saunders B, Contreras R, Petitti DB. The influence of intrapartum antibiotics on the clinical spectrum of early-onset group B streptococcal infection in term infants. *Pediatrics* 2000;**106**(2 Pt 1):244—50.
23. Hoshina K, Suzuki Y, Nishida H, et al. Trend of neonatal group B streptococcal infection during the last 15 years. *Pediatr Int* 2002;**44**:641—6.
24. Mckenna DS, lams JD. Group B streptococcal infections. *Semin Perinatol* 1998;**22**:267—76.
25. American College of Obstetricians and Gynecologists. ACOG Committee Opinion: number 279, December 2002. Prevention of early onset group B streptococcal disease in newborns. *Obstet Gynecol* 2002;**100**:1405—12.
26. Moses LM, Heath PT, Wilkinson AR, Jeffery HE, Isaacs D. Early onset group B streptococcal neonatal infection in Oxford 1985-96. *Arch Dis Child* 1998;**79**:F148—9.
27. Volumenie JL, Fernandez H, Vial M, Lebrun L, Frydman R. Neonatal group B streptococcal infection. Results of 33 months of universal maternal screening and antibioprophylaxis. *Eur J Obstet Gynecol Reprod Biol* 2001;**64**:79—85.
28. Davies HD, Adair CE, Schuchat A, Low DE, Sauve RS, McGeer A. Physicians' prevention practices and incidence of neonatal group B streptococcal disease in 2 Canadian regions. *CMAJ* 2001;**164**:479—85.