

# Eight-Year Outcome of Universal Screening and Intrapartum Antibiotics for Maternal Group B Streptococcal Carriers

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**ABSTRACT.** *Objectives.* To report the outcome of intervention to reduce early-onset group B streptococcal disease (EOGBSD) at a tertiary maternity hospital in Sydney and to review all cases of EOGBSD since intervention to improve outcomes further.

*Methodology.* A prospective study was made of all cases of EOGBSD in the 16 months before and 8 years after an intervention that comprised universal screening and intrapartum ampicillin for all maternal carriers of group B streptococcus. Carriers were detected by screening all women at 28 weeks, or 24 weeks with known risk factors for preterm birth, by low vaginal swab, cultured onto blood agar and treated with intravenous ampicillin in labor, 1 g every 6 hours until delivery. Women with a routine midstream urine test positive for group B streptococcus, a previous neonate with EOGBSD, or preterm labor with an unknown carrier status were also treated. EOGBSD was detected by screening all neonates with maternal and/or neonatal risk factors for sepsis.

*Results.* The incidence of blood culture-positive EOGBSD for all live births before intervention was 1.4 per 1000 compared with a rate after intervention of 0.2 per 1000 live births. The incidence, if there were clinical signs of infection and the urine tested positive for streptococcal antigen, decreased from 3.5 per 1000 before intervention to 0.6 per 1000 live births. There was a statistically significant reduction in neonatal morbidity outcomes after intervention, including requirement for admission and treatment in a neonatal unit and the need for ventilation. An audit indicated that by the 8th year, 90% of all pregnant women were screened by a low vaginal swab at 28 weeks and 10.5% were carriers. After intervention, of the 28 neonates with EOGBSD, 64% were associated with departure from the protocol.

*Conclusion.* The intervention has coincided with a significant decrease in the incidence of blood culture-positive EOGBSD to 0.2 and urine streptococcal antigen-positive disease to 0.6 per 1000 live births. The 84% reduction in EOGBSD has been obtained by treating 244 neonates in labor to prevent disease in one neonate. Additional reduction seems possible by improving the compliance by staff with the protocol. By contrast, before intervention and in maternity units throughout Australia with no intervention, the rates for EOGBSD remain largely unchanged at ~2 per 1000 live births. *Pediatrics* 1998;101(1). URL: <http://www.pediatrics.org/cgi/content/>

*full/101/1/e2; group B streptococcus, maternal carriers, universal screening, incidence, neonatal outcomes.*

ABBREVIATIONS. EOGBSD, early-onset group B streptococcal disease; GBS, group B streptococcus; KGVH, King George V Hospital; CSF, cerebrospinal fluid.

Early-onset group B streptococcal disease (EOGBSD) remains the most common, severe bacterial infection in the newborn, with a mortality of <20%, but with remaining significant morbidity.<sup>1-4</sup> The incidence of EOGBSD is in most series similar to that of sudden infant death syndrome before child care interventions, that is, 2 to 3 per 1000 live births but varying from <1 to >4 per 1000 in some regions.<sup>5</sup> Recognition of EOGBSD as a public health issue has been slower than that of sudden infant death syndrome, although preventive strategies have existed longer.

Recent reviews and recommendations have considered the risk/benefit ratio and the economic costs of either a selective or a universal approach to antepartum screening and intrapartum antibiotic treatment of maternal carriers of group B streptococcus (GBS).<sup>6-9</sup> There is agreement that some intervention is indicated to reduce EOGBSD if it contributes significantly to neonatal mortality and morbidity, although the present options are not ideal.

There are three systematic reviews, two with meta-analyses, that evaluate the efficacy of intrapartum antibiotics in reducing the incidence of EOGBSD.<sup>10-12</sup> All three papers conclude that the incidence of colonization of the neonate of a GBS carrier mother is reduced with intrapartum antibiotic treatment. The two meta-analyses suggest that the incidence of infection is reduced. The third review, by Ohlsson and Myer (1994), argue that poor methodology precludes metaanalysis; however, these authors suggest that individual review of each paper indicates a trend to reduction of EOGBSD. Thus, all three reviews concur that neonatal colonization is reduced significantly by as much as 90%. It has been demonstrated by others in a cohort of 10 000 neonates that if there was no colonization (from any of four sites), no EOGBSD occurred.<sup>13</sup>

This is a descriptive paper reporting the outcome after 8 years and 36 342 live births of a prophylactic strategy of nonselective intrapartum antibiotics for maternal carriers of GBS at King George V Hospital (KGVH), Sydney, Australia. This strategy, aimed at the prevention of EOGBSD in neonates, was intro-

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duced as protocol in March 1988. Preliminary results after 32 months have been published previously.<sup>14</sup> A secondary aim was to review all cases of EOGBSD since intervention to establish whether there are ways to improve further the significantly reduced rate.

## MATERIALS AND METHODS

### Study Methods

A prospective study of all neonates admitted to the neonatal units (levels II and III) at KGVH was undertaken to determine the incidence of EOGBSD before and after introduction of the intervention. KGVH, an urban, tertiary referral and teaching hospital of the University of Sydney, serves a multicultural population in central Sydney. There are ~5000 births each year, with a relatively constant preterm delivery rate of 11% to 12% during the last decade and <1% of mothers with no antenatal care.

The background rate of EOGBSD was determined prospectively over a 16-month period (November 1986 to February 1988) before the intervention, which was introduced as protocol in March 1988. After 3 months of in-service education for the staff, data were collected prospectively on all infants with EOGBSD from June 1988.

The 8-year outcomes after the introduction of the protocol (Fig 1) are reported, including the incidence of EOGBSD and the mortality and morbidity. To indicate the current level of compliance with antenatal screening and the carriage rate of GBS, an audit was conducted of the antenatal records of all mothers giving birth

at KGVH during a 1-month period (November 1995 to December 1995). Staff were unaware of this audit and of the previous audits, performed quarterly during the first 32-months after intervention.<sup>14</sup>

### The Intervention

The intervention strategy was incorporated into a protocol that is outlined as follows: antenatal screening for all women at 28 weeks' gestation, or 24 weeks if known risk factors for preterm birth, by low vaginal swab, cultured onto blood agar, with identification by colony morphology, hemolysis, and positive Christie-Atkins-Munch-Peterson reaction. Maternal carriers were treated with intravenous ampicillin in labor, 1 g every 6 hours until delivery, unless there was a definite history of penicillin allergy, when a cephalosporin was used. In addition, women with a routine midstream urine test positive for GBS at any antenatal visit or a history of a neonate with EOGBSD and women in preterm labor with an unknown carrier status were also treated with intrapartum antibiotics, as outlined in Fig 1.

Strategies to prevent resistance included an antibiotic policy that recommended intrapartum, and not antepartum, treatment of GBS carriers and a postnatal policy that restricted the use of penicillin and gentamicin for suspected early neonatal infection. Cessation of antibiotics was recommended at 48 to 72 hours if blood cultures were negative and the infant was clinically well.

### The Outcome

#### Presence of Disease

The presence of EOGBSD was detected by screening all neonates with maternal and/or neonatal risk factors. The method of screening was dependent on whether the neonate was well, but with maternal risk factors for sepsis, or exhibiting signs of possible sepsis. Clinical indicators to suggest possible neonatal EOGBSD were listed as birth asphyxia and/or the occurrence of any of the following within the first 48 hours of life: unexplained respiratory distress, sustained tachycardia (>160 beats per minute), shock, or abnormal temperature (<36.5°C or >37.5°C).

The maternal risk factors were a positive swab for GBS at 24 to 28 weeks' gestation, spontaneous onset of preterm labor (<37 weeks' gestation), prolonged membrane rupture (>12 hours), intrapartum fever (>37.5 C), and fetal distress. Well neonates with maternal risk factors were initially screened for colonization with GBS as well as a latex particle agglutination test for urine GBS antigen. The urine streptococcal antigen test (Wellcogen, Wellcome Diagnostics, Research Triangle Park, NC) was shown to have a high sensitivity and specificity in the preintervention period.<sup>15</sup> After the reported outcomes at 32 months,<sup>14</sup> well neonates at term were no longer screened by investigation but simply observed on the ward with their mother for 24 hours (Fig 1). This was based on all such neonates remaining well and without evidence of clinical signs to suggest EOGBSD. Additional investigations thus were considered noncontributory and costly.

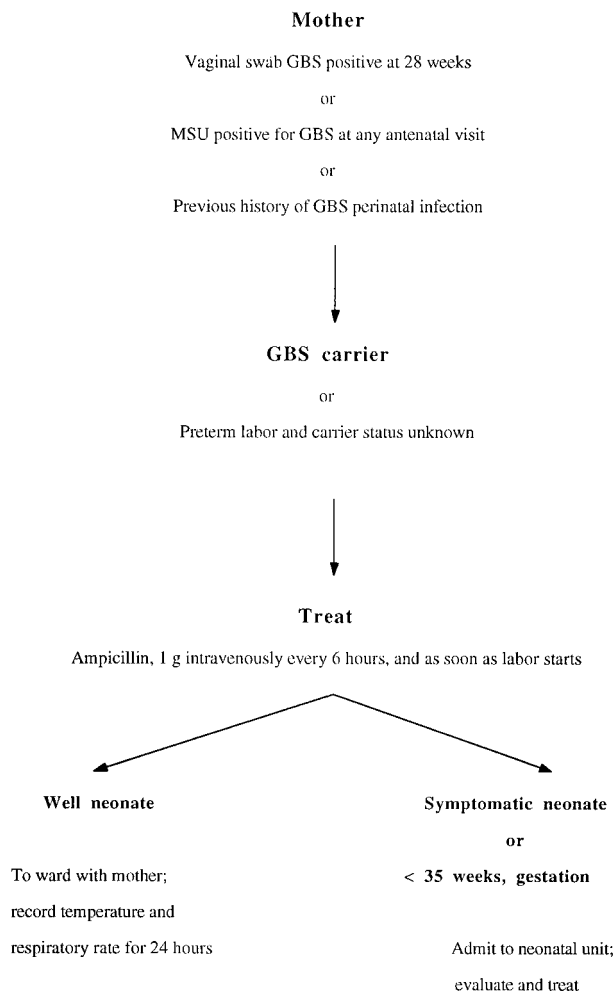
Neonates with any clinical signs to suggest sepsis were investigated by blood culture, blood count, superficial swabs, and a urine streptococcal antigen test. If clinically indicated, cerebrospinal fluid (CSF) and urine were cultured. Histopathologic examination of the placenta of all preterm and most term infants was undertaken using the method of Benirshke,<sup>16</sup> as reported previously for KGVH.<sup>17</sup> All neonates with suspected infection were treated for a minimum of 48 hours with intravenous penicillin and gentamicin.

#### Definition of Disease

EOGBSD was determined prospectively by two methods, both before and after intervention: 1) a positive culture of blood or CSF in a neonate within 48 hours of birth, and 2) a positive urine streptococcal antigen test within 48 hours of birth in a neonate with clinical signs of sepsis. This definition was applied to prevent underenumeration of EOGBSD, because not all neonates will have a positive blood or CSF culture,<sup>18</sup> especially when maternal intravenous antibiotics have been given during labor.

#### Morbidity and Mortality

In infants with EOGBSD, the outcome factors used to determine morbidity were necessity for admission to level II-III neonatal unit



**Fig 1.** Flow sheet of recommendations for antenatal screening and nonselective intrapartum ampicillin for management of maternal GBS carriers and their neonates. MSU indicates midstream urine. Adapted from Jeffery and McIntosh.<sup>14</sup>

for treatment with antibiotics and ventilation. Death that was attributable directly to EOGBSD was determined by a positive blood culture or CSF culture in a sick, colonized neonate, with rapid death occurring usually <48 hours after birth. EOGBSD that contributed to death was defined as a negative blood culture or CSF culture in a sick, colonized neonate with a rapid, fulminating course. Confirmation of GBS pneumonia and/or meningitis was obtained at autopsy. These outcomes were compared with the rates for EOGBSD in the preintervention period.

### Statistics

Preintervention and intervention mortality rates for EOGBSD were compared using the  $\chi^2$  statistic and Fisher's exact test for small numbers where appropriate. Comparison of morbidity was made using confidence intervals and relative risk ratios. A *P* value of <.05 was considered statistically significant.

## RESULTS

The study population included all women who booked at KGVH for delivery or who were referred to KGVH because of a complication with the pregnancy or with labor. Greater than 95% of women were from metropolitan Sydney, principally from the central Sydney area. The losses were relatively few; that is, women who booked but delivered elsewhere. Typically, for the 5 years 1992 to 1996, 95.7% of mothers were booked (96% before 30 weeks' gestation and 84% before 20 weeks' gestation), and only 6% of booked mothers delivered elsewhere. Another 2.9% were transferred to KGVH because of high risk, and 1.4% were unbooked.

### Incidence and Mortality Attributable to EOGBSD

During the 16-month preintervention period, there were 5732 live births and during the 8 years since the introduction of the intervention, a total of 36 342 live births. During the latter period, 19 994 babies were born under universal government insurance coverage and 16 348 babies were born to mothers with private insurance coverage. The patient population under universal government insurance coverage has increased steadily over the period of intervention, from 46% when the study began to 70% during the last year, the highest level recorded for the past decade.

In the 16 months before intervention, 28 infants born at KGVH were admitted with EOGBSD, 8 of whom had positive blood cultures (Table 1). By contrast, there were 28 neonates born at KGVH with

EOGBSD who were admitted to the neonatal unit during the 8 years after intervention, 8 with positive blood cultures. The birth weight distribution of neonates admitted and treated for EOGBSD is illustrated in Table 2.

During the preintervention period, there were three neonatal deaths attributable directly to EOGBSD at 30, 32, and 34 weeks' gestation, all blood culture-positive and confirmed as pneumonia attributable to GBS at autopsy. During the 8-year intervention period, the two neonates who died at 26 and 30 weeks' gestation were blood culture-negative, colonized with GBS, and had a rapid fulminating course characterized by severe neutropenia. Placental histology revealed acute chorioamnionitis in both. Autopsy was performed in one of the two and confirmed hyaline membranes and GBS pneumonia.

### Clinical Signs of Infection and Blood Culture-Positive EOGBSD

The incidence in the 16 months before intervention was 1.4 per 1000, compared with a rate after intervention of 0.2 per 1000 live births (*P* < .0001) (Table 1).

### Clinical Signs of Infection and Urine Streptococcal Antigen-Positive EOGBSD

The incidence of EOGBSD for all live births using this definition of disease decreased from 3.5 per 1000 before intervention to 0.6 per 1000 live births (*P* < .0001) (Table 1). The neonates who were classified in this way were sick infants but, in general, not as ill as those who were blood culture-positive. Nearly all had two or more of the following clinical signs: apnea, respiratory distress, poor perfusion, or temperature <36.5°C or >37.5°C. The total incidence of EOGBSD thus has decreased from 4.9 to 0.8 per 1000 live births. The reciprocal of the difference between these two proportions indicates that 244 neonates must be treated during labor to prevent 1 case of EOGBSD.

After intervention, confirmation of prenatal infection was sought from placental histopathologic examination in 20 of the 28 neonates with EOGBSD. Placentae of 8 neonates were discarded inadvertently. Evidence of chorioamnionitis was found in 18

**TABLE 1.** Group B Streptococcal Incidence and Morbidity Per 1000 Live Births

	Preintervention 16 Months 5732 Livebirths	Intervention 8 Years 36 342 Livebirths	Relative Risk (95% CI)	<i>P</i> Value
Blood culture positive				
All babies*	1.4 (8)†	0.2 (8)†	6.35 (2.38, 16.92)	<i>P</i> = .0001
Ventilated	0.7 (4)	0.1 (4)	6.34 (1.59, 25.35)	<i>P</i> = .013
Urine streptococcal antigen positive				
All babies*	3.5 (20)	0.6 (20)	6.34 (3.41, 11.78)	<i>P</i> < .0001
Ventilated	1.4 (8)	0.2 (9)	5.64 (2.18, 14.60)	<i>P</i> = .0002
Total incidence	4.9 (28)	0.8 (28)	6.34 (3.76, 10.70)	<i>P</i> < .0001

\* All babies treated in a level II–III neonatal unit.

† Number of infants in parentheses.

**TABLE 2.** Distribution of EOGBSD by Birth Weight (in Grams)

	<1000	1000–1499	1500–2499	>2500
<b>Preintervention</b>				
Blood culture-positive	0	1	3	4
Urine strep antigen-positive	0	2	3	15
<b>Postintervention</b>				
Blood culture-positive	0	0	3	5
Urine strep antigen-positive	2	2	9	7

of 20 examinations and umbilical vasculitis in 11 of the 20 infants. Four of the 18 neonates with chorioamnionitis were blood culture-positive; the remaining 14 were urine streptococcal antigen-positive EOGBSD.

In the preintervention period, 40% of mothers of neonates with EOGBSD did not have any of the three published risk factors used for selective intervention, namely spontaneous onset of preterm labor (<37 weeks' gestation), prolonged membrane rupture (>12 hours), or intrapartum fever (>37.5 C).<sup>19</sup> After intervention, this figure was 11%.

### Neonatal Morbidity

There was a statistically significant reduction in neonatal morbidity outcomes after intervention compared with the preintervention group, including requirement for admission and treatment in a neonatal unit and the need for ventilation (Table 1).

### Maternal Morbidity

There was one severe, maternal allergic reaction to ampicillin. This one mother (of ~3400 treated carriers) had no history to suggest sensitization and was treated effectively with steroids and antihistamines but did not require epinephrine. There was no fetal distress, and her term baby was well at delivery and successfully breastfed from the first postnatal day.

### Audit

The audit indicated that by December 1995, 90% of all pregnant women were screened by low vaginal swab at 28 weeks' gestation, and the maternal GBS carriage rate was 10.5%, compared with a 12% rate reported previously.<sup>14</sup>

## CASE REVIEWS

### Not Treated With Intrapartum Antibiotics Per Protocol

Of the 28 neonates with EOGBSD in the 8 years after the introduction of the intervention strategy, 18 (64%) were born to mothers who were not screened and/or treated with intrapartum antibiotics as directed by the protocol. The two neonatal deaths occurred in this group (Table 3). The antenatal screening status of these 18 mothers included 14 who were

not swabbed antenatally and three who had a positive swab at 28 weeks and were not treated in labor. One mother had a negative swab but had a previous neonate with EOGBSD and was not treated in labor.

Twelve of the 14 mothers in the first group went into preterm labor, and the other 2 mothers went on to deliver at term gestation. Of these 2 mothers, 1 developed a fever in labor and was not treated with intravenous antibiotics. Her neonate developed blood culture-positive EOGBSD. The remaining neonate was born to a mother who was not swabbed and not treated with intrapartum antibiotics. A postnatal vaginal swab was positive for GBS.

### Negative Swabs at 28 Weeks' Gestation but EOGBSD

Eight neonates (29%) with EOGBSD, three term and five preterm, were born to mothers who had negative swabs at 28 weeks and therefore were not treated with intrapartum antibiotics. Four of the five preterm neonates whose mothers had a negative swab at 28 weeks' gestation, had a peripartum vaginal swab performed that was positive for GBS.

### EOGBSD Despite Intrapartum Antibiotics Per Protocol

Two neonates developed EOGBSD despite maternal treatment with intrapartum ampicillin at least 4 hours before delivery. The mother of one neonate was transferred in preterm labor at 31 weeks' gestation from a rural center, carrier status unknown. She was treated with three doses of intravenous ampicillin, 1 g at 6 hourly intervals while in labor. Her neonate, who was born after two doses of steroids and failed tocolysis with intravenous salbutamol, was ventilated and had a successful outcome despite a positive blood culture for GBS. The second such neonate was born at term with pneumonia attributable to EOGBSD. His mother was a known vaginal carrier with recurrent bacteruria attributable to GBS and was treated with 1 g of ampicillin 5 hours before delivery. The neonate recovered well with antibiotic treatment.

## DISCUSSION

Routine antenatal screening and nonselective intrapartum antibiotic treatment at KGVH has coincided with a significant reduction in the incidence of EOGBSD. Such a dramatic decrease in incidence and morbidity has not been seen at other maternity units surveyed in Australia, where there is limited or no prophylaxis.<sup>4,20</sup> In the other six maternity units, surveyed prospectively for the second consecutive year in 1993, the incidence of EOGBSD in non-Aboriginal neonates, as defined by blood culture, was 2.2 per 1000.<sup>4</sup> In contrast, at KGVH the rate was 0.4 per 1000 in 1993 and 0.2 for the entire 8 years after intervention. The only published data of the incidence of EOGBSD in Aboriginal neonates found a markedly

**TABLE 3.** Summary of Protocol Failures

Cases	28-Week Swabs	Deaths	Gestational Age (in Weeks)
18	Not performed	14	1
	Positive	3	1
	Negative but previous EOGBSD	1	—
			26, 26, 31, 31, 31, 31, 33, 33, 33, 34, 35, 35, 37, 41, 30, 34, 41, 39

higher rate of 5.2 per 1000 live births.<sup>4</sup> At KGVH, 2% of mothers are Aboriginal, as defined by questionnaire at booking.

The reduction in EOGBSD at KGVH is greater than the earlier reported outcome at 32 months after the intervention was introduced. At this stage, the incidence of EOGBSD was 0.37 per 1000 live births. This is at least partly attributable to evidence of improved compliance with antenatal screening, which has increased from a reported rate of 75% for government-insured mothers and 40% for privately insured mothers at 32 months<sup>14</sup> to a combined 90% rate at 8 years. Other reports of outcome for either universal<sup>21,22</sup> or selective screening<sup>23,24</sup> also suggest a reduction in EOGBSD. Late-onset GBS disease was not measured in our study; however, it is likely that the decreased reservoir of colonized neonates after intrapartum treatment of maternal carriers would contribute to a reduction in late-onset infection in the nursery as well as after returning home.

In reporting the present outcomes, we recognize the limitations of using a historical control group. However, the data were collected prospectively, the disease was determined by two methods to ensure that no case was missed, and the Australian Study Group for Neonatal Infections has provided interstate data in similar settings for comparison.<sup>4,20</sup> The reduction in the incidence of disease at KGVH is similar, although lower, than that reported by the only other Australian tertiary maternity hospitals that have applied universal screening at 32 weeks' gestation, followed by intrapartum treatment of maternal GBS carriers.<sup>21</sup> With a similar carriage rate of 12%, their rate of EOGBSD, determined retrospectively from blood culture-positive neonates, was 0.5 per 1000 live births. No estimate of compliance with the protocol was given, although this may explain the difference in incidence rates.

Another factor that could have decreased the incidence of the disease over the 8 years of intervention is alteration in GBS virulence, but not carriage rate, because this has remained constant between 10% and 12%. However, incidence rates remain unchanged elsewhere in Australia,<sup>4,20</sup> suggesting that virulence has not lessened. Alternatively, referral patterns and therefore risk factors may have changed, but one of the major predisposing factors, preterm birth, has remained relatively constant at 11% to 12% of all births, reflecting the tertiary preference/referral of high-risk obstetric mothers to KGVH. Insurance status, a quasisocioeconomic indicator, has changed in the last 8 years. Despite a significant increase in government-insured mothers and their infants, with higher rates of EOGBSD both before and during intervention, the overall rate of the disease remains significantly reduced.

Another explanation for the reduced incidence of disease after intervention is a possible misclassification bias from nondetection of EOGBSD. This seems unlikely, because the policy concerning level I neonates has remained constant and includes only well infants remaining with their mothers. All others are admitted to level II or III neonatal units. However, some neonates with EOGBSD of borderline viability,

23 to 25 weeks' gestation, who died in the delivery room before admission to the neonatal intensive care unit may have been undetected. Such a bias would have been greater in the preintervention period when such neonates were not routinely offered active intervention and thus would have augmented rather than decreased the pre- and postintervention difference in incidence of EOGBSD.

Several important clinical observations emerged from the maternal and neonatal outcomes. First, no evidence of altered bacterial resistance has been observed in the neonatal flora of inborn neonates either by the hospital microbiologists or independently by pediatric infectious disease specialists. The latter have independently monitored infection disease rates and antibiotic use in the neonatal unit prospectively for the past 7 years<sup>4,20</sup> and reported all GBS fully sensitive to penicillin. Antibiotic use was reported as comparatively low,<sup>25</sup> with exposed neonates receiving antibiotics for a mean of 13.8 days per 100 days of hospitalization. In addition, the pattern of organisms causing early sepsis was similar for babies exposed to intrapartum antibiotics, compared with nonexposed babies.

Second, EOGBSD, despite intrapartum antibiotics for >4 hours before delivery for maternal carriers, is uncommon. There were two such neonates with respiratory distress at birth and confirmed EOGBSD. Their mothers were symptomatic, one with intrapartum fever and the other with prolonged preterm rupture of membranes, and both received intravenous ampicillin. This amounts to 2 in ~3400 maternal carriers who received intrapartum antibiotics and delivered a symptomatic neonate.

Finally, the 8-year outcomes confirm that carrier mothers who received intrapartum antibiotics and delivered an asymptomatic neonate at  $\geq 35$  weeks' gestation remained well, and none developed EOGBSD. Thus, well neonates of treated carrier mothers can safely remain untreated after birth and with their mother.

These findings suggest that tighter adherence to the protocol could reduce further the incidence of EOGBSD. Compliance with screening is high at 90%, but additional improvement is possible because the major cause (64%) of EOGBSD was vertical transmission in mothers either not screened and/or not treated intrapartum as directed by the protocol.

After intervention, there were eight neonates (29%) with EOGBSD whose mothers had a negative swab at 28 weeks' gestation, but who were positive at delivery, representing either a false-negative result or a later acquisition of the organism. This situation has been estimated to occur in 0% to 13% of women.<sup>26,27</sup> This situation could be addressed by the additional use of a composite anal and low vaginal swab and the use of selective media for culture of swabs. Such action would incur added expense, but may prevent infection in these neonates by improving the detection of maternal carriers. A rapid, accurate, inexpensive intrapartum screening test could also provide alternative prevention, assuming maternal recognition of labor and presentation in early labor. However, intrapartum screening tests cur-

rently available are insensitive and detect heavy, but not light, colonization.<sup>7,26</sup>

In conclusion, at KGVH, universal antepartum screening at 28 weeks' gestation and intrapartum antibiotics for maternal carriers of GBS have coincided with a significant decrease in the incidence of EOGBSD to 0.2 per 1000 live births for blood culture-positive disease and to 0.6 per 1000 live births for urine streptococcal antigen-positive disease. The 84% reduction in EOGBSD has been achieved by treating 244 neonates in labor to prevent disease in 1 neonate. Additional reduction seems possible by improving staff compliance with the protocol. In contrast, before the intervention at KGVH and in maternity units throughout Australia with no intervention, the rates for EOGBSD remain largely unchanged at ~2 per 1000 live births.

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## **Eight-Year Outcome of Universal Screening and Intrapartum Antibiotics for Maternal Group B Streptococcal Carriers**

Heather E. Jeffery and Monica Moses Lahra

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