The Relationship of Nosocomial Infection Reduction to Changes in Neonatal Intensive Care Unit Rates of Bronchopulmonary Dysplasia

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Objective To examine whether recent reductions in rates of nosocomial infection have contributed to changes in rates of bronchopulmonary dysplasia (BPD) in a population-based cohort.

Study design This was a retrospective, population-based cohort study that used the California Perinatal Quality Care Collaborative database from 2006 to 2013. Eligible infants included those less than 30 weeks' gestational age and less than 1500 g who survived to 3 days of life. Primary variables of interest were rates of nosocomial infections and BPD. Adjusted rates of nosocomial infections and BPD from a baseline period (2006-2010) were compared with a later period (2011-2013). The correlation of changes in rates across periods for both variables was assessed by hospital of care.

Results A total of 22 967 infants from 129 hospitals were included in the study. From the first to second time period, the incidence of nosocomial infections declined from 24.7% to 15% and BPD declined from 35% to 30%. Adjusted hospital rates of BPD and nosocomial infections were correlated positively with a calculated 8% reduction of BPD rates attributable to reductions in nosocomial infections.

Conclusions Successful interventions to reduce rates of nosocomial infections may have a positive impact on other comorbidities such as BPD. The prevention of nosocomial infections should be viewed as a significant component in avoiding long-term neonatal morbidities. *(J Pediatr 2017;180:105-9).*

Nosocomial infections and bronchopulmonary dysplasia (BPD) are 2 of the most prevalent morbidities in infants with very low birth weight (VLBW) and are closely linked to one another.[1,2] Inflammatory mediators, which are increased during infectious processes, appear to be a significant component of the pathogenesis of BPD.[3-5] Tracheal aspirate analyses of infants who go on to develop BPD have shown increased neutrophils, macrophages, and cytokines, as well as the presence of microbials, compared with those infants who did not have such findings.[3,6-8] Proinflammatory cytokines are thought to disrupt normal late gestational age alveolar and vasculature development with resultant simplified alveoli, dysmorphic vasculature, and impaired gas exchange as seen in postsurfactant BPD ("new" BPD).[4,9-15]

Several studies have demonstrated an increased risk of developing BPD following neonatal late-onset sepsis (ie, nosocomial infections).[16-20] The rates of nosocomial infections remain alarmingly high, affecting up to one-quarter of infants with VLBW in the Neonatal Research Network.[21] Fortunately, the rates of nosocomial infections have declined over the years through successful quality improvements.[22-28] It is unknown whether successful reduction of the rates of nosocomial infection is associated with reduction in the rates of BPD.

The purpose of this study was to determine whether declines in neonatal rates of nosocomial infections also may contribute to neonatal intensive care unit (NICU)-level reductions in BPD in a population-based cohort. We hypothesize that hospitals with successful reductions in the rates of nosocomial infections also will have small, but significant, reductions in the rates of BPD.

Methods

This study used data from the California Perinatal Quality Care Collaborative (CPQCC) from January 2006 to December 2013. CPQCC prospectively collects clinical data on greater than 90% of infants with VLBW admitted to NICUs in California by the use of an expanded Vermont Oxford Network dataset.[29]

BPD	Bronchopulmonary dysplasia
CPQCC	California Perinatal Quality Care Collaborative
NICU	Neonatal intensive care unit
PMA	Postmenstrual age
VLBW	Very low birth weight

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The authors declare no conflicts of interest.

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http://dx.doi.org10.1016/j.jpeds.2016.09.030

For this analysis, eligible infants included those born between $22^{0/7}$ weeks' to $29^{6/7}$ weeks' gestational age with a birth weight between 401 and 1500 g. Infants inborn or outborn transferred to a CPQCC hospital within the first 2 days were included. Exclusion criteria included a major congenital anomaly, delivery room death, or death before 72 hours of life. The 72-hour window allowed us to perform analyses on infants who survived long enough to acquire a qualifying nosocomial infection. Analyses included hospitals with 10 or more infants meeting eligibility criteria during the study period.

The variables of interest were nosocomial infection and BPD. Nosocomial infection was defined as culture-proven infection with bacteria recovered from blood and/or cerebrospinal fluid after day 3 of life. Coagulase-negative *Staphylococcus* isolates were considered as nosocomial infections only if the infant exhibited clinical signs of illness. In the case of multiple infections, only the initial episode was included. BPD was defined by infant's requirement for oxygen at 36 weeks postmenstrual age (PMA), discharge home on oxygen between 34 and 36 weeks' PMA, or transfer to a non-CPQCC hospital receiving oxygen between 34 and 36 weeks' PMA. The definition including infants transferred between 34 and 36 weeks was used because CPQCC did not consistently collect further data on all such infants. If transfer to a non-CPQCC hospital occurred at <34 weeks' PMA, those infants were coded as having an unknown BPD status. This definition approximates the Vermont Oxford Network definition for BPD.[30]

Statistical analyses were performed with SAS (SAS Institute Inc, Cary, North Carolina). A multivariable logistic regression model was used to risk-adjust rates of nosocomial infections with the following variables: maternal race/ethnicity, gestational age, multiple births, small for gestational age, sex, Apgar score at 5 minutes, and postnatal steroids. Using the same methodology as a previously published model,[31] we calculated the rates of risk-adjusted BPD rates, taking into account all risk factors listed in **Table I** (available at www.jpeds.com), along with hospital as a random effect.

Adjusted rates were compared between 2 time periods: years 2006-2010 (period 1) and years 2011-2013 (period 2). These time periods were selected as CPQCC, in conjunction with California Children Services, led a state-wide collaborative quality improvement project to reduce nosocomial infections between 2008 and 2009 based on previous successes.[25,26] Correlation was assessed between changes in hospital rates for BPD and changes in hospital rates of nosocomial infections between period 1 and period 2.

Results

From 2006 to 2010, a total of 22 967 qualifying infants from 129 hospitals were included in the final analyses. Gestational age, birth weight, and sex, were similar between the 2 time periods (**Table II**). From period 1 to period 2, there were significant increases in maternal group B *streptococcus*-positive screening, antenatal corticosteroid administration, and number of inborn patients. There were concurrently significant decreases in multiple gestation births and postnatal corticosteroid administration. Nosocomial infection was associated with increased risk of developing BPD (OR 2.74; 95% CI 2.54-2.94), with a similar association during the 2 time periods (**Table I**).

Table II. Baseline demographics comparing period 1 (2006-2010) and period 2 (2011-2013)

	Period 1, 2006-2010	Period 2, 2011-2013	*P* value
Total number of infants, n	14 866	8101	
Gestational age, d (SD)	189.3 (13.1)	189.3 (13.0)	.98
Birth weight, g (SD)	958.2 (258.9)	959.8 (258.8)	.65
Male sex, n (%)	7941 (53.5)	4298 (53.1)	.52
Maternal ethnicity, n (%)			
Black	1980 (13.4)	1171 (14.5)	<.01
Hispanic	7253 (49.0)	3717 (46.0)	
White	3860 (26.1)	2029 (25.1)	
Other	1710 (11.6)	1158 (14.3)	
Cesarean delivery, n (%)	10 453 (70.3)	5660 (69.9)	.46
Maternal chorioamnionitis, n (%)	1311 (8.8)	790 (9.8)	.02
Maternal GBS positive, n (%)	1205 (10.1)	869 (12.7)	<.01
Received antenatal steroids, n (%)	12 046 (81.0)	6961 (85.9)	<.01
Birth location, n (%)			
Inborn	12 267 (82.5)	6904 (85.2)	<.01
Outborn	2599 (17.5)	1197 (14.8)	
Small for gestational age, n (%)	614 (5.1)	367 (4.5)	.15
Multiple births (%)	3854 (25.9)	1898 (23.4)	<.01
Apgar score at 5 min (%)			
0-4	1479 (10.0)	876 (10.9)	<.01
5-7	4947 (33.5)	2906 (36.1)	
8-10	8329 (56.4)	4259 (53.0)	
Received postnatal steroids, n (%)	3791 (25.5)	1684 (20.8)	<.01

GBS, group B *streptococcus*.

Adjusted rates of nosocomial infections steadily declined from 24.7% in 2006 to 15% in 2013 with an average of 19.1% during the 7-year period (**Figure 1**). There was a 6.9% decrease in adjusted rates of nosocomial infections between the periods from 21.7% in period 1 to 14.8% in period 2. Adjusted rates of BPD declined from 35% in 2006 to 30% in 2013 with an average of 33.8%. There was a 3.4% decrease in adjusted BPD rates between the 2 time periods (35.1% vs 31.7%).

Using the predetermined time periods, we compared changes in hospital-adjusted BPD rate with changes in hospital-adjusted rates of nosocomial infection between the 2 time periods (**Figure 2**). A total of 114 hospitals were included in the analysis. Regression analysis demonstrated a positive relationship with a slope of 0.31 and coefficient of determination (r^2) of 0.08. The coefficient of determination suggests that 8% of the reduction in BPD is attributable to the reduction in the rate of nosocomial infections.

Discussion

Our study found that rates of nosocomial infections have declined steadily during the past decade. Furthermore, we found that adjusted hospital BPD and rates of nosocomial infections were positively correlated, with a calculated 8% reduction of BPD rates attributable to reductions in nosocomial infections. Prevention of nosocomial infections, such as ventilator-associated pneumonias and central line-associated

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