Is Apnea of Prematurity Present at the Time of Neonatal Intensive Care Unit Discharge Associated with Developmental Delay at 3 Years of Age?

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Background: Apnea of prematurity (AOP) occurs commonly in preterm infants, due to immature respiratory control by the central nervous system (CNS) as well as obstructive causes affected by low motor tone.

Objective: To investigate whether very preterm (VPT: ≤ 32 weeks gestation) infants being treated for AOP at the time of Neonatal Intensive Care Unit (NICU) discharge (DC), a potential marker of CNS maturational delay, have delayed development at 3 years adjusted age (AA) as evidenced by lower scores on the Bayley Scales of Infant Development, 3rd edition (BSID-III).

Methods: This is a retrospective cohort study of former VPT infants who were followed at the Regional Neonatal Follow-up Program affiliated with Maria Fareri Children's Hospital at Westchester Medical Center (Valhalla, New York, USA). We compared those infants DC'd home with treatment for AOP (caffeine and/or apnea monitor) to those who were not. Infants with genetic or congenital neurologic conditions known to affect development were excluded. The primary outcome is the comparison of the mean scores as measured with the BSID-III for cognitive, gross and fine motor, and receptive and expressive speech between the two groups at 3 years AA. We further identified conditions of prematurity which were associated with AOP at discharge. Data analysis was performed utilizing SPSS v16.0. Chi-squared tests were used for categorical and t-tests for continuous variables. P value < 0.05 is statistically significant.

Results: In a cohort of 216 patients who had BSID-III evaluations at 3 years AA, 29 were treated for AOP at DC and 187 were not. Comparing those with AOP at DC to those without, gestational age (GA) at birth (27.1±3.0 vs. 27.9±2.9 weeks), birth weight (1109±441 vs. 1174±469 grams), GA at DC (38.6±2.3 vs. 38.1±4.1 weeks), and AA at BSID-III evaluation (32.8±6.5 vs. 35.0±4.6 months) were not different. BSID-III results were similar, except for fine motor composite scores, which were lower in infants with AOP at DC (90.0±11 vs 95.0±11.7) (Table 1). Conditions related to prematurity and their associations with AOP at the time of DC are listed in Table 2.

Conclusion: Although continued treatment for AOP at DC may represent delayed CNS maturational development, such delay did not persist to 3 years AA in domains other than fine motor development. Further, it does not appear that the practice of continued treatment for AOP at home after DC puts infants at significantly greater risk for future developmental delays.

Table 1. BSID-III scores for infants with and without AOP at discharge

BSID-III component	AOP at D/C (n=29)	No AOP at D/C (n=187)
Cognitive composite score[a]	97.1 (±16.1)	97.2 (±11.6)
Receptive language composite score[a]	95.4 (±17.1)	98.8 (±13.6)
Expressive language composite score[a]	94.4 (±13.9)	94.9 (±13.9)

Fine motor composite score[a]	90.0 (±11.0)*	95.0 (±11.7)*
Gross motor composite score[a]	88.0 (±18.2)	92.0 (±12.2)
*P < 0.05; [a]Mean (Standard deviation)		

Table 2. Characteristics and conditions related to prematurity in infants with and without AOP at discharge

.	AOP at Discharge (n=29)	No AOP at Discharge (n=187)
Mother received antenatal steroids[b]	26 (89.7)	150 (80.2)
Mother received antenatal magnesium sulfate[b]	11 (37.9)	75 (40.1)
Infant received postnatal steroids[b]	5 (17.2)	18 (9.6)
Infection (bacteremia, UTI, or meningitis)[b]	5 (17.2)	32 (17.1)
Retinopathy of prematurity[b]	13 (44.8)*	45 (24.1)*
Chronic lung disease[b]	13 (44.8)	62 (33)
Necrotizing enterocolitis[b]	4 (13.8)	14 (7.5)
Intraventricular hemorrhage (Grade 1 & 2)[b]	7 (24.1)	25 (13.4)
Intraventricular hemorrhage (Grade 3 & 4)[b]	3 (10.3)	13 (7.0)

Periventricular leukomalacia[b]	3 (10.3)	12 (6.4)
Patent ductus arteriosus treated[b]	12 (41.4)	66 (35.3)

*P < 0.05; [b] n (%)