Car Seat Screening for Low Birth Weight Term Neonates

Natalie L. Davis, MD, MMSc

abstract

BACKGROUND AND OBJECTIVE: Car seat tolerance screening (CSTS) is a common predischarge assessment of neonates. Almost half of nurseries and NICUs have low birth weight (LBW, <2.5 kg) as an inclusion criterion, regardless of birth gestational age (GA). Little is known about the epidemiology of CSTS in this cohort. The objective of this study was to identify incidence and risk factors for CSTS failure in term LBW infants.

METHODS: This was a retrospective medical record review of 220 full-term LBW infants qualifying for CSTS over a 4-year period between January 2010 to December 2013. We described CSTS results and performed bivariate analyses to evaluate for predictors of failure.

RESULTS: Overall failure incidence was 4.8%. There were no differences between those who passed and those who failed based on birth weight, birth GA, race, gender, Apgar scores, respiratory support requirements, magnesium exposure, corrected GA, or weight at the time of CSTS. Maternal urine toxicology positive for opiates was found to be a significant predictor of CSTS failure. Of the 9 subjects who failed, 2 had a specific diagnosis identified (Prader–Willi syndrome and long QT syndrome) after a failed CSTS prompted closer examination and workup before discharge.

CONCLUSIONS: We found a similar incidence of failure for full-term LBW infants as has been previously reported for preterm infants. The infants who failed were more likely to have mothers who tested positive for opiates before delivery. Epidemiologic data are provided to help guide future CSTS policies and protocol development for this group.

FREE

Division of Neonatology, Department of Pediatrics, University of Maryland School of Medicine, Baltimore, Maryland

www.pediatrics.org/cgi/doi/10.1542/peds.2014-3527

DOI: 10.1542/peds.2014-3527

Accepted for publication Apr 14, 2015

Address correspondence to Natalie L. Davis, MD, MMSc, Department of Pediatrics, Division of Neonatology, University of Maryland Children's Hospital, University of Maryland School of Medicine, 110 S Paca Street, 8th Floor, Baltimore, MD 21201. E-mail: ndavis@peds.umaryland.edu

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

FINANCIAL DISCLOSURE: The author has indicated she has no financial relationships relevant to this article to disclose.

FUNDING: No external funding.

POTENTIAL CONFLICT OF INTEREST: The author has indicated she has no potential conflicts of interest to disclose.

COMPANION PAPER: A companion to this article can be found on page 183, and online at www.pediatrics.org/cgi/doi/10.1542/peds.2015-1292.

WHAT'S KNOWN ON THIS SUBJECT: Almost half of NICUs include low birth weight (<2.5 kg) as an inclusion criterion for car seat tolerance screening (CSTS), formerly car seat challenges. However, little is known about incidence and risk factors for failure in this group.

WHAT THIS STUDY ADDS: This is the largest study to date evaluating the incidence and predictors of CSTS failure in full-term low birth weight neonates. Epidemiologic data are provided to help guide future CSTS policies and protocol development for this group.

Car seat tolerance screening (CSTS), also known as the infant car seat challenge or the car seat test, is one of the most common predischarge tests performed on neonates, having been recommended since the early 1990s for all infants born at <37 weeks' gestational age (GA).[1–3] CSTS consists of a period of observation before discharge to monitor for apnea, bradycardia, and desaturations while the infant sits in a car safety seat. Despite the widespread implementation of this test, many questions remain about inclusion and failure criteria, what to do when an infant fails, and even the utility of this test in identifying infants at risk for adverse cardiopulmonary outcomes after discharge.

The most recent guidelines from 2009 recommend that CSTS entail a period of observation for 90 to 120 minutes or the duration of the car ride home, whichever is longer, for all infants born at <37 weeks' GA.[3] Although the authors note that some hospitals test infants other than those born preterm, namely those with hypotonia, micrognathia, and congenital heart disease, no specific recommendations for testing these groups are made.[3] It is left up to each institution to decide which neonates should undergo CSTS, which remains challenging because the literature available to guide clinicians on performing this test is minimal. One of the most commonly tested groups besides preterm infants are low birth weight (LBW, <2.5 kg) neonates. Almost half of NICUs surveyed have LBW as an inclusion criterion, regardless of birth GA,[4] probably because of early studies speculating that LBW infants fit poorly into conventional car seats, leading them to slouch and have improper fit of straps and harnesses, potentially compromising their safety.[5] Subsequent studies speculated that positioning in conventional car seats designed for larger children may compromise ventilation specifically in premature infants.[6,7] However, LBW full-term infants were not specifically studied.

Although few published studies exist to evaluate this speculation, predischarge weight <2 kg has been linked to higher risk of failure, and those with lower weights at the time of testing have been shown to be at higher risk of poor repeatability of the CSTS result.[8,9] Most studies have focused on preterm infants, who are at higher risk for confounding comorbidities such as chronic lung disease and apnea of prematurity. Little is known about the risk factors for failure in LBW term infants. This group may be at greater risk of adverse cardiopulmonary events in their car seat not only because of their small size but as a function of the timing of testing. Despite small size, term infants are generally discharged earlier than preterm infants. Because younger chronologic age at the time of testing has been shown to be associated with failed CSTS,[10] it is potentially another risk factor for LBW term infants.

This study was performed to determine the incidence of CSTS failure in a large cohort of LBW term infants to characterize demographic and clinical variables that are significantly associated with CSTS failure. We hypothesized that infants who had younger chronologic ages and lower weights at the time of CSTS would be more likely to fail.

METHODS

Study Population

This was a retrospective medical record review of full-term infants born over a 4-year period between January 1, 2010 and December 31, 2013 and admitted to the University of Maryland Children's Hospital in Baltimore, Maryland, who qualified for CSTS because they were born LBW. The study was approved by our institutional review board. Inclusion criteria included birth GA >37 weeks, LBW, and survival to discharge. Exclusion criteria included being discharged on a home ventilator and being deemed inappropriate for CSTS by medical team, including being discharged to hospice care. These dates were chosen because they were after release of the most recent American Academy of Pediatrics (AAP) CSTS guidelines from 2009.[3]

Car Seat Tolerance Screening

At our institution, CSTS involves a period of observation ≥90 minutes or estimated travel time home, whichever is longer. The test is performed on all infants born at <37 weeks' GA and all infants with birth weight <2.5 kg regardless of birth GA and regardless of associated comorbidities. CSTS is performed in the unit of discharge, either the full-term nursery (FTN) or the NICU. Subjects are tested in their personal car seat. Car seat fit assessment is standardized and performed by trained staff before testing. We have a protocol that specifies shoulder harness position, crotch strap distance, and proper versus improper use of blankets or rolls for stability consistent with AAP recommendations.[3] Car seat angle, weight specifications, expiration date, and history of involvement in a motor vehicle accident are all specifically assessed. Certified child passenger safety technicians are available to assist in positioning if necessary. At the time of this study, failure criteria include apnea, cessation of respirations for >20 seconds; bradycardia, any heart rate <80 beats per minute; and desaturation, any drop in saturation to <88%. Under our protocol, when an infant fails the CSTS, the provider is notified and appropriate interventions are performed to ensure immediate safety (eg, removal from car seat, oxygen administration).

Statistical Methods

Baseline characteristics were evaluated between the subjects who

failed and those who passed their initial CSTS. In addition, we evaluated characteristics between subjects who had full documentation and those who did not have a documented CSTS. We used *t* testing, nonparametric Wilcoxon rank-sum testing, χ^2, and Fisher exact testing as appropriate for continuous, binary, and categorical variables. These variables included gender, race, birth weight, birth GA, singleton versus multiple gestation, delivery mode, admission location (NICU versus FTN), Apgar score, maternal anesthesia (general, epidural or spinal, none), maternal analgesics during labor (opiates versus synthetically derived opioid agonist–antagonist analgesic butorphanol tartrate), resuscitation requirements with positive pressure ventilation, group B *Streptococcus* (GBS) status, maternal magnesium exposure, antenatal steroids, respiratory support requirements (intubation, surfactant, mechanical ventilation, continuous positive airway pressure, or nasal cannula), postnatal steroids, caffeine, intraventricular hemorrhage, need for a gastrostomy tube, need for a tracheostomy, and characteristics at the time of CSTS, including weight and corrected GA. Upon admission to our hospital, urinary toxicology is sent for all mothers, and for all infants, which is standard practice at our institution. These results were also analyzed. We performed all statistical analyses by using SAS 9.3 (SAS Institute, Inc, Cary, NC).

RESULTS

A total of 220 subjects were born full term and LBW between 2010 and 2013, of whom 119 spent at least some portion of their admission in the NICU and 101 were admitted entirely to the FTN. Of these, 5 died before testing, 1 was discharged on a home ventilator, and 1 was discharged to home hospice and did not undergo testing (Fig 1). Of those who died before testing, diagnoses included congenital diaphragmatic hernia, trisomy 18, ectodermal dysplasia, multicystic kidney disease with pulmonary hypoplasia, and interstitial lung disease with an Ebstein-like cardiac anomaly. The subject who went home on hospice care had trisomy 18, and the subject discharged on a ventilator had a tracheostomy due to a vascular ring and tracheomalacia.

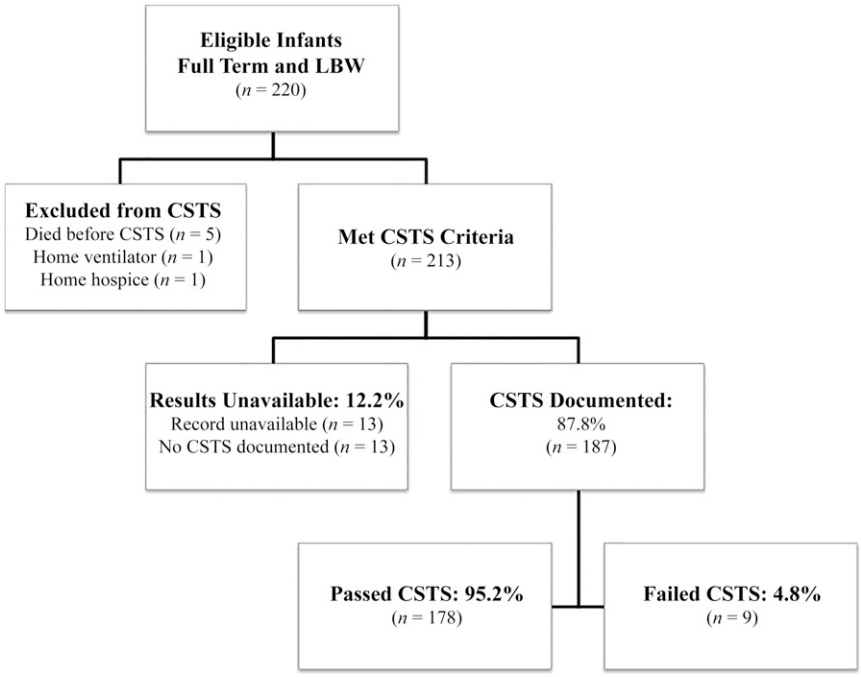

FIGURE 1
Flow diagram of study population.

Therefore, a total of 213 subjects qualified for CSTS, and we had complete data on 87.8% (*n* = 187). The remainder either were transferred to another facility before testing (*n* = 13) or were discharged from our facility without documentation of CSTS result (*n* = 13). Of those without a result, the median birth weight was 2270 g (interquartile range 344), birth GA was 38 + 1/7 weeks (interquartile range 1.8 weeks), 42% were male, 70% were black, and 46% were born via cesarean delivery. There were no statistical differences between those who were and were not tested.

We found an overall 4.8% failure rate (*n* = 9). Seven of the 9 subjects who failed their CSTS spent their entire admission in the FTN, and the remaining 2 spent some time in the NICU. One of the subjects who failed did so based on desaturations at an outside facility and was subsequently transferred to our NICU for care. A repeat test was performed in our institution, and the subject failed. This subject had complete clinical and demographic data available and was included in the final analysis.

All infants with LBW undergo CSTS at our institution regardless of comorbidities or specific diagnoses. There were numerous comorbidities known at the time of CSTS in those who passed their initial CSTS, including trisomy 18, trisomy 21, fetal alcohol syndrome, neonatal abstinence syndrome, Prader–Willi syndrome (PWS), gastroschisis, ventriculomegaly, Dandy–Walker variant, DiGeorge, tracheoesophageal fistula or esophageal atresia repair, and numerous cardiac diagnoses such as tetralogy of Fallot, dysplastic tricuspid valve, and ventricular septal defects. A majority did not have any known specific diagnoses at the time of hospital discharge and CSTS.

On bivariate analysis, there were no significant differences between those who passed and those who failed with regard to gender, race, delivery mode, singleton versus multiple gestation, maternal anesthesia, maternal morphine or nonopiate analgesic use, maternal magnesium treatment, group B *Streptococcus* status, birth weight, birth GA, Apgar score, positive pressure ventilation use, respiratory support, antenatal steroids, surfactant, caffeine, postnatal steroids, or diuretics (Table 1 and Table 2). There were no differences in characteristics at the time of testing, including chronologic age, corrected GA, or weight at the time of CSTS testing. There were no differences in location of admission or whether subjects spent any time in the NICU during their hospitalization. There were no differences in maternal or neonatal urinary toxicology results for marijuana or cocaine.

The only factor found to be statistically different was maternal toxicology results for opiates. Of those who failed, 22.2% (n = 2 of 9) had mothers who tested positive for opiates, compared with 5.6% (n = 10 of 178) of those who passed, P = .0473. A urine sample for all mothers was sent for toxicology at the time of admission before administration of medications during labor, with the exception of 1 mother. No urine sample could be sent for this mother before her stat cesarean delivery, in which she did receive intravenous opiates. Her urine sample was sent after her cesarean delivery and was positive for opiates. It is unclear whether this mother had exposure to opiates before admission, but this baby's urine toxicology was negative, and this baby passed CSTS. Because the mother did have positive urine toxicology for opiates, the pair was included in the bivariate analysis in Table 1. We reran analysis without this mother–infant pair (assuming no prenatal exposure to opiates other than during the cesarean delivery),

TABLE 1 Unadjusted Demographic and Clinical Predictors of CSTS Failure

	CSTS Pass (n = 178), % (n)	CSTS Fail (n = 9), % (n)	P
Gender			.1718
Male	49% (88)	22% (2)	
Female	51% (90)	78% (7)	
Race			.9909
White	8.5% (15)	11.1% (1)	
Black	86.4% (153)	88.9% (8)	
Other	5.1% (9)	0% (0)	
Delivery mode			.9815
Vaginal	66.3% (118)	66.7% (6)	
Cesarean	33.7% (60)	33.3% (3)	
Gestation number			.1472
Singleton	85.4% (152)	66.7% (6)	
Multiples	14.6% (26)	33.3% (3)	
GBS status			.7298
Positive	37.6% (67)	33.3% (3)	
Negative	43.3% (77)	55.6% (5)	
Unknown	19.1% (34)	11.1% (1)	
Maternal anesthesia			.6442
General endotracheal	3.4% (6)	0% (0)	
Spinal or epidural	72% (126)	62.5% (5)	
None	24.6% (43)	37.5% (3)	
Maternal analgesia			
Morphine	2.8% (5)	0% (0)	.8007
Synthetic opioid (Stadol)	27.3% (48)	22.2% (2)	.2954
Magnesium exposure	15.3% (27)	11.1% (1)	.3734
PPV required	14.6% (26)	0% (0)	.6150
Admission location			.2757
FTN	66.9% (119)	88.9% (8)	
NICU	33.1% (59)	11.1% (1)	
Spent time in NICU	42.1% (75)	22.2% (2)	.3114
Medications			
Antenatal steroids	1.7% (3)	0% (0)	.8617
Surfactant	1.1% (2)	0% (0)	.9058
Caffeine	0.6% (1)	0% (0)	.9519
Postnatal steroids	0% (0)	0% (0)	NS
Furosemide	1.1% (2)	0% (0)	.9058
Respiratory support needed			
Ventilator	3.4% (6)	0% (0)	.7408
Continuous positive airway pressure	3.9% (7)	0% (0)	.7039
Nasal cannula	4.5% (8)	11.1% (1)	.3649
Maternal urine toxicology			
Opiates	5.6% (10)	22.2% (2)	.0473
Marijuana	10% (18)	0% (0)	.6038
Cocaine	2.3% (4)	11.1% (1)	.2207
Neonate urine toxicology			
Opiates	3.9% (7)	11.1% (1)	.2991
Marijuana	5.1% (9)	0% (0)	.6351
Cocaine	2.3% (4)	0% (0)	.8196
Intraventricular hemorrhage	2.3% (4)	0% (0)	.8196
Gastrostomy tube	1.1% (2)	11.1% (1)	.1318

GBS, group B *Streptococcus*; NS, not significant; PPV, positive pressure ventilation.

which made the association between positive maternal toxicology and CSTS failure stronger (P = .0335).

Nine infants failed the CSTS, 7 due to desaturations and 2 due to bradycardia. Subject 1 was a singleton who had no prenatal care, had opiate and cocaine in utero exposure (maternal and neonatal positive urine toxicology), was on low-dose enteral morphine for neonatal abstinence syndrome at the time of failure, and was noted to have desaturations to 80% and "small" emesis at the time of failure. It is unclear whether the infant had

TABLE 2 Unadjusted Weight, Age, and Apgar Score Predictors of CSTS Failure

	CSTS Pass (n = 178), Median (IQR)	CSTS Fail (n = 9), Median (IQR)	P
Birth weight, g	2348 (245)	2320 (210)	.8276
Birth gestational age, wk	38 (1.6)	38 + 3/7 (1.3)	.5419
Apgar scores			
1 min	8 (1)	8 (1)	.9696
5 min	9 (0)	9 (0)	.1969
Weight at test, g	2300 (220)	2285 (230)	.4052
Corrected GA at test, wk	38 + 6/7 (1.7)	38 + 4/7 (1.3)	.7852
Age at test, d	2 (4)	2 (1)	.3205

IQR, interquartile range.

emesis and then desaturated or desaturated and then had emesis. The infant was weaned off morphine the next day, with low neonatal abstinence syndrome scores. CSTS was repeated 3 days later, when the infant was off morphine for 48 hours and passed, with notation of a blanket roll on either side of the subject.

Subject 2 was a singleton with negative maternal and neonatal urine toxicology who failed from the FTN twice, continued to desaturate afterward, and needed nasal cannula oxygen. The infant was subsequently noted to have poor feeding and hypotonia and eventually received a diagnosis of PWS and needed a gastrostomy tube. The infant passed the CSTS after 1 month and went home in a car seat.

Subject 3 was a singleton who had in utero opiate exposure (positive maternal and neonatal urine toxicology), was noted to have bradycardia <80 beats per minute during CSTS, and continued to have bradycardia in the 80 seconds after removal from the car seat. Electrocardiography showed a prolonged QTc interval, and follow-up was scheduled with cardiology. This subject was not on QTc-prolonging medications. The infant passed CSTS 24 hours later.

Subject 4 was a singleton with negative maternal and neonatal urine toxicology who had desaturations to 70% for >10 seconds, followed by 60%, which necessitated supplemental oxygen and removal from the car seat. The infant continued to have desaturations to 80% and tachypnea for 7 hours after removal from car seat. Radiographic workup was negative, tachypnea resolved after 7 hours, and a repeat test 24 hours later was passed.

Subject 5 was a twin with negative maternal and neonatal toxicology who had desaturations <88% after 1 hour, repeated the test within 24 hours, and passed.

Subject 6 was a twin with negative maternal and neonatal toxicology who had desaturations <88% without additional notation, repeated the test within 24 hours, and passed.

Subject 7 was a singleton with negative maternal and neonatal toxicology who had bradycardia <80 beats per minute after 15 minutes, repeated the test within 24 hours with adjustments in the shoulder harness and groin straps, and passed.

Subject 8 was a singleton with negative maternal and neonatal toxicology who had saturations near 80% for 1 minute after 45 minutes, repeated the test within 24 hours, and passed.

Subject 9 was a twin with negative maternal and neonatal toxicology who had saturations <88% almost 90 minutes into the test, repeated the test 24 hours later, and passed.

DISCUSSION

Although there have been very few studies on the CSTS in LBW term neonates, up to half of NICUs include this group in their CSTS protocols.[4] It was unclear whether this group should be included in testing because almost no information is available on failure rates or predictors of failure. The aim of this study was to determine the incidence and risk factors for failure of the CSTS in term LBW infants to help guide CSTS protocols and inclusion criteria. We were able to analyze data on 87.8% of eligible LBW term infants born over a 4-year period at our institution. We found an overall failure rate of 4.8%, which is similar to that in published reports on the failure rates in a large cohort of preterm infants.[10] We found that the only significant difference in clinical and demographic characteristics between those who passed and those who failed was maternal urine toxicology positive for opiates.

Previous studies have attempted to identify risk factors for failure of the CSTS to narrow the inclusion criteria for testing and decrease unnecessary resource use. Most of these studies have been of preterm infants and their associated risk factors. In the preterm population, infants who needed respiratory support and those who needed treatment with caffeine for apnea of prematurity were less likely to fail.[10] Because full-term infants are much less likely to need these interventions, it is unclear how these factors would play a role. We assessed important risk factors known to affect neonatal outcomes, focusing on variables from previous studies that were shown to be significant predictors of CSTS failure. A previous study found that a majority of infants who failed CSTS did so while admitted to the FTN, with a lower percentage failing from the NICU.[10] We also found that 78% of CSTS failures occurred in infants admitted to the FTN. One possibility is that the infants in the NICU were on continuous cardiorespiratory monitoring, so vital sign abnormalities before CSTS were easier to detect than in those unmonitored in the nursery. Mode of delivery has been shown to significantly differ in a cohort of preterm infants,[9] although we did not

find this difference in our cohort. Although this finding has not been consistently demonstrated, predischarge weight <2 kg has been linked to increased risk of failure, and those with lower weights at the time of testing have been shown to be at higher risk of poor repeatability of CSTS result, leading to the assessment of birth weight and weight at the time of testing.[8,9] Higher birth GA in preterm infants was associated with higher risk of failure, which may be related to lower chronologic ages at the time of testing.[10] However, neither weight nor GA (at birth or at time of CSTS) were significantly different in our study between those who passed and those who failed.

We did find that maternal urine toxicology positive for opiates was significantly higher in infants who failed than in those passed. Of the infants who failed, 22.2% had mothers who tested positive for opiates (2 of 9), compared with 5.6% of those who passed (10 of 178), $P = .0473$. Effects of maternal medication and drug exposure on CSTS results have not previously been assessed in the literature, but opiate exposure is known to affect respiratory status. Interestingly, neonatal urinary toxicology result was not a predictor. This finding may be related to the timing of obtaining urine samples. Urine from all neonates in our cohort was sent for toxicology, with the goal of obtaining the earliest sample possible. However, we often are unable to get a urinary bag onto the neonate to catch an early urine sample. A later urine sample may not be highly sensitive to detect opiates in the urine despite exposure. Meconium toxicology may be more reliable, but we do not perform this test at our institution.

The association between maternal opiate use and increased odds of CSTS failure in our data are concerning. There is evidence that prenatal cocaine exposure may affect maturation of cardiorespiratory control in term infants, with evidence of increased bradycardia and increased duration of apneic events in these infants.[11,12] However, we did not find an association between urine toxicology positive for cocaine and an increased risk of CSTS failure in our cohort. Prenatal opiate exposure did not seem to have as significant an effect on postnatal apnea and bradycardia when assessed in studies of exposed neonates.[12] However, the prevalence of sudden infant death syndrome (SIDS) is known to be higher in drug-exposed infants, with the highest rates in those exposed to opiates even when we control for prematurity, LBW, and perinatal infections such as HIV.[13,14] There is no evidence linking SIDS and CSTS failure, but the link between opiate exposure and SIDS does raise concern that opiate exposure may have adverse effects on cardiorespiratory status in neonates.

Limitations to this study include the fact that the data are retrospective, so we can evaluate associations but not causality. In addition, complete information was not available on all eligible subjects, although we did have a significant majority (87.8%), and basic clinical and demographic information were similar between those with documented CSTS and those without, making bias less likely in our cohort. In addition, our saturation failure criterion (<88%) is conservative, based on a previous study of failure criteria in hospitals performing CSTS.[4] The most common failure saturation was <90%, with many units using higher saturation limits.[4,10,15–17] Using a higher saturation cutoff would probably lead to a greater CSTS failure rates, and therefore we may be identifying infants with more severe desaturation events. Our conclusions therefore are most generalizable to infants with more severe desaturation events.

The biggest challenge in any study of the CSTS is understanding the meaning of a failed test. Because this test requires placement in the car seat and proper fit assessment, human error can play a role in CSTS failure. Thorough training of staff on proper fit in the car seat is of the utmost importance. The goal of the CSTS is to ensure safety and stability for discharge as outlined by the AAP Committee on the Fetus and Newborn.[3] Although the utility of the CSTS to identify infants at risk for adverse cardiopulmonary events is unclear, a failed CSTS does seem to demonstrate that at the time of testing, the infant had cardiorespiratory instability and was not ready for discharge. In our small cohort of subjects, failing the CSTS prompted additional evaluation of each infant, leading to the identification of 2 infants with significant medical problems that may have been overlooked before discharge if the infant had not failed the CSTS. One had PWS, and 1 had long QT syndrome, and both needed subsequent follow-up. Although the remaining 7 did not have a definite diagnosis explaining their concerning vital sign changes, 6 of the infants were noted to have hypoxic events in the car seat that may not have been noted without the CSTS. One subject was noted to need strap adjustments, and 1 needed blanket rolls to pass, which may have continued to cause problems if vital sign changes had not been noticed on the CSTS.

A large systematic review of literature related to chronic and intermittent hypoxia in children demonstrated an adverse impact on development, cognitive performance, and behavior that should be taken into account in situations where children may be exposed to hypoxia.[18] Another recent study demonstrated that infants have reduced cerebral tissue oxygenation when in the prone position and hypothesized that this reduction in oxygenation may help explain the elevated risk of SIDS while the infant is in this position.[19] Tissue hypoxia clearly leads to increased risk of

morbidity in neonates, which demonstrates the importance of identifying infants at risk for hypoxia before discharge to intervene early. The CSTS is 1 such test, with the goal of identifying at-risk infants. Another test aiming to identify infants at risk for hypoxia is the critical congenital heart disease screen, now widely implemented for newborns. Additional studies should be performed to compare results of the critical congenital heart disease and CSTS tests to identify which infants have predisposing conditions unrelated to car seat positioning and to assess the effect of car seat positioning on desaturation events.

Given that this is a retrospective study, we cannot assess which interventions were performed on the remaining infants who failed because there was no additional documentation. Nor do we have access to the outcomes of these 9 subjects to indicate their risk for future cardiopulmonary events while they are in the car seat. But this study at least confirms the importance of proper assessment of car seat fit in all neonates. The newest guideline was jointly released by the AAP, the National Highway Transportation Safety Administration, the Children's Hospital Association, and the National Safety Council in 2014 and can be used as a resource to help hospitals develop programs to ensure that all children are transported safely.[20]

CONCLUSIONS

The objective of this study was to provide background epidemiologic data on LBW full-term infants undergoing CSTS. Because very little is published on the topic, it is difficult to make evidence-based recommendations related to testing this group of patients. More evidence is needed to support routine testing of all LBW infants, narrowed testing to a specific demographic group, or completely eliminating this test. Right now, it is unclear which of these options is best for our patients. What we noted in this study was that LBW term infants have similar CSTS failure rates as preterms, with a significant association noted with in utero opiate exposure. Performing CSTS on this population remains controversial, although we now have basic epidemiologic data from which to perform future prospective studies to identify the utility of CSTS for LBW term neonates.

ACKNOWLEDGMENTS

The author thanks Kelly Tracey for her assistance in data collection and Jennifer Thompson for her constant support. Thank you to Dr Brian Corwell for his support and assistance in the review of this article. Thank you to Dr Cynthia Bearer and the Division of Neonatology.

ABBREVIATIONS

AAP: American Academy of Pediatrics
CSTS: car seat tolerance screening
FTN: full-term nursery
GA: gestational age
LBW: low birth weight
PWS: Prader Willi syndrome
SIDS: sudden infant death syndrome

REFERENCES

1. American Academy of Pediatrics Committee on Injury and Poison Prevention and Committee on Fetus and Newborn. Safe transportation of premature infants. *Pediatrics*. 1991;87(1):120–122
2. American Academy of Pediatrics. Committee on Injury and Poison Prevention and Committee on Fetus and Newborn. Safe transportation of premature and low birth weight infants. *Pediatrics*. 1996;97(5):758–760
3. Bull MJ, Engle WA; Committee on Injury, Violence, and Poison Prevention and Committee on Fetus and Newborn, American Academy of Pediatrics. Safe transportation of preterm and low birth weight infants at hospital discharge. *Pediatrics*. 2009;123(5):1424–1429
4. Davis NL, Zenchenko Y, Lever A, Rhein L. Car seat safety for preterm neonates: Implementation and testing parameters of the infant car seat challenge. *Acad Pediatr*. 2013;13(3):272–277
5. Bull MJ, Stroup KB. Premature infants in car seats. *Pediatrics*. 1985;75(2): 336–339
6. Willett LD, Leuschen MP, Nelson LS, Nelson RM Jr. Risk of hypoventilation in premature infants in car seats. *J Pediatr*. 1986;109(2):245–248
7. Willett LD, Leuschen MP, Nelson LS, Nelson RM Jr. Ventilatory changes in convalescent infants positioned in car seats. *J Pediatr*. 1989;115(3): 451–455
8. Ojadi VC, Petrova A, Mehta R, Hegyi T. Risk of cardio-respiratory abnormalities in preterm infants placed in car seats: a cross-sectional study. *BMC Pediatr*. 2005;5:28
9. Davis NL, Gregory ML, Rhein L. Test–retest reliability of the infant car-seat challenge. *J Perinatol*. 2014;34(1): 54–58
10. Davis NL, Condon F, Rhein LM. Epidemiology and predictors of failure of the infant car seat challenge. *Pediatrics*. 2013;131(5):951–957
11. Silvestri JM, Long JM, Weese-Mayer DE, Barkov GA. Effect of prenatal cocaine on respiration, heart rate, and sudden infant death syndrome. *Pediatr Pulmonol*. 1991;11(4):328–334
12. Chasnoff IJ, Hunt CE, Kletter R, Kaplan D. Prenatal cocaine exposure is associated with respiratory pattern abnormalities. *Am J Dis Child*. 1989; 143(5):583–587
13. Kandall SR, Gaines J, Habel L, Davidson G, Jessop D. Relationship of maternal substance abuse to subsequent sudden infant death syndrome in offspring. *J Pediatr*. 1993;123(1): 120–126
14. Kahlert C, Rudin C, Kind C; Swiss HIV Cohort Study (SHCS); Swiss Mother & Child HIV Cohort Study (MoCHiV). Sudden infant death syndrome in infants born to HIV-infected and opiate-using mothers. *Arch Dis Child*. 2007;92(11):1005–1008
15. Bass JL, Mehta KA, Camara J. Monitoring premature infants in car seats: implementing the American Academy of Pediatrics policy in a community

hospital. *Pediatrics*. 1993;91(6):1137–1141

16. Degrazia M, Guo CY, Wilkinson AA, Rhein L. Weight and age as predictors for passing the infant car seat challenge. *Pediatrics*. 2010;125(3):526–531
17. Bass JL. The infant car seat challenge: determining and managing an "abnormal" result. *Pediatrics*. 2010; 125(3):597–598
18. Bass JL, Corwin M, Gozal D, et al. The effect of chronic or intermittent hypoxia on cognition in childhood: a review of the evidence. *Pediatrics*. 2004;114(3): 805–816
19. Fyfe KL, Yiallourou SR, Wong FY, Odoi A, Walker AM, Horne RS. Cerebral oxygenation in preterm infants. *Pediatrics*. 2014;134(3): 435–445
20. American Academy of Pediatrics, National Highway Transportation Safety Administration (NHTSA), Children's Hospital Association and the National Safety Council. Hospital discharge recommendations for safe transportation of children. March 2014. Available at: http://cpsboard.org/cps/wp-content/uploads/2014/04/FINAL_dischargeprotocol_2014_logos.pdf

SPARING THE TILL: *While the temperature was only 8 degrees Fahrenheit when I left the house this morning, spring has technically arrived in northern Vermont. We had several days of warmer weather last week and a few of the local farmers had already begun plowing. Most of the farmers in the area plow at least two times a year: once in the spring and once in the fall.*

However, according to an article in The New York Times *(Science: March 9, 2015), frequent plowing may not be very good for the soil. Soil conservation farming, which promotes leaving fields untilled and use of cover crops as manure, has been gaining converts nationwide. The theory is that, while tilling can mix decaying organic material such as dead weeds into the soil, repeated tilling degrades the soil. This repeated tilling kills beneficial earthworms and fungi, and increases reliance on applied fertilizer. The tilled soil no longer traps water easily, and the top layers can be washed or blown away in rainstorms or windstorms. The loss of topsoil further degrades the land and the runoff pollutes ponds and streams. Soil conservation farmers use cover crops all year to help the soil hold water and replace nitrogen without use of synthetic fertilizers.*

A recent study suggested that the widespread use of soil conservation farming could decrease nitrogen pollution in the Upper Mississippi and Ohio River basins by 30 percent. However, not all farmers are enthusiastic about no-till farming. The technique seems to work better for some crops than others. For example, not as many farmers planting corn use the method. Also, special equipment, such as no-till seeders that drill through residue, need to be purchased. Despite these concerns, approximately 35 percent of cropland in the United States is now cultivated using no-tillage techniques.

I am not sure what percentage of cropland in Vermont is cultivated using no-till methods, but I would expect that given our concern over runoff and waterway pollution, it will soon increase.

Noted by WVR, MD

Car Seat Screening for Low Birth Weight Term Neonates

Natalie L. Davis

Pediatrics 2015;136;89
DOI: 10.1542/peds.2014-3527 originally published online June 15, 2015;

PEDIATRICS®
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Car Seat Screening for Low Birth Weight Term Neonates

Natalie L. Davis

Pediatrics 2015;136;89
DOI: 10.1542/peds.2014-3527 originally published online June 15, 2015;