Published in final edited form as:

Infancy. 2008 November 1; 13(6): 570–595. doi:10.1080/15250000802458682.

Contingency Learning and Reactivity in Preterm and Full-Term Infants at 3 Months

David W. Haley,

Department of Psychology, University of Toronto

Ruth E. Grunau,

Centre for Community Child Health Research, Child and Family Research Institute, Department of Pediatrics, University of British Columbia

Tim F. Oberlander, and

Centre for Community Child Health Research, Child and Family Research Institute, Department of Pediatrics, University of British Columbia

Joanne Weinberg

Department of Cellular and Physiological Sciences, University of British Columbia

Abstract

Learning difficulties in preterm infants are thought to reflect impairment in arousal regulation. We examined relationships among gestational age, learning speed, and behavioral and physiological reactivity in 55 preterm and 49 full-term infants during baseline, contingency, and nonreinforcement phases of a conjugate mobile paradigm at 3 months corrected age. For all infants, negative affect, looking duration, and heart rate levels increased during contingency and nonreinforcement phases, whereas respiratory sinus arrhythmia (RSA, an index of parasympathetic activity) decreased and cortisol did not change. Learners showed greater RSA suppression and less negative affect than nonlearners. This pattern was particularly evident in the preterm group. Overall, preterm infants showed less learning, spent less time looking at the mobile, and had lower cortisol levels than full-term infants. Preterm infants also showed greater heart rate responses to contingency and dampened heart rate responses to nonreinforcement compared to full-term infants. Findings underscore differences in basal and reactivity measures in preterm compared to full-term infants and suggest that the capacity to regulate parasympathetic activity during a challenge enhances learning in preterm infants.

More than 60 years ago, Shirley (1938, 1939) reported on a behavioral syndrome found in prematurely born children that included difficulties in motor function, sensory input, speech, social interactions, attention, learning, arousal, and emotion. Since that time, longitudinal studies of infants born prematurely with age-matched, full-term controls have demonstrated a greater incidence of behavioral, cognitive, and learning problems in infants born prematurely that persist into childhood (e.g., Anderson, Doyle, & Victorian Infant Collaborative Study Group, 2003; Grunau, Whitfield, & Davis, 2002; Pharoah, Stevenson, & West, 2003; Rose & Feldman, 1996; Taylor, Klein, Minich, & Hack, 2000) and late

Copyright © Taylor & Francis Group, LLC

Correspondence should be sent to Ruth E. Grunau, Centre for Community Child Health Research, F-6 4480 Oak Street, Vancouver, BC V6H 3V4 Canada. rgrunau@cw.bc.ca.

Copyright of *Infancy* is the property of Lawrence Erlbaum Associates and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.

adolescence (e.g., Grunau, Whitfield, & Fay, 2004; Hack et al., 2002; Saigal, Hoult, Streiner, Stoskopf, & Rosenbaum, 2000). Specifically, preterm infants are slower in the processing of novel (e.g., Gardner & Karmel, 1983; Landry, Leslie, Fletcher, & Francis, 1985; Millar & Weir, 1995; Rose, Feldman, Jankowski, & Caro, 2002; Rose, Feldman, McCarten, & Wolfson, 1988) and contingent stimulation (e.g., Gekoski, Fagen, & Pearlman, 1984; Millar, Weir, & Supramaniam, 1992; Ramey, Heiger, & Klisz, 1972), which may be due, in part, to differences in brain function (e.g., Herbert, Eckerman, Goldstein, & Stanton, 2004) or difficulties in arousal regulation (e.g., Mayes, 2000; Millar & Weir, 2007).

It has been proposed that the preterm infant's impaired capacity for learning and information processing reflects impairments in arousal regulation (Field, 1981; Mayes, 2000; Tronick, Scanlon, & Scanlon, 1990). From this perspective, preterm infants are thought to be either under- or overaroused by stimulation, which limits their capacity to encode and process information. In preterm infants, ability to maintain an optimal arousal state (i.e., being attentive and physiologically aroused but not emotionally upset) may be impaired, overriding the capacity to encode and process novel stimuli. Optimal arousal states, generally, reflect the activation and coordination of multiple neurobiological response systems that enhance the capacity to learn.

Evidence that altered arousal regulation is linked to poorer cognitive performance in preterm infants is found in neonatal studies that have evaluated learning and reactivity concurrently (e.g., Field, Dempsey, Hatch, Ting, & Clifton, 1979; Krafchuk, Tronick, & Clifton, 1983; Lester, Boukydis, & LaGasse, 1996; Rose, Schmidt, & Bridger, 1976). In these studies, preterm neonates showed hypo-responsiveness to low-frequency stimulation (e.g., Gardner & Karmel, 1983; Rose et al., 1976) and hyperresponsiveness to high-frequency stimulation (Krafchuk et al., 1983; Rose et al., 1976) compared to full-term neonates. In both cases, impaired learning (i.e., slower habituation) was evident in preterm infants and was attributed to a lower sensory threshold (e.g., Rose et al., 1976) or a failure of inhibition (Krafchuk et al., 1983; Tronick et al., 1990).

Past the neonatal period, however, few studies have examined learning and reactivity concurrently. Support for the altered arousal regulation characterization of the preterm infant just described has been mixed. Manipulations of arousal level during visual attention tasks (e.g., Gardner, Karmel, & Magnano, 1992) have revealed that increased arousal interferes with visual functioning in preterm infants to a greater degree than it does in full-term infants. With less complex stimuli, such as the presentation of a relatively simple visual stimulus (e.g., toy), preterm infants show similar reactivity patterns compared to full-term controls (Coles, Bard, Platzman, & Lynch, 1999; DiPietro, Porges, & Uhly, 1992), whereas with more complex stimuli, such as social contingency, preterm infants show heightened activation across behavioral and physiological response systems. For example, 3-month-old preterm infants show greater elevations in heart rate (e.g., Coles et al., 1999; Field, 1979), more looking-away behavior (Field, 1979), and greater levels of negative affect (e.g., Brazelton, Koslowski, & Main, 1974) than full-term infants in response to social contingency. In response to contingency learning (nonsocial), altered cardiac responses were observed in the at-risk infants (Millar & Weir, 2007). Whether reactivity differences in preterm and full-term infants' cardiac reactivity, looking behavior, and affect responses are related to learning differences remains an unexamined issue. Evaluation of learning and reactivity in preterm and full-term infants will help elucidate the extent to which impaired arousal regulation may contribute to learning difficulties in general and, in particular, to infants born at risk.

CONTINGENCY LEARNING

The infant's capacity to detect contingencies plays a crucial role in cognitive, social, and emotional development (Watson, 1966). Contingency learning facilitates the development of intentional behavior and more complex forms of social-emotional communication, and involves the dual capacity to maintain an organized state while responding to and interacting with the environment (Tarabulsky, Tessier, & Kappas, 1996). In Rovee-Collier's mobile conjugate reinforcement paradigm (Haley, Weinberg, & Grunau, 2006; Hartshorn & Rovee-Collier, 2003; Rovee & Rovee, 1969; Rovee-Collier, 1997; Rovee-Collier, Sullivan, Enright, Lucas, & Fagen, 1980), infants rapidly associate a motor response with a contingent stimulus (i.e., foot kicking that moves a mobile). During the learning phase, infants show interest and positive facial expressions (Alessandri, Sullivan, & Lewis, 1990; Shapiro, Fagen, Prigot, Carroll, & Shalan, 1998; Sullivan & Lewis, 2003). When the contingency is removed, which is thought to be a source of frustration (Amsel, 1958), infants show greater negative emotion and look away more (Shapiro et al., 1998; Sullivan & Lewis, 2003). Preterm infants have greater difficulty learning to respond to and interact with contingent stimulation, and they require multiple sessions to learn this task (Gekoski et al., 1984). For example, Gekoski et al. found that it was not until the second day of training that preterm infants performed as well as full-term infants. Whether learning difficulties in preterm infants reflect a restricted range in reactivity remains an unexamined issue.

Prior work relating to contingency learning has examined the effects of (a) perinatal risk factors, (b) high-risk populations, and (c) emotion on contingency learning. Greater perinatal risk factors have been linked to contingency learning (Gekoski et al., 1984; Millar et al., 1992) in infants. Within high-risk populations, infants classified as having failure-to-thrive syndrome required two to three 10-min sessions to demonstrate significant increases in contingency learning, whereas controls were able to learn on the first exposure (Ramey et al., 1972). In one of the few studies to examine contingency learning in preterm infants, Gekoski et al. (1984) found that healthy preterm infants needed a second training session before they showed significant increases in responding to contingency compared to full-term infants, who were able to demonstrate learning in the first session. Infants who learn to respond to contingency show increases in facial expressions of interest, joy, and surprise, as well as shifts in attention from baseline to learning (Lewis, Alessandri, & Sullivan, 1990). Greater expressions of negative emotion (e.g., crying) are related to nonlearners' rates of contingency learning (Fagen & Ohr, 1985; Fagen, Ohr, Singer, & Fleckenstein, 1987).

PHYSIOLOGICAL REACTIVITY

Given that attention and emotion are known to play an important role in contingency learning, we expected that the physiological substrates of attention and emotion would also play an important role in contingency learning. The autonomic nervous system supports behaviors involved in maintaining attention and responding to novel stimuli as well as responding rapidly to threats. Multiple nuclei in the brain stem associated with autonomic functions receive projections from the hypothalamus and higher cortical centers. In particular, the nucleus ambiguus (NA) in the brain stem provides parasympathetic projections to the cardiovascular system via the vagus nerve, which inhibits the activation of the heart. Projections from the NA have been proposed to play a critical role in the expression of emotion and the ability to attend to and process information (Porges, 1995). Parasympathetic activation of the heart attributed to the NA can be indexed by respiratory sinus arrhythmia (RSA).

In Porges's (1995) polyvagal theory of emotion, greater parasympathetic activity at rest supports restorative processes, whereas reductions in parasympathetic activity and increases

in heart rate prepare the body for cognitive and social-emotional demands. Reductions in RSA (an index of parasympathetic activity and vagal tone) have been related to better regulation of attention (e.g., Bazhenova, Plonskaia, & Porges, 2001), habituation to novelty (e.g., Bornstein & Suess, 2000), and better developmental outcomes in normal (e.g., Fox & Porges, 1985) and preterm infants (Doussard-Roosevelt, McClenny, & Porges, 2001; Doussard-Roosevelt, Porges, Scanlon, Alemi, & Scanlon, 1997). Suppression of RSA has also been related to greater expression of negative emotion during an emotional challenge (Bazhenova et al., 2001). Given that RSA does play an adaptive role in cognition and emotion, we would expect suppression of RSA during contingency learning to facilitate learning and the expression of negative affect in response to nonreinforcement.

The limbic–hypothalamic–pituitary–adrenocortical (L–HPA) axis is activated more slowly than the autonomic system. It consists of a cascade of hormone responses: Corticotrophin-releasing hormone (CRH), released from the hypothalamus, stimulates the release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary, which in turn stimulates the release of glucocorticoid hormones (primarily cortisol) from the adrenal cortex. Cortisol, in turn, feeds back to regulate hormone release at every level of the axis. Importantly, CRH, which is widely distributed in the central nervous system (CNS), also plays a role not only in stimulating ACTH release but in the activation and coordination of autonomic, metabolic, emotional, and behavioral responses to stress, and thus may modulate social-emotional behavior and cognitive processes (Stansbury & Gunnar, 1994).

Increases in cortisol levels have been related to negative affect during physical challenges (e.g., Gunnar, Broderson, Krueger, & Rigatuso, 1996; Lewis & Ramsay, 1995). Studies of preschool children, however, support the idea that cortisol secretion may serve both adaptive and maladaptive roles, depending on the context and temperament of the child. For example, Gunnar, Tout, de Haan, Pierce, and Stansbury (1997) suggested that temporary elevations in cortisol help bold children meet the challenges of a new school year, whereas continued elevations in cortisol during the year are associated with shy, anxious children. Further examination of the relationships between cortisol and emotion in a learning context is needed to better understand the potentially different roles that cortisol may play in infants who learn rapidly compared to those who are slower to learn new associations.

THIS STUDY

In this study, we examined contingency learning in relation to behavioral and physiological reactivity in preterm and full-term infants at 3 months (age corrected for prematurity). The aim of this study was to evaluate contingency learning in relation to physiological (autonomic and L–HPA) and behavioral reactivity (emotion and looking) in preterm and full-term infants. To examine reactivity (i.e., changes in arousal state), we evaluated look duration, affect, and two physiological systems: the autonomic nervous system and the (L–HPA) axis. Based on the view that preterm infants have altered arousal regulation, we hypothesized that preterm infants would show lower rates of contingency learning and less organized arousal states (i.e., greater expression of negative affect, shorter looking duration, less suppression of RSA, greater increases in heart rate, and greater activation of the L–HPA axis) compared to full-term infants. This hypothesis is consistent with prior work demonstrating that preterm infants have arousal regulation impairments that accompany learning difficulties (e.g., Field, 1979; Mayes, 2000; Mayes & Bornstein, 1997; Rose et al., 1976; Tronick et al., 1990). This hypothesis has not previously been tested beyond the neonatal period.

In most studies of contingency learning, infants who fail to meet the learning criterion are excluded from the study. Because of our interest in the relationship between arousal

regulation and learning, we examined differences in reactivity between learners and nonlearners (i.e., those who reach the learning criterion vs. those who do not). Based on the fundamental assumption that optimal states of arousal facilitate learning, we hypothesized that learners would show greater evidence of optimal arousal states compared to nonlearners, regardless of preterm status. In addition, we examined whether coordination of physiological and emotional reactivity indexes differed among preterm and full-term learners and nonlearners to determine whether such indexes are related to arousal regulation impairment. Recent work indicates that changes in affect and autonomic activity between phases are sensitive to differences in high-risk infants (e.g., Millar & Weir, 2007). Accordingly, we examined whether changes in affect and heart rate measures between conditions (e.g., reinforcement minus baseline or nonreinforcement minus baseline) were related to each other and differed in preterm and full-term infants.

METHODS

Participants were 61 premature infants born at 32 weeks gestational age or earlier and 55 full-term infants seen at 3 months of age corrected for chronological age (CCA); 55 premature and 49 full-term infants completed testing. We tested each preterm infant at his or her corrected age, or age adjusted for weeks of prematurity (Wilson & Cradock, 2004), which is the research and clinical convention. Preterm infants were recruited through the Neonatal Intensive Care Unit (NICU) at Children's and Women's Health Centre of British Columbia to participate in a larger longitudinal program of studies examining the effects of prematurity and early pain-related stress exposure on physiological and cognitive functioning. Neonatal characteristics are presented in Table 1. Full-term infants were recruited through their pediatricians at two major metropolitan nurseries at the Children's and Women's Health Centre of British Columbia and St. Paul's Hospital, both affiliated with the University of British Columbia. Infants with a major congenital anomaly, neurosensory impairment, or maternal drug use during pregnancy were excluded from the study. Mothers were primarily White (80%), married (80%), and college educated (education: $M = 15.9$ years). Mothers were on average 33 years of age, with 1.7 children (range = 1–4).

Procedures

Each infant was tested at home at a time selected by the mother when the infant was likely to be alert and playful. Two silver chloride (AgCl) gel electrodes used for collecting heart rate data were placed on the infant's chest prior to testing (see "Cardiac Measures" section later). The infant was tested using a conjugate reinforcement mobile task using the procedures of Rovee-Collier's laboratory (Rovee & Rovee, 1969; Rovee-Collier et al., 1980). A ribbon was tied to the infant's ankle. The infant was then placed supine in a crib lined with red vertical stripes on a blue background and exposed to a mobile for 3 min (baseline period). Following this baseline period, the free end of the ribbon was attached to the hook supporting the mobile. The infant's foot kicks thus caused the mobile to move (the learning phase). Following the learning phase, which lasted for 9 min, the ribbon was detached from the hook, so that the infant's kicking was no longer reinforced (non-reinforcement phase); this phase lasted for 3 min. At the end of the nonreinforcement phase, the mobile was removed from the infant's view by the research assistant, and the mother removed the infant from the crib.

During this procedure, the research assistants and caregiver stayed out of the infant's view to avoid distracting the infant. However, when the infant became fussy or upset, following the procedures of Rovee-Collier, the research assistant would attempt to soothe the infant by looking over the crib's side, smiling at the infant, and speaking in soothing and reassuring

tones. If these attempts were insufficient, the mother removed the infant from the crib, the experiment was stopped, and the infant's data were excluded from data analyses.

A video camera was positioned on a tripod to the left side of the crib, so that all of the infant's body and the mobile were in the field of view. The video image captured the orientation of the infant's head and eyes toward the mobile as well as his or her facial expressions and leg movements. Video signals from the camera were recorded on an analog VHS tape that was later used for coding. Physiological and video signals were synchronized, and inaudible tones signaled the start of each experimental phase on tape.

Measures

Behavioral coding—Computerized behavioral coding was carried out using Noldus Corporation's Observer software (version 5.0). Two independent raters blind to the group status and all other information about the infants coded the infants' videotaped mobile task behavior. The raters coded infants' behavior for kicking (learning), look duration, and facial expression (negative affect). Approximately 3% to 5% of the time an infant would turn his or her head so far to the right during testing that the infant's facial expressions were obscured; these data were omitted from the total percentages computed for targeted behaviors.

Learning—To assess learning, the frequency of kicks was scored during the baseline, learning, and nonreinforcement phases of the procedure. A kick was defined as a vertical or horizontal movement of the foot that retraces its original path in a smooth, continuous motion (Rovee & Rovee, 1969). Relative rates of kicking for each phase were computed by dividing the total number of kicks for learning and for nonreinforcement by the total number of kicks during baseline. We also did this for each 3-min period of contingency learning (Learning I, II, and III). We employed the commonly used learning criteria to distinguish infants who learned and did not learn the contingency task: a kicking rate that was 1.5 times baseline for any 2 consecutive minutes during the learning phase (Rovee-Collier et al., 1980). Accordingly, two groups of infants were created: learners and nonlearners.

Looking—The infant's direction of gaze was coded in real time using one of four codes: fixation on the mobile, fixation not on the mobile, eyes closed, and eyes unseen. Eyes unseen was used when the infant's eyes were not visible on the video. Average duration of each behavior was computed for each phase of the procedure.

Affect—Affect was scored continuously from videotapes based on the methods of Mayes, Bornstein, and Chawarska (1996). Affect was scored in terms of four categories: positive, negative, neutral, and unseen (Mayes et al., 1996). For the purposes of this study, however, only the negative affect category will be reported. Negative affect was defined as including facial expressions of distress, anger, disgust, or frowns (Mayes et al., 1996). These facial expressions included moments of fussing or crying. This method of coding provided a global measure of negative affect. Average duration of negative affect was computed for each phase of the procedure and then transformed into percentage scores.

Interrater reliability—To evaluate interrater reliability, 20% of the sample was recoded by a different coder. For kicking, average intraclass correlation coefficients were $r = .86$ (baseline), $r = .86$ (learning), and $r = .83$ (nonreinforcement). Some studies report stronger interrater reliability scores using live coding (e.g., Millar and Weir, 2007). Although our coding of behavior from videotapes results in slightly lower interrater reliability than that achieved by live coding, the benefit of video coding is less bias and less contextual influences. In future studies, it may be useful to use two cameras to increase reliability. For

look duration, average intraclass correlation coefficients were $r = .91$ (baseline), $r = .90$ (learning), and $r = .83$ (nonreinforcement). For negative affect, average intraclass correlation coefficients were $r = .85$ (baseline), $r = .87$ (learning), and $r = .85$ (nonreinforcement). Alpha values were the same as the intraclass correlation coefficients.

Cardiac measures—Heart rate data were recorded from gel electrodes on a minimitter (3992/1-IBI Biolog) that was connected by a serial cable to a laptop computer. The two recording electrodes were placed across the chest, one on the upper right and the other on the lower left side of the chest. The 3992/1 Biolog samples the electrocardiogram (ECG) data at 1,000 Hz and performs peak detection (for further information see http://www.minimitter.com/Products/Brochures/900-0103-00_ML.pdf). The data were collected during prebaseline (before the procedure started), baseline (exposure to the mobile), contingency learning (ribbon attached), and nonreinforcement (ribbon detached), and stored offline in heart period format. Heart period files were cleaned and checked for artifacts with MX-Edit software using the methodology of Porges and colleagues (e.g., Doussard-Roosevelt et al., 1997). To determine the parasympathetic component of the heart period data, an estimate of RSA was computed using time series analyses. This analysis extracts the component of the heart period within the respiratory frequency band of young infants (0.30Hz–1.30Hz). The natural logarithm of this variance produces the vagal tone statistic (i.e., RSA). Heart rate and RSA values were calculated in sequential 30-sec epochs, and then average scores were computed for each episode of the contingency learning task.

Salivary cortisol—One of the reasons for conducting the study in the home rather than in the laboratory was to control for the potential effects of traveling to a new environment on baseline cortisol levels. Two saliva samples were collected, a basal sample prior to the start of testing, and a second sample 20 min after the introduction of the mobile. Saliva was obtained using a cotton dental roll, which was then placed into a needleless syringe. No salivary stimulant was used. The saliva was expressed into a vial and stored at -20°C until assayed in the laboratory of J. Weinberg at the University of British Columbia using the Salimetrics High Sensitivity Salivary Cortisol Enzyme Immunoassay Kit for quantitative determination of salivary cortisol (Salimetrics LLC, State College, PA). Intra- and interassay co-efficients of variation were 2.92% and 3.41%, respectively.

Plan of Statistical Analyses

To examine our hypotheses, repeated measures analysis of variance (ANOVA) with group (preterm vs. full-term) and speed (learners vs. nonlearners) as the between-subject factors and with phase (baseline, Learning I, Learning II, Learning III, and nonreinforcement) as the repeated factor were conducted on infant relative kicking rates, negative affect, looking, heart rate, and RSA. For analyses of cardiac data, amount of kicking was included as a covariate. In addition, a repeated measures ANOVA was conducted on cortisol levels with group (preterm vs. full-term) and speed (learners vs. nonlearners) as between-subject factors and with sample (pre and post) as a repeated factor. To examine our prediction that negative emotion and physiology would be correlated in preterm but not full-term infants, bi-variate correlation analyses were used. To examine changes between phases, we computed difference scores and ran correlations on them as well as on the raw scores.

RESULTS

Preliminary Analyses

Attrition—Although the same proportion of preterm (55/61, 90%) and full-term (49/55, 89%) infants completed the procedure, 22% of the preterm infants who completed the

procedure required some facilitation from the experimenter or a parent, whereas only 5% of full-term infants who completed the procedure required facilitation.

Learning—Based on criterion that infants had to kick 1.5 times their spontaneous frequency of kicking during any 2 consecutive minutes relative to the baseline levels, 32 preterm and 26 full-term infants were in the learner group and 29 preterm and 29 full-term infants were in the nonlearner group. After excluding infants who did not complete the nonreinforcement phase, 30 preterm learners (kicking rate: $M = 2.40$, $Mdn = 2.00$, $min = 1.48$, $max = 5.83$) and 25 full-term learners (kicking rate: $M = 4.61$, $Mdn = 3.33$, $min = 1.53$, $max = 16.25$) and 25 preterm nonlearners (kicking rate: $M = .91$, $Mdn = .89$, $min = 0$, $max = 1.47$) and 24 full-term nonlearners (kicking rate: $M = .92$, $Mdn = .93$, $min = 0$, $max = 1.46$) remained. Neonatal characteristics did not significantly differ among nonlearners and learners in each group (see Table 1).

Preterm infants had lower kicking rates overall relative to baseline performance ($M = 1.65$, $SE = .153$) than the full-term group ($M = 2.424$, $SE = .223$) as indicated by a between-subject main effect, $F(1, 97) = 6.282$, $p < .05$, $\eta_p^2 = .06$; however, by definition, the nonlearners had lower kicking rates relative to baseline ($M = .970$, $SE = .225$) than the learners ($M = 3.113$, $SE = .21$), $F(1, 97) = 49.229$, $p < .001$, $\eta_p^2 = .34$. A significant Group $\times$ Learning $\times$ Phase interaction was found for learning, $F(4, 388) = 3.255$, $p < .05$, $\eta_p^2 = .03$. As can be seen in Figure 1, the full-term learners showed greater increases in relative kicking rates than the preterm learners. All learners, regardless of preterm status, surpassed the learning criteria within the first 3-min block of the learning task. However, nonlearners, by definition did not ever reach the learning criterion. Planned contrasts indicated that the full-term learners had greater increases in kicking rate from baseline to Learning I ($p < .05$), and from Learning I to Learning II ($p < .05$), but showed greater decreases from Learning III to nonreinforcement ($p < .01$) compared to the preterm learners. There were no differences between the preterm and full-term nonlearners, and they did not show a significant change in kicking rate during the procedure.

Reactivity

Autonomic: A repeated-measures analysis of covariance (ANCOVA) was carried out on heart rate, controlling for kicking during baseline and learning and heart rate at rest (in mother's lap), to examine the effects of group (preterm vs. full-term), learning (learner vs. nonlearner), and phase (baseline, Learning I, Learning II, Learning III, and extinction). The results (see Figure 2 and Table 2) yielded a Group $\times$ Phase interaction, $F(4, 336) = 2.59$, $p < .05$, $\eta_p^2 = .03$. Planned contrasts indicated that preterm infants showed greater heart rate accelerations from baseline to Learning I ($p < .01$) compared to full-term infants. In addition, full-term infants showed greater heart rate accelerations from Learning III to nonreinforcement ($p < .05$) compared to preterm infants, which was flat (see Figure 2). For RSA, the results yielded a significant Speed $\times$ Phase interaction, $F(4, 336) = 4.76$, $p < .001$, $\eta_p^2 = .05$. The learning group showed greater suppression of RSA from baseline to nonreinforcement compared to nonlearners (Figure 3 and Table 2). To explore this two-way interaction, the same analysis was conducted separately by group. Interestingly, there was a Learning $\times$ Phase interaction for preterm infants ($p < .05$) but not for full-term infants, which indicates that the variability in learning in preterm infants was related to regulation of parasympathetic activity. No other group differences were observed.

L-HPA: To examine whether the procedure would elicit a significant elevation in cortisol, a repeated-subjects ANCOVA was conducted with group and speed as the between-subject factors and sample (pre and post) as the repeated factor, with time of testing (morning vs. afternoon) entered as a covariate. We found that the preterm infants had lower cortisol levels

overall ($M = .18\mu\text{g/dl}$, $SD = .09$) than the full-term infants ($M = .28\mu\text{g/dl}$, $SD = .19$), $F(1, 96) = 15.41$, $p < .001$, $\eta_p^2 = .14$.

Behavior—A repeated-measures ANOVA was carried out on negative affect to examine the effects of group (preterm vs. full-term), learning (learner vs. nonlearner), and phase (baseline, Learning I, Learning II, Learning III, and nonreinforcement). There was a main effect for learning, $F(1, 95) = 5.139$, $p < .05$, $\eta_p^2 = .05$. Across the procedure, learners ($M = 2.43$, $SE = .74$) had lower levels of negative affect than nonlearners ($M = 4.92$, $SE = .80$). There were also significant Group $\times$ Phase, $F(4, 95) = 3.09$, $p < .05$, $\eta_p^2 = .03$, and Speed $\times$ Phase interactions, $F(4, 95) = 2.99$, $p < .05$, $\eta_p^2 = .03$. To follow up the two-way interactions, the same analyses were conducted separately across learning and group. Interestingly, there was a Learning $\times$ Phase interaction for full-term infants ($p < .05$) but not for preterm infants, indicating that the variability in learning in full-term infants was related to the regulation of affect. That is, full-term learners had better regulation of affect than the full-term nonlearners. In addition, we found a Group $\times$ Phase interaction in the learners but not the nonlearners, indicating that full-term learners had better affect regulation than preterm learners (see Figure 4).

For look duration, there was a main effect for group: Preterm infants ($M = 75.94\%$) looked at the mobile less than the full-term infants ($M = 84.78\%$), $F(1, 97) = 6.92$, $p < .01$, $\eta_p^2 = .07$. One-way ANOVAs indicated that preterm infants looked less at the mobile than the full-term infants at baseline ($p < .05$) and Learning II ($p < .05$). There was also a trend for the preterm infants to look less during Learning III ($p = .05$). There were no other group differences.

Coordination of Negative Affect and Physiology

To examine relationships between physiology and negative affect, we carried out correlations separately for Group $\times$ Learning (see Table 3), using change scores (contingency–baseline; nonreinforcement–contingency). For preterm nonlearners, increases in negative affect during learning were associated with increases in RSA (parasympathetic activity) during nonreinforcement. For preterm learners, greater decreases in negative affect during contingency were associated with greater accelerations in heart rate and greater decreases in RSA during nonreinforcement, as well as with greater secretion of cortisol. In addition, greater increases in negative affect during nonreinforcement were associated with greater increases in heart rate during nonreinforcement.

For full-term learners, higher negative affect during contingency was associated with greater accelerations in heart rate during nonreinforcement. For full-term nonlearners, there were no associations between negative affect and physiology. Analyses of negative affect with heart rate and RSA were repeated using partial correlations controlling for the effects of kicking and were consistent with the results of the zero-order correlations.

We also examined relationships between physiology and negative affect during each portion of the experiment using average scores rather than change scores. Greater levels of negative affect at rest were associated with faster heart rates at rest in the preterm nonlearners ($r = .39$, $p < .05$). Among the preterm learners, higher levels of basal cortisol were related to greater levels of negative affect during learning ($r = .43$, $p < .05$). Among the full-term learners, higher levels of negative affect during nonreinforcement were related to greater levels of postcortisol levels ($r = .62$, $p < .001$). No other significant relationships were observed in any of the other groups.

DISCUSSION

This study was conducted to examine contingency learning in relation to behavioral and physiological reactivity in preterm and full-term infants. We tested the hypothesis that learning difficulties would be related to greater evidence of arousal impairment in preterm infants. This hypothesis was based on developmental theories depicting preterm infants as having impaired arousal regulation that adversely affects their capacity to learn (e.g., Field, 1981; Mayes, 2000; Tronick et al., 1990). As expected, we found that preterm infants showed lower kicking rates, looked less at the mobile, and showed greater increases in heart rate in response to contingency compared to full-term infants. We also found that preterm infants showed dampened heart rate responses to violations of contingency (i.e., nonreinforcement) and had lower cortisol levels than full-term infants. Our findings support the idea that learning difficulties in preterm infants may be linked to difficulties in arousal regulation, which we discuss in further detail.

Our hypothesis that preterm infants would show lower rates of learning than full-term infants was partially confirmed. We found that an equal percentage of preterm and full-term infants reached the learning criterion; however, kicking rates were lower in the preterm than in the full-term learners, which replicates previous work (Gekoski et al., 1984). Gekoski et al. (1984) found that preterm infants required a second session to reach a level of contingency learning that was similar to that of full-term infants. Whether the lower rate of kicking in preterm infants is the result of poorer health, faster habituation, temperament, motor delay, cognitive deficits, or other causes is unclear. Interestingly, we also found that preterm learners showed increased kicking rates during nonreinforcement compared to the full-term learners, who decreased kicking. This pattern would suggest that motor delay-, health-, or habituation-related effects are involved. Rather, this pattern is reminiscent of neonatal studies (e.g., Krafchuk et al., 1983) in which preterm infants appear unable to modulate their learned responses to familiar stimuli compared to full-term infants, which raises the question of whether temperament or cognitive deficits are possible contributing factors.

Our data support the view that preterm infants have greater impairment in arousal regulation, which has been reported in previous work in high-risk neonates (e.g., Gardner et al., 1992). Gardner and colleagues (1992) showed that manipulations in arousal level affected visual preferences in brain-injured neonates more than in normal non-brain-injured neonates. We found that preterm infants looked less at the mobile than full-term infants at baseline and at Learning II. This may be the result of fatigue or heightened arousal, which, in turn, may decrease the infant's capacity to encode contingent stimulation. For example, looking less at an exciting stimulus has been shown to decrease heightened states of arousal (Field, 1979; Kopp, 2002). Accordingly, preterm infants may spend less time looking at the mobile because they are more aroused, which may limit learning opportunities rather than reflect underlying deficits in cognition. Alternatively, a fatigue effect may cause infants to look away during the procedure, particularly during the learning. However, differences between groups at baseline would seem to suggest arousal difficulties rather than fatigue per se. Our heart rate data support this perspective. We found that preterm infants had faster basal heart rates compared to full-term infants and showed greater increases in heart rate in response to contingency than full-term infants, which is consistent with prior research examining infant cardiac responses in a social context (Coles et al., 1999; Field, 1979). In short, preterm infants appear less able to attend to the stimulus and show impaired cardiac regulation compared to full-term infants. It may be that heightened arousal evokes the need to behaviorally regulate this arousal via looking away from the mobile. This greater need for regulation may be at odds with learning during the contingency task, which might represent a pattern that contributes to future learning difficulties (Mayes, 2000).

What distinguishes the preterm learner group from the other subgroups? Regulation of cardiac autonomic activity seemed to be linked to the ability of the preterm infants to reach the learning criterion. Specifically, preterm infants who reached the learning criterion showed greater RSA suppression than preterm infants who failed to reach the learning criterion. We also found that preterm learners showed less heart rate acceleration in response to the nonreinforcement phase than full-term learners. This may be taken as further evidence of impaired arousal regulation or suggest that preterm learners were less aware of the change in the experimental condition. Given that preterm infants responded emotionally to the nonreinforcement condition, however, the former would seem correct. In fact, preterm learners were the only infants who showed coordination of increased negative affect and heart rate acceleration in response to nonreinforcement. Although preterm infants showed greater increases in negative affect compared to full-term infants in response to nonreinforcement, it was the preterm nonlearners who showed an increase in negative affect in response to the final 3 min of the contingency learning task (prior to nonreinforcement) compared to the preterm learners (see Figure 4). In contrast, the full-term learners showed the smallest increase in negative affect as well as moderate heart rate responses. These findings are similar to neonatal studies in which preterm infants show behavioral but not cardiac responses to stimulation (e.g., Rose et al., 1976).

We also found that increases in negative affect in response to learning were highly correlated with increases in RSA responses to nonreinforcement in both preterm learners and nonlearners. It is tempting to speculate that in preterm infants negative affect during learning induces a rise in RSA levels in response to nonreinforcement, which then acts to dampen heart rate responses—at least for those infants who show greater levels of negative affect during learning. If so, emotion-induced increases in RSA may inhibit the infant's heart rate response to nonreinforcement, which could help explain why the preterm nonlearners have the apparent disconnect between showing the highest levels of negative affect and dampened heart rate responses to noncontingency. This altered arousal regulation pattern may potentially interfere with information processing (Mayes, 2000). Taken together, the preterm learners differed from full-term learners in that they showed a flattened cardiac response to nonreinforcement, and they differed from the preterm nonlearners in that they showed less negative affect and greater suppression of parasympathetic activity.

Learners Versus Nonlearners

We found that learners regardless of preterm status differed from nonlearners in levels of emotional expression and RSA. We found that learners had lower levels of negative affect overall compared to nonlearners. This finding is consistent with prior research showing that faster contingency learning (Fagen & Ohr, 1985) and habituation (Rose, Futterweit, & Jankowski, 1999) were associated with less expression of negative affect. We also found that learners showed greater vagal suppression (i.e., decreases in RSA) than nonlearners, which is consistent with Porges's (1995) polyvagal theory and previous work showing that greater suppression of RSA is related to faster habituation (e.g., Bornstein & Suess, 2000). However, the relationship of learning to RSA was most significant in preterm infants. Based on the fact that contingency learning is presumably a cognitive challenge whereas nonreinforcement of the contingency is more of an emotional challenge, it is interesting to note that we found greater decreases in RSA from baseline to contingency and from contingency to nonreinforcement in the learners compared to the nonlearners. This finding would suggest that the capacity to regulate RSA plays an important role in learning and in the regulation of emotion.

L-HPA

Although infants showed heightened autonomic reactivity (reflected in increased heart rate) during all phases of the procedure and decreases in RSA from baseline to nonreinforcement, we found that the contingency task was not sufficiently stressful to elicit a cortisol response. Instead, we found that preterm infants showed lower cortisol levels than full-term infants. Clinical studies have found that preterm neonates, while hospitalized in intensive medical care, may be at risk of adrenal insufficiency (e.g., Bolt et al., 2002). Accordingly, our findings suggest that a hyposecretion pattern of cortisol may persist in preterm infants up to 3 months of age (adjusted for gestation) especially those most exposed to neonatal pain (Granaue et al., 2005).

This cortisol pattern is consistent with our recent work showing that alterations in the development of the HPA axis in preterm infants may extend from 3 to 18 months (Granaue, Haley, Weinberg, & Whitfield, 2007). Interestingly, we have also previously shown that greater secretion of cortisol in preterm infants at 3 months of age is related to better 24-hr memory (Haley, Weinberg, & Granaue, 2006). In this study, however, we did not find a relationship between cortisol and learning. In contrast, we found that the secretion of cortisol may be related to the regulation of emotion. Specifically, we found that preterm learners became less upset as they secreted more cortisol. This result supports the notion that cortisol secretion may be adaptive in novel contexts (e.g., Gunnar et al., 1997). Secretion of cortisol in infants with lower levels of negative affect and faster rates of learning (e.g., preterm learners) would appear to constitute an adaptive response pattern. We also found, however, that elevated negative affect during nonreinforcement was associated with greater postcortisol levels in full-term learners, suggesting that the relationship between affect and the L-HPA axis differs between preterm and full-term infants. These findings suggest that alterations in the development of the L-HPA axis in preterm infants may affect learning only indirectly by affecting the relationships between behavior and neurobiology.

Future Directions

This study provides evidence to support the view that difficulties in arousal regulation contribute to learning difficulties in preterm infants. Alternative accounts, however, should be considered. For example, others have found no evidence that arousal affects learning in preterm infants (e.g., Herbert et al., 2004). Although Herbert and colleagues (2004) observed greater variability in the preterm infants' (28–31 weeks gestation) learning rate across three sessions compared to full-term infants at 5 months (CCA), they did not find a mean difference in performance. They argue that this greater variability in performance may be due to delayed or altered brain development. By using specific memory tasks that are dependent on specific cortical regions (e.g., hippocampus), it would be possible to further clarify the extent to which general arousal versus specific brain abnormalities characterize learning difficulties in preterm children.

Based on polyvagal theory, we hypothesized that greater suppression of RSA would enhance learning. We found evidence that greater regulation of vagal tone was associated with less negative affect and greater learning. It should be noted, however, that approximately half of the infants failed to reach the learning criterion, regardless of gestational age. One clear possibility for this is that some infants may not have learned the contingency because they did not like the mobile (e.g., temperament). It has been shown that faster contingency learning is associated with lower negative reactivity (Lemelin, Tarabulsy, & Provost, 2002), longer duration of orientation, faster response to novelty (Fagen et al., 1987), and greater rhythmicity and persistence (Dunst & Lingerfelt, 1985). It may be that after a second day of training, these nonlearners would have reached the learning criterion and, perhaps, demonstrated greater evidence of autonomic regulation as well.

One of the limitations of this study is that we used analytical procedures that examined average scores across relatively large blocks of time. This approach is not sensitive to changes in arousal state that are linked to the infant's response to specific stimuli (e.g., peak attention) but rather reflects a global indicator of arousal state and provides a method for looking at changes in arousal state from one condition to the next. Although our measurement of looking duration and heart rate did not reveal differences between learners and nonlearners, it is possible that more refined measures would reveal such differences. A more in-depth analysis of looking duration and heart rate examining changes in heart rate for each peak looking event (e.g., 5-sec events) might have clarified whether infants failed to learn because they were overaroused or because they are faster habituators. This would be an excellent enterprise in a future study.

We found no differences in perinatal factors (e.g., gestational age) and medical history between learners and nonlearners among the preterm infants. Future efforts to evaluate learning, reactivity, and coordination of emotion and physiological responses in preterm and full-term infants should consider the role of the parent–infant interaction. Specifically, contingency experienced in the parent–infant context has been linked to the infant's contingency learning (e.g., Dunham & Dunham, 1990) and the infant's capacity to regulate affect, attention, and physiology during social interactions (Haley & Stansbury, 2003). In fact, infant reactivity appears relatively stable across social and nonsocial contingency contexts, such as the still-face and mobile paradigms (Shapiro et al., 1998). Are individual differences in parenting or parenting practices (e.g., breast feeding) related to the reactivity patterns of preterm learners and nonlearners? Addressing such questions could further elucidate interventions that might target changes in specific reactivity indexes (e.g., behavior vs. physiology).

Although we examined global measures of negative affect, we did not examine whether specific negative emotions (e.g., sad vs. angry) differed between preterm and full-term infants, or whether the relationship between specific emotions and physiological systems differs between learners and nonlearners among preterm infants; this would be interesting to pursue in future studies. Also, we did not have sufficient samples to evaluate gender differences, which may be relevant and should be examined in future studies. Finally, although we evaluated cortisol 20 min after the onset of the contingency stimulation and found that relationships between cortisol and negative affect during contingency learning were significant in preterm infants, collection of multiple postsaliva samples in a future study would permit evaluation of recovery and delayed peak reactivity of cortisol in relation to negative emotion.

Summary

We found evidence that impairments in arousal regulation are linked to learning difficulties in preterm infants. We found that preterm infants showed slower rates of contingency learning, less attention, lower cortisol levels, and greater heart rate responses to contingent stimulation than full-term infants. We also found that learning, regardless of gestational age, was related to greater suppression of RSA and lower levels of negative affect. Our findings help elucidate relatively specific and subtle differences in reactivity that may contribute to learning difficulties in high- and low-risk infants. Further work is needed to examine the social and developmental contexts that contribute to these reactivity and learning patterns.

Acknowledgments

This work was primarily funded by the National Institute for Child Health and Human Development grant HD39783 to Ruth E. Grunau, with additional funding from the Canadian Institutes for Health Research grant MOP42469 to Ruth E. Grunau and the Human Early Learning Partnership (HELP) grants 20R41341 to Joanne

Weinberg and 2OR31739 to Ruth E. Grunau through the B.C. Ministry for Children and Families, and from the Michael Smith Foundation for Health Research. Ruth E. Grunau is supported by a Distinguished Scholar award from the Child and Family Research Institute, and a HELP Senior Scholar award. We would like to express our appreciation to Dr. Rovee-Collier for providing the test materials and for consultation on the procedures. We are grateful to the families who participated and for the invaluable dedication of the research staff.

REFERENCES

- Alessandri SM, Sullivan MW, Lewis M. Violation of expectancy and frustration in early infancy. *Developmental Psychology*. 1990; 26:738–744.
- Amsel A. The role of frustrative nonreward in noncontinuous reward situations. *Psychological Bulletin*. 1958; 55:102–119. [PubMed: 13527595]
- Anderson P, Doyle LW. Victorian Infant Collaborative Study Group. Neurobehavioral outcomes of school-age children born extremely low birth weight or very preterm in the 1990s. *Journal of American Medical Association*. 2003; 289:3264–3272.
- Bazhenova OV, Plonskaia O, Porges SW. Vagal reactivity and affective adjustment in infants during interaction challenges. *Child Development*. 2001; 72:1314–1326. [PubMed: 11699673]
- Bolt RJ, Van Weissenbruch MM, Popp-Snijders C, Sweep FGJ, Lafeber HN, Delemarre-Van De Waal HA. Maturity of the adrenal cortex in very preterm infants is related to gestational age. *Pediatric Research*. 2002; 52:405–410. [PubMed: 12193676]
- Bornstein MH, Suess PE. Physiological self-regulation and information processing in infancy: Cardiac vagal tone and habituation. *Child Development*. 2000; 71:273–287. [PubMed: 10834463]
- Brazelton, TB.; Koslowski, B.; Main, M. The origins of reciprocity: The early mother–infant interaction. In: Lewis, M.; Rosenblum, LA., editors. *The effect of the infant on its caregiver*. New York: Wiley; 1974. p. 49-76.
- Coles CD, Bard KA, Platzman KA, Lynch ML. Attentional response at eight weeks in prenatally drug-exposed and preterm infants. *Neurotoxicology and Teratology*. 1999; 21:527–537. [PubMed: 10492387]
- DiPietro JA, Porges SW, Uhly B. Reactivity and developmental competence in preterm and full-term infants. *Developmental Psychology*. 1992; 28:831–841.
- Doussard-Roosevelt JA, McClenny BD, Porges SW. Neonatal cardiac vagal tone and school-age developmental outcome in very low birth weight infants. *Developmental Psychobiology*. 2001; 38:56–66. [PubMed: 11150061]
- Doussard-Roosevelt JA, Porges SW, Scanlon JW, Alemi B, Scanlon KB. Vagal regulation of heart rate in the prediction of developmental outcome for very low birth weight preterm infants. *Child Development*. 1997; 68:173–186. [PubMed: 9179997]
- Dunham P, Dunham F. Effects of mother–infant interactions on infants' subsequent contingency task performance. *Child Development*. 1990; 61:785–793. [PubMed: 2364753]
- Dunst CJ, Lingerfelt B. Maternal ratings of temperament and operant learning in two- to three-month-old infants. *Child Development*. 1985; 56:555–556.
- Fagen JW, Ohr PS. Temperament and crying in response to the violation of a learned expectancy in early infancy. *Infant Behavior and Development*. 1985; 8:157–166.
- Fagen JW, Ohr PS, Singer JM, Fleckenstein LK. Infant temperament and subject loss due to crying during operant conditioning. *Child Development*. 1987; 58:497–504. [PubMed: 3829790]
- Field T. Visual and cardiac responses to animate and inanimate faces by young term and preterm infants. *Child Development*. 1979; 50:188–194. [PubMed: 446203]
- Field, T. Infant arousal, attention, and affect during early interactions. In: Lipsitt, L.; Rovee-Collier, C., editors. *Advances in infancy research*. Vol. 1. Norwood, N.J.: Ablex; 1981. p. 58-100.
- Field TM, Dempsey JR, Hatch J, Ting G, Clifton RK. Cardiac and behavioral responses to repeated tactile and auditory stimulation in preterm and term neonates. *Developmental Psychology*. 1979; 15:406–416.
- Fox NA, Porges SW. The relation between neonatal heart period patterns and developmental outcome. *Child Development*. 1985; 56:28–37. [PubMed: 3987407]

- Gardner, JM.; Karmel, BZ. Attention and arousal in preterm and full-term neonates. In: Field, T.; Sostek, A., editors. *Infants born at risk: Physiological, perceptual, and cognitive processes*. New York: Grune & Stratton; 1983. p. 69-98.
- Gardner JM, Karmel BZ, Magnano CL. Arousal visual preference interactions in high-risk neonates. *Developmental Psychology*. 1992; 28:821–830.
- Gekoski MJ, Fagen JW, Pearlman MA. Early learning and memory in the preterm infant. *Infant Behavior and Development*. 1984; 7:267–275.
- Gunnar MR, Broderson L, Krueger K, Rigatuso J. Dampening of adrenocortical responses during infancy: Normative changes and individual differences. *Child Development*. 1996; 67:877–889. [PubMed: 8706532]
- Gunnar MR, Tout K, de Haan M, Pierce S, Stansbury K. Temperament, social competence, and adrenocortical activity in preschoolers. *Developmental Psychobiology*. 1997; 31:65–85. [PubMed: 9222117]
- Grunau RE, Haley DW, Weinberg J, Whitfield MF. Re-programming of hypothalamic–pituitary–adrenal response in preterm infants. *Journal of Pediatrics*. 2007; 150:151–156. [PubMed: 17236892]
- Grunau RE, Holsti L, Haley DW, Oberlander T, Weinberg J, Solimano A, et al. Neonatal procedural pain exposure predicts lower cortisol and behavioral reactivity in preterm infants in the NICU. *Pain*. 2005; 113:293–300. [PubMed: 15661436]
- Grunau RE, Whitfield MF, Davis C. Pattern of learning disabilities in extremely low birth weight children at age 8½ years. *Archives of Pediatrics and Adolescent Medicine*. 2002; 156:615–620. [PubMed: 12038896]
- Grunau RE, Whitfield MF, Fay TB. Psychosocial and academic characteristics of extremely low birth weight (800 g) adolescents who are free of major impairment compared with term-born control subjects. *Pediatrics*. 2004; 114:e725–e732. [PubMed: 15576337]
- Hack M, Flannery DJ, Schluchter M, Cartar L, Borawski E, Klein N. Outcomes in young adulthood for very-low-birth-weight infants. *New England Journal of Medicine*. 2002; 346:149–157. [PubMed: 11796848]
- Haley DW, Stansbury K. Infant stress and parent responsiveness: Regulation of behavior and physiology during still-face and reunion. *Child Development*. 2003; 74:1534–1546. [PubMed: 14552412]
- Haley DW, Weinberg J, Grunau RE. Cortisol, contingency learning, and memory in preterm and full-term infants. *Psychoneuroendocrinology*. 2006; 31:108–117. [PubMed: 16122876]
- Hartshorn K, Rovee-Collier C. Does infant memory expression reflect age at encoding or age at retrieval? *Developmental Psychobiology*. 2003; 41:283–291. [PubMed: 12621654]
- Herbert JS, Eckerman CO, Goldstein RF, Stanton ME. Contrasts in infant classical eyeblink conditioning as a function of premature birth. *Infancy*. 2004; 5:367–383.
- Kopp CB. Commentary: The codevelopments of attention and emotion regulation. *Infancy*. 2002; 3:199–208.
- Krafchuk, EE.; Tronick, EZ.; Clifton, RK. Behavioral and cardiac responses to sound in preterm neonates varying in risk status: A hypothesis of their paradoxical reactivity. In: Field, T.; Sostek, A., editors. *Infants born at risk: Physiological, perceptual, and cognitive processes*. New York: Grune & Stratton; 1983. p. 99-128.
- Landry SH, Leslie NA, Fletcher JM, Francis DT. Visual attention skills of premature infants with and without intraventricular hemorrhage. *Infant Behavior and Development*. 1985; 8:309–321.
- Lemelin J, Tarabulsy GM, Provost MA. Relations between measures of irritability and contingency detection at 6 months. *Infancy*. 2002; 3:543–554.
- Lester BM, Boukydis CFZ, LaGasse L. Cardiorespiratory reactivity during the Brazelton scale in term and preterm infants. *Journal of Pediatric Psychology*. 1996; 21:771–784. [PubMed: 8990723]
- Lewis M, Alessandri SM, Sullivan MW. Violation of expectancy, loss of control, and anger expressions in young infants. *Developmental Psychology*. 1990; 26:745–751.
- Lewis M, Ramsay DS. Developmental change in infants' responses to stress. *Child Development*. 1995; 66:657–670. [PubMed: 7789193]

- Mayes LC. A developmental perspective on the regulation of arousal states. *Seminars in Perinatology*. 2000; 24:267–279. [PubMed: 10975433]
- Mayes, LC.; Bornstein, MH. Attention regulation in infants born at risk. In: Burack, JA.; Enns, JT., editors. *Attention, development, & psychopathology*. New York: Guilford Press; 1997. p. 97–122.
- Mayes LC, Bornstein MH, Chawarska K. Impaired regulation of arousal in 3-month-old infants exposed prenatally to cocaine and other drugs. *Development & Psychopathology*. 1996; 8:29–42.
- Millar WS, Weir CG. A comparison of fixed-trial and infant control habituation procedures with 6- to 13-month-old at-risk infants. *Infant Behavior and Development*. 1995; 18:263–272.
- Millar WS, Weir CG. Heart rate reactivity during contingency learning in 5-to 10-month-old at-risk and non-risk babies. *British Journal of Developmental Psychology*. 2007; 25:135–146.
- Millar WS, Weir CG, Supramaniam G. The influence of perinatal risk status on contingency learning in six- to thirteen-month-old infants. *Child Development*. 1992; 63:304–313. [PubMed: 1611935]
- Pharoah PO, Stevenson CJ, West CR. General certificate of secondary education performance in very low birth weight infants. *Arch Dis Child*. 2003; 88:295–298. [PubMed: 12651749]
- Porges SW. Orienting in a defensive world: Mammalian modification of our evolutionary heritage. A polyvagal theory. *Psychophysiology*. 1995; 32:301–318. [PubMed: 7652107]
- Ramey CT, Heiger L, Klisz D. Synchronous reinforcement of vocal responses in failure-to-thrive infants. *Child Development*. 1972; 43:1449–1455. [PubMed: 4643780]
- Rose SA, Feldman JF. Memory processing speed in preterm children at eleven years: A comparison with full terms. *Child Development*. 1996; 67:2005–2021. [PubMed: 9022226]
- Rose SA, Feldman JF, Jankowski JJ, Caro DM. A longitudinal study of visual expectation and reaction time in the first year of life. *Child Development*. 2002; 73:47–61. [PubMed: 14717243]
- Rose SA, Feldman JF, McCarten CM, Wolfson J. Information processing in seven-month-old infants as a function of risk status. *Child Development*. 1988; 59:589–603. [PubMed: 3383669]
- Rose SA, Futterweit LR, Jankowski JJ. The relation of affect to attention and learning in infancy. *Child Development*. 1999; 70:549–559. [PubMed: 10368909]
- Rose SA, Schmidt K, Bridger WH. Cardiac and behavioral responsivity to tactile stimulation in premature and full-term infants. *Developmental Psychology*. 1976; 12:311–320.
- Rovee CK, Rovee DT. Conjugate reinforcement of infant exploratory behavior. *Journal of Experimental Child Psychology*. 1969; 8:33–39. [PubMed: 5804591]
- Rovee-Collier C. Dissociations in infant memory: Rethinking the development of implicit and explicit memory. *Psychological Review*. 1997; 104:467–498. [PubMed: 9243961]
- Rovee-Collier CK, Sullivan MW, Enright M, Lucas D, Fagen JW. Reactivation of infant memory. *Science*. 1980; 208:1159–1161. [PubMed: 7375924]
- Saigal S, Hoult LA, Streiner DL, Stoskopf BL, Rosenbaum PL. School difficulties at adolescence in a regional cohort of children who were extremely low birth weight. *Pediatrics*. 2000; 105:325–331. [PubMed: 10654950]
- Shapiro B, Fagen J, Prigot J, Carroll M, Shalan J. Infants' emotional and regulatory behaviors in response to violations of expectancies. *Infant Behavior and Development*. 1998; 21:299–313.
- Shirley M. Development of immature babies during their first two years. *Child Development*. 1938; 9:347–360.
- Shirley M. A behavior syndrome characterizing prematurely-born children. *Child Development*. 1939; 10:115–128.
- Stansbury, K.; Gunnar, MR. Adrenocortical activity and emotion regulation. In: Fox, NA., editor. *Monographs of the Society for Research in Child Development*. Vol. 59. 1994. The development of emotion regulation: Biological and behavioral considerations
- Sullivan MW, Lewis M. Contextual determinants of anger and other negative expressions in young infants. *Developmental Psychology*. 2003; 39:693–705. [PubMed: 12859123]
- Tarabulsky GM, Tessier R, Kappas A. Contingency detection and the contingent organization of behavior in interactions: Implications for socioemotional development during infancy. *Psychological Bulletin*. 1996; 120:25–41. [PubMed: 8711014]
- Taylor HG, Klein N, Minich NM, Hack M. Middle-school-age outcomes in children with very low birth weight. *Child Development*. 2000; 71:1495–1511. [PubMed: 11194251]

- Tronick E, Scanlon KB, Scanlon JW. Protective apathy: A hypothesis about the behavioral organization and its relation to clinical and physiologic status of the preterm infant during the newborn period. *Clinics in Perinatology*. 1990; 17:125–154. [PubMed: 2318013]
- Watson JS. The development and generalization of “contingency awareness” in early infancy: Some hypotheses. *Merrill-Palmer Quarterly*. 1966; 12:123–155.
- Wilson SL, Cradock MM. Review: Accounting for prematurity in developmental assessment and the use of age-adjusted scores. *Journal of Pediatric Psychology*. 2004; 29:641–650. [PubMed: 15491987]

FIGURE 1.
Mean values of relative kicking for preterm and full-term groups across phases.

FIGURE 2.
Mean values of heart rate by Phase $\times$ Group $\times$ Speed.

FIGURE 3.
Mean values of RSA by Phase $\times$ Group $\times$ Speed.

FIGURE 4.
Mean values of negative affect by Phase $\times$ Group $\times$ Speed.

TABLE 1

Demographic Information of Sample by Group (Preterm Versus Full-Term) and Speed (Learners Versus Nonlearners)

	Group × Speed			
	Preterm Infants		Full-Term Infants	
	Nonlearners ^a	Learners ^b	Nonlearners ^c	Learners ^a
Gestational age at birth (weeks)	29.17 (2.51)	29.13 (2.46)	39.98 (1.14)	40.01 (1.30)
Range	24.00–32.86	22.86–32.86	37.29–42.14	37.29–41.77
Age corrected for prematurity (weeks)	13.22 (.88)	13.39 (1.35)	12.90 (.78)	13.20 (1.03)
Size for gestational age (small/normal/large)	4/19/2	4/25/1	0/24/0	1/22/2
Sex (male/female)	13/12	18/12	15/9	9/16
Birth weight (g)	1224.76 (435.30)	1311.30 (514.32)	3503.13 (373.60)	3488.80 (452.43)
Days on mechanical ventilation	12.86 (20.42)	11.97 (19.26)	—	—
Number of skin-breaking procedures	116.96 (86.62)	133.07 (42.96)	—	—

^a $n = 25$.

^b $n = 30$.

^c $n = 24$.

TABLE 2

Descriptive Information on Study Measures for Entire Sample and by Group

	Entire		Preterm Infants		Full-Term Infants		
Variable	M	SD	M	SD	M	SD	p Value
Looking (%)							
Baseline	80.52	22.03	73.82	27.45	82.72	17.09	.04*
Learning I	86.02	19.05	81.06	24.48	86.21	17.34	.19
Learning II	82.54	23.66	73.83	30.65	84.38	18.73	.03*
Learning III	81.40	23.27	69.19	31.88	79.69	26.17	.05
Nonreinforcement	68.20	27.05	61.76	31.76	69.59	25.12	.16
Negative affect (%)							
Baseline	1.38	4.14	2.10	5.45	1.07	3.77	.24
Learning I	1.90	5.78	2.72	5.88	1.92	6.43	.48
Learning II	2.13	5.71	2.99	6.17	3.21	8.10	.87
Learning III	4.47	10.06	8.42	14.20	4.38	9.12	.07
Nonreinforcement	8.26	12.52	9.92	13.55	7.54	11.25	.32
Heart rate (bpm)							
Baseline	147.59	10.58	152.71	12.43	147.61	10.06	.02*
Learning I	148.31	12.95	149.79	13.28	146.31	11.85	.16
Learning II	150.81	13.02	151.18	13.28	149.93	12.11	.61
Learning III	152.43	12.80	153.23	13.85	151.34	12.20	.46
Nonreinforcement	153.47	12.31	153.48	12.82	153.22	11.82	.85
Respiratory sinus arrhythmia (ln/msec)							
Baseline	3.46	1.12	3.04	1.27	3.15	1.07	.66
Learning I	3.44	1.15	3.43	1.20	3.39	1.06	.85
Learning II	3.35	1.18	3.36	1.19	3.28	1.09	.70
Learning III	3.33	1.18	3.31	1.18	3.32	1.13	.96
Nonreinforcement	3.26	1.30	3.27	1.27	3.23	1.31	.89
Cortisol (µg/dl)							
Pretesting	.26	.23	.20	.15	.32	.28	.004**
Posttesting	.24	.17	.19	.12	.30	.20	.001**

*
 $p < .05$.**
 $p < .001$.

TABLE 3
Bivariate Correlation Analyses Between Negative Affect and Physiological Reactivity for Learners and Nonlearners, Preterm and Full-Term Infants

	Negative Affect							
	Preterm Nonlearners				Preterm Learners			
	Base- Learning	Learning- Nonreinforcement	Base- Learning	Learning- Nonreinforcement	Base- Learning	Learning- Nonreinforcement	Base- Learning	Learning- Nonreinforcement
Heart rate (bpm)								
Baseline- Learning	.14	.01	-.28	-.11	.02	-.02	.30	.63**
Learning- Nonreinforcement	-.25	.10	-.55**	.41*	-.12	-.03	.10	.18
Respiratory sinus arrhythmia (ln/msec)								
Baseline- Learning	.08	-.12	-.16	.01	-.03	-.18	-.13	-.26
Learning- Nonreinforcement	.50*	.20	.62**	-.26	.08	-.20	.03	-.05
Cortisol (ug/dl)								
Pre-post	.07	-.09	-.51**	-.12	-.19	-.20	-.04	.18

* $p < .05$.

** $p < .001$.