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The Effects of Noise on Neonates in the NICU

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ABSTRACT *Preterm infants in the neonatal intensive care unit (NICU) are constantly exposed to ambient noise that often exceeds recommended levels. There is a growing concern that such noise puts preterm infants at high risk for adverse health effects. This review looks at the effects of NICU noise on the cardiovascular, respiratory, auditory and nervous systems. Loud transient noise has negative short-term effects on the cardiovascular and respiratory systems of preterm infants, although direct evidence linking noise to neonatal pathology is still unclear. Further controlled trials with larger sample sizes are needed to determine the effects of more extensive exposure to NICU noise on early brain maturation and long-term developmental outcomes.*

BACKGROUND

In the past two decades, the survival rate of very low birth weight (VLBW) infants has dramatically improved. However, as more of these preterm infants are reaching school age, the high incidence of neurodevelopmental problems is becoming more apparent. There is growing concern that such problems may in part stem from an un favorable neonatal intensive care unit (NICU) environment, particularly excessive noise exposure.

The NICU is often characterised by loud, unpredictable noise from extraneous sources such as alarms, ventilators, phones and staff conversation to which preterm infants are especially vulnerable. In addition, the self-generated sound of infant crying can be a significant source of noise as loud sounds tend to be amplified within the incubator. In 1997, the American Academy of Pediatrics determined that safe sound levels in the NICU should not exceed an hourly level of 45 decibels on an A-weighted scale (dBA). It is well established that noise levels in the NICU often exceed these recommendations, potentially resulting in numerous adverse noise induced health effects. However, the effects of excessive noise exposure on the brain and long-term developmental outcomes are not well established.

Preterm infants have decreased autonomic and self-regulatory abilities, and are vulnerable to high noise levels due to their inability to filter and process noxious stimuli. By 26–28 weeks gestational age (GA), the preterm infant's auditory system is sufficiently mature for loud noise to produce physiological changes in heart rate, blood pressure, respiration and oxygenation. Therefore, maintaining a stable physiological state is crucial, especially during this critical time for development of the central nervous system (CNS) when the most rapid neural formation is taking place.

EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM

Heart rate

One of the earlier studies in the field looked at 60 preterm infants (28–32 weeks GA) randomly assigned to an experimental or control group. The experimental group was exposed to a recording of their mother's voice playing for 30 min daily throughout their hospitalisation. At 36 weeks corrected gestational age (CGA), all infants were exposed to 10 s of an 85 dB noise in a drowsy state and a 30 s recording of a female's voice in an active crying state. In the drowsy state, the noise stimulus elicited heart rate acceleration in both groups ($p<0.01$). However, in the crying state, the noise stimulus elicited heart rate deceleration, which was more pronounced in the experimental group ($p<0.01$). Results suggest that an infant's response to noise may not only be dependent on the infant's behavioural state but also on prior exposure to sounds.

Another early study exposed two preterm infants (34–35 weeks GA) to sudden loud NICU noise at 70–75 dBA caused by doors closing, diaper pails and staff conversation. Although all of these environmental stimuli were associated with a transient increase in heart rate, the small sample size of this study clearly limits its applicability. Zahr and Balian looked at 55 preterm infants (23–37 weeks GA) and their heart rate responses to nursing interventions and environmental noise. Sound levels were not measured; a bedside observer recorded any loud noises and the infant's response. Although a slight increase in heart rate was found in response to all the noise stimuli, these results were not statistically significant. The lack of sound level measurements and the extreme variability in gestational age and age at testing limit the impact of this study. Schulman looked at 31 infants (29–40 weeks GA) at high and low risk for neurological damage. All infants were exposed to an 80 dB low frequency buzzer for 3 s in either a quiet alert or quiet asleep state. The high risk infants had heart rate acceleration in both states. However, the low-risk infants had heart rate acceleration only in the quiet sleep state. Thus, both the behavioural state of the infant and predisposing conditions of the CNS affect the cardiac response. However, this study included both preterm and full-term infants, with significant variation in the age at testing. Williams *et al* looked at the effects of noise on 11 extremely low birth weight (ELBW) infants (<1000 g) during the first week of life. All infants were exposed to routine incubator noise at baseline levels of 50–60 dBA over a 2 h period. The lower birth weight infants (454–694 g) responded with an increase in heart rate to loud noises. However, the higher birth weight infants (766–910 g) experienced a bi-phasic heart rate response, showing an initial decrease in heart rate followed by heart rate acceleration.

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The authors concluded that the higher birth weight newborns had more mature CNS control over heart rate.

Blood pressure

Jurkovicova and Aghova exposed 30 low birth weight infants (mean 1613 g) to a 63–250 Hz (79–85 dBA) low frequency noise (generated by the closing of incubator doors) five times over a 30 s interval. The infants were also exposed to 30 s of high frequency 4000 Hz continuous noise from the incubator alarm system. In 85% of infants, all noise stimuli resulted in a 10 mm Hg increase in systolic blood pressure by and a 9 mm Hg increase in diastolic blood pressure, both of which returned to baseline after 5 min. However, all of the blood pressure measurements were within the normal range. Williams *et al* looked at the effect of noise on mean arterial blood pressure (MABP) in 11 ELBW infants (401–1000 g) during the first week of life. Infants were exposed to baseline levels of 50–60 dBA incubator noise over a 2 h period. The higher birth weight infants demonstrated a bi-phasic blood pressure response to noise; they first responded with a decrease in blood pressure followed by an increase. The lower birth weight infants also experienced some decreases in blood pressure after the noise stimulus. The correlations between heart rate and MABP were stronger in higher birth weight newborns. However, none of these blood pressure changes were statistically significant.

EFFECTS OF NOISE ON THE RESPIRATORY SYSTEM

Unlike the cardiovascular system, there has been limited investigation into the effects of noise on the respiratory system, and results are varied and inconsistent. Wharrad and Davis compared the respiratory rate response of 22 full-term and 20 preterm infants (mean 32 weeks GA) to a 5 s white noise stimulus of 80, 90 or 100 dBA versus no stimulus. Decreased respiratory rate was observed in response to the acoustic stimulus in all infants, with respiratory rate decreasing more as the intensity of the noise increased. The results were statistically significant only for the preterm infants with the 100 dBA stimulus ($p<0.05$). In another study, 65 preterm infants (26–32 weeks GA) were evaluated. Sound levels, oxygen saturation and infant states were recorded in a pre-study state with the infant in the incubator, in a study state with the infant in the incubator with acoustic foam in place, and in a post-study state with the acoustic foam removed. With the foam in place, there was an average decrease in noise levels of 3.27 dBA. Oxygenation improved by more than 1% for all infants with the acoustic foam, and was sustained for 10 min following removal of the foam ($p<0.01$).25 However, this could be due to normal fluctuations in oxygen saturation and the fact that the majority of these infants were on supplemental oxygen therapy. Zahr and Balian looked at 55 preterm infants between 23 and 37 weeks GA, exposing them to common NICU environmental noises such as alarms, phones and loud conversations. Average oxygen saturations were significantly lower during noisy periods (90% vs 93%; $p<0.01$). There was no significant change in respiratory rate with noise stimuli. However, the significant variation in gestational ages and age at testing, baseline oxygen requirement and underlying lung disease, and the overall medical stability of the infants were not accounted for.

EFFECTS OF NOISE ON INFANT SLEEP

Sleep is important for healthy neurodevelopment, and disruption of normal sleep cycles may be detrimental. The adverse effects of noise on sleep patterns have been primarily illustrated in full-term rather than in preterm infants. One of the few studies in this area looked at the sleep patterns of 55 preterm infants (23–37 weeks GA) in response to NICU environmental noise, including loud conversations, alarms, phones and infant crying. The Anderson Behavioral Scale was used to assess the infant's behavioural and sleep states. Results indicated that 43% of the preterm infants were negatively affected by the noise, going from a sleep state to fussy or crying. Strauch also evaluated six preterm infants before and after implementation of a NICU quiet hour protocol. Sounds levels were on average 5.5 dB lower during the quiet hour when compared with control conditions ($p<0.0005$). During the quiet hour, 84.5% of the infants were in a light/deep sleep compared with 33.9% in the control setting ($p<0.0005$).

EFFECTS OF NOISE ON BRAIN PERFUSION

One case report looking at two preterm infants (34–35 weeks GA) showed that sudden loud noise in the NICU can lead to agitation, crying, hypoxia and subsequently to an increase in intracranial pressure (ICP). However, the clinical significance of this ICP increase is uncertain. There have been no further studies to confirm this anecdotal observation or to more rigorously examine the question of whether or not excessive noise exposure directly contributes to the development of intracranial hemorrhages. The negative impact of noise on apnoea, heart rate and hypoxia may subsequently lead to decreased perfusion of critical brain tissue; however, this direct effect of noise has not yet been evaluated.

CONCLUSIONS

This review suggests that elevated noise levels in the NICU have potentially adverse effects on infant physiologic stability and future neurodevelopment. Loud transient noise has been shown to cause immediate physiological changes, such as increased heart rate, blood pressure and respiratory rate, and decreased oxygen saturation. Such changes increase the likelihood of subsequent apnoea and bradycardia episodes. The existing evidence regarding the effects of noise on NICU patients does not yet allow us to make definitive conclusions. Many of the studies in this area of research are limited due to lack of randomisation, small sample sizes and large variations in the gestational ages of the infants and the experimental techniques used. In addition, the chronological ages of the infants at testing varied significantly, and confounding medical problems were not adequately accounted for in most of the studies. There is also a lack of recent studies in this field, with the majority of evidence coming from research performed decades ago. Further research and more rigorous clinical investigations are needed to directly link noise exposure to pathology and to examine its effects on early brain development and long-term developmental outcome**.** Maintaining safe noise levels within the NICU will enhance the capacity of critically ill infants to cope with the unexpected transition from the protective uterine environment to the overwhelming world of the NICU.

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