

The Effect of Ambient Noise in the NICU on Cerebral Oxygenation in Preterm Neonates on High Flow Oxygen Therapy

Kramarić, Karolina; Šapina, Matej; Milas, Vesna; Milas, Krešimir; Dorner, Sanja; Varžić, Darije; Šerfezi, Josip; Adelson, Phillip David

Source / Izvornik: **Signa vitae : journal for intensive care and emergency medicine, 2017, 13, 52 - 56**

Journal article, Published version

Rad u časopisu, Objavljena verzija rada (izdavačev PDF)

<https://doi.org/10.22514/SV133.062017.11>

Permanent link / Trajna poveznica: <https://um.nsk.hr/um:nbn:hr:239:481455>

Rights / Prava: [Attribution 4.0 International](#)/[Imenovanje 4.0 međunarodna](#)

Download date / Datum preuzimanja: **2024-11-23**



Repository / Repozitorij:

[Repository UHC Osijek - Repository University Hospital Centre Osijek](#)

The effect of ambient noise in the NICU on cerebral oxygenation in preterm neonates on high flow oxygen therapy

KAROLINA KRAMARIĆ^{1,2}, MATEJ ŠAPINA^{1,2}, VESNA MILAS^{1,2}, KREŠIMIR MILAS^{1,2}, SANJA DORNER¹, DARIJE VARŽIĆ³, JOSIP ŠERFEZI³, PHILLIP DAVID ADELSON^{4,5,6,7,8,9,10}

1 Paediatric Clinics, University hospital Osijek, J. Huttlera 4, Osijek, Croatia

2 Medical Faculty Osijek, Osijek, Croatia, Cara Hadrijana 11a, Osijek, Croatia

3 Institute for Advancement of Safety, Trg Lava Mirskog 3, Osijek, Croatia

4 Director, Barrow Neurological Institute at Phoenix Children's Hospital

5 Diane and Bruce Halle Endowed Chair for Paediatric Neurosciences

6 Chief, Paediatric Neurosurgery /Children's Neurosciences

7 Professor and Chief, Neurological Surgery, Department of Child Health, University of Arizona, College of Medicine, Phoenix

8 Professor, Mayo Clinic Medical School

9 Adjunct Professor, Ira A. Fulton School of Biological and Health Systems Engineering, Arizona State University

10 Paediatric Neurosurgery Fellowship Program (Director)/ Barrow Neurological Institute

Corresponding author:

Karolina Kramarić

Cara Hadrijana 11a

31 000 Osijek, Croatia

Phone: 00 385 91 470 40 20

E-mail: karolina.kramaric@gmail.com

ABSTRACT

In this early pilot study, we sought to determine if the alteration in these physiologic effects in premature infants in response to ambient noise in the NICU could be assessed evaluating cerebral blood saturation. Three premature infants, on high flow nasal cannula oxygen support (HFNC), at less than 34 weeks of gestation were included in the study. Three variables were used to evaluate sound levels due to AAP and EPA guidelines; Leq,1h, L10,1h and Lmax,1min.

All of the patients studied were found to be exposed to statistically significant noise levels (above recommendation) throughout all of the time periods measured. Noise levels were found to be similarly elevated during the 1 am and 3 pm time periods as well, though not as much as compared to the 7 am measure. A statistically significant difference was found within every patient's rSO₂ levels in both hemispheres, but also in the absolute differences of rSO₂. Positive significant statistical correlations were found between the average rSO₂ and Leq,1h ($\rho=0.14$), Lmax,1min ($\rho=0.18$), L10,1h ($\rho=0.15$). Significant negative correlations were found between the absolute difference levels and Lmax,1min ($\rho=-0.3$), and L10,1h ($\rho=-0.18$)

This data highlights the need for further study as to the potential impact of noise

on the cerebral physiology of premature infants. Further research is needed to assess the potential long-term side effects of environmental noise on the premature infant's brain.

Key words: noise, infant, new-born, neonatology, noise measurement

INTRODUCTION

Throughout their lifetimes, humans interact with their environment. The in utero foetus is well protected by the womb and experiences low frequency sounds (as the womb has a protective and adaptive function), meaning full term infants have the opportunity and possibility to identify the maternal voice against background sounds. In such a nurturing and confined environment, they achieve proper neurosensory development. (1) In contrast, the premature infant, prior to 37 weeks of gestational age, spends their first months in the Neonatal Intensive Care Unit (NICU) environment, which is often a harsh, invasive and noisy environment at a time of physiological limitation and central nervous system immaturity, making them especially vulnerable to NICU environmental stress that can often continue. (2) They also have an increased need for long term intensive care, which makes them more sensitive to

the negative effects of the NICU. (1)

According to its definition, noise is undesirable sound and sound is vibration in a medium, usually air. When these vibrations occur at high levels sound may become harmful. "Level of sound are characterized by intensity, loudness, frequency, periodicity and duration; the loudness of sound can be measured in units called decibels (dB)." (1) The United States (US) Environmental Protection Agency (EPA) recommends sound levels for hospital environments at a maximum of 45 dB during daytime hours and 35 dB during night time hours. Additionally, to protect the wellbeing and health in hospitals, the American Academy of Pediatrics (AAP) Sound Study Group established criteria for noise in the NICU, recommending that overall continuous sound in bed space and patient care area should not exceed an hourly loudness equivalent (Leq,1h) of 45 dB(A), hourly sound levels should not exceed 50 dB(A) 10% of the time (L10,1h) and should sound not exceed a one-second maximum level (Lmax,1min) of 65 dB(A) [3]. "These recommendation have been reviewed and endorsed by the Council of International Neonatal Nurses as well as the Institute for Family Centred Care." (1)

Despite these recommendations, previous research has shown that NICUs and Intensive Care Units sound levels frequently exceed the EPA and AAP recommended

guidelines. (4) Vital sign monitors, oxygen supply, infusion pumps, suction equipment, ventilators and even incubators all are sources of noise, contributing to increased sound levels. Additionally, human factors such as visitors, parents, medical staff, nursing staff, other members of the healthcare team, noisy alarms, telephones and mobile phones all contribute to ambient noise levels. Previous studies have shown that conversations near incubators, patient areas, entrances to the units and computer areas are the most likely places of high sound levels. During physician rounds, nurse reports and visitations people are always engaged in conversation, which contributes to making noise, so those are the time periods when we can expect increasing noise levels. (5) Incubators provide many positive attributes but also can be a source of noise; if they are used as a surface for writing, an object striking plastic material, or carelessly closing incubator doors can all create sound levels in excess of 100 dB. Unfortunately, many NICUs are not designed for noise reduction due to a lack of space and crowding, ventilation systems, and buffering wall and floor coverings.

Negative effects of increased sound levels can have long implications for the neonate, especially since the capability of shutting off of environmental stimuli differs between premature and full-term infants. Previous research studies have shown that premature infants change their behavioural state in response to noise. Environmental noise can cause agitation and be a major source of stressful stimulations. (6) The preterm infant is more likely to abrupt fluctuation of blood pressure, heart rate, respirations, oxygen saturation, decreases in cerebral oxygenation, going from sleep to wake, periods of apnoea. (7) Noise also can contribute to cochlear damage, language and hearing delays. Often these conditions result in behavioural changes that can be detected long after discharge as developmental or neurological delays. (8) Near infrared spectroscopy (NIRS) is a non-invasive technique relying on the relative tissue transparency to near-infrared light. It is used to assess microcirculatory oxygenation through the measurement of oxyhaemoglobin and de-oxyhaemoglobin concentrations. Cerebral oximetry is based on NIRS technology to monitor regional cerebral oxygen saturation (rSO₂). (9, 10) Although there is a similarity to pulse oximetry, cerebral oximetry monitors measure the nonpulsatile signal component reflecting tissue circulation. (11) A great advantage of NIRS monitoring is in pro-

ducing real time data at the bedside, which allow continuous monitoring of the changes in cerebral oxygenation. In the paediatric population, a promising use has been found in various surgical and anaesthesiology procedures, monitoring neurodevelopmental outcomes etc. (12-16)

In this early pilot study, we sought to determine if the alteration in these physiologic effects in premature infants in response to ambient noise in the NICU could be assessed evaluating cerebral blood saturation.

The aims of the study are stated in the following hypotheses:

1. Premature infants are exposed to elevated levels of noise beyond standard recommendations
2. Elevated noise levels in the NICU will result in changes in rSO₂, indicating increased stress to the child

MATERIALS AND METHODS

Our research was accepted by our institution's ethical committee, and informed consents were obtained from all research participant's parents or guardians.

This pilot study is a part of an ongoing larger project on the effect of noise on premature infants. Three male premature infants, on high flow nasal cannula oxygen support (HFNC), born with less than 34 weeks of gestation were included in the study. A brain ultrasound excluded congenital brain malformations. The first subject was a premature infant born with 29+5 weeks of gestation, birth weight 1,395 g and APGAR 10/10. The other two were twins, gestational age of 33 weeks, APGAR score 6/6, birth weight 2,466 g, and 1,994 g, APGAR score 2/6, respectively. The study was performed in the NICU of the Paediatric Clinic, University Hospital in Osijek. All infants were in the same incubator model Dräger Caleo.

Sound was measured using sound meter Bruel&Kjaer, Denmark, model 2250 L. The instrument was certified to meet the specification of the manufacturer and was calibrated in a controlled environment using a standard with values traceable to the National Institute of Standards. The sampling rate of noise collection was 10 seconds, which were further averaged to achieve one minute data. Readings were taken using A-weighted levels and slow meter responses, a procedure that filters sound according to the frequency response of a human ear, which helps the sound meter recording to map more closely the sound

pressure.

The study protocol required that sound levels be measured for 24 hours. For this study purpose, three different time intervals lasting one hour were chosen for analysis. The first time period was the one with the most intensive work, at the beginning of the day shift, between 7 AM and 8 AM. The second one during the early afternoon between 3 PM and 4 PM, and the third one during the night hours between 1 AM and 2 AM.

Three variables were used to evaluate sound levels due to AAP and EPA guidelines. The first variable Leq,1h, measures the steady state dB(A) noise during a 1-hour period. Leq,1h is the measure for evaluating the average sound level. L10,1h measures the dB(A) sound level that is exceeded 10 percent of the time over the upper limit of the set range during a 24-hour period. Lmax,1min measures the maximum sound level of one second duration during a one minute period.

To monitor the cerebral oxygenation, a INVOS 5100c (INVOS oximeter, Covidien Corporation, Boulder, CO) was used. INVOS is device which allows non-invasive monitoring placing sensors to the skin surface. The obtained values were recorded every 6 seconds, which were further averaged for a 1 minute time interval to allow a consistent analysis with the sound levels data.

The data was analysed with the R software (www.r-project.org). The normality of the distributions of numerical variables was assessed using the Kolmogorov Smirnov test. If the data did not meet the assumption of normality, the values were descriptively presented with median and interquartile range. Differences between more groups were assessed with the Friedman's and Kruskal Wallis test, followed by Tukey's post hoc test. The Spearman's rank correlation test was used to test the associations between variables. P values less than 0.05 were considered statistically significant.

RESULTS

The first objective of this research was to assess different noise levels and brain oximetry in premature children according to three different time levels. The results of our findings are summarized in Tables 1. and 2. All of the patients studied were found to be exposed to statistically significant noise levels (above recommendation) throughout all of the time periods measured. The medians of the highest Leq,1h

Table 1.

	Patient 1	p*	Patient 2	p*	Patient 3	p*	All	p†
Leq (dB)	1 a.m. 54.1 (52.5-58)	<0.001	46.8 (46.6-47.7)	<0.001	56.7 (55.1-57.7)	<0.001	53.4 (47.7-56.9)	<0.001
	7 a.m. 55 (53.4-56.4)		57 (54.8-58.5)		57.9 (56.3-59.4)		56.6 (54.3-58.5)	<0.001
	3 p.m. 53.3 (52.1-55.1)		50 (48.7-54.8)		52.9 (51.9-54.9)		52.7 (51.5-55)	<0.001
Lmax (dB)	1 a.m. 83 (76.1-86.6)	<0.001	69.1 (65.3-73.4)	<0.001	83.5 (79.9-87.4)	<0.001	79.2 (71.5-85.3)	<0.001
	7 a.m. 85.6 (80.6-90.9)		88.6 (86-91.5)		89.1 (84.1-92.9)		87.7 (83.1-92.3)	0.016
	3 p.m. 79.4 (75.4-84.1)		78.6 (75-85.3)		76.2 (71.9-80.8)		78.6 (74.1-83.8)	0.001
L10 (dB)	1 a.m. 56.2 (53.1-60.6)	<0.001	47.1 (46.8-48.8)	<0.001	59.7 (56.1-62.3)	<0.001	54.5 (48.8-59.9)	<0.001
	7 a.m. 56.6 (54.9-59.4)		59.8 (57.5-61.7)		60 (58.8-62.7)		59.2 (56.7-61.4)	<0.001
	3 p.m. 55.8 (54.2-58)		51.5 (49.9-55.3)		54.5 (52.5-57.4)		54.5 (52-57.4)	<0.001
Left rSO2 (%)	1 a.m. 79.55 (77.67-84.55)	<0.001	85.6 (83.8-86.4)	<0.001	91.4 (90.6-92.7)	<0.001	86.38 (82.8-90.6)	
	7 a.m. 77.8 (76-81.55)		94.9 (94.3-95)		87.6 (86.5-90.8)		87.6 (81.55-94.3)	
	3 p.m. 83.7 (80.3-86.2)		93.7 (92.7-94.8)		86.7 (85.9-87.8)		87.3 (85.4-92.7)	
Right rSO2 (%)	1 a.m. 78.9 (77.2-80.9)	<0.001	91.8 (88.3-94)	<0.001	94.8 (94-95)	<0.001	91.7 (80.9-94.7)	
	7 a.m. 78.9 (76.6-79.9)		93.1 (92.3-93.9)		95 (95-95)		93.1 (79.9-95)	
	3 p.m. 89.5 (80.9-90.7)		94.2 (93-95)		94.9 (94.2-95)		93.5 (90.7-95)	
Absolute difference (%)	1 a.m. 0.2 (0.1-1.6)	<0.001	6.1 (4.6-8.27)	<0.001	3.2 (1.8-3.9)	<0.001	3.75 (2-5.9)	
	7 a.m. 2.8 (1.6-4.9)		1.3 (0.8-2.2)		7.4 (4.2-8.5)		1.9 (1.1-4.2)	
	3 p.m. 2.1 (1-3.9)		0.7 (0.1-1.2)		7.4 (6.4-8.4)		4.2 (1-7.3)	
Average rSO2 (%)	1 a.m. 79.05 (77.38-83.28)	<0.001	88.9 (85.95-90.2)	<0.001	93.05 (92.15-93.55)	<0.001	88.97 (83.55-92.15)	<0.001
	7 a.m. 78.45 (76.35-80.7)		93.85 (93.2-94.45)		91.3 (90.75-92.9)		91.3 (80.7-93.6)	<0.001
	3 p.m. 86.9 (80.6-88.2)		93.85 (92.8-94.75)		90.75 (90.1-91.4)		90.75 (88.2-92.9)	<0.001

*within p values, †between p values, Leq – equivalent sound levels, Lmax - maximum sound level of one second duration during one minute period, L10 – sound level that is exceeded 10 percent of the time, rSO2 – regional cerebral blood saturation

(56.6 dB (54.3-58.5)), Lmax,1min (87.7 dB (83.1-92.3)) and L10,1h (59.2 dB (56.7-61.4)) levels were measured at 7 am. Noise levels were found to be similarly elevated during the 1 am and 3 pm time periods as well, though not as much as compared to the 7 am measure. (Table 1.)

Our second objective was to determine the correlation of noise elevations to cerebral physiology as measured by rSO2. A statistically significant difference was found within every patient's rSO2 levels in both hemispheres, but also in the absolute differences of rSO2. When measured together, the lowest median difference levels were 1.9% at 7 am. Respectively, differences in the average rSO2 were found, the lowest were observed at 1 am. (88.97 (83.55-92.15)), and the highest at 3 pm. (91.3% (80.7-93.6)).

Further analysis was to assess the cor-

relations between noise levels and rSO2. Positive significant statistical correlations were found between the average rSO2 and Leq,1h ($\rho=0.14$), Lmax,1min ($\rho=0.18$), L10,1h ($\rho=0.15$). Significant negative correlations were found between the absolute difference levels and Lmax,1min ($\rho=-0.3$), and L10,1h ($\rho=-0.18$). (Table 2.)

DISCUSSION

The aim of an NICU is to provide an environment that replaces the womb as much as possible, and insure optimal conditions for normal development. According to previous research, the sound levels in NICUs are above recommended standards. (17) The purpose of this study was to examine noise levels during day and night shifts, and to evaluate the impact on cerebral

physiology. We hypothesized and found a significant relationship between environmental noise and cerebral oximetry. The main findings of this study would suggest that there is a positive correlation between noise levels and brain oximetry. All variables Leq,1h, L10,1h and Lmax,1min were above recommended standards suggested by the AAP and EPA. We can assume that Leq,1h exceeded the recommended <50 db(A) at 7 am because that is the time of nursing and physician reports. Precedent work monitoring cerebral oxygenation has been done on infants undergoing extracorporeal membranous oxygenation, cardiac surgery and resuscitation. To our best knowledge, this is the very first study relating cerebral oxygenation and environmental noise in the NICU.

Elevated brain oximetry is an indirect measure of increased brain blood flow and

Table 2. Correlation table

	Leq	Lmax	L10	Left rSO2 (%)
Leq	1			
Lmax	0.75*	1		
L10	0.96*	0.68*	1	
Left rSO2 (%)	0.14*	0.21*	0.16*	1
Right rSO2 (%)	0.10*	0.07*	0.09*	0.66*
Absolute difference (%)	-0.19	-0.30*	-0.20*	-0.18*
Average rSO2 (%)	0.14*	0.18*	0.15*	0.96*

Leq – equivalent sound levels, Lmax - maximum sound level of one second duration during one minute period, L10 – sound level that is exceeded 10 percent of the time, rSO2 – regional cerebral blood saturation, *p<0.05

may be an indication of increased stress. Moreover, a change in cerebral oxygenation was shown in the reduction of the absolute differences between the left and right hemispheres where there should be normal variability. (18, 19) Acute exposure to higher noise causes a stress response which increases heart rate, blood pressure and cardiac output. (20 – 22) Premature infants are especially at risk for intraventricular haemorrhage, due to their lack of

cerebral blood flow autoregulation. (23) We found that increased noise levels lead to decreased variability highlighting a worsening of an already compromised autoregulation.

CONCLUSION

Although this research lacks the sample size for a better generalization of our re-

sults, these preliminary findings suggest that more attention may need to be given to noise reduction in the NICU. This data highlights the need for further study as to the potential impact of noise on the cerebral physiology of premature infants. Questions still remain and further research is also needed to assess the potential long-term side effects of environmental noise on a premature infant's brain.

REFERENCES

1. Darcy AE, Hancock LE, Ware EJ. A descriptive study of noise in the neonatal intensive care unit ambient levels and perceptions of contributing factors. *Advances in Neonatal Care*. 2008;8(3):165-75.
2. Williams AL, Sanderson M, Lai D, Selwyn BJ, Lasky RE. Intensive care noise and mean arterial blood pressure in extremely low-birth-weight neonates. *American journal of perinatology*. 2009;26(05):323-9.
3. Health CoE. Noise: a hazard for the fetus and newborn. *Pediatrics*. 1997;100(4):724-7.
4. Wachman EM, Lahav A. The effects of noise on preterm infants in the NICU. *Archives of Disease in Childhood-Fetal and Neonatal Edition*. 2011;96(4):F305-F9.
5. Brandon DH, Ryan DJ, Barnes AH. Effect of environmental changes on noise in the NICU. *Advances in Neonatal Care*. 2008;8(5):S5-S10.
6. Maroney DI. Recognizing the potential effect of stress and trauma on premature infants in the NICU: how are outcomes affected? *Journal of Perinatology*. 2003;23(8):679-83.
7. Berg AL, Chavez CT, Serpanos YC. Monitoring noise levels in a tertiary neonatal intensive care unit. *Contemp Issues Commun Sci Disord*. 2010;37(1):69-72.
8. Freudenthal A, Van Stuijvenberg M, Van Goudoever J. A quiet NICU for improved infants' health, development and well-being: a systems approach to reducing noise and auditory alarms. *Cognition, Technology & Work*. 2013;15(3):329-45.
9. McCormick PW, Stewart M, Goetting MG, Dujovny M, Lewis G, Ausman JI. Noninvasive cerebral optical spectroscopy for monitoring cerebral oxygen delivery and hemodynamics. *Critical care medicine*. 1991;19(1):89-97.
10. Jobsis FF. Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. *Science*. 1977;198(4323):1264-7.
11. Tortoriello TA, Stayer SA, Mott AR, Dean McKenzie E, Fraser CD, Andropoulos DB, et al. A noninvasive estimation of mixed venous oxygen saturation using near-infrared spectroscopy by cerebral oximetry in pediatric cardiac surgery patients. *Pediatric Anesthesia*. 2005;15(6):495-503.
12. Sood ED, Benzaquen JS, Davies RR, Woodford E, Pizarro C. Predictive value of perioperative near-infrared spectroscopy for neurodevelopmental outcomes after cardiac surgery in infancy. *The Journal of thoracic and cardiovascular surgery*. 2013;145(2):438-45. e1.
13. Bronicki RA, Checchia PA, Anas NG, Adams GJ, Penny DJ, Bleiweis MS, et al. Cerebral and somatic oxygen saturations after repair of tetralogy of Fallot: Effects of extubation on regional blood flow. *The Annals of thoracic surgery*. 2013;95(2):682-6.
14. Rais-Bahrami K, Rivera O, Short B. Validation of a noninvasive neonatal optical cerebral oximeter in veno-venous ECMO patients with a cephalad catheter. *Journal of Perinatology*. 2006;26(10):628-35.
15. Hoffman GM, Mussatto KM, Brosig CL, Tweddell JS, Ghanayem NS. Cerebral oxygenation and neurodevelopmental outcome in hypoplastic left heart syndrome. *Anesthesiology*. 2008;109(A7):A7.
16. Elser HE, Holditch-Davis D, Levy J, Brandon DH. The effects of environmental noise and infant position on cerebral oxygenation.

Advances in neonatal care: official journal of the National Association of Neonatal Nurses. 2012;12(Suppl 5):S18.

17. Philbin MK, Gray L. Changing levels of quiet in an intensive care nursery. *Journal of Perinatology*. 2002;22(6):455.
18. Geschwind N, Galaburda AM. Cerebral lateralization: Biological mechanisms, associations, and pathology: I. A hypothesis and a program for research. *Archives of neurology*. 1985;42(5):428-59.
19. Perlmutter JS, Powers WJ, Herscovitch P, Fox PT, Raichle ME. Regional asymmetries of cerebral blood flow, blood volume, and oxygen utilization and extraction in normal subjects. *Journal of Cerebral Blood Flow & Metabolism*. 1987;7(1):64-7.
20. Van Kempen EE, Kruize H, Boshuizen HC, Ameling CB, Staatsen BA, de Hollander AE. The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. *Environmental health perspectives*. 2002;110(3):307.
21. Baker CF. Discomfort to environmental noise: heart rate responses of SICU patients. *Critical Care Nursing Quarterly*. 1992;15(2):75.
22. Babisch W. Stress hormones in the research on cardiovascular effects of noise. *Noise and health*. 2003;5(18):1.
23. Vesoulis ZA, Mathur AM. Cerebral Autoregulation, Brain Injury, and the Transitioning Premature Infant. *Frontiers in Pediatrics*. 2017;5