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Maternal sounds elicit lower heart rate in preterm newborns in the first month of life

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ABSTRACT

Background: The preferential response to mother's voice in the fetus and term newborn is well documented. However, the response of preterm neonates is not well understood and more difficult to interpret due to the intensive clinical care and range of medical complications.
Aim: This study examined the physiological response to maternal sounds and its sustainability in the first month of life in infants born very pretermaturely.
Methods: Heart rate changes were monitored in 20 hospitalized preterm infants born between 25 and 32 weeks of gestation during 30-minute exposure vs. non-exposure periods of recorded maternal sounds played inside the incubator. A total of 13,680 min of HR data was sampled throughout the first month of life during gavage feeds with and without exposure to maternal sounds.
Results: During exposure periods, infants had significantly lower heart rate compared to matched periods of care without exposure on the same day ($p < .0001$). This effect was observed in all infants, across the first month of life, irrespective of day of life, gestational age at birth, birth weight, age at testing, Apgar score, caffeine therapy, and requirement for respiratory support. No adverse effects were observed.
Conclusion: Preterm newborns responded to maternal sounds with decreased heart rate throughout the first month of life. It is possible that maternal sounds improve autonomic stability and provide a more relaxing environment for this population of newborns. Further studies are needed to determine the therapeutic implications of maternal sound exposure for optimizing care practices and developmental outcomes.

1. Introduction

The fetus has a substantial capacity for sound recognition and auditory learning in the intra-uterine environment [1]. As an indication of this, newborn infants show a clear preference for their mother's voice shortly after birth [2,3]. The fetus, however, does not begin its auditory experience by hearing sounds, but rather by sensing them through the bones of the skull [4]. Hearing begins at approximately 25–26 weeks of gestational age (GA) as cochlear hair cells first translate acoustic vibrations and then airborne sound stimulation into coded electrical signals that are sent to the brainstem for additional processing [5]. Consistent responses to vibroacoustic stimuli have been observed in the fetus from approximately 27–28 weeks onwards [6], particularly in response to low frequency sounds [7]. These basic auditory skills are known to be a prerequisite for subsequent processing of human speech sounds, beginning with mother's voice.

Fetal response to mother's voice has been mainly identified by measuring heart rate (HR) changes. This response, beginning at 32 weeks of gestational age (GA, has been robustly demonstrated by Kisilevsky and colleagues [8]. Over two minutes of voice exposure, fetuses from 32 to 37 weeks of GA showed an initial HR decrease for 30 s, followed by a HR increase until the end of the stimulus. By the time fetuses reached term age, however, the response shifted to an immediate HR increase which was sustained for the full 2 min of voice exposure [9]. Overall, fetal HR at near-term has been shown to increase in response to the mother's voice and decrease in response to a stranger's voice [10]. Studies have suggested that this preferential response is modulated by HR variability and cardiac vagal tone, reflecting a pre-attentional form of reaction [11]. The neural basis of this response was recently revealed using fMRI in normal pregnancy fetuses at 33–34 weeks' gestation [12].

Pregnancy complications and atypical prenatal development can significantly restrict auditory recognition abilities, dampening the perception of sounds in utero. For example, iron-deficient infants born to diabetic mothers [13] demonstrated shorter event-related potentials (ERP) in response to acoustic stimulation of their mother's voice. Similarly, growth-restricted fetuses and newborns showed significantly

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weaker HR response to their mother's voice compared to healthy controls who were appropriately grown for gestational age [14]. Thus, the integrity of the intra-utero environment seems to be important for securing optimal auditory development.

The prenatal response to mother's voice continues postnatally. Term newborns show perceptual sensitivities in response to familiar speech stimuli [15]. This preferential response has been demonstrated in full-term infants by several measurements, including increased non-nutritive sucking [16] and reactive movement towards to the source of the sound [17]. Interestingly, newborn infants also show a preference to the type of language used (i.e., native vs. foreign) based on their individual language experience in utero [18,19]. Most recently, Beauchemin and colleagues showed that exposure to maternal voice activated language-related cortical areas, whereas a stranger's voice activated more generic voice-specific areas [20]. The authors interpreted their findings as evidence for an innate auditory-motor speech loop, specifically tailored to the mother's voice. Similarly, Partanen and colleagues [21] demonstrated that term newborns react differentially to familiar vs. unfamiliar sounds they were exposed to as fetuses, revealing a direct correlation between the amount of prenatal exposure and brain activity. The above studies suggest that auditory attention, learning, and memory originate before birth to allow proper priming of language centers of the brain.

Whereas the newborn and fetal response to mother's voice has been well studied, the sustainability of this response following a premature birth is not fully understood. Chronologically, a typically-developing fetus at 28 weeks of gestation and an infant born 12 weeks prematurely are exactly of the same age; however, developmentally, they may show very different HR responses when presented with their mother's voice. The ability of preterm newborns to show a preference for their mother's voice while still in the Neonatal Intensive Care Unit (NICU) may be constrained by several factors including their immature nervous system, their hypersensitivity to loud noise, the sudden demands for hearing through air (instead of amniotic fluid), and their limited capacity to hear human speech sounds clearly in the noisy NICU environment. Thus, it is unclear whether HR changes observed in preterm infants are solely indicative of their physiological response to mother's voice or possibly a reflection of cardiorespiratory instability or side-effects of caffeine treatment [22].

A number of studies have examined the impact of maternal voice in the preterm population (for review see [23]); however, only a few studies have specifically utilized HR analysis to measure the infant's response. An early study identified decreased HR in this population with exposure to maternal voice compared to white noise [24]. Recent studies have identified increased HR in response to live maternal speech [25], but no difference in response to recorded maternal speech [26]. However, the response of preterm infants to recordings which include both mother's voice and heartbeat in an attempt to simulate the intra utero environment has not yet been studied. Additionally, the nature of this response in the first critical month of life and the extent to which it might be affected by the infant's age, health status, respiratory support, and caffeine therapy remain unstudied and would be an important contribution to our current understanding. The present study aimed to fill these gaps in knowledge by examining the effects of exposure to mother's voice on HR in hospitalized preterm newborns in the first month of life. It was hypothesized that exposure to mother's voice and heartbeat would result in decreased HR compared to matched periods of care without exposure on the same day.

2. Methods

2.1. Study population

Twenty preterm infants participated in this study. The study was approved by the Institutional Review Board (IRB), and parents gave written informed consent within approximately the first week of life.

Table 1 Population Characteristics.

Total subjects	20
Female sex, %	35
Maternal age (y)[a]	33 ± 5.6 (18–42)
GA at birth (wks)[a]	29 ± 2.4 (25 5/7–32 4/7)
Birth weight (g)[a]	1231 ± 302.4 (700–1710)
Apgar 5 min[a]	8 ± 1.1 (6–9)
PMA at study onset (wks)[a]	30 ± 2.5 (26 2/7–33 4/7)
Required respiratory support attesting, %	63
Full gavage feeding at testing %	75
Caffeine treatment, %[b]	90

[a] Values are shown as Mean ± SD (range).
[b] Average dose of 6.38 mg/kg/day in the first month of life.

The mean gestational age at birth was 29 weeks ($SD = 2.4$) and the mean post menstrual age (PMA) at study onset was 30 weeks ($SD = 2.5$). A detailed description of the study population is given in Table 1. Inclusion criteria included: birth GA between 25 and 32 weeks. Exclusion criteria included: chromosomal anomalies;; major congenital anomalies; symptomatic infections; congenital hearing loss; perinatal brain lesions; small for gestational age; anemia of prematurity (Hgb ≤10 g/dL); history of significant maternal deprivation, abuse or malnutrition; history of maternal alcoholism or use of illicit drugs; and smoking during pregnancy in light of evidence for impaired auditory discrimination of speech sounds in infants of smokers mothers [27]. All infants passed their hearing test prior to NICU discharge.

2.2. Maternal sound recording

Mother's voice and heartbeat were recorded individually for each infant in a specialized recording studio. Voice recording was done in a standardized fashion via a large-diaphragm condenser microphone (KSM44, Shure, USA), capturing three types of vocalizations (speaking, reading, and singing) from each mother. The maternal recordings were attenuated using a low-pass filter with a cutoff of 400 Hz, and were subsequently mixed with individualized recording of the mother's heartbeat via a digital stethoscope (ds32a, Thinklabs Digital Stethoscopes, USA). The maternal voice was overlaid with the recording of the maternal heartbeat so that the infant could hear them simultaneously. This was done in an attempt to simulate the auditory experience in utero. The maternal recordings were loaded onto an MP3 player (Phillips Electronics, SA2RGA04KS, Netherlands) for playback via microaudio speakers at the bedside. Maternal sounds were played at a mean LAeq of 57.2 ± 3.4 dBA (A-weighted). Loud peaks were attenuated to achieve a safe level of sound delivery <65 dBA to approximate normal human conversation [28] as would otherwise occur when a mother speaks to her infant at the bedside. This sound attenuation protocol was administered individually for each infant by a sound level meter (Bruel & Kjaer, 2250, Denmark) as validated in a previous safety and feasibility study [29] and was successfully used in recent studies from our group [30,31].

2.3. Study procedure

Nurses were instructed to coordinate the maternal sounds with the infant's routine care, 4×/day, avoiding playing the sounds during parental visits and clinical exams. The exact time maternal sounds were played was denoted by nurses on a study timesheet at the bedside. Maternal sounds were always played after the care session, when the infant was tucked in and put to sleep, as gavage feeding was initiated. HR data was collected from the infants' cardiac monitor four times a day, twice a week, over a 30-minute period during two feeds *with* and two feeds *without* exposure to maternal sounds on the same day. This approach allowed us to compare the infant's HR during clinically-comparable periods in the infant's NICU routines, resulting in a total of 24 data collection sessions per infant throughout the first month of life. Analysis was based on 13,680 min of data, with 720 data points per infant (three infants had missing data contributing 480 data points