

Sleep disruption due to hospital noises

Jeffrey M. Ellenbogen*^{1,2}, Orfeu M. Buxton *^{1,3}, Wei Wang^{1,3}, Andy Carballeira^{4,5}, Shawn O'Connor³, Dan Cooper³, Scott M. McKinney², Jo M. Solet^{1,6}

- Division of Sleep Medicine, Harvard Medical School, 221 Longwood Avenue, Boston, MA 02115
- Department of Neurology, Massachusetts General Hospital, 22 Fruit Street, Boston, MA 02114
- Department of Medicine, Brigham and Women's Hospital, 221 Longwood Avenue, Boston, MA 02115
- Cavanaugh Tocci Associates, 327F Boston Post Road, Sudbury, MA 01776
- Berkelee School of Music, 1140 Boylston Street, Boston, MA 02215
- Department of Medicine, Cambridge Health Alliance, 1493 Cambridge Street, Cambridge MA 02139
- * jeffrey_ellenbogen@hms.harvard.edu

ABSTRACT

Background: Sleep plays a critical role in maintaining health and well-being. Yet hospitalized patients are frequently exposed to noise that can disrupt sleep. Efforts to attenuate hospital noise have been limited by incomplete information on the interaction between sounds and sleep physiology. Objective: We examined the cortical (electroencephalographic; EEG) arousal responses during sleep to typical hospital noises by sound type, sleep stage, and sound level in order to determine profiles of acoustic disruption of sleep. Design: Three-day polysomnographic study with a baseline (sham) night followed by two intervention nights. Settings: Soundattenuated sleep laboratory. Participants: Volunteer sample of 12 healthy subjects. Intervention: We systematically administered 14 recorded hospital sounds at calibrated, rising decibel levels (total=3,147) during specific sleep stages. Measurements: EEG arousals, using established criteria, during Rapid Eye Movement (REM) sleep, and non-REM stages 2 (N2) and 3 (N3). Results: Sound presentations yielded arousal response curves that varied due to sound level, sound type, and sleep stage. Higher sound levels led to a greater probability of sleep disruption. Electronic sounds tested were more arousing than other sounds, including human voices. The effects of sound level and sound type were modified by sleep physiology, producing unique arousal probability profiles in each sleep stage. In REM sleep, the range of responses across sound types was narrowest, suggesting less differentiation of sounds. Limitations: Results in healthy people may underestimate effects of noise in hospitalized patients. Conclusions: This study systematically quantifies the disruptive capacity of sounds on sleep. These arousal-probability profiles can be expected to drive innovation in design, construction, engineering, building materials, equipment and care-giving protocols, with the goal of providing environments conductive for sleep among hospitalized patients.

INTRODUCTION

Sleep is essential for the restoration of health and well-being (Institute of Medicine 2006). However, in hospitals, where healing is paramount, noise frequently disrupts patients' sleep. In a recent national survey, patients identified the noise levels around rooms at night as the quality of care factor with the most room for improvement (Jha et al. 2008). Acoustic measurements from a major urban hospital document a crescendo of nighttime hospital noise over the last 45 years from an average level of 42 decibels to over 55 decibels in 2005 (Busch-Vishniac et al. 2005). Hospitals are exposed to external noise sources such as traffic and airplane sounds known to disrupt

sleep (Griefahn et al. 2008) with documented dose-related consequences for next day cognitive performance (Elmenhorst et al. 2010). Patient care also produces noise specific to treatment and protection, such as IV and cardiac monitor alarm signals. Improving acoustics in environments of care to protect sleep and enhance outcomes for the more than 37 million patients admitted annually to hospitals in the United States (Carpenter 2010) has become a trans-disciplinary priority (Facility Guidelines Institute 2010; Basner et al. 2006; Bartick et al. 2009; Montague et al. 2009).

The goal of this study was to provide essential information about the impact of sound on sleep to guide architectural, technological and programmatic advances to facilitate sleep for hospitalized patients. We tested the hypothesis that rising levels of hospital sounds increasingly disrupt sleep with responses differing by sleep stage. We designed a protocol exposing subjects to hospital noises and using polysomnographic assessments of cortical arousals, a standard (Bonnet & Arand 2007; Basner 2010; Kohlschütter 1863) and sensitive (Basner et al. 2008; Halasz et al. 2004) measure of sleep disruption due to nighttime noise strongly correlated with awakenings (Basner et al. 2008). These data function as a cornerstone for positive interventions across three potential targets for improving sleep among hospitalized patients: sound level, sound stimulus types, and sleep physiology.

METHODS

Pre-study conditions

Healthy, young subjects were recruited by flyers, website postings, and word of mouth then screened by questionnaire, physical exam, and laboratory tests. Subjects slept at home on a regular schedule for at least four days prior to their participation, confirmed by wrist actigraphy.

Sleep laboratory conditions

Participants stayed at the MGH Sleep Laboratory for three days. On the second and third nights, sounds were presented throughout sleep, as described below. Sleep stages and arousals were identified—by sleep technician and physician—using current criteria (lber et al. 2007).

Acoustic stimuli

Recordings of hospital sounds were captured at a medical unit of Somerville Hospital, Somerville, Massachusetts. Each sound stimulus corresponds to a specific category (external to building, within hospital and within or outside patient room) identified as salient in the AIA Guideline on Sound and Vibration in Healthcare Facilities (Facility Guidelines Institute 2010). Sounds were normalized to 10 seconds in duration.

Sound presentation

Hospital noises were presented with three-dimensional verisimilitude (e.g., airplane sounds move across space) using four studio monitor loudspeakers. Once steady sleep stage was reached—assessed in real-time by a technician—noises were presented starting at 40 $L_{AEQ,10\text{-sec.}}$ (dB) in steps of 5 $L_{AEQ,10\text{-sec.}}$ (dB) until sleep was disrupted by an arousal or 70 $L_{AEQ,10\text{-sec.}}$ (dB) was reached. Because both the integrated equivalent sound level and the length of the noise were held constant, all stimuli were effectively normalized for their "noise dose", an integration of sound intensity over time (De Gennaro et al. 1995). Stimulus trains were presented on a background of

measured average of 34-35 $L_{AEQ,10\text{-sec.}}$ (dB) due to continuous air flow (as required in a hospital setting). During sound stimulus presentation, the sound level in the patient room was logged in 1-second increments using an environmental sound monitor (Rion NL-31, with Type 1 microphone) installed roughly 10 inches above the head of the sleeping participants, and programmed to output a DC voltage proportional to the A-weighted fast response sound level ($L_{A10,10\text{-sec.}}$ (dB).

Polysomnography was examined by a certified sleep technician and physician for the classification of sleep stages, and the identification of cortical arousals indicating a disruption of sleep quality and continuity (Bonnet et al. 2007).

Statistical analysis

Generalized linear mixed models evaluated the effects of hospital noises on probability of sleep disruption. Four separate models are shown for sounds presented during stages N2, N3, REM and all of these stages combined. Fixed factors included stimulus type, sound level, and sleep stage. Subject was treated as a random effect, incorporating subject-specific intercepts into the model. We assumed a piecewise linear relationship between adjacent stimuli levels and the resultant arousal probabilities. To adjust for two ears exposed versus either ear against the pillow, supine position (two ears exposed) served as the referent category. The model was organized to render the results more accessible by comparing all stimuli to the human voice; the disruptive impact of any particular sound (i.e., the regression coefficient associated with that stimulus) was presented in reference to the combined results of the three vocal stimuli.

RESULTS

Twelve healthy subjects (7F, mean age±SD of 27±7 years) successfully completed this study in which hospital noises were systematically presented during sleep to quantify the disruptive effects of noises on sleep.

We observed a main effect of sound level on arousal probability: louder sounds were more apt to lead to sleep disruption. This effect was modified by the type of sound presented (e.g., alarm compared to closing door) and the stage of sleep during which the sound occurred (e.g., REM compared to stage N3).

The arousal probability curve segments significantly differ from a line with a slope=0 (p<0.05) with the exception of the segment spanning 70 $L_{A10,10\text{-sec.}}$ (dB). We observed a significant effect of sleep stage on noise-evoked arousal probability (Figure 1). Separate model results are shown in Table 1 for stimuli presented during non-REM stages N2 and N3, and REM sleep, and all stages of sleep combined.

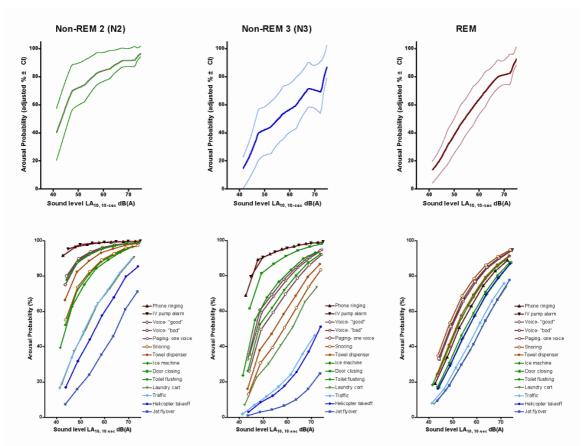


Figure 1: Sleep disruption due to noise stimuli presented during sleep, by stage of sleep Top row is overall effect, bottom row depicts each sound individually.

Table 1: Multiple regression models comparing the effect of different hospital noises on EEG arousal probabilities. Positive coefficients are more likely to cause EEG arousals than voice stimuli (the reference level), whereas negative coefficients reflect stimuli less likely to cause EEG arousals than voice stimuli. Significant differences are in bold. *p<0.01; ***p<0.001; ***p<0.0001

Acoustic stimulus type	stage non- REM2 stimuli only	stage non- REM3 stimuli only	stage REM stimuli only	all stages combined
	β	β		β
IV pump alarm	1.60***	1.90***	0.09	0.95***
Phone ringing	1.37***	1.91***	-0.37	0.69***
Voice	Reference level			
Door closing	-0.07	1.17**	-0.76***	-0.12***
Towel dispenser	-0.49*	-0.75**	-0.17	-0.38**
Snoring	-1.03***	-1.09***	0.27	-0.57***
Ice machine	-1.23***	0.28	-0.23	-0.65***
Toilet flush	-1.11***	-0.16	-0.64**	-0.66***
Laundry cart	-2.41***	-1.37***	-1.35***	-1.50***
Traffic	-2.37***	-2.47***	-1.15***	-1.77***
Helicopter	-2.84***	-2.65***	-0.75***	-2.15***
Jet plane	-3.72***	-3.77***	-1.35***	-2.43***

DISCUSSION

Mitigating noise for hospitalized patients

Approaches to mitigating noise for sleeping patients can be grouped into three broad categories: Eliminating or controlling the source, blocking the sound path, and shifting subjects' awareness. Efforts to control sound at the source include public policy restrictions on acceptable night noise such as aircraft over-flights (Basner et al. 2006), as well as substitution of quieter technologies, for example, PDA's for eliminating overhead paging, and telemetry from nurses' station for staff intrusive oversight (Montague et al. 2009; Kahn et al. 1998). One hospital intervention study that included altered night care routines reduced sound levels from staff voices and patient awakenings, resulting in approximately 25 % reduction of unit-wide sedative medication use (Bartick et al. 2009).

The second approach to mitigating noise focuses on blocking the *path* of the sound or attenuating or masking sound waves before they reach patients. Examples range from design configurations, advanced construction materials (Berg 2001), closing doors and supplying earplugs that block sounds. For example, an intervention study combining alterations to nighttime nursing care routines lead to reduced nighttime sound levels and improved patient satisfaction (Cmiel et al. 2004). We expect these interventions to have direct benefit for sleep.

The third approach to mitigate noises from disrupting a sleeping patient attempts to attenuate noise *perception* by the patient. In this situation, the sound exists, and the barriers of the sound to reaching a sleeping person are limited. And so the focus becomes attempts to alter the brain itself by creating physiologic states in which sound is less apt to be perceived. This can be achieved, for example, through inducing physiological brain states that are less predisposed to disruption. A recent study demonstrated that the presence of sleep spindles indicates the brain's capacity to block sound perception, possibly through a gating mechanism in the thalamus (Dang-Vu et al. 2010). A device or pharmaceutical that could enhance sleep spindles, thereby altering the brain's response to sound could be sleep-protective in noisy environments. Further, because our data demonstrate that individuals in stage N3 sleep are the least likely to experience sound-evoked disruption, innovations that can enhance amount of sleep time spent in N3 might similarly be protective.

These findings point to three critical factors that are important when assessing the detrimental consequences of noises for sleep: the types of sounds, the sound levels, and patient sleep physiology. These factors can be weighed against each other when considering anticipated functional programs and clinical populations served, architectural and requirements such as site-specific building urban building envelopes, codes and local policies, and cost constraints. Improved acoustic environments directed specifically toward protecting sleep, consistent with current guidelines in the United States (Facility Guidelines Institute 2010) and European Union (WHO 2009) should produce multiple benefits for patients including reduced over-sedation, shorter hospital stays, and improved clinical outcomes (Johnson 2003; Cmiel et al. 2004; Hagerman et al. 2005; Basner et al. 2006, 2008; Griefahn et al. 2008; Bartick et al. 2009), and these should be reflected in enhanced patient satisfaction ratings.

The effect of noise is influence by the type of sound:

As expected, the most potent disruptors of sleep were electronic sounds intentionally designed to be alerting (Bruck et al. 2009; McNeer et al. 2007). Inspection of the arousal probability curves in Figure 1 corresponding to these sounds (i.e., phone and IV alarm) reveals that these devices might not be suitably attenuated to spare sleep when set to a 'quiet' setting: even within their lowest audible ranges in this study, they produce insults to sleep with more than 50 % probability. Instead, these sounds could be managed by better targeting intended recipients without needlessly alerting patients, and limiting sound level and duration of signals.

Staff conversations and voice paging were also highly alerting, producing a 50 % chance of arousal at 40 $L_{A10,10\text{-sec.}}$ (dB). Voice transmission can be modified behaviorally and blocked through design and construction solutions. Simple strategies include planning for and directing conversation to designated consulting spaces. In many healthcare settings, policy still includes keeping patient doors open to allow for visual monitoring and easy accessibility for care-givers. This policy carries a tradeoff, however, as it exposes the patient to excess noise from nurses' station and other sources. Better patient-monitoring technology may help minimize need for this open door policy, at least at night, balanced against unique patient-safety concerns. Proper door hardware could decrease the sounds generated by door closing. Door gasket installation could limit sound transmission between patient rooms and halls.

Numerous other hospital-based sounds emanating from sources external to a patient room can disrupt sleep, for example, ice machines, laundry carts, and overhead paging (Yinnon et al. 1992; Southwell & Wistow 1995; Topf et al. 1996). These sounds were, as a group, arousing at relatively low sound levels. Clearly ice machines should be architecturally isolated from patient areas or re-engineered. Improving the selection and maintenance of carts and organizing the schedule of use and routing to reduce noise is a low-tech contribution to protecting patients' sleep.

Exterior-to-building noises (jet, helicopter, traffic) were the least arousing among our group of stimuli, yet similar to previously studies of sleep disruption by airplane overflights, we observed a 32 % probability of arousal at 45 dBA (Basner et al. 2008). It appears that these most continuous of our stimuli are less arousing as compared with more intermittent stimuli like a phone ringing or IV alarm, in which higher peaks and faster rise times are more likely to induce cortical arousals. Previous work in aircraft noise has determined that the average sound level ($L_{\rm eq}$) is inadequate for describing the sleep disruptive effects of aircraft noise. Instead, a maximum sound level exposure, as administered in the current study, is a more appropriate exposure metric (Basner et al. 2006). (A recent study examined several objective and subjective parameters of sleep after road, rail or air traffic noise exposures. For details see Basner et al 2011.)

The effect of noise is influenced by the stage of sleep

Sleep is not a uniform state of reduced consciousness, but rather a cyclic orchestration of physiologic states. Indeed the architecture and composition of sleep can vary from person to person (Dang-Vu et al. 2010). Older people tend to have less stage N3 non-REM sleep (Chayon et al. 2004; Landolt et al. 1996) and stage distributions can be influenced by various medications (e.g. antidepressants suppress REM sleep) (Mayers & Baldwin 2005). We therefore report our data by sleep stages, addressing

differential stage vulnerabilities to noise for application to a range of individuals and populations. Our data confirm that stage N3 sleep is the deepest stage of sleep with respect to capacity to withstand disruption by noise. The probability of arousal by any given stimulus, controlling for decibel level, was lowest in stage N3.

During REM sleep, we observed a narrow range of cortical arousals, relative to other non-REM stages of sleep, and given a wide range of sounds administered in this experiment. This might demonstrate that the brain during REM sleep has less capacity to differentiate among sounds compared to non-REM sleep. This finding is unexpected because REM sleep has an abundance of cerebral activity relative to stage N3 sleep, including in auditory areas of the brain (Braun et al. 1997). Auditory evoked potentials elicited by saying a subject's name during REM sleep also appear similar in morphology to those observed during wakefulness (Perrin et al. 1999), implying some higher-order processing in REM. This supports the broader notion that, in REM sleep, cerebral resources are dedicated to internal processing—such as dream content—rather than to differentiating external sound sources. A useful next study would examine the dreams of people hearing these sounds to see if they are, in fact, incorporated into the dream content preferentially.

This information concerning the differential resistance of certain stages of sleep to acoustic disruption might inform research exploring brain-based approaches to mitigating sensory disruption of sleep. Such approaches might include novel drugs or devices that maximize the more resilient features of sleep physiology such as stage N3 sleep, or sleep spindles (Dang-Vu et al. 2010).

Although ecologically valid, this experiment has some limitations that might cause an underestimation of the effects of noise on sleep. First, we intentionally calibrated sounds based on their A-weighted scale, which emphasizes frequencies in the audible range for humans. The second limitation is that stimuli were presented individually for 10-second exposures and were halted if an EEG arousal resulted. Finally, we studied only young, healthy adults. In that context, our data is best viewed as providing the minimum sleep disruption caused by these common noises, across a range of sound-pressure levels, and should therefore be used to set a minimum standard for noise-attenuating techniques.

SUMMARY

Protecting sleep from acoustic assault in hospital settings is a key aim in advancing quality of care for inpatient medicine. Toward this end, we have characterized the vulnerability of sleep to commonly encountered hospital sounds by deriving unique arousal probability profiles to enable customized target thresholds and interventions to limit noise-induced sleep disruption. This research has already informed the first acoustic standards in the Guidelines for the Design and Construction of Health Care Facilities (Facility Guidelines Institute 2010). With the leading edge of baby boomers turning 65 this year and an aging healthcare infrastructure, billions of dollars of healthcare facility renovation and new construction are anticipated in the coming decade (Carpenter 2010). Improving the acoustics environment in existing and new facilities will be critical to ensuring future healthcare environments enable the highest quality care.

REFERENCES

Bartick MC, Thai X, Schmidt T et al. (2009). Decrease in as-needed sedative use by limiting nighttime sleep disruptions from hospital staff. J Hosp Med 5: E20-E24.

Basner M (2010). Arousal threshold determination in 1862: Kohlschutter's measurements on the firmness of sleep. Sleep Med 11: 417-422.

Basner M, Samel A, Isermann U (2006). Aircraft noise effects on sleep: application of the results of a large polysomnographic field study. J Acoust Soc Am 119: 2772-2784.

Basner M, Glatz C, Griefahn B et al. (2008). Aircraft noise: effects on macro- and microstructure of sleep. Sleep Med 9: 382-387.

Basner M, Muller U, Elmenhorst EM (2011). Single and combined effects of air, road, and rail traffic noise on sleep and recuperation. Sleep 34: 11-23.

Berg S (2001). Impact of reduced reverberation time on sound-induced arousals during sleep. Sleep24: 289-292.

Bonnet MH, Arand DL (2007). EEG arousal norms by age. J Clin Sleep Med 3: 271-274.

Bonnet MH, Doghramji K, Roehrs T et al. (2007). The scoring of arousal in sleep: Reliability, validity, and alternatives. J Clin Sleep Med 3: 133-145.

Braun AR, Balkin TJ, Wesensten NJ et al. (1997). Regional cerebral blood flow throughout the sleep-wake cycle an H215O PET study. Brain 120: 1173-1197.

Bruck D, Ball M, Thomas I et al. (2009). How does the pitch and pattern of a signal affect auditory arousal thresholds? J Sleep Res 18: 196-203.

Busch-Vishniac IJ, West JE, Barnhill C et al. (2005). Noise levels in Johns Hopkins Hospital. J Acoust Soc Am 118: 3629-3645.

Carpenter D (2010). Proceed with caution: lean year expected for hospital construction. Health Facilities Management 2010 Feb: 11-18.

Cmiel CA, Karr DM, Gasser DM et al. (2004). Noise control: a nursing team's approach to sleep promotion. Am J Nurs 104: 40-48

Dang-Vu TT, McKinney SM, Buxton OM et al. (2010). Spontaneous brain rhythms predict sleep stability in the face of noise. Curr Biol 20: R626-R627.

De Gennaro L, Casagrande M, Violani C et al. (1995). The complementary relationship between waking and REM sleep in the oculomotor system: an increase of rightward saccades during waking causes a decrease of rightward eye movements during REM sleep. Electroencephal Clin Neurophysiol 95: 252-256.

Dijk DJ, Duffy JF, Czeisler CA (2001). Age-related increase in awakenings: Impaired consolidation of nonREM sleep at all circadian phases. Sleep 24: 565-577.

Elmenhorst EM, Elmenhorst D, Wenzel J et al. (2010). Effects of nocturnal aircraft noise on cognitive performance in the following morning: dose-response relationships in laboratory and field. Int Arch Occup Environ Health 83: 743-751.

Facility Guidelines Institute (2010). Guidelines for design and construction of health care facilities.

Griefahn B, Brode P, Marks A et al. (2008). Autonomic arousals related to traffic noise during sleep. Sleep 31: 569-577.

Hagerman I, Rasmanis G, Blomkvist V et al. (2005). Influence of intensive coronary care acoustics on the quality of care and physiological state of patients. Int J Cardiol 98: 267-270.

Halasz P, Terzano M, Parrino L et al. (2004). The nature of arousal in sleep. J Sleep Res 13: 1-23.

Iber C, Ancoli-Israel S, Chesson A et al. (2007). The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester, IL: Americal Academy of Sleep Medicine.

Institute of Medicine (2006). Sleep disorders and sleep deprivation: An unmet public health problem. Washington, DC: National Academies Press.

Jha AK, Orav EJ, Zheng J et al. (2008). Patients' perception of hospital care in the United States. N Engl J Med 359: 1921-1931.

Johnson AN (2003). Adapting the neonatal intensive care environment to decrease noise. J Perinat Neonatal Nurs 17: 280-288.

Kahn DM, Cook TE, Carlisle CC et al. (1998). Identification and modification of environmental noise in an ICU setting. Chest 114: 535-540.

Kohlschütter E (1863). Messungen der Festigkeit des Schlafes. Zs Rationelle Med 17: 209-253.

Landolt H-P, Dijk DJ, Achermann P et al. (1996). Effect of age on the sleep EEG: Slow-wave activity and spindle frequency activity in young and middle-aged men. Brain Res 738: 205-212.

Mayers AG, Baldwin DS (2005). Antidepressants and their effect on sleep. Hum Psychopharmacol 20: 533-559.

McNeer RR, Bohorquez J, Ozdamar O et al. (2007). A new paradigm for the design of audible alarms that convey urgency information. J Clin Monit Comput 21: 353-363.

Montague KN, Blietz CM, Kachur M (2009). Ensuring quieter hospital environments. Am J Nurs 109: 65-67.

Ohayon MM, Carskadon MA, Guilleminault C et al. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. Sleep 27: 1255-1273.

Perrin F, Garcia-Larrea L, Mauguiere F et al. (1999). A differential brain response to the subject's own name persists during sleep. Clin Neurophysiol 110: 2153-2164.

Southwell MT, Wistow G (1995). Sleep in hospitals at night: are patients' needs being met? J Adv Nurs 21: 1101-1109.

Topf M, Thompson S (2001). Interactive relationships between hospital patients' noise-induced stress and other stress with sleep. Heart Lung 30: 237-243.

Topf M, Bookman M, Arand D (1996). Effects of critical care unit noise on the subjective quality of sleep. J Adv Nurs 24: 545-551.

WHO (2009). Night noise guildelines for Europe. Copenhagen; WHO Regional Office for Europe.

Yinnon AM, Ilan Y, Tadmor B et al. (1992). Quality of sleep in the medical department. Br J Clin Pract 46: 88-91.