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# Sleep Disruption due to Hospital Noises

## A Prospective Evaluation

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**Background:** Sleep plays a critical role in maintaining health and well-being; however, patients who are hospitalized 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:** To determine profiles of acoustic disruption of sleep by examining the cortical (encephalographic) arousal responses during sleep to typical hospital noises by sound level and type and sleep stage.

**Design:** 3-day polysomnographic study.

**Setting:** Sound-attenuated sleep laboratory.

**Participants:** Volunteer sample of 12 healthy participants.

**Intervention:** Baseline (sham) night followed by 2 intervention nights with controlled presentation of 14 sounds that are common in hospitals (for example, voice, intravenous alarm, phone, ice machine, outside traffic, and helicopter). The sounds were administered at calibrated, increasing decibel levels (40 to 70 dBA [decibels, adjusted for the range of normal hearing]) during specific sleep stages.

**Measurements:** Encephalographic arousals, by using established criteria, during rapid eye movement (REM) sleep and non-REM (NREM) stages 2 and 3.

**Results:** Sound presentations yielded arousal response curves that varied because of sound level and type and sleep stage. Electronic sounds were more arousing than other sounds, including human voices, and there were large differences in responses by sound type. As expected, sounds in NREM stage 3 were less likely to cause arousals than sounds in NREM stage 2; unexpectedly, the probability of arousal to sounds presented in REM sleep varied less by sound type than when presented in NREM sleep and caused a greater and more sustained elevation of instantaneous heart rate.

**Limitations:** The study included only 12 participants. Results for these healthy persons may underestimate the effects of noise on sleep in patients who are hospitalized.

**Conclusion:** Sounds during sleep influence both cortical brain activity and cardiovascular function. This study systematically quantifies the disruptive capacity of a range of hospital sounds on sleep, providing evidence that is essential to improving the acoustic environments of new and existing health care facilities to enable the highest quality of care.

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Sleep is essential for the restoration of health and well-being (1). However, in hospitals, where healing is paramount, noise frequently disrupts patients' sleep. In a recent national survey, patients identified the noise levels in and around rooms at night as the quality-of-care factor with the most need for improvement (2). Acoustic measurements from a major urban hospital document a crescendo of nighttime hospital noise over the past 45 years from an average level of 42 dBA (decibels, adjusted for the range of normal hearing) to more than 55 dBA in 2005 (3). Hospitals are exposed to external noise sources known to disrupt sleep, such as traffic and airplane sounds (4), with documented dose-related consequences for next-day cognitive performance (5). Patient care also produces noise specific to treatment and protection, such as intravenous and cardiac monitor alarm signals (6). Improving acoustics in environments of care to protect sleep and enhance outcomes for the more than 37 million patients who are hospitalized annually in the United States (7) has become a transdisciplinary priority (8–11).

The goal of this study was to provide essential information about the effect of sound on sleep to guide architectural, technological, and programmatic advances to facilitate sleep and improve clinical outcomes for patients who are hospitalized. We hypothesized that the capacity for sleep disruption varies by the type of sound and increases for each type as the sound level increases. We further hypothesized that the stages of sleep, characterized by diverse cortical activity patterns, are differentially vulnerable to disruption by noise. Sleep stages cycle through the night and vary in their relative proportions with age (12), medications (13), and certain medical and psychiatric disorders (14), among other factors. Therefore, a useful exploration of the responses of sleep to noises must include sleep stage during noise exposure.

Sleep stages include 2 brain state categories: rapid eye movement (REM) sleep and non-REM (NREM) sleep stages. The range of NREM sleep stages includes progressively deepening levels from drowsiness to deep sleep, termed *N1*, *N2*, and *N3* (15): N1 is the brief transition between wakefulness and sleep, N2 is typically the most

*See also:*

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Summary for Patients . . . . . 1

 

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***Context***

The negative effects of hospital noise on sleep are among the most common concerns of inpatients and their families.

***Contribution***

During sleep laboratory studies of healthy volunteers, investigators found that the disruptive effect of recorded hospital noises varied by the type and level of sound emitted and by the volunteer's stage of sleep. Electronic sounds designed to be alerting were most disruptive, as were staff conversations and voice paging.

***Caution***

Volunteers were young and healthy. Sounds were not administered together.

***Implication***

Reduction of hospital noise through policies, procedures, and building design may lead to improved patient sleep and, thus, quality of care.

*—The Editors*

abundant stage of sleep in adults, and N3 (or slow-wave sleep) is the deepest level. These REM and NREM brain states seem to be driven by different nuclei and neurotransmitters (16). They can be readily discerned through distinct patterns appearing on electroencephalograms (EEGs) (15) and neuroimaging (17). In addition, behavioral evidence demonstrates that the sleeping brain responds to auditory stimuli differently during REM than during NREM sleep (18). We sought to elaborate on this evidence by examining differential responses to hospital noise exposures between REM and NREM states and exploring variability in sleep disruption within the deepening stages of NREM.

We designed a protocol to examine the influence of graded noise exposures during all stages of sleep, through polysomnographic (PSG) assessments (combined EEG, electrooculogram, and electromyogram), a standard (19, 20) and sensitive (21, 22) system to measure sleep strongly correlated with awakenings (21). Because elevations in heart rate are known to occur during full EEG-documented awakenings from sleep (23), we used the electrocardiogram to detect the presence of clinically relevant heart rate responses to noise-induced arousals. We predicted heart rate elevations during these EEG-documented sleep arousals in participants who were exposed to common hospital sounds.

## METHODS

### Design Overview

All study procedures were approved by the human research committees of the involved institutions. The design is a 3-day PSG study, beginning with a baseline (sham) quiet night followed by 2 noise exposure intervention nights, during which EEG arousals and electrocardiogram heart rate accelerations were documented.

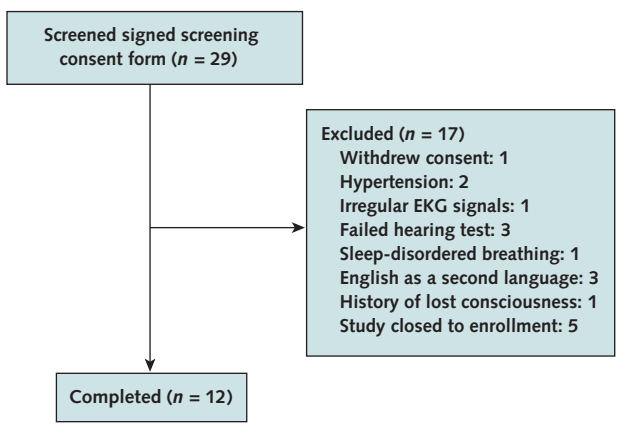
### Participants and Setting

Participants were recruited through flyers, Web site postings, and word of mouth and then screened by questionnaire, physical examination, and laboratory testing. Participants who reported medical or psychiatric conditions or use of substances or medications that potentially affect sleep were excluded from the study. Criteria for exclusion included history of drug or alcohol abuse; depression; anxiety; posttraumatic stress and obsessive compulsive disorders; neurologic or sleep disorders; infectious diseases; diseases of the cardiovascular system; or treatment with antidepressants, neuroleptics, or major tranquilizers. Urinalysis confirmed the absence of caffeine, nicotine, and alcohol. Standard audiometric screening confirmed normal hearing (that is, exceeding 20 dBA in both ears). The first 12 eligible and available participants were enrolled (**Figure 1**). F1

Participants slept at home on a regular schedule for at least 4 days before participation in the study. They reported sleeping a mean of 7.72 hours (SD, 0.27) over a mean of 6.5 days (SD, 1.1) through a time-stamped phone-answering system that was confirmed through wrist actigraphy (AW-64, Philips Respironics, Murrysville, Pennsylvania), which demonstrated a mean of 7.16 hours (SD, 0.29) of sleep over a mean of 6.7 monitored days (SD, 0.9).

Participants stayed at the Massachusetts General Hospital Sleep Laboratory for 3 days. Each night, participants were given an 8.5-hour sleep opportunity, which began at their normal bedtimes. Continuous video observation and wrist actigraphy confirmed that participants did not nap during the day. Light levels were maintained at less than 1 lux (darkness) during sleep periods and approximately 90 lux (ordinary daylight in room) during waking periods. Because of continuous air exchange (required in health care

*Figure 1.* **Study flow diagram.**

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EKG = electrocardiogram.



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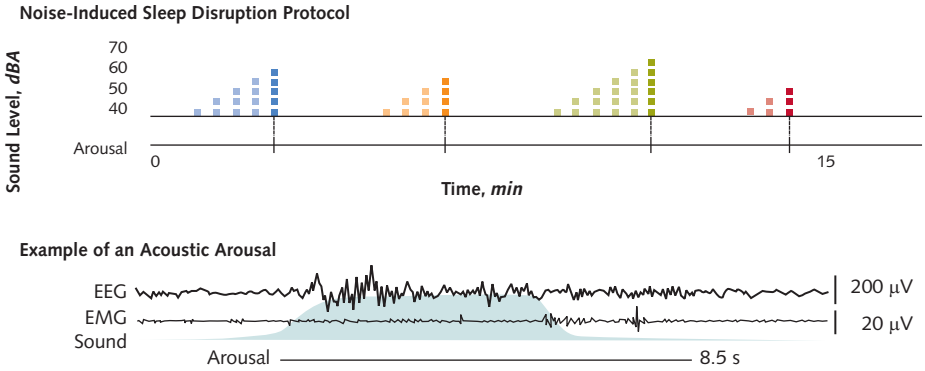
*Figure 2.* **Schematic diagram of study protocol.**

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*Top.* The solid vertical lines along the *x*-axis indicate stimuli evoking EEG arousals, and a sample of 4 noises is shown. Each color represents a different sound type. Ten-second noises were evaluated for their probability to induce a cortical arousal at increasing sound levels in varying stages of sleep and presented once per 30-second sleep epoch (while sleep stage was stable) until an arousal occurred, sleep stage changed, or the 70-dBA maximum was reached. *Bottom.* Shown here is a typical sound-induced arousal from stage N2 sleep, as measured by polysomnography. Arousals are defined by their appearance on the EEG (the right frontal lead F3 shown here), characterized by an abrupt shift of frequency that lasts at least 3 seconds. Arousals during rapid eye movement sleep require a concurrent increase in submental EMG activity. This transient arousal lasted for approximately 8.5 seconds before sleep resumed. dBA = decibels, adjusted for the range of normal hearing; EEG = electroencephalogram; EMG = electromyogram.

facilities), background ambient sound levels averaged between 34 and 35 dBA ($LA_{EQ,\ 10\text{-}s}$ [equivalent continuous A-weighted scale {adjusted for the range of normal human hearing} sound pressure level, averaged over the 10-second stimulus duration]). On the first night, participants slept undisturbed to allow adaptation to the PSG equipment and laboratory environment, confirm absence of sleep disorders (including sleep apnea), and establish baseline sleep recordings. On the second and third nights, recorded hospital sounds were presented to participants throughout sleep.

### Intervention: Acoustic Stimuli

Recordings of hospital sounds were captured on a medical unit of Somerville Hospital, Cambridge Health Alliance, Somerville, Massachusetts. Each sound stimulus fit within 1 or more of the categories identified as salient in the American Institute of Architects Guideline on Sound and Vibration in Healthcare Facilities: external to building, within hospital, and within or outside patient rooms (8). Fourteen noise stimuli were selected: "good" conversation, which was defined as 1 male and 1 female voice discussing a positive patient outcome; "bad" conversation, which was defined as the same voices discussing a negative patient outcome; male voice from an overhead paging system calling a physician by name; door opening or closing; telephone ringing; toilet flushing; ice machine disgorging; IV alarm sounding; laundry cart rolling; automatic paper towel machine dispensing; helicopter takeoff; jet engine flyover; and outside traffic flow. Samples of acoustic stimuli are available at www.annals.org. To control for differences in duration across stimuli, sounds were normalized to 10 seconds (**Appendix Table 1**, available at www.annals.org).

Hospital noises were presented as stimuli with 2-dimensional verisimilitude (for example, airplane sounds moved across space) through use of 4 studio monitor loudspeakers (PS6, Event Electronics, Silverwater, Australia) arrayed about the head of the sleeping participants (a modified pattern from the ITU-R BS 775-1 Recommendation, omitting the center loudspeaker). Sound levels in the participants' room were logged in 1-second increments by using an environmental sound monitor (NL-31, with type 1 microphone [Rion, Tokyo, Japan]) installed roughly 10 inches above the head of the sleeping participants and programmed to output a direct current voltage proportional to the A-weighted fast-response sound level.

Once a steady sleep stage of at least 90 seconds was recorded, as assessed in real time by a technician, stimuli were systematically presented once per 30-second sleep epoch, starting at an exposure level ($LA_{EQ,\ 10\text{-}s}$) of 40 dBA in increasing steps of 5 dBA (**Figure 2**, *top*) until either sleep F2
was disrupted by an arousal (**Figure 2**, *bottom*), sleep stage changed, or the 70-dBA maximum exposure level was reached. Because both the equivalent sound level and the duration of the noise stimuli were held constant, all stimuli were normalized to deliver an equal "noise dose," an integration of sound intensity over time (24). All stimuli were presented in a computer-generated random order within each sleep stage on both exposure nights for every participant.

### Outcomes

Standard PSG recordings (Comet XL, Grass Technologies, West Warwick, Rhode Island) were collected on all 3 nights through skin surface electrodes. Sleep stages and arousals were identified by using current criteria (15). **Figure 2** (*bottom*) depicts a standard arousal, as defined by an



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abrupt shift of EEG frequency lasting at least 3 seconds. Arousals during REM sleep also require a concurrent increase in submental electromyogram activity (**Figure 2**). Body position was scored from infrared video to allow for statistical adjustment based on whether either ear was occluded, potentially attenuating arousal responses. Body position (supine or not) was assessed continuously by a sleep technician viewing the infrared video on the same screen as the EEG signals they were using to score sleep stages in real time at the initiation of each acoustic stimulus.

Experimental tasks were coordinated by 2 researchers; a sleep technician maintained PSG signal quality classification of sleep stages and identification of cortical arousals indicating sleep disruption (25), along with documentation of body position. A second technician or investigator maintained the acoustic equipment and initiated the programmed, semiautomatic presentation of escalating noise stimuli. Discrepancies with the real-time scoring were resolved by a board-certified sleep physician.

### Statistical Analysis

The probability of arousal by stimulus, sound level and type, and sleep stage was examined descriptively (graphically). Generalized linear mixed models were applied to evaluate the effects of hospital noises on the binary outcome (arousal from sleep) with a logit link, by using PROC GLIMMIX in SAS software, version 9.2 (SAS Institute, Cary, North Carolina), for determining differences by sleep stage, with factors of study night and body position. Because of large interperson differences, the participant was treated as a random effect, incorporating participant-specific intercepts into the model. We assume that, between 2 adjacent presented stimuli levels (for example, 50 and 55 dBA), the arousal probability increases linearly for the intervening stimuli levels (for example, 51 to 54 dBA). Because an ear against the pillow could attenuate the administered sound level, body position served as a covariate in the model where supine position (reference category) corresponded to having both ears exposed. This model was used to estimate the probability of arousal while accounting for stimulus, sound level, sleep stage, and body position. We separately estimate the additional effect of the night of study (see the Results section).

To assess the effects of noise on heart rate during sleep by sleep stage, we calculated the profile of instantaneous heart rate during each arousal relative to the average heart rate during the 10 seconds preceding each corresponding sound onset. To quantify the temporal dynamics of the heart rate response, we calculated the median durations from the sound onset to the time of peak heart rate during each arousal and to the time of arousal onset.

### Role of Funding Source

Nonprofit entities, the American Architects Health Foundation, Facilities Guidelines Institute, and The Center for Health Design, contributed resources to this investigator-initiated study. They did not play a role in the study design, conduct, reporting, or the decision to submit a manuscript.

## RESULTS

Twelve healthy, white participants (8 women; mean age, 27 years [SD, 7]; mean body mass index, 21.8 kg/m$^2$ [SD, 3.7]) successfully completed this study.

As expected, louder sounds were more apt to cause sleep disruption (**Figure 3**). Effects varied by the type of sound stimulus (for example, IV alarm vs. voices) and by the stage of sleep during which the sound stimulus was presented (for example, REM vs. N3). F3

We saw an effect of sleep stage on sound stimulus–evoked arousal probability (**Figure 3**, *top panels*); N2 differed from N3 and REM (both $P < 0.001$, Bonferroni-adjusted), but N3 and REM did not differ overall, using model-based probability estimates. The pattern of arousal probabilities from stages N2 to N3 were relatively consistent in terms of sound stimulus order from most to least arousing, but shifted to overall lower arousal rates during N3 compared with N2 (**Figure 3**, *middle panels*). In marked contrast, arousals from REM sleep revealed a more homogeneous and monotonic pattern across sounds presented than NREM stages (**Figure 3**, *middle panels*) not readily apparent from the mean curves alone (**Figure 3**, *top panels*). Arousals occurred at lower sound levels on the third study night compared with the second study night ($P < 0.001$). Testing for the stage–by–study-night interaction only showed a slight difference across nights among sleep stages ($P = 0.020$, adjusted for sound levels [**Appendix Table 2**, available at www.annals.org]; body position was not significant and was not included in the final interaction model). The significant interaction suggests that the arousal probability was lower on the third night for all sleep stages, but the magnitude of the difference varied across stages and may reflect some degree of sensitization of arousals to sound presentation. Depiction of arousal probabilities for individual sound stimuli by stage and sound level revealed considerable heterogeneity in the responses to the various stimuli (**Figure 4**). F4

We studied the change in heart rate during stimulus-induced arousals by subtracting the instantaneous heart rate from the average heart rate of the 10 seconds preceding the sound onset. The stage in which the arousal occurred substantially predicts the magnitude of the heart rate increase ($P < 0.0001$); the greatest responses occurred during REM, followed by N3 and N2 (**Figure 3**, *bottom panels*). All pairwise comparisons are significant at an $\alpha$ level of $0.05/3 = 0.017$. Baseline (prearousal) HR does not predict the magnitude of the response ($P = 0.94$). Study night is not significant ($P = 0.83$), reflecting a lack of habituation of the electrocardiogram heart rate response.

Heart rate responses are aligned by their peaks in **Figure 3** (*bottom panels*). The stage in which the arousal occurred significantly predicts the duration of time from the



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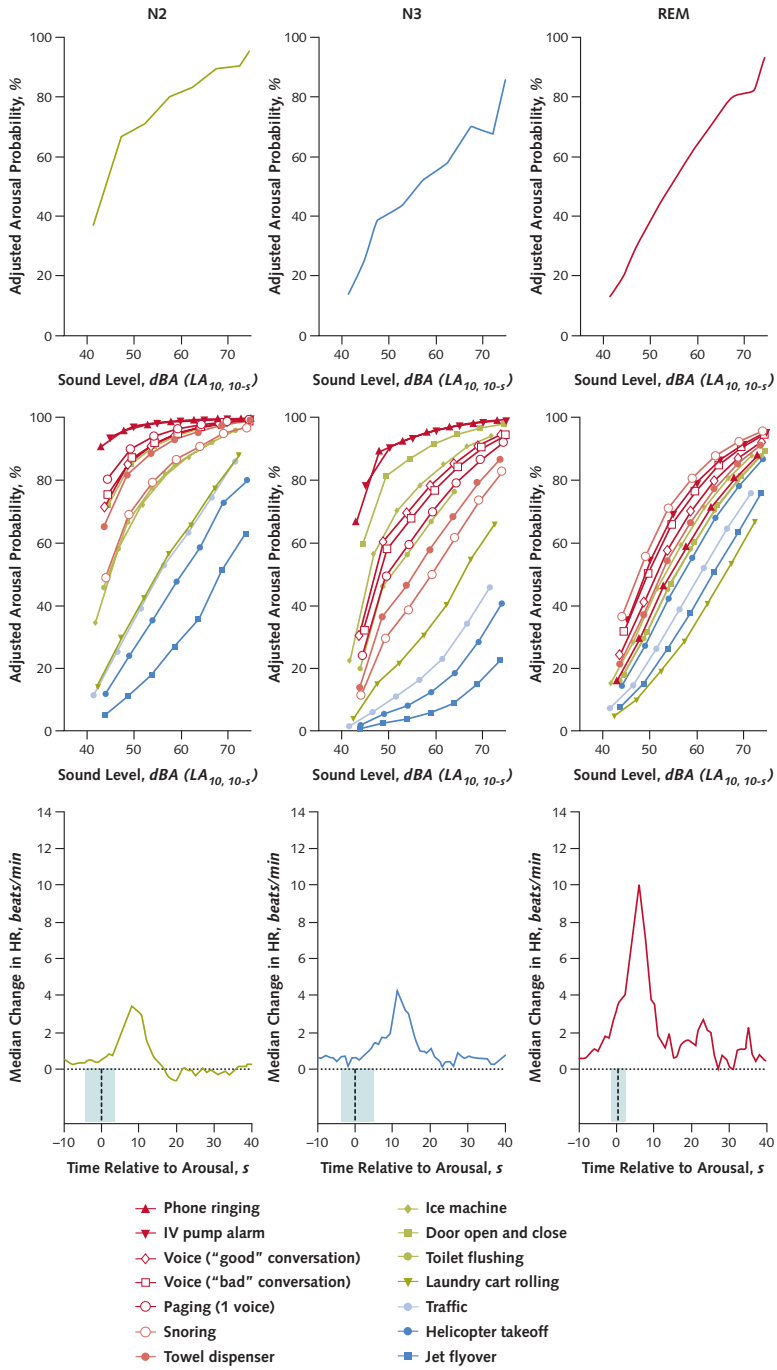
*Figure 3.* **Sleep disruption due to noise stimuli presented during sleep, by stage of sleep.**




Arousal probability of sound stimuli presented in sleep stages N2, N3, and REM. Ten-second noises were introduced during sleep stages N2, N3, and REM to evaluate their propensity to disturb sleep. Once a stable stage of at least 90 seconds was reached, noises were initiated at sound levels of 40 dBA (equivalent continuous A-weighted [adjusted for the range of normal human hearing] sound pressure level, averaged over the 10-s stimulus duration) and presented every 30 s in 5-dBA increments until an arousal occurred or the 70-dBA exposure level was reached. dBA = decibels, adjusted for the range of normal hearing; HR = heart rate; $LA_{10,\ 10\text{-}s}$ = sound pressure level, averaged over the 10-s stimulus duration, exceeded 10% of the time; N2 = non-REM sleep stage 2; N3 = non-REM sleep stage 3; REM = rapid eye movement. **Top panels.** Mean arousal probabilities are depicted for stimuli presented during stages N2, N3, and REM versus presented sound level and adjusted for stimulus and body position (see Methods section). **Middle panels.** Mean arousal probabilities for individual noise stimuli by sleep stage, adjusted for body position. **Lower panels.** Changes in the median HR during nonspontaneous, noise-induced arousals are aligned by the time of the peak HR response and expressed relative to the average HR in the 10 seconds preceding the arousals in stages N2, N3, and REM. The vertical lines represent the median time of arousal onset (with CIs) before that peak.

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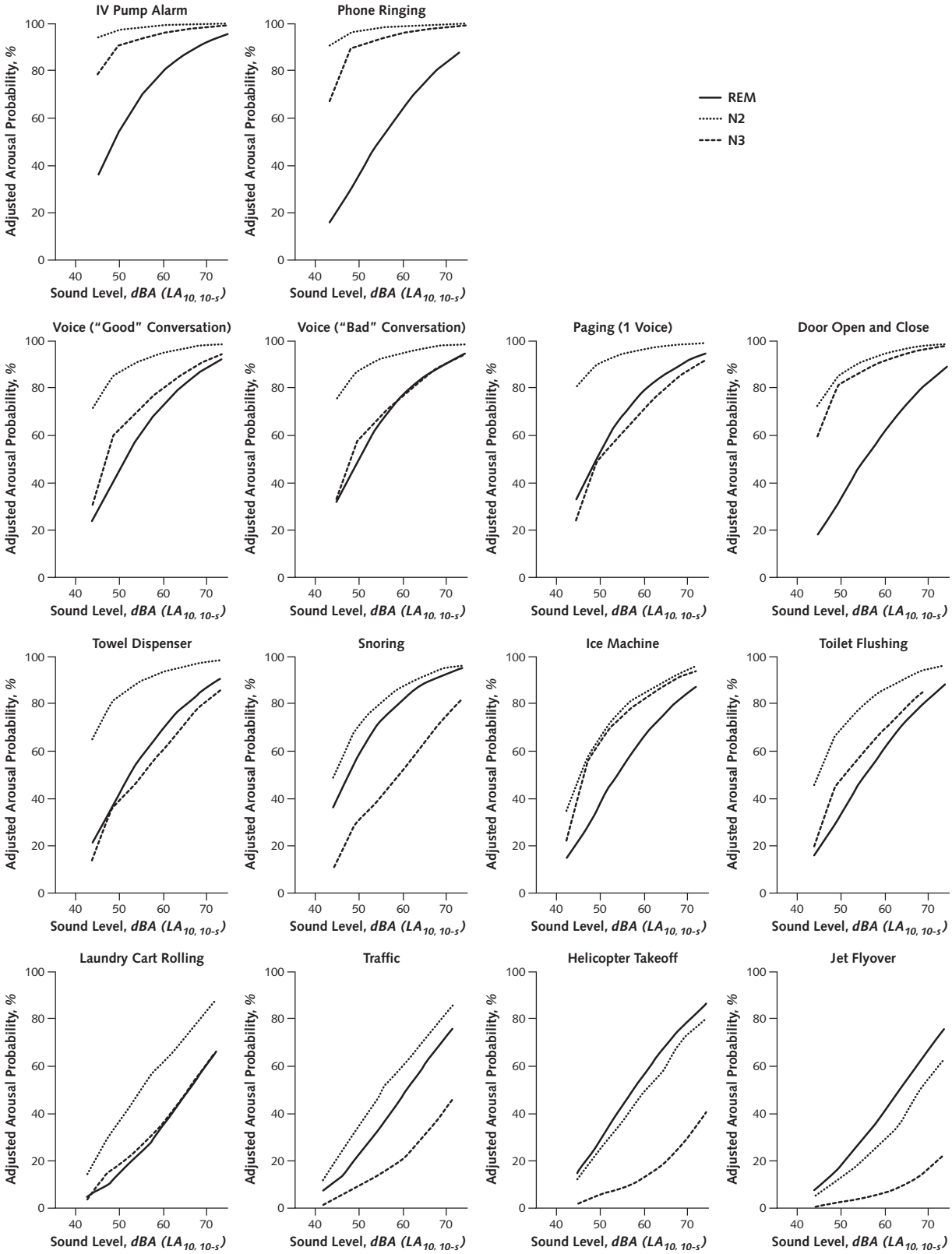
*Figure 4.* **Electroencephalogram arousal probabilities for noise stimuli presented, adjusted for body position (see Methods section).**




dBA = decibels, adjusted for the range of normal hearing; $LA_{10,\ 10\text{-}s}$ = sound pressure level, averaged over the 10-second stimulus duration, exceeded 10% of the time; N2 = non-REM sleep stage 2; N3 = non-REM sleep stage 3; REM = rapid eye movement.



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start of the arousal to the peak of the HR increase ($P < 0.001$); the fastest response times to peak were found during REM, then during N2 and N3. The median time from the start of the arousal to peak HR in REM is significantly shorter than N2 and N3; N3 is not significantly different from N2 adjusted for several comparisons at an $\alpha$ level of 0.017. No differences were seen in baseline HR across sleep stages ($P = 0.53$).

## DISCUSSION

This study systematically quantifies the disruptive capacity of hospital-recorded sounds on sleep. Sound presentations during sleep yielded arousal response curves that varied due to sound type and level and sleep stage. As predicted, for each stimulus higher sound levels led to a greater probability of sleep disruption. Electronic sounds, such as an IV alarm designed to alert medical staff, were consistently more arousing than other sounds at the same noise dose. Overall, the effect of sound level and type were modified by sleep stage physiology, producing unique arousal probability profiles for each sleep stage. We further demonstrate that the arousal effects of noise on sleep include heart rate elevations, even when disruptions are brief and frequent. Heart rate effects may be particularly relevant to critical care settings, in which monitor alarms are very frequent (6). These arousal probability profiles have the potential to drive needed innovation in design, construction, engineering, building materials, monitoring and communication equipment, and care-giving protocols to preserve sleep and enhance environments for healing. Improved acoustic environments consistent with current guidelines in the United States (8) and European Union (26) could deliver several clinical benefits, including reduced sedation and shorter hospital stays (4, 9, 10, 21, 27–29).

Disrupted sleep is known to be associated with hypertension (30), incidence of cardiovascular and coronary heart disease (31), impaired immune function (32), elevated stress hormone responses (33), attention and memory deficits (34), and depressed mood (35). Preservation of patients' sleep should be a priority for contributing to improved clinical outcomes for patients who are hospitalized (36). Spontaneous arousals are known to accelerate HR (36–39). Full awakenings evoked by noise lead to HR elevations of approximately 10 beats/min (36). We demonstrate that evoked arousals elicit HR acceleration from all stages of sleep, but a greater magnitude (10 beats/min) and faster onset of HR accelerations from REM, with lesser magnitude and less rapid accelerations in stages N2 and N3. Our data demonstrate that the effect of noise on sleep includes HR elevations, even when the disruption is brief and frequent, as might be seen in an intensive care unit setting. A recent synthesis of hospital soundscape surveillance data described a pattern of intensive care unit noise exceeding a "peak" of 60 dBA more than 50% of the time at night (40) and, thus, the frequency of sleep disruptions may be high in typical intensive care or other inpatient units, as described by patient self-reports (2), and in other units, such as the neonatal intensive care unit (41). A study of patients in the cardiac intensive care unit demonstrated that adverse acoustic environments are associated with higher pulse amplitude at night and elevated use of $\beta$-blockers. The patient group exposed to the acoustically unmodified environment also demonstrated higher rates of rehospitalization and poorer ratings of quality of care (29).

More broadly across the hospital, patients who are hospitalized frequently have delirium with immediate and long-term consequences, including an association with increased mortality rates. Sleep disruption has been proposed as a modifiable target for delirium interventions (42). A prospective and multifaceted delirium intervention study of older patients on a general medical ward—a study that included a sleep component and noise reduction—successfully reduced delirium symptoms and the rate of sleep medication use (43). Sleep is a cyclic orchestration of stages that alters with aging and can vary from person to person with specific medical, psychiatric, and situational differences (44). Older persons tend to have less N3 sleep (12, 45), and various medications can influence stage distributions (for example, antidepressants suppress REM sleep) (46). Our data provide a framework for implementing targeted strategies to mitigate noise-induced sleep disruption, which potentially contributes to delirium among patients who are hospitalized.

Approaches to mitigating noise for sleeping patients include eliminating or controlling the source of sound or blocking its path. The first approach, controlling sound at the source, includes public-policy restrictions on acceptable night noise, such as aircraft flyovers (9); substitution with quieter technologies, such as personal digital assistants in place of overhead paging; and telemetry from nurses' stations to limit intrusive oversight (11, 47). The night care intervention study at 1 hospital established a "quiet time" period, altered intrusive medication routines, and reduced sound level exposures from staff voices. This protocol resulted in a 25% reduction of unit-wide sedative medication use and improved patient satisfaction ratings (10). The second approach to mitigation focuses on "blocking" or attenuating sound along the transmission path, including hospital unit design configurations; application of advanced construction materials, such as acoustic surfaces (48); closing doors; and even supplying earplugs to patients.

As expected, the most potent sleep disruptors were electronic sounds intentionally designed to be alerting (49, 50). The arousal probability curves in **Figure 4** corresponding to these sounds (that is, phone ringing and IV alarm) reveal that these devices may not be suitably attenuated to spare sleep, even at their quietest settings: within the lowest tested ranges in this study, these sounds produced sleep disruption more than 50% of the time. Alarm signals have proliferated in health care settings. Monitor alarms could



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be better managed through enhanced algorithms, more careful patient assignment and clinically relevant configuration standards, and targeting intended responders with technologies using nonauditory channels (6).

Staff conversations and voice paging were also found to be highly alerting, producing a 50% chance of arousal at 50 dBA (sound level exceeded 10% of the time) in N2 and REM sleep. Voice transmission can be modified behaviorally (10) and diminished through design and construction solutions. Simple strategies include planning for and directing conversation to designated consulting spaces. In many health care settings, policy still includes keeping patient doors open to allow for visual monitoring and easy accessibility by caregivers, which exposes patients to excess noise from the nurses' station and other sources. Centralized patient-monitoring technology may help minimize the need for this policy—at least at night—while still addressing patient-safety concerns. Proper door hardware and gasketing could decrease the sounds generated by door closing and limit sound transmission from halls.

Other tested hospital sounds (for example, ice machines, laundry carts, and overhead paging) that emanate from sources external to patient rooms (51–53) were, as a group, arousing at relatively low sound levels. Ice machines should be architecturally isolated from patient areas or reengineered. Modifying procedures and equipment, such as selection and maintenance of carts and organizing the schedule of use and routing, is a low-tech, low-cost contribution to reducing noise. Exterior-to-building noises (for example, jet, helicopter, traffic) were the least arousing among our group of stimuli, and our findings were consistent with other studies of sleep and airplane flyovers (21). The previous work determined that statistical description of average sound level ($LA_{EQ}$) over 24 hours is an inadequate measure for describing the sleep-disruptive effects from noise. Examining disruption at different sound levels is the more appropriate exposure metric (9), especially for sounds with broad ranges that peak. It is, therefore, not surprising that we determined that continuous stimuli (for example, traffic noise) are less arousing than intermittent stimuli (for example, phone ringing or IV alarm). At the same adjusted noise dose, higher transient sound levels and faster rise times are more likely to induce cortical arousals. In light of our findings, broad sleep-preserving steps should include changes in the design of health care facilities, construction materials chosen for acoustic properties, improved monitoring and alerting technology, sleep-protective night care routines, and education and retraining of health care personnel on the effect of noise on patient arousal and cardiac responses to such sleep disruptions (4, 9, 10, 21, 27–29).

During REM sleep, we saw a narrower range of cortical arousals, relative to NREM stages of sleep, and across the wide range of sounds administered in this experiment. This may demonstrate that the brain, during REM sleep, has less capacity to differentiate among sounds compared with NREM 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 (17). Auditory-evoked potentials elicited by saying a participant's name during REM sleep also seem similar in morphology to those seen during wakefulness (18), implying that there is some higher-order processing in REM sleep. 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.

Although ecologically valid in many aspects, this experiment has some limitations that may cause an underestimation of the effects of noise on sleep. We presented noises individually for up to 10 seconds and halted if arousal occurred. This procedure minimized full awakenings and increased sleep time available for more stimulus presentations. In a hospital setting, sounds often last longer than 10 seconds and several sounds occur simultaneously. We do not account for relative proportion or intermittency of stimuli in a hospital setting; our data are intended to provide a framework by which a specific unit could assess the sleep-disrupting effects of a specific hospital environment. We studied only 12 young, healthy adults. The typical patient who is hospitalized is older, with generally less of the most protected deep sleep, N3 (54). Medical and psychiatric conditions, as well as pain and medication use, compromise sleep in patients who are hospitalized, presumably rendering deep sleep, N3, more difficult to achieve. Noise can be expected to interact with these other sleep-disrupting stressors associated with hospitalization (55). Therefore, we judge our arousal probability profiles for N2 sleep to be most relevant for predicting acoustic disruption of sleep in inpatient populations. Future studies should assess the effect of noise on sleep disruption and HR changes in older participants to confirm generalizability and document effects on sleep stage proportions and architecture. Together, these limitations may cause our data to underestimate the effects of noise on sleep for patients who are hospitalized. Our data should be viewed as providing reference points that demonstrate sleep disruption caused by these common hospital noises, across a range of sound levels, and should be used to set a minimum for noise-attenuating standards.

In summary, protecting sleep from acoustic assault in hospital settings is a key goal in advancing the quality of care for inpatient medicine. We 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 (8). With the leading edge of baby boomers turning 66 this year and an aging health care infrastructure, billions of dollars in health care facility renovation and new construc-



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tion are anticipated in the coming decade (7). Improving the acoustics in health care facilities will be critical to ensuring that these environments enable the highest quality care and the best clinical outcomes.

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rich4/zai-aim/zai-aim/zai01212/zai4110d12z | xppws | S=1 | 5/10/12 | 6:24 | 4/Color Figure(s): F1-3 | Art: M11-1159

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## *Appendix Table 1.* Acoustic Descriptors of Sound Stimuli*

| Acoustic Descriptor | Voice ("Bad" Conversation) | Voice ("Good" Conversation) | Door Open and Close | Helicopter Takeoff | Ice Machine | IV Pump Alarm | Jet Flyover | Laundry Cart Rolling | Overhead Paging (1 Voice) | Phone Ringing | Snoring | Toilet Flushing | Towel Dispenser | Traffic |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| $L_{01}$ | 76 | 78 | 78 | 76 | 73 | 76 | 74 | 73 | 77 | 73 | 74 | 75 | 74 | 72 |
| $L_{10}$ | 74 | 74 | 74 | 74 | 72 | 75 | 74 | 72 | 74 | 73 | 74 | 74 | 73 | 71 |
| $L_{50}$ | 67 | 68 | 67 | 69 | 70 | 66 | 69 | 71 | 67 | 71 | 68 | 69 | 67 | 70 |
| $L_{90}$ | 52 | 56 | 55 | 54 | 69 | 47 | 52 | 60 | 46 | 35 | 35 | 45 | 45 | 57 |
| $L_{99}$ | 50 | 41 | 36 | 36 | 46 | 42 | 36 | 36 | 35 | 35 | 35 | 35 | 37 | 35 |
| $L_{max}$ | 76 | 78 | 79 | 76 | 73 | 76 | 74 | 74 | 78 | 73 | 74 | 76 | 74 | 72 |
| $L_{min}$ | 35 | 35 | 35 | 35 | 35 | 35 | 35 | 35 | 35 | 35 | 35 | 35 | 35 | 35 |
| $L_{EQ}$ | 70 | 70 | 70 | 70 | 70 | 70 | 70 | 70 | 70 | 70 | 70 | 70 | 70 | 70 |
| $L_{10}$–$L_{EQ}$ | 4 | 4 | 4 | 4 | 2 | 5 | 4 | 2 | 4 | 3 | 4 | 4 | 3 | 1 |
| $L_{max}$–$L_{EQ}$ | 6 | 8 | 9 | 6 | 3 | 6 | 4 | 4 | 8 | 3 | 4 | 6 | 4 | 2 |
| $L_{01}$–$L_{EQ}$ | 6 | 8 | 8 | 6 | 3 | 6 | 4 | 3 | 7 | 3 | 4 | 5 | 4 | 2 |
| $L_{10}$–$L_{90}$ | 22 | 17 | 20 | 20 | 3 | 28 | 22 | 13 | 28 | 38 | 39 | 28 | 29 | 14 |
| $L_{01}$–$L_{99}$ | 26 | 37 | 42 | 40 | 26 | 34 | 38 | 37 | 42 | 38 | 39 | 40 | 37 | 37 |

IV = intravenous; $L_{01}$ = sound level exceeded 1% of the time; $L_{10}$ = sound level exceeded 10% of the time; $L_{50}$ = sound level exceeded 50% of the time; $L_{90}$ = sound level exceeded 90% of the time; $L_{99}$ = sound level exceeded 99% of the time; $L_{max}$ = maximum A-weighted root-mean square sound level; $L_{min}$ = minimum A-weighted root-mean square sound level; $L_{EQ}$ = equivalent continuous sound level.
* Sound stimuli = 70 dBA (adjusted for the range of normal human hearing).

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## *Appendix Table 2.* Time Spent in Stages of Sleep and Wakefulness During 8.5-Hour Sleep Periods

| Variable | Night 1 | Night 2 | Night 3 |
|---|---|---|---|
| Mean sleep stage (SD), *min* | | | |
| N1 | 58.0 (16.7) | 63.6 (16.9) | 59.3 (22.3) |
| N2 | 232.3 (26.3) | 247.3 (36.3) | 238.4 (28.7) |
| N3 | 90.5 (37.2) | 69.2 (30.9) | 80.5 (33.6) |
| REM | 99.1 (23.6) | 104.6 (19.2) | 101.0 (18.7) |
| Mean wakefulness (SD), *min* | 28.2 (14.9) | 23.5 (18.9) | 30.8 (14.9) |

N1 = non-REM sleep stage 1; N2 = non-REM sleep stage 2; N3 = non-REM sleep stage 3; REM = rapid eye movement.



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