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Noise Pollution in Hospitals: Impact on Patients

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ABSTRACT

- *Objective:* To review the epidemiology of noise pollution in hospitals and its effects on patients.
- *Methods:* Review of the literature.
- *Results:* Using academic search engines such as PubMed, JSTOR, and JASA, as well as common internet search engines, 36 papers were selected that focus on noise as it relates to patient sleep disturbances, cardiovascular response, length of hospital stay, pain management, wound healing, and physiological reactions. Results generally show the potential for negative physiological effect when patients are exposed to noise; however, conflicting studies are also reported. This review attempts to define the research chain in the collected articles by determining which acoustic characteristics were examined, what type of acoustic intervention (if any) was used, and what the patient outcomes were.
- *Conclusion:* The effects of hospital noise on patients are generally negative but sometimes inconclusive. Information on specific acoustic metrics/methodologies used is often limited, few studies examine the impacts of acoustic interventions, and some patient outcomes were studied in a limited number of articles or via small subject sample sizes, highlighting areas of potential future research.

Florence Nightingale recognized noise as a health hazard in 1859 when she wrote "Unnecessary noise is the most cruel abuse of care which can be inflicted on either the sick or the well" [1]. Since then, a growing body of research highlights the potential negative impacts of noise pollution in hospitals. For example, links have been developed between sleep disturbances, such as reduction of sleep depth, continuity, or duration, cardiovascular response, wound healing, pain management, and other patient responses due to hospital noise. There is also concern for staff and visitors; for example, noise in general has been shown to alter staff stress levels, impact job performance, induce hearing loss at high noise levels, generate annoyance, and cause an increased rate of burnout [2–6]. However, this paper will focus specifically on patients. For additional information on staff response, refer to other sources including [7] and [8]. In addition to the direct connections between noise and patient physiology described in this paper, other secondary relationships between hospital noise and patient health could be possible. For example, some have voiced concern over the potential of noise to degrade the ability of staff to orally communicate [7,8]; this is an issue potentially related to patient safety but such secondary effects are not the focus of this review article.

It is important to note that the conclusions drawn by the authors in this paper are limited due to the concerns about some of the previous literature, including (1) the limited subject population both in size and condition type, (2) the lack of detail about acoustic methodologies, (3) the presence of some conflicting results, and (4) very few studies published on some topics (eg, pain management).

NOISE OVERVIEW

Noise is often referred to as unwanted sound. Kryter states that noise evaluations are useful to "assess, or predict the unwantedness, disturbance, objectionableness, undesirability, unacceptability, perceived noisiness, or simply the noisiness of the sound environment in real life" [9]. Noise can be measured in many ways. A common unit of measurement is the decibel (dB) which is a measurement of the energy contained in noise relative to the very minimum amount of energy average humans can detect. Often weightings (ie, filters) are applied to better simulate the human ear's perception of loudness across frequency. A-weighting (dBA) is the most com-

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mon example and works well for a large range of average noise levels, whereas C-weighting (dBC) is sometimes applied to very loud noises. Sound frequency, measured in hertz (Hz), is related to human perception of pitch. For example, a piccolo is a high-frequency instrument and a tuba is a low-frequency instrument. Bandwidth refers to the range of frequencies included in a particular sound. If sounds are comprised of a wide range of frequencies (such as air-conditioning noise), they are referred to as broadband. On the other hand, a single note, or tone, would be referred to as narrow-band.

Human perception of loudness is highly complex; however, some approximations of typical sounds given for reference are: quiet residence (40 dBA), private office (50 dBA), conversational speech (60 dBA), vacuum cleaner (70 dBA), heavy traffic (80 dBA), pneumatic hammer (100 dBA), jet aircraft (120 dBA) [11,12]. The World Health Organization (WHO) recommends average noise levels are no more than 35 dBA in rooms where patients are treated or observed and no more than 30 dBA in ward rooms [10], although a recent landmark study showed that no hospital noise results published since 1960 meet these guidelines [7].

The noise normally occurring inside rooms is often referred to as "background noise." In hospitals, background noise can result from a variety of sources including air-conditioning systems, medical devices such as respirators, and occupant sounds such as conversation. Impulsive noises, or very loud, short duration events, are also commonly found in hospitals (eg, doors slamming, metal-to-metal contact, alarms). Two other types of noise sometimes used in acoustics experiments are white noise and pink noise. White noise has a constant amount of energy across frequency whereas the energy in pink noise changes with frequency. Both types of noises sound similar to TV or radio static (ie, turned on but not tuned to a station). Another important measure of sound in rooms is reverberation time (RT). RT is a measure of energy decay. It is related to volume and absorption; generally larger spaces with less absorption (harder, reflective surfaces) have longer RTs.

SLEEP DISTURBANCE

Numerous articles show negative relationships between hospital noise and sleep [13–29]. Methods include polysomnography (PSG), structured questionnaires, interviews, and electroencephalography (EEG) [13–26]. Although the percentage slightly varies, it has generally been shown that roughly 11% to 20% of arousals and awakenings are due to noise [13]. In the experiments involving sleep disturbance, researchers have often focused on disturbances that occur within 3 seconds of a measurable increase in noise, eg, greater than 10 to 15 decibels (dBA).

As early as 1976, investigators studied how the quality and quantity of sleep in a respiratory intensive care unit (ICU) was affected by noise [14]. Ten patients were monitored using PSG, interviews, and observations for 48 hours and exposed to sounds such as speech, equipment noise, alarms, phones, tapping of chairs and rails, radios, construction noise, and heating, ventilation, and air-conditioning (HVAC) noise. None of the 10 patients completed 1 undisturbed sleep cycle, and only 1 patient had sufficient sleep time for even the possibility of a complete sleep cycle. This study described an average normal night's sleep as consisting of 4 to 5 sleep cycles, with each cycle lasting 90 to 120 minutes. The study concluded that patients would have difficulty sleeping normally due to interruptions such as noise caused by personal and environmental noises. Aurell and Elmqvist studied 9 subjects in a postoperative ICU using PSG, EEG, and interviews [13]. A reduction of environmental noise was also made to compare a quiet environment to the noisy environment. The degree of change was unreported, but with these acoustical alterations all 9 subjects experienced sleep deficiencies.

Topf et al studied the interaction between noise and the suppression of rapid eye movement (REM) sleep with a sample of 70 women, comparing quiet conditions with noisy conditions [16]. Approximately one-quarter of sleep time is normally spent in REM, which consists of episodic bursts of rapid eye movements along with heart rate, respiration irregularities, and paralysis of major muscle groups other than some respiratory muscles [15]. REM sleep is believed to help with cognitive factors such as memory retention and learning capabilities. Although the Topf et al study took place in a sleep lab, the subjects were exposed to a recording of nighttime coronary care unit (CCU) noise at 84 dB [16]. Noise affected the quality of REM sleep compared to patients in quiet conditions; specifically, the subjects exposed to noisy CCU sounds experienced shorter REM periods and less REM activity. The authors concluded that noise acts as a suppressor to REM.

Another study by Carley et al tested 5 adults to see if acoustic stimulation can cause a sleep disturbance [17].

The acoustic signals consisted of 2 binaural tone bursts: a 0.5-second 4 kHz tone at 85 dB SPL and a 99-second interstimulus interval produced by a tone generator. An EEG arousal could be evoked in non-REM sleep with an acoustic stimulation. Furthermore, respiratory activity increased after an acoustic stimulation that was independent of general electrocortical arousal.

Aaron et al sought to determine the level and number of noise peaks needed in order to create a sleep arousal [18]. In this study, an EEG arousal was defined as awake state or alpha rhythm of at least 3 seconds' duration, occurring following at least 30 seconds of sleep [18]. This study took place in an intermediate respiratory care unit and utilized 24-hour PSG measurements. Although this study only had a sample size of 6 subjects, the researchers concluded that noise peaks greater than 80 dB(A) can correlate to sleep arousals. The authors noted that follow-up studies were needed to identify the sources of the noise peaks. Freedman et al used a larger sample size of 22 patients in a medical ICU where 20 of the patients were mechanically ventilated [19]. The mean noise level was measured as 59.1 dBA in the day and 56.8 dBA at night. They found that environmental noise was not the main reason for sleep fragmentation, but noise was partially responsible. Specifically, 11.5 and 17% of the total arousals and awakenings were related to noise, respectively. The exact sources of noise were not described.

There has also been investigation into whether the differences between peak and average background noise levels are important. Stanchina et al tested 5 subjects and recorded 1178 arousals using PSG [20]. Subjects were exposed to recorded sounds from an ICU that included patient-staff interactions, alarms, ventilators, equipment noise, and others. Another recording combined these sounds with white noise. The average level of the ICU noise-only was 57.9 dB and the combined ICU and white noise was 61.1 dB. The addition of white noise lowered the number of sleep arousals caused by ICU noise even though the average noise level increased.

Another study compared the different contributions of ICU noise and patient-care activity noise to sleep disruption [21]. Six healthy male patients and 7 mechanically ventilated male patients were studied using a 24-hour PSG and a structured questionnaire was administered to the healthy subjects. The patient population was already admitted to an ICU, whereas the healthy population consisted of subjects who volunteered to spend a 24-hour period in an ICU. For the mechanically ventilated subjects, the average daytime and nighttime noise levels were 56.2 and 53.9 dB, respectively. For the healthy subjects in an open ICU, the average daytime and nighttime levels were 55.6 and 51.4 dB, respectively. For the healthy subjects in a single room, the average daytime and nighttime levels were 44.3 and 43.2 dB, respectively. Results showed ICU noises and patient-care activities accounted for less than 30% of the sleep disruptions. The remaining arousals and awakenings were caused by opening and closing the ICU main door, which was located near the patients. The patients generally slept worse, meaning they generally exhibited more awakenings and arousals per hour and a shorter sleep time as compared to the healthy subjects. For example, patients slept an average of 6.2 hours total, whereas healthy subjects slept an average of 8.2 to 9.5 hours total depending on if they were in an open bed location or a single room. They also determined that about half of the patient's sleep occurred during the day.

Questionnaire-only studies have also analyzed the relationships between noise and sleep disturbances in lieu of direct physiological measurements. Topf et al exposed 60 females to an 8-hour audiotape of coronary care unit (CCU) noise from monitoring devices, ventilators, suction machines, drains, oscilloscopes, and staff [22]. The subjects reported a general negative impact on sleep, including longer time to fall asleep, more awakenings, and fewer hours sleeping. Another survey of 50 general ICU patients used the Intensive Care Unit Environmental Stressor Scale (ICUESS) and revealed that high stressors such as pain and noise were also factors in patients' inability to sleep [23]. Another study of 203 patients revealed that patients felt their sleep was significantly worse in an ICU than at home [24]. Specifically, staff communication and alarms were the most disruptive to sleep, whereas telephones, televisions, beepers, and equipment noise were not as disruptive.

Observational studies also provide insight. Dlin et al observed ICU patients and recorded interruptions to sleep [25]. All but 3 of the patients in the ICU participated, although specific sample size was not given. Patients were interviewed and asked about sleeping patterns at home, in previous hospitalizations, and in the current hospitalization. Additionally, staff members observed the patients as "frequently as possible" without structured questioning, and one final staff observation following release from the ICU. Each staff observation consisted of logging interruptions such as discrete events which impacted the patient in a direct manner (such as the tak-

ing of blood pressure) and noted the nature, duration and response of the interruption. They observed that the main deterrent to sleep was activity and noise.

Monson and Edéll-Gustafsson implemented various changes in a neuro-ICU including noise-reducing medical and nursing routines and afternoon and night non-disturbance periods [26]. Two patient groups were analyzed: 9 before and 14 after. The total number of sleep disturbance factors over a one-week span before the behavioral modification program averaged 194.3 sleep disturbances, as compared to 162.1 after. Another program by Walder et al implemented 5 guidelines including systematic closure of doors, a reduction of intensity of alarms, efforts for low conversation, and coordination and limitation of nursing interventions in sleeping hours [27]. Results showed a lowering of average and peak noise levels after the changes, but reported that sleep patterns could still be disrupted. A similar study by Kahn et al attempted to reduce peak noise sources in both medical and respiratory ICUs [28]. An observer was present in the measurements to note the noise sources. They recorded noises such as HVAC, medical equipment, televisions, telephones, intercoms, beepers, and conversations. The mean peak levels were 80.0 and 78.1 dBA, before and after behavioral modification, respectively. There was significant reduction in the total number of peaks exceeding 80 dBA, from 1363 periods out of 2880 possible periods to 976 periods out of 2811 possible periods. Specifically, between 6 am and 12 am, there was a significant reduction of noise peaks exceeding 80 dBA. The authors suggested that sleep can be improved due to this reduction of noise peaks, but they left the topic for a future study.

Studies show that factors of the acoustic environment besides noise can correlate to sleep disruption. For example, sleep has been related to reverberation time (RT). A study by Berg, which took place in a refurbished former surgical ward, focused on the effects of RT on noise-induced sleep arousals using EEG for 12 subjects [29]. RT was reduced by an average 26% after the installation of sound absorptive ceiling tiles. Noise ranged from 27 to 58 dB(A), coming from both continuous and impulsive sources such as dropped plates, traffic noise, fan noise, machine noise, doors closing, and radios. Results showed that the installation of the sound absorptive ceiling tiles did not significantly change noise levels, but did significantly reduce the number of sleep arousals.

CARDIOVASCULAR RESPONSE

Cardiovascular response is also related to the acoustical environment [30–36]. Some of the earliest studies revealed that heart rate, blood pressure, and other cardiovascular measures can be affected by noise. Falk et al studied the relationship between vasoconstriction and noise intensity and bandwidth [31]. At noise levels greater than 70 dB there exists a linear relationship between increases of noise intensity and increases in vasoconstriction. Further, an exposure to 90 dB of white noise could cause an immediate vasoconstriction with a recovery time of about 25 minutes after the white noise was turned off. The severity of vasoconstriction was a function of bandwidth of the noise—as the bandwidth increases, the vasoconstriction worsens.

Conn related heart rate, frequency of arrhythmias, and state of anxiety during quiet and noisy periods in a coronary care unit (CCU) [32]. Twenty-five male patients were exposed to 1-minute noise recordings between 3 to 4 pm and 7 to 8 pm. The results showed that anxiety was heightened and the number of ventricular arrhythmias rose significantly during the "noisy" periods, defined to be periods of noise greater than 55 dB.

Further research has related changes in heart rate with types of noise source. One study found that the average heart rate increased due to the presence of human sounds (talking) [33]. Another study of 28 patients in a surgical ICU showed an increase in heart rate due to talking inside a patient's room [34]. The average noise levels ranged from 49.1 to 68.6 dBA. When there were noise events that caused an increase of 3 dBA or greater in overall noise level, 89% of the tests showed an increase in heart rate more frequently than a decrease in heart rate. For 46% of the tests, this increase was statistically significant. When sound pressure levels showed a 6 dBA increase, the heart rate also rose from two to twelve beats per minute. Additionally, heart rate significantly increased for impulse noise. A similar study compared ambient stressors of equipment sounds to social stressors like conversation in a CCU [35]. Measurements were taken 3 times a day over 2 days with 20 subjects. 55% of the hospital noise was conversation in the room, 20% of the noise originated from background sound, 15% from hall conversation, and 10% from environmental sound. Although noise did not significantly affect blood pressure, heart rate was elevated during social stressor conversations compared to quiet ambient conditions. Also, heart rate was about 3 beats per

minute faster during conversational sounds than during environmental sounds.

Similar to the sleep study with variable acoustics described earlier [29], Hagerman et al examined blood pressure and heart rate in a "good" and "bad" acoustical environment [36]. Subjects were 94 patients in an intensive coronary heart unit. Absorptive acoustical ceiling tiles were added that decreased reverberation time by 50% in the main work area and by 56% in the patient rooms. Also, the absorption decreased overall noise levels by 5 to 6 dB in the patient rooms. Even though there was no significant difference across the entire group in heart rate, blood pressure, or pulse amplitude, there were significant differences when analyzing the data by type of disease. Pulse amplitude is the difference between systolic and diastolic blood pressure [36]. In acute myocardial infarction and unstable angina pectoris groups, the heart pulse amplitude was higher with the "bad" (ie, less absorptive) acoustical setting at night. Additionally, patients in the "bad" acoustic setting were re-hospitalized more frequently at 1- and 3-month follow-up.

GASTRIC ACTIVITY

Effects of noise on gastric activity are somewhat unclear. Sonnenberg et al examined the link between cardiovascular response, mental stress, gastric acid secretion, and noise [37]. One phase tested 10 male subjects and exposed them to 90 dBA of broadband noise for 1 hour. Blood pressure, heart rate, and respiratory rate were measured. The second phase tested the gastric response of 14 male subjects after exposing them to 90 dBA of broadband noise and a gastric stimulation. Results showed that both diastolic and systolic blood pressure increased by 4 and 8 mm Hg, but heart rate and respiratory rate were not affected. However, the noise did not affect gastric acid secretion. Another experiment where 50 dyspeptic subjects were exposed to 95 dB pink noise for 15 minutes also showed no relationship between gastric secretion and noise [38]. Sonnenberg et al also reported previously unpublished results by J.F. Erckenbrecht that found that noise significantly increased small bowel transit time, stool frequency, and stool volume [37].

In another study [39] 21 male subjects were exposed to a 110-minute recording that simulated different noise sources—hospital noise at 87.4 dBA, conversation at 91.3 dBA, and traffic at 85.6 dBA. Gastric activity was studied via gastric myoelectrical activity (GMA), which controls stomach motility. GMA was measured via an electrogastrogram (EGG) using standard electrocardiogram (EKG) electrodes on the upper abdominal wall. Three cycles per minute (CPM) GMA is common for humans. Results showed that hospital and traffic noise exposure significantly decreased the percentage of 3 CPM activity. There was also a nonsignificant decrease in percentage of 3 CPM activity with respect to conversation noise. The authors concluded that loud noise can alter gastric myoelectrical activity.

WOUND HEALING

Wound healing and noise has thus far primarily been studied in animals [40–43]. Although some potential effects on humans can be surmised, it is important to acknowledge that the response of humans to noise in general is more complex. One wound healing experiment exposed rats to 80 dB of rock music for a 22-hour time period and then measured changes of leukocyte function [40]. The rock music was turned off periodically to prevent habituation. Lymphocyte function remained unchanged in the presence the noise. However, short-term noise exposure did cause an alteration of the superoxide anion and interleukin-1 secretion of neutrophils and macrophages, thus decreasing wound healing.

Wysocki measured wound surface area and found that the wounds healed slower in a group of rats exposed to random white noise at 85 dB [41]. The noise was played intermittently for 15 minutes for 19.5 days. Additionally, the average weight of the exposed group of rats was lower even though food intake was the same between the exposed and unexposed group. In another study, 119 mice exposed to temperature and noise stressors were inflicted with a small wound [41]. The noise stressor consisted of 99 dBC white noise. Results showed noise slowed down healing rate (ie, reduction of wound area) but noise affected the healing rate less than temperature stressors.

Healing rate can also be measured by the hormone secretion of the suprarenal cortex [43]. 124 albino rats had a patch of skin removed from part of the back. Healing rate was measured by 2 methods: size reduction of the wound and weight of the suprarenal gland. The rats were exposed to combined environmental stressors that included flashes of light, a ringing bell, and scraping metal wheels. The stressors slowed wound healing in male but not female rats. Within the male group, the average difference in total healing was about 8 days.

Table 1. Categorical Breakdown of Papers Reviewed

Outcome	Authors	Year
Sleep	Aurell & Elmquist	1985
	Parthasarthy& Tobin	2004
	Freedman et al	2001
	Gabor et al	2003
	Cooper et al	2000
	Topf & Davis	1993
	Carley et al	1997
	Aaron et al	1996
	Stanchina et al	2005
	Topf et al	1996
	Novaes et al	1997
	Freedman et al	1999
	Dlin et al	1971
	Berg	2001
	Monsen & Edell-Gustafsson	2005
	Walder et al	2000
	Kahn et al	1998
Pain	Minkley	1968
	Gardner et al	1960
Cardiovascular	Falk & Woods	1973
	Cantrell	1979
	Storlie	1976
	Conn	1981
	Marshall	1972
	Baker	1992
	Baker et al	1993
	Hagerman et al	2005
	Sonnenberg et al	1984
Hospital stay	Fife & Rappaport	1976
Wound healing*	McCarthy et al	1992
	Wysocki	1996
	Cohen	1979
	Toivanen et al	1960
Other responses	Castle et al	2007
	Tomei et al	1994

*Studies conducted on animal subjects.

OTHER RESPONSES

Only one study relates noise to average length of hospital stay [44]. The study compared the hospital stay length of 416 cataract patients while the hospital was and was not under construction. Hospital stay was longer for patients during the louder construction periods, with average length increasing about 1 day.

Only one study links hospital noise to patient pain management [45]. The range of noise levels was related to the number of patients requiring pain medication, such as narcotics, in a 10-bed recovery room. Results showed an increase in noise was related to an increase in pain medication. On the other hand, noise can potentially be used into reduce pain sensations in certain cases. Gardner et al exposed 1000 dental patients to ordinary dental office sounds such as dental drills [46]. The patients wore earphones and they were able to adjust the level of either orchestral music or white noise, noting that the white noise sounded similar to a waterfall. Sixty-five percent of the patients were able to use this audio-analgesic effectively for procedures that usually elicit the use of nitrous oxide or local anesthesia. The authors theorized that the music and white noise acted as analgesics due to their relaxing, soothing nature and because they masked sounds of dental drills, therefore reducing patient anxiety during procedures.

DISCUSSION OF RESULTS

It is clear from this literature review that hospital noise is a serious issue potentially linked to several types of negative reactions in patients. **Table 1** summarizes the studies described in this review by author and outcome.

Patient sleep has been shown to be negatively affected by the sound environment. A number of research methods, including PSG, EEG, patient questionnaires, and observational studies have confirmed relationships between noise and sleep length, quantity, or quality. The occurrence rate of sleep arousals tends to rise with exposure to heightened background or peak levels. There have been many studies conducted on the number of noise-induced sleep arousals within different sleep stages. Previous studies have also determined which noise sources, such as staff talking and telephone noise, arouse the patients most frequently and these sources typically become the targets for noise reduction programs. One of the major concerns with these studies is that generally small sample sizes are used in the initial experiments (eg, as few as 5 patients); thus, the applicability of these results to larger populations must be scrutinized.

Cardiovascular response has been related to noise exposure. Occurrences of vasoconstriction and increases in heart rate and blood pressure due to the presence of acoustical stimuli have been shown. Additionally, anxiety and arrhythmia episodes are more frequent in noisy scenarios. The addition of acoustic absorption can

potentially help offset the levels of noise and reduce the negative effects of noise on heart pulse amplitude and incidence of re-hospitalization. However, as a whole, results are not entirely consistent, with some studies showing changes and others not. Additionally, it is not yet known in general whether the presence of noise is directly linked to the onset of heart disease or other chronic cardiovascular problems.

Research on other health effects has been limited. For example, there are no known studies of noise and wound healing in humans. Relationships between noise and pain management are also unclear. The studies that do exist suggest the presence of noise may increase the need for pain medication but conversely, patient control over sound can potentially limit the need for certain medications. The field of music therapy is ripe with evidence suggesting the benefits of pleasing sounds, but was not the focus of this literature review. Rigorous studies are needed to relate noise to wound healing and pain.

The literature taken as a whole can be organized in a way that studies the entire research chain: from the acoustic metrics being tested through the mechanism being introduced to the occupant outcomes. For example, Berg [29] focused on how a change in reverberation time affects patient sleep; thus the acoustic metric is reverberation time, the mechanism is the addition of absorptive tiles, and the occupant outcome is the change in patient sleep arousals. The occupant outcomes shown in Table 2 were generally negative when the subjects were exposed to noise, ie, sleep quality was reduced, cardiovascular response was heightened, and healing rates were lowered. When environmental changes or behavioral changes were made to reduce noise or reverberation time, the outcomes were generally positive.

It is important to note in Table 2 under the "Metric" column several studies show blank acoustic metrics. This does not mean that acoustics were not considered in the studies; rather, it reveals that specific acoustic metrics were not systematically altered. For example, in Carley et al [17], 85 dB tones were used in the study for noise exposures. A change in decibel value was not specifically studied; instead, it is the exposure to noise that was of interest. Alternatively, in the Hagerman study [36], the acoustic metric that was altered and controlled for was reverberation time (value reduced by 50%–56%). The mechanism was that they added absorptive panels. The outcome was that patients were re-hospitalized at a higher incidence when the reverberation time was longer. Hence, the Hagerman study [36] systematically altered reverberation time, as opposed to Carley et al [17] that drew their conclusion from the exposure itself, not from an acoustic metric.

CONCLUSIONS

This review reveals that hospital noise is a serious issue linked to several potential negative reactions in patients. The clinical significance of these results is not entirely known, but some hypotheses are possible. Sleep is fundamental to human health in general and critical to patient recovery. Alertness, mood, behavior, coping abilities, respiratory muscle function, ventilatory control, healing time, and length of stay are just a few of the potential impacts of patient sleep disturbance or deprivation [25,28,47]. Cardiovascular or other arousals due to noise stressors can also impede patient recovery. As Baker states, "stress results in compensatory biological changes, thus redirecting or exhausting resources that might otherwise be available to combat the original disease process" [35]. Additional research is needed to determine the chronic implications of noise on patient health.

Very few studies examine the entire research chain (acoustic metrics to mechanism to outcome). Thus, the conclusions drawn by the authors in this paper are limited due to concerns such as subject population, lack of detail about acoustic methodologies, the presence of some conflicting results, and limited number of publications on some topics. Regardless, this literature review concludes that hospital noise is a serious issue that can negatively affect patient physiology and more research is needed.

The ultimate goal should be to identify ways to improve the acoustic environment but generally only rudimentary measures (dBA) have been reported. These acoustic metrics may be overly simplistic for hospital environments [7,8]. Additionally, a number of "mechanism" studies evaluating changes in the acoustic environment are needed in order to optimize the effectiveness of acoustic or behavioral alterations. Already, the use of absorptive ceiling tiles has been shown to positively impact patients. Other acoustical variables such as room shape or equipment selection should also be investigated in detail. With a better understanding of how interventions impact the acoustics and therefore occupant outcomes, strides can be made in filling the holes in the research chain and providing a healthier atmosphere for patients, staff, and visitors.

Table 2. Papers that Fully or Partially Highlight the Research Chain of Metrics, Mechanisms, and Outcomes

Patient Outcome	Article	Acoustic Metric(s)	Mechanism*	Outcome(s)
Sleep				
	Hilton 1976		A	No undisturbed sleep cycles
	Aurell & Elmquist 1985		B-	Sleep deficiencies seen in all subjects
	Parthasarthy & Tobin 2004		A	11-20% of arousals and awakenings due to noise
	Topf & Davis 1993		A	REM sleep quality changed--shorter REM periods and less REM activity
	Carley et al 1997		A	EEG arousal evoked in non-REM sleep. Respiratory activity was increased.
	Stanchina et al 2005	Peak levels	A	Increased number of sleep arousals
	Topf et al 1996		A	Reported a negative impact on sleep
	Berg 2001		C	Significantly reduced the number of arousals during sleep
	Monsen & Edell-Gustafsson 2005		D	Reduced number of sleep disturbances
	Walder et al 2000	L_{Aeq} and Max Levels	D	Sleep patterns altered
	Kahn et al 1998	Peak levels	D	Number of noise peaks reduced but no physiological component to study
Cardiovascular				
	Storlie 1976		A	Determined that change in heart rate possible
	Conn 1981		A	Anxiety was heightened and arrhythmias rose
	Marshall 1972		A	Heart rate increased by 2-12 BPM
	Baker et al 1993		A	Heart rate increase ~3bpm primarily during in conversations only
	Hagerman et al 2005	Reverberation time	C	Speech intelligibility improved. Patients re-hospitalized at higher incidence in "bad" acoustics.
	Sonnenberg et al 1984		A	Blood pressure increased
Hospital Stay				
	Fife & Rappaport 1976		A	Hospital stay length increased
Wound Healing				
	McCarthy et al 1992		A	Leukocyte function altered
	Wysocki 1996		A	Weight loss
	Cohen 1979		A	Slower healing area rate
	Toivanen et al 1960			Healing rate lowered
Others				
	Castle et al 2007		A	Decreased gastric myoelectrical activity
	Tomei et al 1994		A	No link to physiology

*A = exposure to noise; B(+ or -) = increase or decrease in noise level; C = installed absorptive panels; D = administrative behavioral changes.

Hospital caregivers and administrators can begin acting on current knowledge to improve the hospital noise environment. A combination of administrative strategies (eg, behavioral modifications, quiet zones, changing alarm settings) and design strategies (eg, sound absorbing materials, architectural layout) are needed. As described earlier in this paper, administrative approaches such as behavioral modification programs have been

shown to be effective in improving sleep and reducing noise levels [26–28]. Other studies discussed earlier have shown that design strategies such as adding sound absorption can improve sleep [29] and reduce incidence of rehospitalization [36]. Caregivers and administrators can work with a building acoustical consultant who is versed in evidence-based hospital design. Ryherd and Zimring present information on some design strategies available and provide basic recommendations about how to facilitate collaborations with acoustic consultants [48].The consultant can help incorporate design strategies such as reducing noise from building systems like air-conditioning, changing communication systems to reduce overhead paging, using sound absorbing materials that meet hospital safety requirements, and incorporating floor plan layouts that are conducive to good acoustics.

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