Minimal Olfactory Perception During Sleep: Why Odor Alarms Will Not Work for Humans

Mary A. Carskadon, PhD[1]; Rachel S. Herz, PhD[2]

[1]Department of Psychiatry and Human Behavior, Sleep Research Laboratory, Brown Medical School/Bradley Hospital and [2]Department of Psychology, Brown University, Providence, RI

Study Objectives: To examine olfactory arousal threshold during sleep in comparison to an auditory tone.

Design: On night 1, participants rated odor intensity when awake and experienced olfactory stimuli during stage 1 sleep. Night 2 involved stage 2, stage 4, and rapid-eye-movement (REM) sleep trials using the "staircase" threshold-detection method. Electroencephalogram, electrooculogram, electromyogram, electrocardiogram, and respiration were recorded along with behavioral response. An 800-Hz tone was given on trials when odors failed to arouse.

Setting: Participants slept in individual rooms. Stimulus-delivery systems were operated from a separate room, where an experimenter observed physiologic recordings and behavioral responses.

Participants: Three healthy men and 3 women aged 20 to 25 years (mean, 22 years).

Interventions: Two odorants, peppermint and pyridine, at 4 concentrations were presented through nasal cannulas using an air-dilution olfactometer. Tones were played over a speaker.

Measurements: Behavioral (button press and oral) responses, electroencephalographic activation, and changes in breathing and heart rate were assessed.

Results: Participants responded to odors on 92% of stage 1 sleep trials. Peppermint was ineffective in stages 2, 4, and REM sleep. Pyridine produced behavioral threshold on 45% of stage 2 trials, none in stage 4, and one third of REM sleep trials. Tones were effective on at least 75% of trials. Heart rate increased significantly only following behavioral responses to odors or tones across sleep stages.

Conclusions: The data indicate that human olfaction is not reliably capable of alerting a sleeper.

Key Words: arousal threshold, olfaction, sleep, autonomic response

Citation: Carskadon MA; Herz RS. Minimal olfactory perception during sleep: why odor alarms will not work for humans. *SLEEP* 2004;27(3):402-5.

INTRODUCTION

MANY STUDIES HAVE SHOWN AUDITORY PROCESSING DURING SLEEP AND SLEEP DISRUPTION BY AMBIENT NOISE.[1-4] Furthermore, simple auditory stimuli during waking provide significant informational content without need for contextual mediation. By contrast, conclusive data on olfactory detection during sleep are scarce,[5,6] and waking olfaction is regarded as the sense most heavily influenced by external cognitive cues for informational mediation.[7,8] These differences in attentional and cognitive mediation suggest that olfactory and auditory processing will be differentially affected during human sleep.

Several basic features of olfaction are well understood. Most current theories point to combinatorial coding mechanisms where molecules elicit unique temporal and spatial patterns of olfactory-receptor firing that are centrally interpreted as specific odors.[9] Individual differences in olfactory sensitivity (eg, sex and age) are also well known, and such factors as menstrual-cycle phase and training are mitigating variables.[10,11] Detection thresholds for individual odorants vary widely; for example, humans detect tertiary butyl mercaptan at concentrations of less than 1 part per billion in air versus an everyday odor such as isopropyl alcohol at 10 parts per million. Most odors stimulate the trigeminal nerve to some degree, which is relevant to detection; anosmics unable to detect weak trigeminal odors can fully detect strong trigeminal odor.[12] Familiarity also influences odor sensitivity; recognition and detection are better with more experience and with ability to name odors.[13] Individuals who fail to detect an unusual ambient odor are able to perceive the scent after a label is provided.[14] Finally, odors eliciting strong hedonic responses may be detected at lower thresholds than hedonically 'neutral' odors. Thus, odor detection by humans is strongly mediated by cognitive, experiential, and affective factors that are independent of peripheral physiology.

In contrast, simple auditory detection functions in physiologic accord with stimulus intensity relatively free from contextual mediation.[15] Nevertheless, auditory-arousal threshold is altered by sleep. Indeed, increased arousal threshold is a hallmark of sleep. Zepelin and colleagues[16] showed in young adults that auditory-arousal thresholds to tones (40 to 115 dB) were elevated in sleep, more so for stage 4 than for stage 2 or rapid eye movement (REM) sleep. Less is known about olfaction during sleep; 1 olfaction study has shown demonstrable behavioral responsiveness to peppermint odor during sleep, though at levels markedly lower than during waking.[5]

We selected 2 odors of comparable trigeminal strength but of opposite hedonic valence as our olfactory stimulants: peppermint is a pleasant odor and pyridine is unpleasant and aversive at high concentrations. Equivalence of trigeminal strength was inferred from the findings of Doty and colleagues[12] whose anosmic patients were able to detect pyridine and menthol (from which peppermint oil is partly derived). Thus, individuals unable to "smell" the odors were able to detect both with 100% accuracy (15 of 15 participants) through trigeminal activation. The present study used arousal-threshold detection methods to examine the sleep-related response to olfactory stimuli of opposite hedonic valence. A moderate auditory stimulus was used in trials when the odorant failed to produce arousal to identify whether another modality would produce arousal. Behavioral, electroencephalographic (EEG) activation, and autonomic responses were evaluated.

METHODS

Participants were 3 men and 3 women aged 20 to 25 years (mean = 22 years), screened by telephone interview and sleep questionnaire for the absence of sleep or other health problems and normal olfactory function.

Disclosure Statement
No significant financial interest/other relationship to disclose.

Submitted for publication August 2003
Accepted for publication December 2003
Address correspondence to: Mary A. Carskadon, PhD, E.P. Bradley Hospital Sleep Research Lab, 300 Duncan Drive, Providence, RI 02906; Tel: (401) 421-9440; Fax: (401) 453-3578; E-mail: mary_carskadon@brown.edu

They kept a regular approximately 8-hour sleep schedule for 1 week, documented by sleep-wake diaries. Women were studied during the late follicular menstrual phase because waking olfactory sensitivity is greatest during this phase.[10] The project was approved by the Institutional Review Board of the E.P. Bradley Hospital, and participants received modest compensation.

Stimulus Presentation Apparatus

Odors were presented using an air-dilution olfactometer based on the Dravnieks design.[17] An aquarium pump passed a constant flow of air (minimally perceptible when awake) to the subject via intranasal cannulas, with valves directing air through 1/8-inch diameter Teflon tubing into 1 of 9 thirty-milliliter glass tubes, 1 empty and 8 containing 15 mL of pyridine or peppermint oil (International Flavors and Fragrances, Union Beach, NJ), at 100% concentration and at 3 successive one-third dilutions prepared in diethly phthalate. No attempt was made to alter the odor of the nasal cannulas. The experimenter and olfactometer were in a separate room from the sleeper, with Teflon tubing connecting the olfactometer to the sleepers' intranasal cannulas through a wall conduit. Sleep-related olfactory trials began with airflow through the cannulas for 2 minutes at a constant rate until a trial series ended. The 800-Hz tones were presented through a speaker approximately 3 feet from the participant's head. Stimulus intensities were predetermined at a distance 3 feet from the speaker; no attempt was made to fix the participant's head position. To equate auditory signals across trials, intensity was based on mean levels effective to wake young adults in the Zepelin[16] study (stage 2, women = 78 dB and men = 85 dB; stage 4, women = 97 dB and men = 105 dB; REM, women = 85 dB and men = 80 dB).

Polysomnography

Participants were instrumented for central (C3/A2 or C4/A1) and occipital (O1/A2 or O2/A1) referential EEG leads according to the international 10-20 system[18]; electrooculogram from right and left outer canthi; mental/submental electromyogram; anterior tibialis electromyogram (night 1); nasal airflow (thermistor); abdominal respiratory effort (mercury-filled capillary strain gauge); and electrocardiogram (modified lead II). They wore intranasal cannulas for odorant presentation on both nights with an elastic chinstrap to encourage nose breathing by gentle chin pressure. A small response button was taped to the dominant hand connected to a separate polysomnography channel. The experimenter signaled trials on the record with a similar switch connected to another channel. Verbal responses were heard through a speaker in the room with the experimenter.

Procedures

To ensure that changes in olfactory perception did not occur overnight, we first tested whether the odors were detected equally in the evening and morning. Waking odorant assessments occurred on night 1 before applying electrodes and in the morning before removing electrodes. Participants made numeric assessments of stimulus strength using the Stevens[19] method. Randomly ordered 5-second exposures (1 per minute) were completed twice for all strengths of each odorant while awake. Sleep recordings began at participants' habitual sleep-onset times. Odorant trials were presented during stage 1 sleep no longer than 30 minutes after lights out, followed by undisturbed sleep and rising at usual wake-up times. Participants were instructed on both nights to press the button and state "I smell something" whenever they detected the presence of either odorant or to press the button and state "I hear something" if they heard the tone.

On night 2, sleep-related olfactory arousal-threshold trials occurred after at least 20 minutes of uninterrupted sleep. A contingency table was used to determine the timing of trials; for example, the next trial after an awakening had to follow at least 12 minutes of uninterrupted sleep; after a movement, at least 3 minutes; after a transient arousal, at least 2 minutes; after no arousal to an odorant, at least 2 minutes of sleep, etc. Arousal threshold for a given odorant was assayed within each of 3 sleep stages—stage 2 sleep, stage 4 sleep, and REM sleep—as determined using standard sleep-staging criteria[20] and as confirmed by subsequent evaluation of the polysomnography records. We measured odor threshold using the staircase method,[16] where trials begin with the weakest stimulus stepping up to stronger stimuli in the absence of a behavioral response and back to a weaker stimulus when a behavioral response is elicited. Thus, 2 behavioral responses to the same odor strength designates "threshold." To ensure that participants did not have extraordinarily deep sleep, when an olfactory arousal threshold was not achieved, we ended the series with an 800-Hz auditory tone. Four trials intended for stage 4 were subsequently determined as stage 3 sleep but were combined with stage 4 trials because no response was observed.

Odor trials lasted up to 15 seconds or until a behavioral arousal (button press or verbal response) occurred. Auditory trials lasted up to 5 seconds or behavioral arousal. EEG activations were scored off line if alpha rhythm was detected during the presentation of the odorant or tone or during 15 seconds after the presentation. Heart rate was evaluated for 15 seconds before and after stimulus presentations, converted to beats per minute (heart rate × 4), and differences were assessed with repeated-measures analysis of variance. Respiration was monitored to ensure that breathing continued during odor trials.

RESULTS

All participants detected both odors when awake. The more highly concentrated odors elicited higher intensity ratings, and pyridine consistently garnered higher ratings than peppermint. We found no presleep-to-postsleep difference in rated intensity (Table 1) nor any sex difference.

During stage 1 sleep in the first 30 minutes of night 1, participants responded behaviorally (button press, verbal response, or both) and manifested EEG activation (alpha rhythm) on 21 of 24 trials (12 for peppermint, 9 for pyridine); 1 pyridine trial showed EEG activation without behavioral response. Thus, 91% of odor trials in the early transition to stage 1 sleep elicited a response.

This report includes all *completed* (ie, to odorant threshold or to tone) arousal threshold *series* that were performed in non-REM (NREM) sleep stages 2 and 4 and in REM sleep. Thus, individual trials from series that were not finished, due to insufficient time in a particular sleep stage or end of night, are not included in this report. Inspection of those few individual trials showed findings consistent with those reported below. Figure 1 shows representative odor and tone trials.

For stage 2 sleep, we performed 8 complete peppermint threshold series, including 32 individual odor presentations without achieving threshold (Figure 2a, squares). No peppermint strength elicited a single behavioral response; however, EEG activation occurred twice at the lowest strength and 3 times at the highest. By contrast, 6 of 8 tone trials elicited behavioral responses (Figure 2b, squares), and EEG activation

Table 1—Magnitude Estimation Summary

Pyridine Presleep Trials				Pyridine Postsleep Trials			
Strength	No.*	Mean Rating	SD	Strength	No.*	Mean Rating	SD
1	5	3.60	2.07	1	4	3.75	1.26
2	12	8.33	3.50	2	12	7.21	3.24
3	12	8.04	3.65	3	12	7.29	3.99
4	9	7.94	2.98	4	8	7.25	3.53
Peppermint Presleep Trials				**Peppermint Postsleep Trials**			
Strength	No.*	Mean Rating	SD	Strength	No.*	Mean Rating	SD
1	9	1.83	0.71	1	10	2.30	0.95
2	8	2.44	1.78	2	7	2.57	1.51
3	12	3.17	1.57	3	10	2.80	1.21
4	11	3.14	1.03	4	11	3.05	1.19

*Number of ratings. All subjects received 2 trials of each stimulus at every strength in presleep and postsleep trials; failure to rate the stimulus was excluded from the data set.

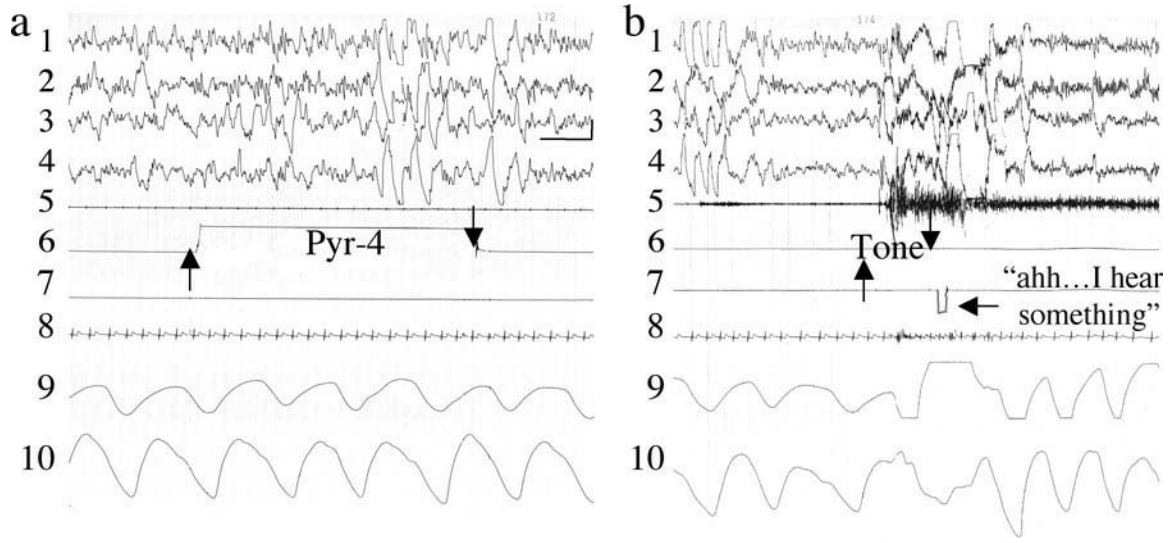

Figure 1—Two consecutive trials in a man. For both, 1=C3/A2; 2=O2/A1; 3=ROC/A1; 4=LOC/A2; 5=EMG; 6=odor presentation signal; 7=participant signal; 8=heart rate; 9=nasal thermistor; 10=abdominal strain gauge. The amplitude calibration (50μV) is appropriate for EEG channels; the time calibration is 3 seconds. **(a)** Pyridine trial during stage 2 sleep with undiluted pyridine (Pyr-4). Up arrow indicates start and down arrow end of stimulus presentation. No behavioral, EEG, or autonomic response occurred. **(b)** Subsequent tone trial (105 dB) during which the participant moved, awoke, signaled, gave a verbal response, and manifested autonomic changes.

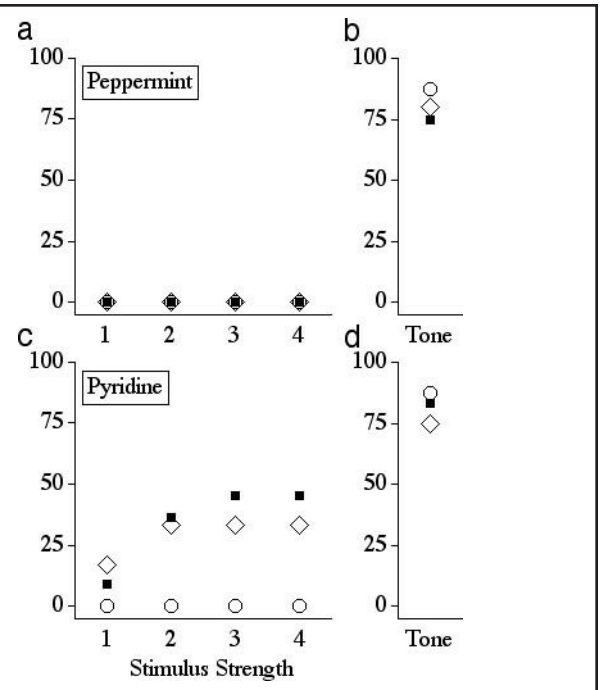

Figure 2—Threshold determinations for peppermint and pyridine in NREM stages 2 and 4 and REM sleep. For all panels, ■=stage 2, ○=stage 4, and ◇=REM sleep; strength 1=most diluted, strength 4=undiluted odor. **(a)** Cumulative percentages of arousal threshold series ending with behavioral arousals for peppermint and **(c)** pyridine. **(b)** Percentage of arousal to tones ending peppermint and **(d)** pyridine series.

alone occurred on 1. Eleven complete pyridine series in stage 2 sleep included 52 presentations. Threshold occurred at the lowest strength in 1 series, at strength 2 in 3 series, and at strength 3 in another (Figure 2c, squares). No other pyridine trials during stage-2 sleep evoked a behavioral response. Five of 6 tone trials elicited behavioral responses (Figure 2d, squares), and 1 elicited only EEG activation.

Stage 4 sleep is generally considered to be "deeper" than stage 2, and our results confirmed this notion for odor detection. We completed 8 stage-4 peppermint series with 32 trials without achieving threshold (Figure 2a, circles); no behavioral responses and only 1 EEG activation (strength 4) occurred. On the other hand, behavioral responses to tones ended 7 series (Figure 2b, circles), with EEG activation alone on the eighth. The 8 completed pyridine series in stage 4 sleep included 34 individual trials without achieving threshold (Figure 2c, circles) and showed behavioral response (pyridine strength 3) only once and EEG activations at strengths 1, 2 (twice), and 4. The tone produced behavioral responses on 7 trials (Figure 2d, circles), with EEG activation alone on the eighth.

REM sleep, commonly associated with dreaming, may show variable sensory response due to dream incorporation of stimuli.[16] We completed 5 peppermint olfactory arousal-threshold series, including 20 individual trials in REM sleep. Neither threshold (Figure 2a, diamonds) nor single behavioral responses occurred, although EEG activation following peppermint administration occurred during 1 or 2 trials of each strength. The tone, on the other hand, produced a behavioral response on 4 of 5 occasions (Figure 2b, diamonds), with an EEG activation on the fifth. Finally, we completed 6 REM-sleep pyridine series, including 22 individual trials. Threshold was achieved once at strength 1 and once at strength 2 (Figure 2c, diamonds); EEG activation in the absence of behavioral response occurred once at strengths 2, 3, and 4 in other series. Behavioral responses occurred 3 times in 4 tone trials (Figure 2d, diamonds), the fourth eliciting only EEG activation.

As to autonomic response (Table 2), heart rate did not change when odorants failed to elicit behavioral responses, even if EEG activation occurred. On the other hand, heart rate increased significantly in the 15 seconds following behavioral responses to odors or tones across sleep stages. Our measure of breathing revealed no apneas or slowed breathing rate during odor trials, although behavioral responses were often accompanied by movement artifact and variable breathing rate following arousal.

DISCUSSION

The observed sleep-related responses to olfactory stimuli of prominent trigeminal activity and graded strengths indicate significant state-related alteration of olfactory sensitivity in young adults. Odorants were easily detected when awake and in the early transition into sleep (88% of stage 1 trials). By contrast, no behavioral responses to peppermint were observed in other sleep stages, and behavioral responses to pyridine were infrequent. A previous study[5] showed behavioral responses to a 3-minute "maximal" intensity peppermint stimulus in stage 2 sleep on about 16% of trials and EEG "speeding" slightly more frequently. In our hands, peppermint elicited EEG activation in the absence of behavioral response for 15% of stage 2 sleep trials, 20% of REM sleep trials, and only 1 time (3%) in stage 4 sleep. The greater behavioral response rate to peppermint for the Badia et al study[5] may be accounted for by longer stimulus presentation, use of maximal strength, and trials only in stage 2 sleep. Even so, peppermint did not reliably elicit responses. Our data

also showed that, while detection of pyridine occurred more often during stage 2 and REM sleep than during stage 4 sleep, pyridine responses were inconsistent. In contrast to minimal sleep-related olfactory response, a 5-second 800-Hz tone elicited behavioral responses (82%) or EEG activation on 97% of sleep trials.

When subjects were awake, peppermint was perceived as a pleasant stimulus and pyridine as both noxious and stronger. If sleep-associated responses to pyridine resulted from the odor's foul nature, then discrimination based on salience might be postulated for the olfactory system. Our data provide some support for this possibility, since participants who responded to pyridine during sleep did so at lower stimulus strengths, comparable in rated intensity to the stronger peppermint samples. Salience has been noted to increase sleep response rates in several studies[6,21] using other sensory modalities. A full appraisal of this salience hypothesis for sleep-related olfactory detection, however, will require further study with greater control of odor strength and hedonic valence.

Our results indicate significant alteration of perceptual processing as a function of sleep state. Whether this alteration represents a change in cognitive mediation of the stimuli or alteration of the neural pathways linking olfactory and arousal circuits is unclear. Event-related-potential studies indicate that certain processes of attention and memory-related operations involved in auditory processing remain operative during sleep.[22] The relative lack of olfactory response may indicate that, as with microsomatic animals, audition is the sensory system that remains most active during sleep in humans.[6] On the other hand, relative loss of olfactory sensitivity during sleep may result from loss of contextual and cognitive cues. Studies that systemically compare waking cognitive-contextual mediation and sleeping stimulus responsiveness across sensory systems are warranted.

Some suggest that the human olfactory system during sleep is sufficiently well tuned to ensure arousal to such threatening stimuli as odors associated with smoke from fire.[23] Our results strongly suggest otherwise. The intensity, strength, and noxiousness of the pyridine stimulus elicited behavioral arousal or EEG activation on fewer than half of stage 2 trials, less than one third of REM-sleep trials, and virtually no stage-4-sleep trials. This is a nontrivial lack of response, since pyridine is a component of coal tar and is also used as a herbicide for firewood,[24] and thus is a likely by-product of many real fires. In practical terms, therefore, olfactory awareness in humans is low to absent during sleep, and human olfaction appears insufficiently sensitive and reliable to act as a sentinel system. We further note that auditory arousal threshold is highest in young and sleep-deprived individuals,[6,16,25] increasing the likelihood that olfactory processing is even worse for children and sleep-deprived adults.

Although arousals to olfactory stimuli appear inconsequential during sleep, our data do not address whether odors may have other effects. One might access "unconscious" odor perception through analysis of dream mentation retrieved from REM sleep, particularly since spontaneous reporting of dreams containing olfactory sensations is low.[26,27] One could also investigate whether odors presented in sleep demonstrate habituation or if shifted attentional state resets the ability to perceive an odor. Although showing limited overt response to olfactory stimuli, sleep provides other opportunities to assess olfactory perception.

Table 2—Autonomic Response: Heart Rate (beats per minute)

	Prestimulus Mean (SD)	Poststimulus Mean (SD)
Stage 2 Sleep Odor Trials		
No response	60 (7)	60 (7)
EEG activation alone	61 (8)	70 (16)
Behavioral response*	59 (8)	88 (12)
Stage 4 Sleep Odor Trials		
No response	62 (8)	62 (9)
EEG activation alone	67 (9)	72 (14)
Behavioral response*	63 (8)	92 (16)
REM Sleep Odor Trials		
No response	62 (9)	63 (9)
EEG activation alone	59 (7)	63 (13)
Behavioral response*	61 (9)	82 (13)
All Stages Tone Trials		
No behavioral response	60 (7)	55 (13)
Behavioral response*	59 (8)	90 (14)

Heart rate (beats per minute) for 15 seconds before and after stimulus presentation.
* Heart rate significantly greater after stimulus ($P < .01$). EEG refers to electroencephalogram; REM, rapid eye movement.

ACKNOWLEDGEMENTS

We thank Garrett Etgen, Pamela Bigler, Jayson Carr, Joan Gelin, Stephen S. Davis, and Kate B. Herman for their assistance and a Grass Foundation Trustee Grant for funding.

REFERENCES

1. Thiessen GJ. Disturbance of sleep by noise. J Acoust Soc Amer 1978;64:216-22.
2. Horne JA, Pankhurst FL, Reyner LA, Hume K, Diamond ID. A field study of sleep disturbance: effects of aircraft noise and other factors on 5,742 nights of actimetrically monitored sleep in a large subject sample. Sleep 1994;17:146-59.
3. Aaron JN, Carlisle CC, Carskadon MA, Meyer TJ, Hill NS, Millman RP. Environmental noise as a cause of sleep disruption in an intermediate respiratory care unit. Sleep 1996;19:707-10.
4. Carter N, Henderson R, Lal S, Hart M, Booth S, Hunyor S. Cardiovascular and autonomic response to environmental noise during sleep in night shift workers. Sleep 2002;15:457-64.
5. Badia P, Wesensten N, Lammers W, Culpepper J, Harsh, J. Responsiveness to olfactory stimuli presented in sleep. Physiol Behav 1990;48:87-90.
6. Velutti RA. Interactions between sleep and sensory physiology. J Sleep Res 1997; 6:61-77.
7. Herz, R.S. The effect of verbal context in olfactory perception. J Exp Psychol Gen; In press.
8. Engen T. The Perception of Odors. New York: Academic Press; 1982.
9. Malnic B, Hirono J, Sato T, Buck LB. Combinatorial receptor codes for odors. Cell 1999;96:713-23.
10. Doty RL, Snyder P, Huggins G, Lowry LD. Endocrine, cardiovascular and psychological correlates of olfactory sensitivity changes during the human menstrual cycle. J Comp Phys Psychol 1981;95:45-60.
11. Rabin MD. Experience facilitates olfactory quality discrimination. Percep Psychophys 1988;44:532-40.
12. Doty RL, Brugger WE, Jurs PD, Orndorff MA, Snyder PJ, Lowry LD. Intranasal trigeminal stimulation from odorous volatiles: Psychometric responses from anosmic and normal humans. Physiol Behav 1978;20:175-85.
13. Rabin MD, Cain WS. Odor recognition: Familiarity, identifiability and encoding consistency. J Exp Psychol Learn Mem Cogn 1984;10:316-25.
14. Van Toller C, Kirk-Smith MD, Wood N, Lombard J, Dodd GH. Skin conductance and subjective assessments associated with the odour of 5-a-androstan-3-one. J Biol Psychol 1983;16:85-107.
15. McAdams S, Drake C. Auditory perception and cognition. In: Pashler H, Yantis S, eds. Stevens Handbook of Experimental Psychology, 3rd ed. Vol 1. Sensation and Perception. New York: John Wiley & Sons, Inc, 2002:397-452.
16. Zepelin H, McDonald CS, Zammit GK. Effects of age on auditory awakening thresholds. J Gerontol 1984;39:294-300.
17. Dravnieks A. Instrumental aspects in olfactometry. In: Moulton DG, Amos T, Johnson JW, eds. Methods in Olfactory Research, New York: Academic Press; 1975:1-58.
18. Jasper HHCC. The ten-twenty electrode system of the International Federation. Electroencephalogr Clin Neurophysiol 1958;10:371-5.
19. Stevens SS. Psychophysics. New York: Wiley; 1975.
20. Rechtschaffen A, Kales A. A Manual for Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Subjects. Washington, DC: Public Health Service, US Government Printing Office; 1968.
21. Portas CM, Krakow K, Allen P, Josephs O, Armony JL, Frith CD. Auditory processing across the sleep-wake cycle: simultaneous EEG and fMRI monitoring in humans. Neuron 2000;28:991-9.
22. Bastuji H, Garcia Larrea L, Franc C, Mauguiere F. Brain processing of stimulus deviance during slow-wave and paradoxical sleep: a study of human auditory evoked responses using the oddball paradigm. J Clin Neurophysiol 1995;12:155-67.
23. Schiffman S. Use of olfaction as an alarm mechanism to arouse and alert sleeping individuals. Aromachology Rev 1996;4:8-9.
24. Bush PD, Neary DG, McMahon CK, Taylor JW. Suitability of hardwoods treated with phenoxy and pyridine herbicides for use as firewood. Arch Environ Contam Toxicol 1987;16:333-41.
25. Busby K, Pivik RT. Failure of high intensity auditory stimuli to affect behavioral arousal in children during the first sleep cycle. Pediatr Res 1983;17:802-5.
26. Carskadon MA, Wyatt J, Etgen G, Rosekind MR. Nonvisual sensory experiences in dreams of college students. Sleep Res 1989;18:159.
27. Zadra A, Nielsen TA, Donderi DC. Prevalence of auditory, olfactory, and gustatory experiences in home dreams. Percept Mot Skills 1998;87:819-26.