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Comparison of Cardiovascular Responses to Noise During Waking and Sleeping in Humans

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Summary: Eighty subjects, 40 men and 40 women, were allocated to one of two groups according to their self-estimated high or low sensitivity to noise. In the first part of the experiment, they were exposed to sequences of common noises during the morning or the afternoon. The heart-rate and finger-pulse responses were measured and recorded in relation to sensitivity, sex of subjects, and time of day. The different types of noise were compared for both responses. The heart-rate response showed differences between sensitivity groups but not between noises. In contrast, no significant differences were obtained between sensitivity groups when using the finger-pulse response, but clear differences were observed between noises. In a second part of the experiment, 10 men and 10 women subjects were selected from the previous two sensitivity groups. These 20 subjects were exposed during sleep to the same noises as during the daytime. Heart-rate and finger-pulse responses during sleep were significantly greater than during waking, and they did not differ significantly with respect to sensitivity to noise or gender. These two autonomic responses showed differences between noises that appeared to be related to their noise-equivalent-level value. Compared with the silent baseline night, the sleep pattern showed no significant modification in the night of noise disturbance, except for the frequency of transient activation phases, which was significantly increased in the latter. **Key Words:** Noise sensitivity—Sleep disturbance—Heart rate—Finger-pulse response.

The effect of noise on humans is well documented. It has been demonstrated that noise has an effect on the autonomic nervous system in both waking and sleeping humans (1). The existence of physiological modifications, such as heart-rate response (HRR) (2,3), finger-pulse response (FPR) (4,5), sleep disturbances (6,7) and hormonal changes (8), is well established. Keefe and Johnson (9) and Muzet and co-workers (10) demonstrated that noise induces a rapid biphasic cardiac response and a phasic peripheral vasoconstrictive response.

These cardiovascular responses to auditory stimulation have often been analyzed with respect to the noise characteristics. During waking, the autonomic responses to noise are greatly influenced by the stimulus intensity (11), rise time, in the case of a

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progressive increase of the noise intensity (12), duration (13), spectral composition (14), and background noise level (15). Furthermore, meaning and affective aspects of stimuli (16) have been shown to interact with physiological responses. During sleep, both the physical characteristics and the psychological aspects (20,21) of the stimuli are also of major importance (17,18,19). In the present study, we decided to use a variety of noises that commonly occur during both daytime and nighttime. Each type of noise was different in intensity, duration, rise time, and spectral composition, and so we could expect some differences between noises that would permit us to examine the possible relationship between physiological reactions to noise and noise-equivalent level—a physical measure integrating variables such as intensity and duration.

Whereas studies performed during solely waking or solely during sleeping are numerous, investigations conducted during both vigilance states are very rare. Hatton (22) has studied HRR in infants (6 to 12 weeks old), using as stimulation pure tones of 750 and 1,000 Hz. The HRR observed during waking was a deceleration, whereas during sleeping it was an acceleration. Keefe and associates (23) have studied HRR and FPR during waking and sleeping states in adults, using pure-tone stimuli at 1,000 Hz. These authors observed a larger accelerative response during sleeping than during waking. One of the aims of the present study was to compare the cardiac and vasomotor modifications induced by noise in waking and sleeping humans. Our experiment differed from the two previously cited studies in several points. We used different commonly occurring noises instead of pure-tone stimulations. In this way, our experimental conditions were close to a real situation. In Hatton's experiment the subjects were infants, which involved large differences compared with our adult studies, especially in the sleep pattern. In Keefe and colleagues' study the intensity of the stimuli used during waking was just at the level of the auditory threshold, and it was increased during sleeping. In our experiment the noises were higher in intensity during daytime and lower during the night.

Another goal of this experiment was to study the cardiovascular responses to noise in relation to self-estimated noise sensitivity. Indeed, a psychological study made by Langdon (24) showed that people who judged themselves to be very sensitive to noise were more annoyed by noise than those who considered themselves as not sensitive to noise. Another study, by Thomas and Jones (25), showed that subjects with low sensitivity estimated the threshold of uncomfortable loudness at a higher intensity than that set by highly sensitive subjects. Our question was whether or not these two types of subjects had different cardiovascular responses to noise. The use of commonly occurring noises was adopted in the present case because subjects generally estimated their noise sensitivity in relation to commonly occurring noises.

The last question that we intended to answer concerned sex of the subjects and time-of-day differences. One of the few studies considering this question was undertaken by Ohkubo and co-workers (4). They analyzed the finger-pulse response to train noise and showed that female subjects had a greater reactivity than male subjects. A psychological study done by Baker and colleagues (26) suggested that arousal induced by noise was related to sex and time-of-day factors. In this experiment, we sought to determine if the cardiovascular response to noise differed not only between men and women but also between morning and afternoon.

In the first part of the present experiment, we studied HRR and FPR to noise during waking, and in the second part, during sleep. The effects of noise were considered in

relation to self-estimated sensitivity to noise and gender. The different types of noises were compared for both physiological measures and both waking and sleeping states.

METHODS

Daytime Experiment

Subjects. A total of 255 students (age range, 21.45 years ± 2.64) of both sexes (125 men, 130 women) were recruited at the University of Strasbourg through an announcement. They were simply informed about a noise experiment with electrophysiological recordings, for which they would be paid for their participation. All signed an informed consent form and completed a questionnaire prior to participating in the experiment. They were asked to make self-estimates of sensitivity to noise, each subject giving a score on a scale ranging from 1 (not sensitive to noise) to 12 (very sensitive to noise). In addition, they were asked to choose on a single self-rating schedule one of three statements: "I am not very sensitive to noise," "I am sensitive to noise," "I am very sensitive to noise." The distributions of the sensitivity score among men and women, compared by using a contingency table, did not differ significantly [$\chi^2 = 12.84$; degree of freedom (df) = 9; not significant (NS)]. The results for male and female subjects were therefore pooled, and the distribution of the noise sensitivity score obtained (Fig. 1) did not differ significantly from the theoretical calculated normal distribution [$\bar{x} = 7.6$; standard deviation (SD) = 2.1; $\chi^2 = 7.91$; df = 9; NS]. This total population was divided into three groups with about the same number of subjects: low sensitivity, high sensitivity, and medium sensitivity. Only subjects who indicated low sensitivity, with a score of 6 or below, or high sensitivity, with a score of 9 and above, were assigned to the extreme groups. To verify the validity of this classification based on the sensitivity scale, we compared our results with those obtained from the three self-rating statements mentioned earlier. We never found people in the low sensitivity group stating "I am very sensitive to noise," and we never found in the high sensitivity group people stating "I am not very sensitive to noise."

From these two groups, we randomly selected 80 subjects. On the basis of sex and

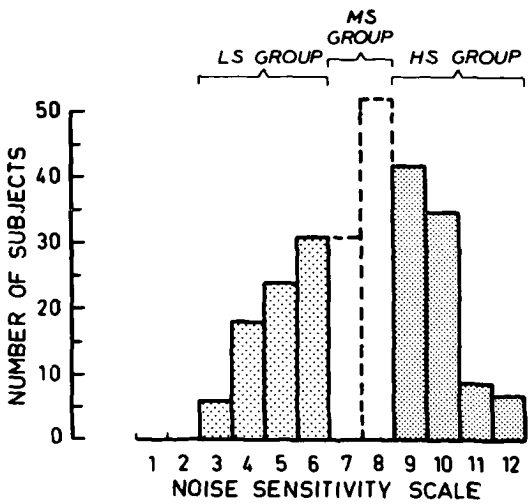

FIG. 1. Distribution of the noise-sensitivity score for the 255 volunteers. LS, low sensitivity; MS, medium sensitivity; HS, high sensitivity.

self-estimated sensitivity, subjects were separated into four groups whose characteristics are given in Table 1.

All subjects stated that they were in good health and were not taking any kind of medication. Their hearing was tested on an automatic audiometer. For the 10 frequencies tested between 250 and 8,000 Hz, the hearing loss observed was always less than 15 decibels [dB (A)].

Ambient Conditions. The noises used in this study were five commonly occurring noises: jet air plane (AIR.), truck (TRU.), motorcycle (MOT.), train (TRA.), and telephone ring (TEL.) (Fig. 2). The characteristics of these noises are given in Table 2. The maximum intensities were measured with a time constant of 125 ms. The noise-equivalent level (Leq) reflects the amount of noise that integrates both intensity and time duration. Leq values were measured for 60 noises/h.

Each noise was reproduced six times on a tape to present 30 noises during an experimental sequence lasting for 40 min. The order in which noises were presented and the intervals between noises (varying from 50 to 130 s) were semirandomly determined. Thus, the time of the noise presentation, as well as the type of the noise presented, was unpredictable.

Experiments were made in two similar rooms where ambient conditions were kept constant; background noise (BGN) was produced by monotonous and nonfluctuating traffic noise recorded at a distance from a busy highway. The dynamic profile was free of peaks (Fig. 2), and it was not possible to recognize distinctively any particular noise. The intensity of noise was set at an average level of 45 dB(A); the ambient temperature was 21°C; the relative humidity was 50%; and the air velocity was 0.2 m $s.^{-1}$

Physiological Recordings. Physiological measures continuously recorded on a polygraph included electrocardiogram with electrodes placed on the chest; finger-pulse amplitude by plethysmography of one finger-tip of the nondominant hand; respiratory movements by plethysmography with the strain gauge placed on the abdominal skin 5 cm above the navel; and body movements, using a radar system.

In addition to paper recording, heartbeat intervals were continuously recorded on a digital computer. Beat-by-beat instantaneous heart rate was then calculated by the computer and time-related to the noises. The amplitude of the HRR to each noise was calculated as the difference between the highest and the consecutive lowest heart rate occurring during the noise presentation (Fig. 3).

TABLE 1. *Characteristics of the groups of subjects (mean and standard deviation)*

Sex	Self-estimated sensitivity to noise	Age	N
a. Daytime experiment			
Female	LS = 5.12 ± 0.78	21.7 ± 2.3	20
Male	LS = 5.06 ± 1.11	23.3 ± 2.6	20
Female	HS = 10.05 ± 0.97	21.3 ± 1.7	20
Male	HS = 9.86 ± 0.96	23.3 ± 3.3	20
b. Nocturnal experiment			
Female	LS = 5.00 ± 1.00	22.8 ± 2.8	5
Male	LS = 5.40 ± 0.55	23.6 ± 4.3	5
Female	HS = 9.60 ± 0.89	22.6 ± 2.3	5
Male	HS = 9.60 ± 0.89	22.6 ± 2.3	5

LS, low sensitivity; HS, high sensitivity.

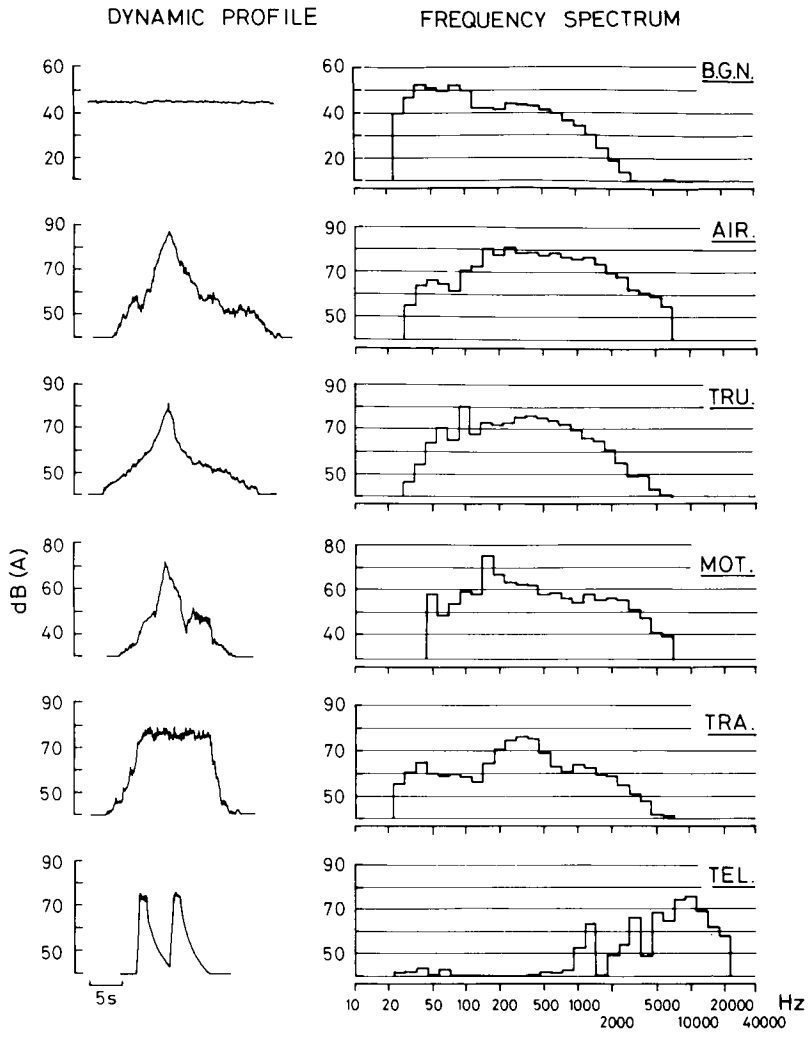

FIG. 2. Dynamic profiles and frequency spectra in one third of octave for the background noise and the five experimental noises.

The FPR to noise was customarily a vasoconstriction. We measured the amplitude of the FPR by dividing the amplitude of the smallest finger pulse occurring during the noise by the average amplitude of the 10 finger pulses recorded just before the occurrence of the noise.

Experimental Protocol. Each of the four groups of subjects was randomly divided into two half-groups. One half-group was tested in the morning from 0900 to 1100, and the second half-group was tested during the afternoon, from 1500 to 1700, to study the time-of-day effect. All noises were presented by a loud speaker placed 1 m behind the subject.

During the noise stimulation, the subjects were seated at a small desk, and they concentrated on written questionnaires and mathematical games. This activity was strictly monitored by visual observations through a closed-circuit television.

TABLE 2. *Maximum intensity, time duration, Leq values (60 noises/h), and Leq differences compared with train noise*

Type of Noise	Maximum Intensity [dB(A)]	Time duration (s)	Leq value [dB(A)]	Leq difference [dB(A)] vs. train noise
Airplane	86	21.4	67.7	−0.5
Truck	81	20.4	61.9	−6.3
Motorcycle	71	10.2	52.7	−15.5
Telephone	74.5	10.0	62.0	−6.2
Train	76.6	16.8	68.2	—

Leq, noise-equivalent level.

Nocturnal Experiment

Subjects. From the 80 subjects of the daytime experiment, we retained five subjects in each of the four experimental groups whose characteristics are given in Table 1. The five subjects in each group were selected because they had about the same mean amplitudes for the HRR and the FPR as the original group from which they were drawn.

Ambient Conditions. The noises used as stimuli were the same as those used for the diurnal experiment, except for the telephone ring, which was excluded because it could have awakened the subjects too easily. For the same reason, noise intensities were reduced by 15 dB(A) from those of the daytime noises. The four different noises were presented semirandomly at a rate of 8/h. The intervals between noises (varying from 4.5 to 10.5 min) were also determined semirandomly.

The ambient conditions were kept constant: the background noise was the same as in the daytime experiment, and the level was set at 35 dB(A); ambient temperature was 19°C; relative humidity was 50%; and air velocity was 0.2 m s^{-1}.

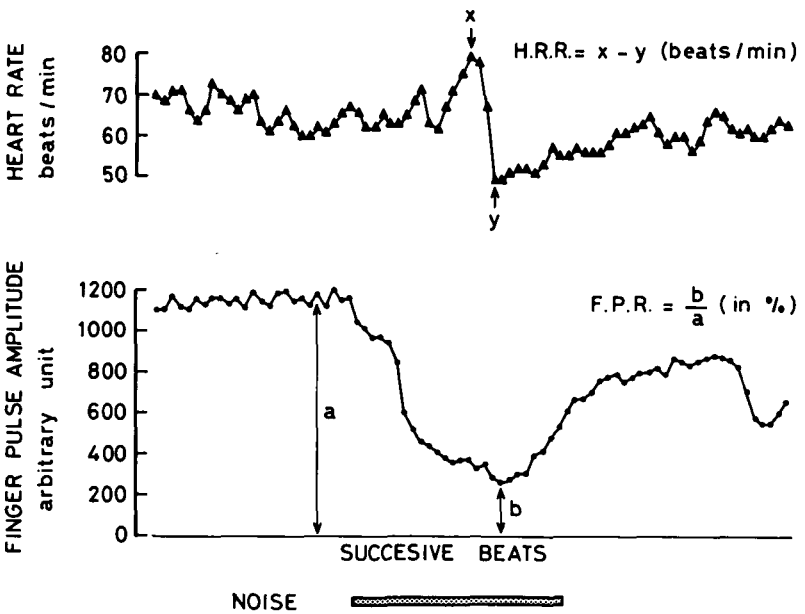

FIG. 3. Examples of heart-rate response (HRR) and finger pulse response (FPR) induced by noise.

Physiological Recordings. The physiological measurements that were recorded continuously during the night were the same as for the daytime experiment. In addition, two electroencephalograms (Cz-A2, F3-A2), right and left electro-oculograms, and an electromyogram of the mentalis muscle were recorded.

Sleep stage scoring, for each 30-s period of the night, followed the Association of Professional Sleep Societies (APSS) manual (27). Sleep onset was defined as the time of the occurrence of the first well-defined spindle.

Experimental Protocol. Each subject slept in the laboratory for three consecutive nights (N1 = habituation, N2 = baseline, N3 = noise-disturbed). Lights-out time was 2300, and the subjects were awakened at 0700. The third night was disturbed by the 64 noises evenly distributed throughout the 8 h allocated to sleep. The noises were presented during all sleep stages, and the distribution of the noise over the different sleep stages was directly related to the duration of each sleep stage. The results obtained for both HRR and FPR were calculated over all sleep stages, excluding the rare awake episodes.

Statistical Analysis

Statistical analysis was made by analysis of variance (ANOVA). For technical reasons, we had to exclude some of the records of the daytime experiment. Therefore, to reach the requirement of equal numbers of subjects imposed by the statistical analysis, a few subjects were eliminated at random in some groups. However, the characteristics of the groups tested did not differ from those shown in Table 1a for the total population.

Multivariate ANOVA for repeated measures (MANOVA) was used to compare the different types of noise according to the approach described by Vasey and Thayer (28). This procedure uses the Geisser and Greenhouse (29) conservative values from the BMDP 4V program. When overall F values were significant, the Bonferroni procedure was used. The Bonferroni procedure reduced the accepted level of significance (α) as a function of the number of comparisons (n). The Bonferroni corrected significance was α/n. In our study, 10 F tests were performed to compare the five types of noise for the daytime experiment, requiring a much rigorous $p = 0.005$ ($\alpha/10$) to be used as representing a 5% level of confidence. Similarly, six F tests were performed to compare the four types of noise for the nocturnal experiment; $p = 0.008$ ($\alpha/6$) represented the 5% level of confidence.

RESULTS

Cardiovascular Variables in the Daytime Experiment

1. *Sensitivity, Sex, and Time-of-day Effects*. A three-factor ANOVA, including 56 subjects, allowed us to compare low- and high-sensitivity groups, male and female groups, and morning and afternoon groups.

1.1. *Average Amplitude of HRR:* Considering the average amplitude of HRR, we found a significant group effect in response to the airplane noise [$F(1,48) = 6.54$, $p \leq 0.05$], the telephone ring [$F(1,48) = 5.06$, $p \leq 0.05$], and the train noise [$F(1,48) = 5.61$, $p \leq 0.05$]. The low-sensitivity group did indeed show smaller average amplitude of HRR in comparison to the high-sensitivity group. For the truck and motorcycle noises, a similar difference between the two groups was observed, but it did not reach significance. No significant difference between sexes or time of day was found. However, we observed a significant interaction between sex and time of day for the airplane noise

[F(1,48) = 4.53, $p \leq 0.05$], the train noise [F(1,48) = 4.05, $p \leq 0.05$], and the truck noise [F(1,48) = 4.40, $p \leq 0.05$]. The male subjects showed a higher average amplitude of HRR in the morning than in the afternoon for all these noises, whereas the reverse was true for female subjects. The same tendency was observed for the motorcycle and telephone noises, but the interactions were not significant.

1.2. *Average Amplitude of FPR:* The average amplitude of FPR showed a sex difference for the telephone ring only [F(1,48) = 7.41, $p < 0.01$]. The female subjects had larger FPR responses to that noise than did male subjects. For the four other noises, there was no significant difference between the groups in terms of sensitivity, sex, and time-of-day groups.

2. *Comparison Between the Different Types of Noise.* To compare the five different noise reactions, we used a MANOVA that included 37 subjects. When the overall F values were significant, the differences between the five types of noise were tested by F-tests.

2.1. *Average Amplitude of HRR:* The MANOVA showed no significant difference between noises [F(3.5, 124.6) = 0.73; NS]. In other words, the average amplitude of HRR did not differ significantly from one noise to another (Fig. 4A).

2.2. *Average Amplitude of FPR:* The MANOVA indicated that between-noise differences were present [F(3.5,126.2) = 5.06, $p \leq 0.001$]. The results of the a priori comparisons represented in Fig. 4B indicate that motorcycle noise elicited the smallest vasoconstrictive response. The average amplitude of FPR for the motorcycle noise differed significantly from that observed for the train [F(1,36) = 12.4, $p \leq 0.001$]; airplane [F(1,36) = 21.73, $p \leq 0.001$], and telephone [F(1,36) = 14.73, $p \leq 0.001$] noises but did not differ significantly from the truck noise [F(1,36) = 1.28, NS].

Cardiovascular Variables in the Nocturnal Sleep Experiment

1. *Sensitivity and Effects of Sex:* For each noise, we compared low- and high-sensitivity groups and female and male groups. ANOVA (subjects × sensitivity × sex) failed to reveal any significant effect either on the average amplitude of HRR or on the average amplitude of FPR.

2. *Comparison of Daytime and Nocturnal Results.* The comparison of the daytime (pooled data for the two times of day) and nocturnal results obtained for the 20 selected subjects (Fig. 5) showed large differences for each of the noises studied. Three-way

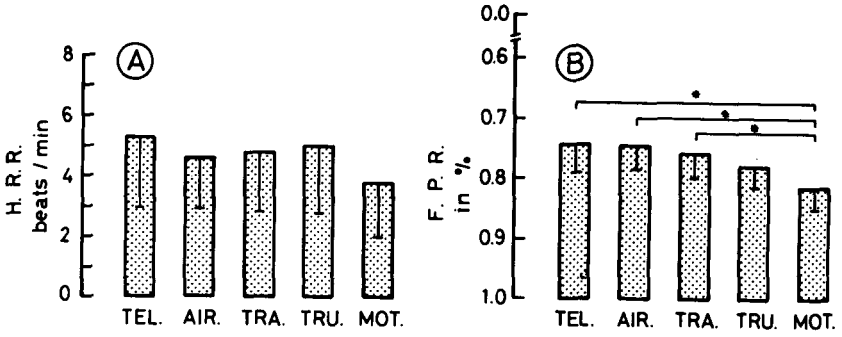

FIG. 4. Between-noise comparisons for the HRR (A) and FPR (B) obtained in the awake state (mean ± standard deviation). Asterisk = $p \leq 0.005$.

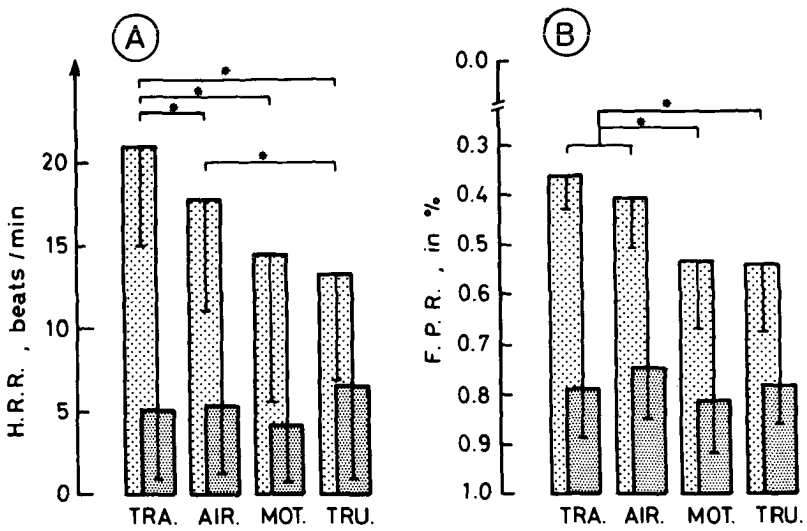

FIG. 5. HRR (A) and FPR (B) obtained for the 20 subjects during sleep and in the awake state. Between-noise comparisons are given for the nocturnal values (mean ± standard deviation). Asterisk = $p \leq 0.008$. ⬚, nocturnal results; ▨, daytime results.

ANOVA (20 subjects; two states: waking in daytime experiment and sleeping in nocturnal experiment; and four noises) was used to test the significance of the differences observed between waking and sleeping.

2.1. *Average Amplitude of HRR:* The ANOVA showed significant differences between waking and sleeping states [$F(1,76) = 119.6$, $p \leq 0.001$]. The average amplitude of HRR was 10% greater during sleeping than during waking, despite the fact that noise intensities were lowered by 15 dB(A) at night. A significant interaction between day or night and type of noise [$F(3,76) = 3.57$, $p \leq 0.05$] indicates the presence of a difference between the various types of noise in the nocturnal experiment.

2.2. *Average Amplitude of FPR:* For the average amplitude of FPR, we obtained the same result as for the HRR. There was a significant difference between daytime and nighttime [$F(1,76) = 277.3$, $p \leq 0.001$], as well as the interaction between day or night and type of noise [$F(3,76) = 4.77$, $p \leq 0.05$].

3. *Comparison of the Different Types of Noise.* The results obtained for the two physiological measures are presented in Fig. 5.

3.1 *Average Amplitude of HRR:* The MANOVA used to compare the effects of the four noises revealed that the average amplitude of HRR was not the same for each noise [$F(2.0, 37.6) = 16.55$, $p \leq 0.001$]. The F tests (Fig. 5A) showed that the train noise induced an average amplitude of HRR significantly ($p \leq 0.001$) larger than the other three noises [airplane: $F(1,19) = 18.60$; truck: $F(1,19) = 35.24$; motorcycle: $F(1,19) = 28.18$]. The average amplitude of HRR for the airplane noise was significantly higher than that measured for the truck noise [$F(1,19) = 9.56$, $p < 0.006$].

3.2. *Average Amplitude of FPR:* The MANOVA [$F(2.9,55.8) = 30.76$, $p \leq 0.001$] and the F tests indicated similar results to those we obtained for the average amplitude of HRR (Fig. 5B). The train noise elicited a stronger vasoconstrictive response that did the truck [$F(1,19) = 46.70$, $p \leq 0.001$] or motorcycle [$F(1,19) = 54.42$, $p \leq 0.001$]

noises. The airplane noise evoked significantly ($p \leq 0.001$) larger vasoconstrictive responses than the truck [$F(1,19) = 27.33$] and motorcycle noises [$F(1,19) = 34.40$].

The average amplitudes of FPR observed for these two last noises did not differ significantly from each other.

Effect of Noise on Sleep Parameters

The sleep variables calculated in baseline (N2) and in noise-disturbed (N3) nights are shown in Table 3. None of these parameters was significantly affected by nocturnal noises. The goal of this experiment was to study the phasic cardiovascular responses to noise in sleeping humans, without creating sleep disturbances. The noises were loud enough to induce phasic autonomic responses, but too weak to induce significant sleep structure modifications.

The frequencies of transient activation phases (TAP) calculated under the two experimental conditions and for the different sleep stages are presented in Table 4. These activation phases have been described previously by Schieber and co-workers (30). They are characterized primarily by concomitant and reversible modifications on the electrophysiological records, as follows: on electroencephalogram (EEG), the short replacement of usual activities by fast frequency activities; on electromyogram (EMG), the increase of muscular tone and the occurrence of bursts of muscle potentials on the other records; heart-rate increase; and decrease of finger-pulse amplitude. In addition, body movements were often, but not always, associated with these signs. During the noise-disturbed night, the frequency of transient activation phases was significantly greater for the total population in total sleep time [$F(1,19) = 15.4$, $p \leq 0.01$], in stage 2 [$F(1,19) = 8.32$, $p \leq 0.01$], and in rapid eye movement (REM) sleep [$F(1,19) = 4.78$, $p \leq 0.05$]. In slow-wave sleep, TAP increase did not reach the threshold for significance. For low- and high-sensitivity groups, we observed the same tendency, but the level of significance level was obtained only for the high-sensitivity group.

DISCUSSION

In the daytime experiment, we observed differences in the average HRR amplitude that were related to subjective sensitivity to noise. The low- sensitivity group had a lower average amplitude of HRR, but there was no corresponding difference in terms of the average amplitude of FPR. As suggested by Keefe and associates (23), the

TABLE 3. *Sleep variables in baseline (N2) and noise disturbed (N3) nights*

	Duration (min)		Number of stages	
	N2	N3	N2	N3
TST	439.9 ± 44.9	433.7 ± 27.2	—	—
L	17.7 ± 14.4	19.0 ± 13.2	—	—
W	22.8 ± 39.1	26.2 ± 20.2	7.8 ± 5.9	9.2 ± 6.0
Stage 1	16.6 ± 6.1	16.4 ± 5.9	17.2 ± 7.2	17.1 ± 6.8
Stage 2	229.3 ± 40.9	235.7 ± 27.6	24.4 ± 6.4	25.8 ± 6.2
Stage 3	42.3 ± 25.9	37.4 ± 20.8	7.6 ± 3.2	8.2 ± 3.5
Stage 4	57.8 ± 28.4	47.5 ± 23.3	3.5 ± 1.6	4.1 ± 1.9
REM	94.2 ± 27.8	96.9 ± 16.2	5.2 ± 2.0	5.9 ± 2.8

TST, total sleep time; L, sleep latency; W, wakefulness, excluding the sleep latency; REM, rapid eye movement.

TABLE 4. *Frequency of transient activation phase (TAP) in baseline (N2) and noise-disturbed (N3) nights*

TAP	N2	N3	N2 vs. N3 [F(1,19)]
Total sleep	0.074 ± 0.021	0.095 ± 0.029	15.40[a]
Stage 2	0.072 ± 0.028	0.089 ± 0.026	8.32[b]
SWS	0.037 ± 0.016	0.045 ± 0.015	2.58
REM	0.113 ± 0.062	0.146 ± 0.088	4.78[c]

SWS, slow-wave sleep; REM, rapid eye movement.
[a] $p \leq 0.001$.
[b] $p \leq 0.01$.
[c] $p \leq 0.05$.

dissociation of HRR and FPR may be due to the differences in innervation in the control of these responses: FPR is controlled solely by sympathetic nerves, whereas HRR is controlled by both sympathetic and parasympathetic nerves. Our results suggest that the activation of the autonomic nervous system owing to noise was the same in both low-sensitivity and high-sensitivity groups, because FPR does not discriminate between these two groups. The observed difference in HRR would then be related to a difference in the parasympathetic inhibitory activity. In the low-sensitivity group, the parasympathetic regulation of heart-rate acceleration would be more efficient than in the high-sensitivity group, which would explain the observed difference.

Significant interactions between sex of subject and time of day were observed for HRR during waking. Although the reasons for such differences are not clear, these results corroborate the findings of Baker and colleagues (26) that the activation induced by noise differed significantly in relation to gender and time of day. Here again, we obtained a dissociation between HRR and FPR, because such an interaction was not observed for FPR.

Another dissociation of HRR and FPR was obtained when comparing the different types of noise. Although no difference between noises was observed for HRR, FPR exhibited significant differences from one noise to another. A possible explanation for the differences observed is that HRR did not differ between noises because the parasympathetic regulation prevented the heart-rate acceleration from reaching its maximum amplitude. However, we failed to establish a clear relationship during the waking state between the two autonomic responses studied and the noise-equivalent level.

These results suggest that, during waking, FPR could be an index of the activation, whereas HRR was a more complex response resulting from arousal and inhibitory mechanisms.

Before comparing results for waking and sleeping, we attempted to ascertain whether habituation occurred during these two states. We found no habituation in the awake state or in the sleeping state across noise presentations. Some studies reported habituation across stimuli presentations in awake humans (13,15). In these experiments, either intervals between stimuli were constant or the stimulus presented was always the same. Thus, the novelty of the stimulus decreased across trials, and habituation occurred. In contrast, in our experiment, because of the semirandom distributions, it was impossible to predict either the time of the noise presentation or the type of noise presented, which could explain the absence of habituation. Lack of habituation during sleeping was in agreement with other reports. Muzet and co-workers (10), using traffic

noises, obtained habituation neither during a single night nor from night to night. Griefahn (31) has stimulated sleeping humans with tank fire and she also reported that no habituation took place during the night.

The comparison between sleeping and waking states showed that during sleep, cardiovascular responses to noise were greater than during waking. This difference is the more remarkable because the intensities of the noises used in the nocturnal experiment were reduced by 15 dB(A). A possible explanation is that the cortical inhibition exerted on the reticular formation during waking (32) is removed during sleeping and thus the autonomic responses to noise would be enhanced. Furthermore, as suggested by Baust and Bohnert (33), it is possible that HRR during sleep could be initiated by a phasic inhibition of the parasympathetic cardioinhibitory center.

No significant effects of noise sensitivity or sex were found during the sleep state; the difference in HRR observed between low-sensitivity and high-sensitivity groups during the awake state, disappeared during sleep. This result could be related to the difference in the sympathetic and parasympathetic balance in waking and sleeping states. However, whether the differences between sensitivity groups were predominantly psychologic, the state of consciousness would be necessary and thus, during sleeping, differences would not occur.

During sleep, the effects of noises on HRR and FPR were both in agreement with Leq levels seen between different types of noise. The train and airplane noises, which had the highest Leq, induced the highest physiological responses, whereas the truck and motorcycle noises, which had the lowest Leq, elicited the lowest HRR and FPR. This result is in direct contrast to the absence of such an effect in the awake state. The relation between noise-Leq and cardiovascular responses suggests that this physical measure could give an indication of the cardiovascular effects of traffic noise during sleep.

In the awake state, the sounds do not become noise until processed by the brain in terms of their meaning for the subject. Therefore, autonomic reactions such as HRR and FPR could be filtered by mental processing. During sleep, information processing can occur with some particularly meaningful stimuli, such as subject's name (20,21). Traffic noises used in our experiment had no particular personal significance; thus, during sleep the physiological responses to noise would not be modulated by mental processing, and they could directly reflect the magnitude of the auditory stimuli. This finding is supported by the fact that during sleep, the recorded HRR and FPR were much greater than in the awake state.

The nocturnal noise level was purposely kept low so as not to affect sleep structure. Only the frequency of the transient activation phases was increased in the noise-disturbed night. These arousal reactions are under the control of the reticular formation (30). The increase of TAP, like that of HRR and FPR, is directly related to reticular arousals induced by the noise. The reticular activations, transmitted through ascending pathways to the cortex, produce TAP as they elicit HRR and FPR through descending pathways.

In contrast to the waking state, there is no dissociation during sleep between HRR and FPR, whose magnitudes both seem to be related to noise Leq values. These cardiovascular responses to noise are much greater during sleep, which suggests that the adverse effects of noise on the cardiovascular system are especially important during this vigilance state. Special attention must be given to this finding, and prevention of exposure to noise must be reinforced to protect the restorative function of sleep.

REFERENCES

1. Kryter KD. *The effect of noise on man.* New York: Academic Press, 1985.
2. Libert JP, Amoros C, Muzet A, Ehrhart J, Di Nisi J. Effect of triazolam on heart rate level and on phasic cardiac response to noise during sleep. *Psychopharmacology* (in press).
3. Kryter KD, Poza F. Effect of noise on some autonomic system activities. *J Acoust Soc Am* 1980;67:2036–44.
4. Ohkubo C, Miyazaki K, Osada Y. Response of finger pulse amplitude to intermittent noise. *Bull Inst Publ Health* 1976;25:1–8.
5. Muzet A, Ehrhart J. Amplitude des modifications cardiovasculaires provoquées par le bruit au cours du sommeil. *Coeur Med Int* 1978;17:40–56.
6. Griefahn B, Muzet A. Noise induced sleep disturbances and their effects on health. *J Sound Vib* 1978;59:99–106.
7. Eberhardt JL, Strale LO, Berlin MH. The influence of continuous and intermittent traffic noise on sleep. *J Sound Vib* 1987;116:445–64.
8. Brandenberger G, Follenius M, Trémoliéres C. Failure of noise exposure to modify temporal pattern of plasma cortisol in man. *Eur J Appl Physiol* 1977;36:193–205.
9. Keefe FB, Johnson LC. Cardiovascular response to auditory stimuli. *Psychon Sci* 1970;19:335–7.
10. Muzet A, Ehrhart J, Eschenlauer R, Lienhard JP. Habituation and age differences of cardiovascular response to noise during sleep. 5th Congress on Sleep Research, Amsterdam. In: *Sleep*. Basel: Karger, 1980:212–5.
11. Berg WK, Graham FK. Reproductible effects of stimulus intensity on heart rate response curves. *Psychophysiology* 1970;6:653–4.
12. Turpin G, Siddle DAT. Effects of stimulus intensity on cardiovascular activity. *Psychophysiology* 1983;20:611–24.
13. Smith DBD, Strawbridge PJ. Stimulus duration and the human heart rate response. *Psychon Sci* 1968;10:71–2.
14. Eves FF, Gruzelier J. Individual differences in the cardiac response to high intensity auditory stimulation. *Psychophysiology* 1984;21:342–52.
15. Lobstein T, Webb B, Cort J. Background noise level and heart rate orienting: response detection using time series analysis. *Psychophysiology* 1978;15:316–9.
16. Gang MJ, Teft L. Individual differences in heart rate responses to affective sound. *Psychophysiology* 1975;12:423–6.
17. Muzet A. Modifications végétatives entraînées par le bruit au cours du sommeil. *Rapport Ministère de l'Environnement et du Cadre de Vie,* Paris: Ministère de l'Environnement et du Cadre de Vie. 1980.
18. Berg WK, Jackson JC, Graham FK. Tone intensity and rise-decay time effects on cardiac responses during sleep. *Psychophysiology* 1975;12:254–61.
19. Levere TE, Morlock GW, Thomas LP, Hart D. Arousal from sleep; the differential effect of frequencies equated for loudness. *Physiol Behav* 1974;12:573–82.
20. Mc Donald DG, Schicht WW, Frazier RE, Schallenberger HD, Edwards DJ. Studies of information processing in sleep. *Psychophysiology* 1975;12:624–9.
21. Oswald I, Taylor AM, Treisman M. Discriminative response to stimulation during human sleep. *Brain* 1960;83:440–53.
22. Hatton H. Developmental change in infant heart rate response during sleeping and waking states. *Develop Psychol* 1973;8:35–41.
23. Keefe FB, Johnson LC, Hunter EJ. EEG and autonomic response pattern during waking and sleep stages. *Psychophysiology* 1971;8:198–212.
24. Langdon FJ. Noise nuisance caused by road traffic in residential area: Part I. *J Sound Vib* 1976;47:243–63.
25. Thomas JR, Jones DM. Individual differences in noise annoyance and the uncomfortable loudness level. *J Sound Vib* 1982;82:289–304.
26. Baker MA, Holding DH, Loeb M. Noise, sex and time of day effects in a mathematics task. *Ergonomics* 1984;27:67–80.
27. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Washington DC: US Government Printing Office, 1968.
28. Vasey MW, Thayer JF. The continuing problem of false positive in repeated measures ANOVA in psychophysiology: a multivariate solution. *Psychophysiology* 1987;24:479–86.
29. Geisser S, Greenhouse SW. An extension of Bor's results on the use of the F distribution in multivariate analysis. *Ann Math Stat* 1958;29:885–91.
30. Schieber JP, Muzet A, Ferrière PJR. Les phases d'activation transitoire spontanées au cours du sommeil normal chez l'homme. *Arch Sci Physiol* 1971;25:443–65.
31. Griefahn B. Cardiac responses caused by shots of tanks during sleep. *J Sound Vib* (in press).
32. Zimmermann M. Neurophysiology of Sensory System. In: Schmidt RF, ed. *Fundamentals of sensory physiology* New York: Springer, 1978;31–8.
33. Baust W, Bohnert B. The regulation of heart rate during sleep. *Exp Brain Res* 1969;7:169–80.