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0014-4754/84/050425-05$1.50 + 0.20/0

Sleep disturbance due to transportation noise: ear plugs vs oral drugs*

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Introduction

From 1970 onwards, an increasing proportion of the research on noise has been oriented toward assessing its influence on sleep. Notwithstanding some interesting results which will be summarized in this paper, a major problem arises from the fact that such research is aimed at studying the impact of a physical phenomenon – noise – on a physiological phenomenon – sleep – while the exact definition and nature of both phenomena are still controversial. For the vast majority of human beings sleep quality is reflected only by subjective feelings upon awakening and, in so far as they later may lead to a complaint, they must be considered as being important. Despite this and with the exception of a few caricatural situations, there is though little correlation between these subjective reports and 'measurements'. Instead of reviewing all the data on the relationships between noise(s) and sleep perturbations as presented in several symposia and published in reviews[15,38,61], we will attempt here to approach this problem from a more general point of view, and pose 4 questions:

The first question we shall address will be epidemiological: are there any differences in the patterns of drug consumption between populations living in areas with different noise levels?

In view of the fact that some adaptation may occur in the course of long periods will then consider the effects

of reducing night-time noise levels on the sleep patterns of subjects who have slept for years in a noisy environment.

And conversely, what are the effects of creating noise during the night on the sleep patterns of normal subjects and, under these conditions, what are the physical characteristics of noise that appear to be the most sleep-disturbing?

Last, but not least, are the sleep patterns of individuals who sleep in a noisy environment and who are subjectively well adaptated to it, completely 'normal'?

General problems

How much noise can a subject tolerate before his sleep becomes disturbed? This was one of the questions studied in the early period of research into the relationship between sleep and noise, when noise was used to assess the differences in sleep 'depth' as a function of sleep stage[44]. In these experiments, artificial noises such as clicks were the most commonly used stimuli, and no attention was paid to the information content of the noise in the ususal environment. With the appearance of jet planes and especially supersonic aircraft a number of laboratory experiments were instigated to study the impact of such loud noises on sleep. As reviewed by Lukas[31], sleep disturbances were at that time defined only in terms of EEG arousal reactions.

The rapid development of urban traffic brought for the experimentator the considerable difficulty of having to specify a single noise event against background noise and, as a consequence, of having to global sleep changes occurring in those persons exposed to traffic noise. Moreover, even though sleeping with electrodes on the scalp cannot be considered as being entirely natural, a progressive change from laboratory studies to field studies took place. This allowed research on subjects in their home environment after prolonged exposure to noise and, at the same time, on their possible adaptation, whether instrumental (double glazing), behavioral (moving the bedroom) or physiological (habituation).

Principal results

Noise environment and drug-taking habits: ear plugs vs oral drugs

Correlations found in epidemiological studies do not a priori imply the existence of causal relationships. Despite this and the fact that a number of factors may account for the chronic use of neurotropic drugs, their rate of prescription to residents exposed to noisy environments might well reflect a response to this perturbation. Interestingly, the consumption of hypnotics in relation to noise levels is not as well documented as that of antihypertensive drugs[22] and the results are far from being unequivocal. In contrast to the results of the Langdon survey[26] which showed that noise does not influence hypnotic taking habits Lambert[25] and Wehrli[59] have found some positive correlations between these habits and traffic noise levels. These results are supported by more recent studies related to the influence of aircraft noise on medical treatments for sleep disorders[9,21]. These studies reported that prescription of sedative, tranquilizing, hypnotic and antidepressant medications varies from 15% to 27% according to the environmental noise intensity. No habituation appears to take place. François[10], using the Taylor test, reports an increase in anxiety ratings from 1975 to 1981 around Roissy Airport in Paris. There is a similar problem near Copenhagen Airport (and also at Schipol Airport in Amsterdam-Knipschild[22]) where, according to Relster[45], among the residents exposed to Leq (equivalent energetic level) ranging from 60 to 70 dB(A), 25% usually take tranquillizers, 19% have been referred to a psychiatrist or a psychologist, and 4% have been hospitalized in a psychiatric clinic.

The more optimistic results from some of the studies related to road traffic are probably questionable since in most of them the use of ear plugs was not accounted for. According to Wehrli[59], ear plugs use ranges from 0% in quiet areas to 14% in noisy zones.

Psychophysiological correlates of night-time noise reduction

From the data in table 1 it can be concluded that, whatever the experimental design, statistically significant modifications occur both in sleep stage durations and in phasic responses to isolated noise events after moderate reductions (by 6–14 dB(A) Leq) of night-time noise levels. Results from 3 different experiments[20,55,60] are also consistent and show that there is a positive correlation between noise levels (expressed in Leq per min) and heart rate, independent of specific sleep stages. Noise reduction is usually followed by an overall increase in REM and/or delta sleep, (the latter being relatively unaffected by isolated noise events[28]), together with a reduction in intermittent awakenings. According to data from Friedmann[11] on the effects of night-time noise reduction after a long-lasting noise exposure, the maximum increase in delta sleep takes place one week after the initiation of noise limitation and plateaus after about month. It is interesting to note that in a quieter sleeping environment there is an increase in REM sleep in older people, where as there is an increase in delta sleep in younger subjects[5]. In addition to these polygraphic findings, an improvement in subjective sleep quality and morning performance takes place. It would thus seem that not only is there a global improvement whenever night-time noise is reduced, but also that apparently no complete physiological adaptation to night-time noise occurs in the long term.

Psychophysiological correlates of night-time noise intrusion

An increase in night-time noise levels leads to sleep pattern modifications which apparently vary in magnitude as a function of the new noise intensity (table 2). Following a slight increase in noise levels, Griefhan[16] observed only moderate sleep alterations while Ehrenstein[6] described an initial delta sleep reduction followed by a REM sleep decrement. The results of a field study of Vallet and Blanchet[52] clearly demonstrate that, under

natural circumstances, noise intrusion is followed by shorter REM sleep latencies, reduced delta sleep time and by an increase in the time spent awake. It also appears that irregular noises induce sleep changes that are more marked than those occuring after a monotonous increase in night-time noise levels[40].

Since a decrement in both subjective appreciation of sleep and specific performance occurs after night-time noise exposure[20,60] there is general agreement about the pejorative effects of noise during night sleep. Of interest here are results from Muzet[32] who showed that there was rapid habituation of polygraphic and subjective responses to repeated noises while cardio-vascular reactions persisted.

Dissociation between EEG, cardiovascular and subjective effects of noise

Some of the studies reported in table 2 are particularly interesting in that they show a dissociation between subjective habituation to noise level variations and persisting objective sleep pattern and cardiovascular modifications[18,20].

Experiments by Muzet[37] have been oriented towards answering the question whether the phasic cardiovascular reactions that occur in response to isolated noises habituate over 2 weeks of noise exposure. 26 subjects were investigated in sound-attenuated recording rooms and submitted to noise stimulations which consisted of 90 isolated traffic noises per hour with a peak level varying from 40 to 65 dB(A). The amplitude of the cardiovascular responses (CVR), i.e. heart rate acceleration and peripheral vasoconstriction, followed by heart rate (HR) deceleration and peripheral vasodilatation, was correlated with noise intensity (fig. 1) and persisted over the two-week exposure period, whereas EEG and subjective modifications disappeared after 3–5 nights. These repeated phasic HR responses are likely to be responsible for the more global correlations found, on a 1-min time base between mean HR and Leq[20] and for the whole night duration between HR variability and mean energetic noise level variations. Such correlations have however not been observed in another study[60].

Whereas in Muzet's experiments obvious physiological reactions triggered by noise were still present in individuals subjectively well adaptated to night-time noise, the

Table 1. Effects of decreasing noise levels

Authors, year and reference	Laboratory or at home	Noise sources; number of noise events	Noise levels; background and noise level variation	Assessment criteria	Number of subjects age and sex number of nights	Physiological and psychological after effects of noise reduction
Levere and David, 1977[28]	Laboratory	Recorded aircraft noise; 15 noises of 20 sec per night	80 dB(A) and 65 dB(A) ↓ = 15 dB(A)	EEG desynchronisation Questionnaire	12 ss, male 18-23 years 4 nights	Less sleep disruption only during stages I & II; little effect on Δ sleep, no subjective improvement
Friedmann and Globus, 1974[11]	At home, telemetry	Years os exposure to aircraft noise, cessation of late flights (Los Angeles)	External noise Leq: before 77 after 51 dB(A) inside peak levels 52 before 39 after	Sleep stages duration	12 ss (couples) 45 years ± 7 3 blocks of a week each	All sleep stages increase, especially Δ sleep; maximum 1 week after noise reduction
Wilkinson and Campbell, 1983[60]	At home	Years of exposure to traffic noise, reduction of noise by thermopane windows	mean Leq 47 dB(A), mean Leq after: 41 dB(A) Δ = 6dB(A)	Sleep stages duration Heart rate (HR) Questionnaire Reaction time	12 ss (couples) 1 group < 45 years 1 group > 45 years 20 nights in 2 blocks	↑ Δ sleep, ↑ REM latency. On a 1 min time base heart rate positively correlates with noise better sleep quality reported, simple reaction time improved
Vallet and Gagneux, 1983[55]	At home, telemetry	Years of exposure to traffic noise, reduction of levels by moving the bed to a quiet room	Noisy Leq: 52 42 dB(A) 'Quiet' Leq 42 27 dB(A)	Sleep stages duration Heart rate Questionnaire Reaction time	26 ss (10 couples) 4 ss < 30 years 10 ss < 50 years 12 ss > 50 years 11 consecutive nights	↑ REM sleep, ↓ awakenings; correlation between noise levels and heart rate. Improved sleep quality, shorter reaction the next day
Jurriens and Kumar, 1983[20]	At home	Years of exposure to traffic noise, thermopane windows	'Noisy' Leq: 52 41 'Quiet' Leq: 42 37 dB(A)	Sleep stages duration Heart rate Questionnaire Reaction time	12 ss (4 couples) 3×4 ss: 20, 30, 50 years 20 nights in 2 blocks	↑ REM sleep time, ↓ Δ sleep, ↓ awakenings; correlation between noise and heart rate, Better subjective quality of sleep
Griefahn and Gros 1983[16]	At home	Years of exposure to traffic noise, chance by earplugs	Leq 48 35 dB(A) Δ 9 dB(A) peak (L1)56-41	Sleep stages duration Questionnaire Reaction time	10 ss (5 couples) 25 65 (= 39 ± 10) years, 12 consecutive nights	↑ Δ sleep, ↓ Δ sleep latency, better performance (less errors)
Eberhardt, 1983[5]	At home, telemetry	Years of exposure to traffic noise, temporary thermopane windows	Leq 38 dB(A) Δ Leq: 6 to 11 dB(A). peak levels: 50 55 dB(A)	Sleep stages duration Body movements Single events effects	13 ss, male 1 group 63 73 years, 1 group 21-27 years 7 nights non consecutive	↑ Δ sleep for the young subjects, ↑ REM sleep for the aged ↓ time spent awake group

L1: noise level exceeded for 1% of the time.
dB(A): frequency-filter weighted according to ear sensitivity (with attenuation in low frequency range).

Table 2. Effects of increasing noise levels

Authors, year and reference	Laboratory or at home	Noise sources, number of events/nights	Noise levels, background and noise level variation	Assessment criteria	Number of subjects number of nights age and sexe	Physiological and psychological results after noise increasing
Jurriens, 1980[18]	Laboratory	Recorded traffic noise	Background: 34 dB(A) Leq Noise 40–60 dB(A)	Sleep stage duration Subjective sleep quality Reaction time test	6 ss, male 18–30 years 10 nights quiet 20 nights noisy	↓ △ sleep time, lower sleep subjective quality, no clear alteration of reaction time (RT)
Ehrenstein and Muller-Limmroth, 1980[6]	Laboratory	Recorded urban traffic noise + pile driver and air hammers	Background: 38 dB(A) Leq 50–70 Leq 76–86 dB(A) from pile driver	Sleep stages duration + mood scale + heart rate + hormones + performance	6 ss, male 19–23 years 8 consecutive nights	↓ △ sleep time during the first 2 nights with noise, ↓ REM sleep (nights 3 to 8) worsening of mood
Muzet, 1977[32]	Laboratory	Simulated noise; temperature variation; 160 trucks noises for each 2h period, 10 or 20sec duration for each noise	Backround: 40 dB(A) Leq Peak levels: 80, 65, 55, 40 dB(A)	Sleep stages shifts nb of movements, Cardiovascular questionnaire Performance	18 ss, male 19–27 years 5 consecutive nights	↑ in stage shifts nights 1 ↓ subjective quality and 2 Persisting cardiovascular reactions
Ohrstrom, 1982[40]	Laboratory	Recorded noise Continuous noise	51 dB(A) Leq	Number of movements Performance	6 ss, 2 female, 4 male	↑ Number of movements (+ 16%)
		Intermittent noise Truck peaks events	80 dB(A) peack ≃ Leq 35–43 dB(A)	Mood	12 ss, 3 female, 9 male 18–30 years 6 nights	↑ Number of movements (+ 22%) poorer subj. sleep quality in the 2 conditions (the poored with intermittent noise)
Vallet and Blanchet, 1977[52]	At home, telemetry	Road traffic noise before/after new expressway	before Leq ≃ 30 dB(A) after Leq = 45 dB(A) peack 55.60	Sleep stages duration Questionnaire	12 ss, male 27–50 years 9 nights = 3 before and 6 after	↓ △ sleep (−28 min, −6% TST) shorter REM latency
Griefahn and Gros, 1982[16]	At home	Road traffic noise, opening the windows	Leq = 35 to 48 dB(A) mean △ Leq = 6,8 dB(A) peak (L1) 41–56 △ peak (L1) 9,5	Sleep stages duration Questionnaire Reaction time (4 choices)	10 ss (5 couples) 25–63 years 12 consecutive nights	Slight modification of sleep stages, poorer sleep quality, more tired in the morning

converse has also been demonstrated. Two independent studies have been conducted in the Los Angeles International Airport area following cancellation of night flights. In the first study, based on interviews[7], no subjective sleep improvement was noted, but Friedmann[11] who performed field recordings during the same period found that delta sleep increased from 12 to 17% of total sleep time one week after the flight schedule changes. The same dissociation has also been reported by Vallet and François[54] who concluded that, whereas physiological sleep modifications level off rather rapidly after noise appearance, changes in subjective appreciation follows a slower time course.

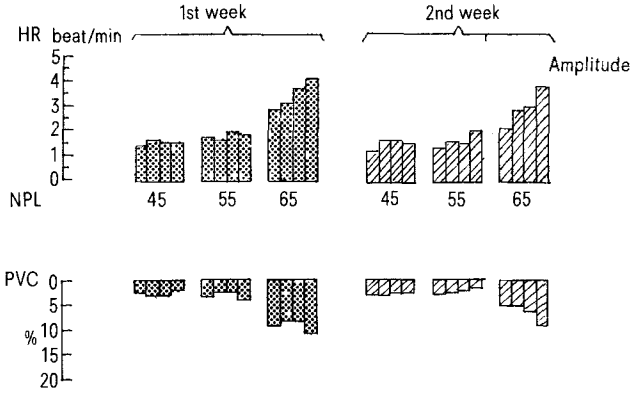

Figure 1. Mean heart rate (HR) and peripherical vaso constriction (PVC) responses to different noise peak levels (NPL) in 18 subjects over a 2-week period. Each column corresponds to a 2-h period: from left to right and for each NPL: 23.00–01.00 h; 01.00–03.00 h; 03.00–05.00 h, and 05.00–07.00 h; from Muzet[39].

Discussion: Methodological considerations

The difficulty in drawing clear cut general conclusions, from these studies stems from methodological differences. For instance, the criteria used to assess 'objective' sleep alterations extend from EEG frequency change[28] to reported awakenings[49] HR modifications[39] or body movement[41]. The EEG scoring itself is subject to interexperimental differences. With both visual or automatical scoring, the analysis is made on a time-base scale which varies from 20 sec to 1 min. In addition, some discrepancy between REM scores has been demonstrated[23] depending on the techniques of scoring used. The experimental designs themselves also vary widely from one study to another. In comparison with labora-

tory studies where it is possible to balance the order of noise or no-noise situations, the field studies depend on imposed situations like the opening of a new highway. When noise changes are introduced by the experimenter himself they include different techniques such as installing thermopane windows, moving the bedroom to a quieter place in the house, wearing ear plugs etc. Moreover, the delay between the changes in noise and the physiological recordings are not identical and these differences may account for some discrepancies in the reported results[19].

It is also obvious that the influence of the physical characteristics of noise stimulation: the frequency and level of the stimuli, as well as that of individual factors (sex, age) should also be considered.

Physical characteristics of noise

Compared with field studies, laboratory experiments provide better control of noise stimulation. However this methodological advantage is balanced by the physical problems related to either the synthesis of the noises[32], or replaying recorded noise. In spite of attempts aimed at the control of reverberation time, simulating noise source displacement[46] and improving the quality of the replay apparatus, laboratory noise production is still hampered by some degree of spectral distortion which is perceived by the experimental subjects[24].

Laboratory studies, however, bring the advantage that a number of other critical factors can be controlled, such as the number of noise events per night.

For instance it was found on the basis of interview data and physiological recordings, that car or truck[36] noises presented at a rate of 1.8/min lead to a higher percentage of EEG changes than a presentation at a rate of 4.3/min. The global rate of presentation is, however, not sufficient because the time-interval between 2 individual noises also influences the probability of an EEG reaction, which reaches a maximum for a noise interval of 40 min[14]. This might not be the case when residents are exposed to 2 different noise sources, such as trucks plus railroad traffic, which can produce a measured noise level of 70 dB(A) Leq at the building frontage. Under these circumstances, and in spite of peak levels from both sources being identical and the frequency of trucks higher than that of trains, more sleep disturbances are induced by the former[56]. Noises do not occur in a 'vacuum', and they emerge over a background noise level, the existence of which modulates the physical significance of peak levels. In most laboratory recording rooms the monotonous background noise due to the air conditioning ranges from 32 to 38 dB(A), a level at which fluctuations in noises induce some sleep pattern alterations[42]. This peak/background ratio is clearly important when results from field studies, comparing physiological reactivity to isolated noises under quiet to those under noisy background conditions, are considered: during the quiet nights the physiological reactions to noise are less frequent but they occur for noise peak levels which trigger no specific reactions during the noisy nights[55] (table 3).

It would thus appear that physiological reactivity is subject to some degree of adaptation as long as the information content of noises remains the same.

Table 3. EEG effects of the background level and the peaks levels from Vallet[55]

	Noisy condition (Leq 52–42 dB(A))			Quiet condition (Leq 42–27 dB(A))			Difference (Student's t-test)
	dB(A) peak level	σ	N	dB(A) peak level	σ	N	
Awakenings	52.56	7.86	333	43.85	5.85	114	$p < 0.01$
Sleep stage shift	51	7.37	641	42.38	5.23	299	$p < 0.01$
Transient effect	50.57	6.29	584	41.99	5.33	310	$p < 0.01$

Specific meaning of noise

It is well known from everyday experience that the specific meaning of a given noise is as important a factor as is its intensity in inducing arousal reactions. This was demonstrated experimentally by Oswald[43] who played a tape recorded list of 560 names to sleeping subjects and observed that the EEG and EMG reactions of a given subject were triggered only by his own name. When the tape recording was played in reverse no reaction occurred despite the fact that the physical characteristics of the sounds were maintained. These interesting results have been replicated by Langford[27].

This meaningfulness of noises may, conversely, induce a better habituation. This is implied by the results of a field study by Rylander et al.[48] who showed that, after sonic booms of 60 N/m^2, only 10% of the military population is awakened as compared to 56% of the civilian population.

The acquired hyporeactivity might be related not only to the specific meaning of some noises but also possibly to an habituation process taking place before birth. This is strongly suggested by results from Ando and Hattori[2] who studied the way in which infants reacted to aircraft noise as a function of the time since their mothers had moved near Osaka International Airport. Over 48% of the babies whose mothers moved here either before or during the first 5 months of pregnancy slept soundly in the presence of aircraft noise whereas in the group of infants whose mothers moved to the airport surroundings either during the last 5 months of pregnancy or after childbirth less that 15% slept and 50% awakened and cried at the noise. Awakenings and cryings were observed in only 13% of the former population.

Individual factors

Age and sex. Two basic factors, age and sex, influence the individual reaction to noise. When young subjects (21–27 years old) are moved to a quiet sleeping room they fall asleep faster and experience an increase in delta sleep. In contrast, older subjects (63–73 years old), an age group in which there is a physiological decrease in delta sleep, show an increase in REM sleep. In both groups however, the reduction in body movements is identical[5]. The increased sensitivity of sleep to simulated sonic booms as a function of age is clear when data from Lukas[29] (table 4) and Rice[33] are considered. This author reports that for sonic booms with an external

Table 4. Response frequencies to simulated sonic booms. Age effect from Lukas[29]

Age and sex	No response (%)	Sleep stage shift (%)	Awakening (%)
5–8 years (2 males, 2 females)	94.1	5.9	0.0
45–57 years (4 males)	80.7	6.31	4.0
69–75 years (4 males)	61.7	23.4	14.9

Table 5. Response frequencies to jet noise. Sex effect (in 10–24-year-old subjects) during sleep stages, from Lukas[30]

Sleep stages	Sex	Sleep responses		
		No effect (%)	Sleep stage shifts (%)	Awakening (%)
2	Male	51.8	20.7	27.5
	Female	36.4	10.9	52.7
3 and 4	Male	41.8	30.6	27.6
	Female	17.9	37.2	44.9
SP or REM	Male	61.9	13.3	24.8
	Female	29.5	16.8	53.7

pressure of 25–300 N/m^2, babies show little reaction but 30% of the adult population is awakened.

A reversed age-linked dependence of the response to noise seems to apply for cardiovascular reactions (CVR) during sleep. Muzet[39] studied CVR to noise in subjects from 3 age groups (6–12 y, 18–29 y, 56–66 y). There was a positive correlation between peak noise intensity and the magnitude of CVR in the three groups, but the correlation was lowest in the oldest group and highest in the youngest whatever the sleep stage, except during REM sleep where CVR of the intermediate group was identical to that of the infants. Moreover, whereas in the older subjects, the magnitude of CVR decreases late in the night, it remains the same, or increases, as the night progresses in the other two groups. Furthermore, women appear to be more sensitive to noise than men; awakening reactions occur at lower peak levels in women[30]. The same tendency has been demonstrated in adults following aircraft noises (68, 79, 84 dB(A)) (table 5) but not following sonic booms. It also appears that female subjects are most particularly susceptible to awakenings caused by noise during sleep stage IV[60].

Sleep habits. A number of experiments have shown that the sleep structure follows circadian modifications[57]. As compared to field recordings, laboratory investigations often make it necessary to change sleep habits (e.g., time of going to bed) which in addition to the independent variable (noise) have an influence on sleep structure. This is important since, as demonstrated for train engineers[8], noise is much more disturbing when people sleep under abnormal schedules. This source of variation often is not well controlled.

Noise exposure duration

Globus and co-workers[13] were among the first to investigate whether chronic noise stimulation influences normal sleep. They studied Los Angeles residents who lived for at least 6 years either close to L.A. International Airport or in a quiet area. Those residents in the airport neighborhood had less deep slow wave sleep (delta sleep), more stage 1 sleep and more intra-night wakefulness than the residents in quieter areas.

Chronic exposure to noise resulting from a residential situation is psychologically or physiologically completely different from any chronic laboratory noise stimulation which is known to be of limited duration and independant of the subject's usual environment. It is thus unlikely that reactivity to chronic noise will be the same under laboratory and field conditions. This appears to be true on the basic of the data in figure 3 which show that after a 1-year exposure to aircraft noise the probability of stage shifts or awakenings in response to a given noise level is reduced but still persists even for noises with very low peak levels[53]. Studying 16 subjects during 12–24 consecutive nights, Thiessen[50] reported that the frequency of awakenings was reduced by about 50% at the end of a 2-week period, whereas the frequency of sleep stages shifts in response to truck noises (60 dB(A)) did not decrease over time. However, when young male subjects were exposed for 30 days to loud noises emitted at 22-sec intervals around the clock, no habituation of CVR occurred and delta sleep remained reduced over the entire experimental period[51]. This lack of habituation is likely to be related to the experimental situation and the particular physical characteristics of the noise because habituation of EEG and EMG reactivity together with persisting CVR to night-time isolated noise has been described by Muzet[39]. We must recall here, however, that an overall noisy night-time ambiance first leads to a decrease in delta sleep and then to a decrease in REM sleep[6]. 'Adaptation' might thus correspond to overall sleep pattern differences and not only to alterations in a single sleep stage. Moreover the differences in adaptation that occur according to whether noise is delivered either over the entire 24-h period or only during the night raise the possibility that day-time noise exposure possibly modulates night-time reactivity to noise.

Effect of day-time noise and activity on sleep patterns

In view of the well known influence of physical activity on sleep patterns[3] it is somewhat surprising that the exact nature of the usual day-time activity of the sub-

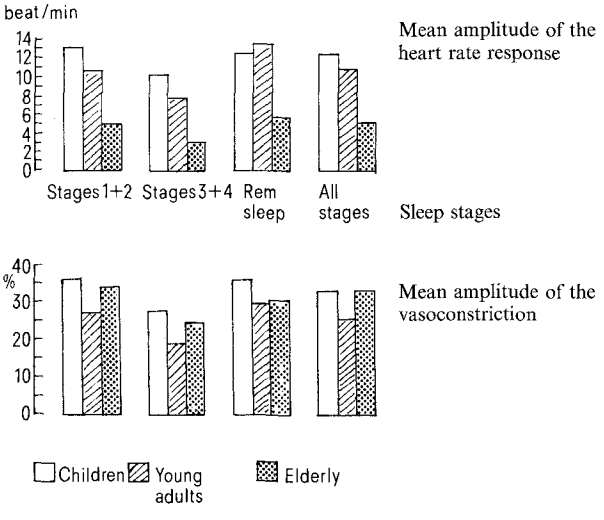

Figure 2. Cardiovascular responses to noise (mean values for different age-groups and sleep stages) from Muzet[39].

jects in the experiments reported above has been completely neglected. The fact that day-time noise was not taken into account for studies into the relation between night-time noise and sleep might represent a serious methodological weakness. This is implied by the results of Mouret[4] who showed that when young subjects are required to sleep in a sound-attenuated room, there is a negative correlation between REM sleep time and the noise energy to which the subjects were exposed during the day, whatever the source of noise.

Neurophysiological interpretations

One of the most consistent experimental results is that noise induces a reduction of delta sleep which does not habituate[18, 60]. Such a long-lasting modification probably cannot be attributed to aging since delta sleep time increases when the subjects are moved to a quiet bedroom[5, 55]. This implies that night-time noise leads to a chronic reduction in delta sleep as evaluated by polygraphic recordings. This long-lasting effect is puzzling since not only are delta sleep requirements identical in short, long and average sleepers[58] but also experimental deprivation of delta sleep is much more difficult to achieve than REM deprivation[1]. As, in long investigation term, the same noise levels still induce a decrease in this sleep stage a possible interpretation might be that, physiologically, the same noise becomes more and more disturbing.

Furthermore Vallet's studies[52] demonstrated that the increase in noise level related to the opening of a new turnpike induced changes in sleep patterns which were very similar to those described in unipolar depressive patients, namely reduced REM sleep latency, reduced delta sleep and frequent awakenings[34]. Even though REM eye movement density was not calculated, the similarities between the sleep patterns of these normal subjects and those of depressive patients are striking and might be related to alterations in cortisol and temperature circadian rhythms, created by the acoustic stress.

The lack of habituation of the phasic cardiovascular responses to isolated noises during sleep, as demonstrated by Muzet[39] may also possibly be related to experimental data from Jacobs and his collaborators[17], who showed that cells in the serotonergic nucleus raphe dorsalis (RD) fail to habituate in response to auditory or visual stimuli. The excitatory responses to the stimulations persist during sleep and are independent of behavioral arousal which habituates together with the responses of cells in the classical reticular formation[17]. It is known that, among other functions, the RD plays an important role in cardiovascular regulation, especially during stress[12], and this lack of habituation might therefore represent the neurophysiological basis for the data of Muzet.

It is a must point whether this lack of habituation could also be responsible for the increased prevalence of depressive states reported by Knipschild[21] in noisy areas. A decrease in the serotonin content of the RD is one of the most consistent findings in the brain of depressive suicides and hypersensitivity to noise also represents a common feature in depressed patients. It could be, therefore, that the level of metabolic exhaustion linked to depression might be precipitated in some borderline subjects by this constant responsiveness to environmental stimulation (see review in Mouret[35]).

Conclusion

Sleep is not only the output of an EEG machine but a global phenomenon with unique physiological, environmental and psychological features occurring in a given individual. There is no question that statistical evaluation of noise-induced sleep disturbances in non homogeneous groups of subjects has resulted in the construction of a magnificent neurophysiological edifice by assembling a few individual bricks. In this respect, whether subjectively disturbed or not, we now know that when we sleep in noisy areas our delta sleep is reduced, our heart rate fails to habituate to individual

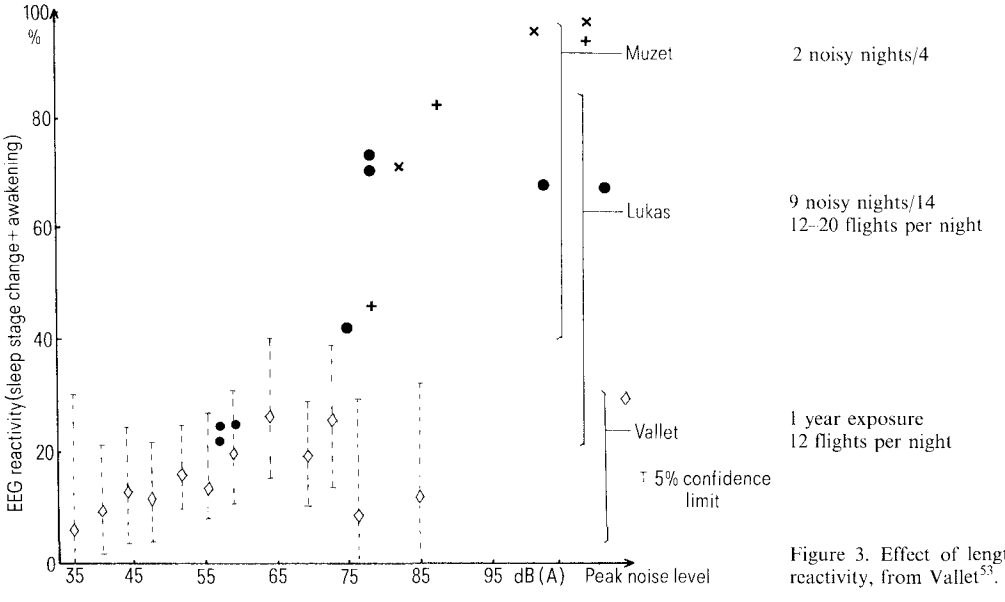

Figure 3. Effect of length of exposure on sleep EEG reactivity, from Vallet[53].

noises, and we are more likely to develop psychic disturbances than residents in quiet areas. Data from these studies are however interspersed with results from a number of experiments during which the subject has been looked upon as nothing but a sleeper, i.e., experiments in which day-time stresses and noises were considered as irrelevant for the night-time study. It seems important therefore that future studies should gather more longitudinal data based on a limited number of subjects selected according to their day-time habits and psychological (e.g., extroverts/introverts) and physiological (e.g., short or long sleepers) characteristics. The knowledge about these situational factors and personality traits might provide new insights into individual biological strategies developed to cope with noise stress.

The difficulties in assessing the effect of noise on sleep are particularly obvious if we consider another finding: deaf subjects spend significantly less time in delta sleep than do control subjects[47] and they present sleep pattern alterations remarkably similar to those which are most consistently described in noisy conditions.

* Research by the authors has been supported by grants from the Ministère de la Qualité de la Vie et de l'Environnement (France) and Commission of European Communities.

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Homeostatic and adaptive roles of human sleep

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In order to respond to the demands of life and reproduction, a living organism must develop multiple functions. The more these functions are integrated by and into higher levels of the central nervous system, the more complex they become. This is the case for sleep function since sleep has biological as well as psychological aspects, and interacts with two environments:

1. *internal,* characteristic of an individual with regard to similar functions both of lower level of integration and/or a more general type such as the circadian function, and

2. *external,* corresponding to the physiological and psychological environment.

Recent advances in the knowledge of the biological aspects of sleep have stimulated the development of studies concerning the interrelationship between its biological function (mainly related to the internal environment) and its psychological function (more related to the external environment). Where the latter is concerned the individual must integrate himself in the psychosocial world with the pressures, demands and needs it exerts upon him.

Phylogenetic studies of sleep in mammals have shown that during the nycthemeron the total duration of sleep, the number of its episodes as well as their composition vary from one species to another[4, 40]. In farm animals[34] and in monkeys[6] sleep varies with the environment. Similarly, human sleep behavior depends upon at least 2 categories of factors, internal and external. The internal factors modulate the innate rhythmic alternation of waking and sleeping while the external ones may change from time to time during a lifespan. The precise degree of contribution of either factor to actual sleep behavior is not yet known. However it would seem that sleep depends mainly on the 'internal state'.

Before describing these factors in greater detail, it is interesting to recall a few descriptive principles of the general living systems theory as reviewed by Miller[25]. Such a theory has allowed a new approach to the complex biological systems which have multiple levels of integration. This theory describes 2 different types of systems, closed and open. The closed systems are governed solely by the cybernetic rules of homeostasis and by feed back controls. The open systems are ruled in a similar manner but in permanent interaction with a complex enviroment in which relationships are generated for a specific, evolutive, adaptative organization in the framework of reciprocal, flexible and integrative processes. As for the relationship between sleep and biological functions, it appears to be more of a mixed but stabilized system called 'steady state'. This can be studied either from the point of view of homeostatic