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HEART RATE AND SLEEP LATENCY IN YOUNG MEN

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SEVERAL investigations of healthy subjects or of patients suffering from a variety of psychiatric disorders have shown that their usual delay before falling asleep at night (sleep latency) is related to their personality and the degree of their psychiatric impairment [1–6]. The concept of hyperactivation of the central nervous system, produced by the ascending reticular activating system and the limbic system, has often been invoked, explicitly or implicitly, to explain such a relationship.

Support for this hyperactivation theory of insomnia has come from reports that sleep latency is related to the heart rate, the rectal temperature and the frequency of peripheral vasoconstrictions at night [2], to mean rates of urinary excretion of cortisol and its metabolites [7, 8] and to the frequency of the subject's EEG during wakefulness [9]. By contrast, other investigators have reported that sleep latency is not significantly related either to the heart rate or to the level of integrated EMG activity measured at rest during the day [10–12].

The purpose of the present experiment was to determine whether sleep latency was related to the heart rate measured before sleep onset at night in healthy young men.

METHODS

Subjects

Fifteen healthy, male university students, aged 20–22 yr, volunteered to be subjects in an experiment concerned with the quality of sleep. They were asked not to take any drugs during the experiment, but in other respects their daily activities, including sporting activities in some subjects, continued as usual. They described their usual sleep habits in a sleep questionnaire [13] and none were insomniacs, although their usual sleep latencies varied between 5 and 30 min.

Recordings

The subjects came to the sleep laboratory about an hour before their usual bed-time and each slept for 3 consecutive nights in separate, warm and dark bedrooms. All-night recordings were made of the electroencephalogram (EEG, electrode positions C_4–A_1), electrooculogram, electrocardiogram (ECG) and, in some cases, the submental EMG. The recordings were made on a standard EEG machine and sleep stages were scored visually in periods of 40 seconds using standardized criteria [14]. Sleep latency was taken as the time between lights out and the appearance of the first spindle in the EEG of stage 2 sleep. The heart rate, calculated from the time taken for 20 R-waves in the ECG, was measured every 1–3 min throughout the recording, except when there were gross body movements which produced characteristic changes in the other channels of the recording and an increase in heart rate. Mean heart rates were calculated for each night's recording for the period (a) before sleep onset, and (b) during the first 10 minutes of sleep. The difference between (a) and (b) was used as a measure of the change in heart rate with sleep onset. Of the 45 nights involved initially, the heart rate data were not available for the first night in Subject 5. Subject 15 was studied when he was well and also for a further 3 nights beginning on the day after the onset of malaise, fever and membranous tonsillitis, later diagnosed as being caused by infectious mononucleosis. Results from the latter recordings were considered separately.

Statistical analysis

After normalizing the distribution of sleep latencies by $\log_e$ transformation, the results were analysed by 2-way analysis of variance, by product–moment coefficients, and by the paired *t*-test.

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Geometric means were calculated from the log transformed sleep latencies; arithmetic means from the other data which were not transformed. In the analysis of variance, a missing value for the heart rate was estimated by interpolation from all the other results.

RESULTS

Sleep latency

The mean duration of sleep for 45 nights was 439 (SD 50) min. Sleep latencies varied from 3 to 186 min in different subjects (Table 1). The (geometric) mean latency for all subjects was 14·8 min: the median and modal latencies were both 13 min. By contrast, the arithmetic mean of the untransformed data was 22·4 (SD 33·6) min: hence the need for transformation.

There were highly significant differences, demonstrated by the analysis of variance, between the sleep latencies of different subjects ($F = 9{\cdot}03$; $df = 14, 27$; $p < 0{\cdot}001$) and between the latencies on different nights ($F = 5{\cdot}78$; $df = 2, 27$; $p < 0{\cdot}01$). In general, latencies became shorter on successive nights (Table 2): the longest latency was on the first night in 10 subjects, on the second night in 4 subjects, and on the third night in 1 subject.

The mean sleep latency for each subject, after the first night in the laboratory, was similar to his usual latency at home during the preceding few weeks, as reported in the sleep questionnaire ($r = 0{\cdot}463$; $p = 0{\cdot}05$, 1-tailed test).

TABLE 1.—THE MEAN HEART RATE BEFORE SLEEP, THE CHANGE IN HEART RATE WITH SLEEP ONSET, AND THE SLEEP LATENCY FOR 3 NIGHTS IN EACH SUBJECT

Subject No.	Heart rate before sleep (bpm)	Change in heart rate with sleep onset (bpm)	Sleep latency (min)	
			Geometric mean	Range
1	76.9	-10.1	105	40-186
2	66.6	-2.9	15	5-27
3	54.8	-4.8	9	6-13
4	51.9	-2.5	14	13-17
5	59.9	-3.4	16	13-20
6	65.0	-6.7	11	8-13
7	44.0	-0.6	6	5-7
8	62.1	-6.1	35	23-39
9	65.6	-5.6	12	8-16
10	66.6	-5.9	12	9-17
11	64.6	-6.3	16	15-17
12	57.9	-7.1	9	7-12
13	55.9	-6.2	15	9-32
14	60.3	-1.0	27	24-34
15	61.0	+2.0	7	3-11
15, febrile	78.6	+1.4	9	4-20

Heart rate

The overall mean heart rate before sleep was 61·1 (SD 8·0) beats/min (bpm). There were highly significant differences between subjects (Table 1. $F = 11{\cdot}50$; $df = 14, 27$; $p < 0{\cdot}001$). The differences between nights (Table 2) were not significant ($F = 1{\cdot}43$; $df = 2, 27$; $p > 0{\cdot}1$). The change in heart rate with sleep onset varied from a decrease of 14·6 to an increase of 4·4 bpm, the mean being a decrease of 4·5 bpm ($p < 0{\cdot}001$, paired t-test). This change in heart rate with sleep onset varied significantly between subjects ($F = 3{\cdot}18$; $df = 14, 27$; $p < 0{\cdot}01$), but not between nights ($F = 1{\cdot}41$; $df = 2, 27$; $p > 0{\cdot}1$) although the greatest changes did tend to occur on the first night.

Considering all 44 nights for which data were available, there was a highly significant correlation between the sleep latency and the heart rate measured before sleep ($r = 0{\cdot}566$, $p < 0{\cdot}001$) and and between sleep latency and the change in heart rate with sleep onset ($r = 0{\cdot}400$, $p < 0{\cdot}01$). Because much of the data variance was derived from differences between subjects rather than between nights, correlation coefficients were calculated also between the mean sleep latency and the mean heart rates for each subject. These, too, were significant: $r = 0{\cdot}633$, $p < 0{\cdot}02$ for the heart rate before

sleep and $r = 0{\cdot}528$, $p < 0{\cdot}05$ for the change in heart rate with sleep onset. Thus, the higher a subject's heart rate was at night the longer he took to fall asleep and the more his heart rate decreased with sleep onset.

Fever in Subject 15 increased his sleep latency, but not as much as it increased his heart rate. This subject's heart rate increased with sleep onset, even in the presence of febrile tachycardia.

TABLE 2.—THE MEAN HEART RATE BEFORE SLEEP, THE CHANGE IN HEART RATE WITH SLEEP ONSET, AND THE SLEEP LATENCY FOR EACH NIGHT

Night	Heart rate before sleep (bpm)	Change in heart rate with sleep onset (bpm)	Sleep latency: Geometric mean (min)
1	62.9	-5.3	18
2	60.7	-3.5	16
3	60.8	-4.6	11

DISCUSSION

The results appear to support the hyperactivation theory of insomnia, at least in so far as difficulty in falling sleep is concerned, although this is only one of several dimensions of variation in the characteristics of a night's sleep [15].

The progressively shorter sleep latencies on successive nights were almost certainly due to decreasing levels of behavioural arousal as the subjects adapted to the laboratory and its procedures—the "first-night effect" [16]. Differences in behavioural arousal on successive nights may have affected the heart rate, as evidenced by the highest heart rate tending to occur on the first night, but that effect was overshadowed by other influences on heart rate, perhaps as a result of the day's activities (e.g. sporting activities). The relationship between heart rate and sleep latency was based mainly on the highly significant differences in both variables between different subjects; differences which were maintained across nights. However, sleep latency and heart rate were not related directly because a racing heart caused by fever did not markedly prolong sleep onset.

In healthy young adults whose resting heart rates are relatively low, as in the present investigation, the degree of parasympathetic (vagal) control is more important than that of sympathetic tone as a determinant of their heart rate; and the two controls can change independently, at least to some extent [17]. Similarly, the cardiac slowing which usually accompanies sleep onset is caused by an increase in vagal tone at that time as much as, and perhaps more than by a decrease in sympathetic tone [18, 19]. Low levels of vagal control of the heart and relatively long sleep latencies may, each in their own way, reflect a relatively low capacity for organized sleep-promoting activity in the central nervous system at night which may be partially independent of the level of arousal-promoting activity at the time. This could explain why insomnia occurs in ischaemic heart disease, in which parasympathetic control of the heart is known to be decreased [20], and why frontalis muscle tension, and by implication sympathetic tone, measured before sleep onset is not consistently related to sleep latency [11].

The failure of Freedman and Papsdorf [12] to find a significant relationship between sleep latency and heart rate in insomniacs may have been because their measurements of heart rate were made earlier in the day, not just before sleep onset. There is a circadian rhythm of heart rate which does not have the same phase in relation to the time of day in all subjects [21]. There may also be a circadian rhythm of sleep latencies which becomes manifest after allowance is made for differences in the duration of the preceding period of wakefulness [22].

If difficulty in falling asleep at night can be caused by a decrease in sleep-promoting activity in the central nervous system which is not simply a consequence of a high level of arousal-promoting activity, then our present conceptual basis for the treatment of insomnia may have to be changed.

SUMMARY

The interaction between sympathetic and parasympathetic control of the heart rate at rest may reflect, at least indirectly, the complex interactions between arousal-promoting and sleep-promoting activities in the central nervous system which determine the delay before falling asleep at night (sleep latency). The heart rate was measured before and during the first 10 min of sleep in 15 healthy men, aged 20–22 yr, who slept for 3 consecutive nights, with EEG monitoring, in a sleep laboratory. The sleep latencies and heart rates varied significantly between different subjects ($p < 0{\cdot}01$). The longer the sleep latency the higher the heart rate before sleep and the greater was the decrease with sleep onset.

Differences between the relatively low resting heart rates (around 60 bpm) of healthy subjects reflect differences in vagal tone as much as, and perhaps more than, differences in sympathetic tone. Similarly, consistent differences in sleep latency may reflect differences in the activity of sleep-promoting system which may be partially independent of the activity in arousal-promoting systems.

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