Short Report

Heart Blocks During Sleep: A Case Report in a Healthy Subject

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Summary: Cardiac arrhythmia was fortuitously discovered during an experimental sleep recording in a healthy 50-year-old man. A 24 hr electrocardiographic (ECG) monitoring revealed the prevalence of these abnormalities during sleep and their absence during the awake period. Sleep and ECG were recorded during 4 consecutive nights. Numerous (500–1,100 events/night) heart blocks and sinus pauses occurred 60–80 min after going to bed. They were never observed during awakenings and were exacerbated during paradoxical sleep. The atrioventricular blocks were of the Wenckebach-Luciani type. No other abnormality was found. These conduction abnormalities are discussed as possibly due to phasic increases in vagal firing, which during paradoxical sleep would be superimposed on the tonic increase in heart rate. Key Words: Sleep—Electrocardiography—Cardiac arrhythmia.

In recent years the prevalence of cardiac arrhythmias which occur during sleep has brought attention to their possible role in sudden death. Premature atrial or ventricular contractions have been reported during sleep (Rosenblatt et al., 1973) and bigeminy has been observed during paradoxical sleep (PS) (Jordan and Grice, 1977). Sinoatrial (SA) and atrioventricular (AV) blocks were observed during sleep in patients with sleep-induced obstructive apnea (Tilkian et al., 1977). In healthy students during 24 hr electrocardiographic (ECG) monitorings, Brodsky et al. (1977) observed frequent sinus pauses but rare AV blocks at night while sleeping. However, these authors did not record sleep parameters. Also interesting is the case reported by Nevins (1972), who studied a 56-year-old patient with congestive heart failure and found episodes of first- and second-degree AV blocks only during PS; other cardiac arrhythmias such as premature atrial and ventricular contractions were not specifically related to this stage of sleep.

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The case we are reporting is original in that it concerns a healthy subject experiencing SA and AV conduction abnormalities related to the different stages of sleep.

**METHODS**

The study was performed in a healthy 50-year-old man who exhibited SA and AV conduction abnormalities fortuitously during a previous experimental nocturnal sleep recording.

A 24 hr ambulatory ECG monitoring was obtained with a portable ECG tape recorder (Oxford Medilog 4-24) using a bipolar electrode lead system ($V_1-V_2$). During this recording, the subject followed his normal daily routine and slept from 0030 to 0730 hr. The tapes were analyzed using a rapid scanner for arrhythmia detection and data reduction (Medicat Electronic) at high speed ($\times 60$).

Four consecutive nights were recorded in a sound-proof room at 20°C ambient temperature. The subject lay on a hospital bed with blankets at his convenience. Polygraphic sleep tracings (electroencephalogram, electromyogram, electro-oculogram) were recorded at a paper speed of 15 mm/sec (Beckman Accutrace 200) and analyzed as previously described (Buguet et al., 1980). An ECG was recorded using bipolar montages from electrodes placed so as to record well-defined P waves. Nasal and buccal breathing was monitored with two thermocouples from one nostril and the mouth. Heart rate was calculated per minute by counting P waves, and all arrhythmias were counted and analyzed individually. P-P intervals were measured during the second sleep cycle (between the end of the first and second PS phases) both in periods with and without conduction blocks. These measurements were related to sleep stages and to the inspiratory-expiratory cycle. Data were compared with the Student's $t$-test.

**RESULTS**

A resting 12-lead ECG did not reveal any electrical abnormality in this healthy subject and showed a P-R interval within normal range ($177.2 \pm 1.5$ msec). An ambulatory ECG was performed for $22\frac{1}{2}$ hr. The subject had 84,368 cardiac complexes. The heart rate ranged from 92 to 45 beats/min (mean, 70) during the waking period and from 65 to 42 beats/min (mean, 60) during sleep (Fig. 1). The distribution of the R-R intervals (Fig. 1) was normal, with the left peak representing awake intervals and the right peak representing intervals during nocturnal sleep. Conduction abnormalities occurred only during sleep.

During the night, starting 60–80 min after going to bed, numerous (500–1,100/night) conduction abnormalities occurred. These were of two types and were superimposed on a background of a sinus arrhythmia. The first type corresponded to sinus pauses (average P-P interval 1.68 times as long as normal) and represented 5% of the conduction abnormalities. Definite SA blocks, with a P-P interval twice as long as the preceding normal interval, occurred in only 4%. The second type (95% of the abnormalities) was represented by second-degree AV block with the Wenckebach-Luciani phenomenon (Fig. 2).

Figure 3 shows that these conduction abnormalities occurred mainly during PS
FIG. 1. Analysis of the 24 hr electrocardiographic recording. Top: Histogram of the R-R intervals (msec) on 84,368 complexes. Bottom: Time course of the heart rate in beats per minute (bpm) averaged every 10 min.

(71%; 8.8 ± 0.45 events/min), although they were present during the other sleep stages (29%; 0.97 ± 0.10 events/min). They never appeared during awakenings intervening within the sleep period. During PS, 7% of the AV blocks were of the 2/1 type. They were not related to the occurrence of rapid eye movements, and there was no relationship with the phase of the respiratory cycle. No other type of cardiac arrhythmia was found. The respiratory recordings did not reveal any irregularity such as apnea.

FIG. 2. Examples of sinus pauses (A), sinoatrial blocks (B), and atrioventricular blocks (A, B, and C) on the electrocardiogram (ECG) occurring during a polygraphic recording of sleep (EOG, electro-oculogram; EMG, electromyogram; EEG, electroencephalogram, with bipolar montages O₁ - Cz on the upper trace and O₂ - Cz on the lower channel; R.R., respiratory rate, with buccal breathing on the upper channel and nasal breathing on the lower trace; i, inspiration; e, expiration). In A, there is a sinus pause with a P-P interval 1.68 times longer than the preceding normal P-P interval, followed with a 2/1 rate by an atrioventricular block. In B, a definite sinoatrial block (P-P interval twice as long as normal) is immediately followed by an atrioventricular block. Because of the short P-R interval following the atrioventricular block, one could question the existence of a nodal event. In C, atrioventricular blocks of the Wenckebach-Luciani period type reach the 2/1 rate. All these examples were taken during paradoxical sleep.
FIG. 3. Distribution of the sleep stages throughout 1 of the 4 nights and time course of heart rate, sinus pauses, and sinoatrial and atrioventricular blocks. Sleep stages are represented as a hypnogram (W: wakefulness; 1, 2, 3, 4: stages 1, 2, 3, and 4; PS: paradoxical sleep) in which vertical bars below the hypnogram represent body movements, horizontal bars over the hypnogram represent the presence of eye movements (in bursts of at least 2 sec duration in a 20 sec period), and horizontal dark bars in the hypnogram mark the periods of muscular atonia during the different stages of sleep. In this subject, sleep stages have a normal progression throughout the night. The different stages of sleep reach normal values for the age group of the subject (Feinberg and Carlson, 1968). The heart rate decreases progressively throughout the night, except for the awakenings, to reach 47 beats/min during the sixth hour of sleep. It also increases during paradoxical sleep, especially during the fourth phase. Heart blocks occur after about an hour of sleep and are exacerbated during paradoxical sleep.

The atrial rate, calculated from the measurement of the P-P intervals, was studied during stages 2 and 4 and PS. There was no progressive increase in the P-P intervals prior to an AV block. In each stage of sleep, there was a sinus arrhythmia related to the respiratory cycle (Table 1), with the atrial rate higher during inspiration than during expiration \( (p < 0.001) \). The atrial rate was also higher during PS than during the other stages of sleep \( (p < 0.001) \). However, it was similar in stages 2 and 4 in either phase of the respiratory cycle.

In all sleep stages, during the Wenckebach-Luciani phenomena, the P-P intervals did not vary with the respiratory phase. The P-P intervals were always greater in stage 2 \((1,132 \pm 5 \text{ msec})\) and stage 4 \((1,117 \pm 4 \text{ msec})\) than in PS \((1,086 \pm 3.8 \text{ msec})\). During PS, the atrial rate with the Wenckebach-Luciani phenomenon was always slower than in normal cardiac cycles \( (p < 0.001) \). During stages 2 and 4, the atrial rate with the AV block was slower than normal with inspiration \( (p < 0.001) \), but it did not differ from the normal expiratory rate.

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<th>TABLE 1. P-P intervals during different sleep stages as related to the respiratory phases (means ± SEM)</th>
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<td>P-P interval (msec)</td>
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DISCUSSION

In this healthy 50-year-old man, nocturnal sleep, especially PS, was accompanied by the occurrence of sinus pauses, few SA blocks, and numerous AV blocks of the Wenckebach-Luciani type. Such conduction abnormalities during sleep, along with other arrhythmic manifestations, were described by Brodsky et al. (1977) in 6% of their healthy subjects during 24 hr ECG monitorings. However, the highest amount of abnormalities rated only 17 events in one night. Tilkian et al. (1977) also described second-degree AV blocks in 2 and SA blocks in 5 of their 15 patients with sleep-induced obstructive apnea. Also, Nevins (1972) observed such conduction abnormalities in a patient with congestive heart failure. However, we did not observe any pathological sign nor any sleep apnea in our subject.

The 22½ hr ambulatory ECG recording showed that the conduction abnormalities occurred only between 0030 and 0730 hr and that the awake period was characterized by the absence of conduction abnormalities. Our subject also performed submaximal exercise (70% of his VO₂ max) on an ergocycle under ECG monitoring. No abnormality was recorded before, during, or after (20 min periods) the exercise session. The fact that the conduction blocks occurred selectively during sleep argues against the existence of an intrinsic depression of the AV transmission (Rosenblueth, 1958) but favors a vagal action mediating a depression of AV nodal conduction, which Brodsky et al. (1977) stated may be a normal variation of cardiac activity. The exacerbation during PS could be part of the phasic autonomic variations characterizing this stage of sleep. The intensity of the vagal firing would increase phasically during PS, superimposed on a background of tonically increased heart rate. This phasic occurrence of conduction blocks mainly during PS is in agreement with the findings of Baust and Bohnert (1969) that the phasic variability of heart rate during PS is under vagal control. This hypothesis is also supported by the work of Tilkian et al. (1977), in which atropine prevented AV blocks in 2 patients with sleep apnea syndrome.

The other findings of this study are in agreement with other works regarding the variability of heart rate with the respiratory cycle (Bond et al., 1973), as well as the values of heart rate and its increase during PS (Snyder et al., 1964).

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REFERENCES


RÉSUMÉ

Troubles du rythme cardiaque pendant le sommeil chez un homme en bonne santé