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Effects of Acute Sleep Deprivation On Repolarisation Parameters

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ABSTRACT

Acute sleep deprivation is associated with various adverse physiological consequences, such as dysregulation of the autonomic nervous system and adverse cardiovascular events. Markers of ventricular repolarisation are widely accepted in clinical practice to assess the risk of developing malignant ventricular arrhythmias. The aim of this study was to investigate the arrhythmogenic potential of acute sleep deprivation.

This was a prospective, observational study conducted in a single centre to investigate the relationship between acute sleep deprivation and ECG parameters. The included subjects did not have any acute or chronic diseases and ECG abnormalities. A 12-lead ECG was performed after a sleepless period of 24 hours after the night shift and after seven days of normal sleep following the night shift. The 12-lead ECG was recorded using a Philips PageWriter TC20 at a rate of 50 mm/sec and ECG parameters evaluated included Tp-e/QT and Tp-e/QTc ratios, frontal QRS-T angle, PR, QT, QTc and Tp-e intervals.

Fifty-nine nurses were included in the study. Qt and QTc duration, mean Tp-e and corrected Tp-e intervals were prolonged after acute sleep deprivation. In addition, Tp-e/Qt ratio and Tp-e/QTc were found to be longer after acute sleep deprivation compared to after normal sleep.

The results of our study suggest that acute sleep deprivation has a significant effect on ECG parameters, especially on repolarisation-related ECG parameters, but not on depolarisation parameters.

Keywords: Sleep deprivation, repolarisation, electrocardiography, Tp-e, QTc interval

Introduction

Sleep deprivation (SD) is a significant public health concern which harms the cardiovascular, and neurological systems (1, 2). Aging, lifestyle, stress, and shift work are all factors that contribute to insufficient and disturbed sleep (1, 3). SD is particularly common among shift workers, including medical professionals (4). SD is linked to several negative physiological outcomes, such as an increased risk of adverse cardiovascular events and autonomic dysregulation. Previous studies have shown that acute SD can reduce parasympathetic cardiac modulation and/or an increase in sympathetic activity, as determined by heart rate variability analysis (5). Getting sufficient sleep and maintaining good sleep quality can help regulate metabolism and reduce the incidence and mortality of certain diseases (1). The surface electrocardiogram (ECG) is a cost-effective, easily accessible, and non-invasive diagnostic tool commonly used in cardiology. It can be used to predict the risk of ventricular arrhythmias, particularly in clinical trials, by assessing certain parameters. Markers of ventricular repolarisation, which can be evaluated using surface ECGs, include the QT interval, corrected QT interval (QTc), QT dispersion (QTd), T peak-T end (Tp-e), and Tp-e/QT ratio which is widely accepted in clinical practice when assessing the risk of developing malignant ventricular arrhythmias (6).

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A number of studies have indicated that acute SD is associated with ECG abnormalities, including prolonged QT intervals and increased QT and P-wave dispersion (7, 8). However, only a limited number of studies have evaluated the relationship between Tp-e value in ECG and SD (9).

The objective of this study was to investigate the arrhythmogenic potential of acute SD. To achieve this, we compared various repolarisation parameters in ECGs taken from the same group of healthy adults after normal sleep and after 24 hours of SD.

Materials and Method

Study population: This is a prospective, observational study conducted at a single center to investigate the association between acute SD and ECG parameters. Written informed consent was obtained from all volunteers before the study. The individuals included in the study did not have any acute or chronic disease. Individuals with a history of pregnancy, those who use regular medication including sleeping pills, those who use stimulant substances, those who consume alcohol regularly, and those with sleep disorders including sleep apnea syndrome, and malignancies were excluded from the study. Furthermore, individuals whose ECG did not exhibit normal sinus rhythm and so analyses could not be performed, those who slept for a duration of six hours or less, and those who slept for a duration of nine hours or more were excluded from the study.

A physical examination and 12-lead ECG were conducted following a night shift, a period of 24 hours without sleep, and seven days of normal sleep following the night shift. Physical examination and ECGs were performed between 08.00 and 09.00 am. In the physical examination, the resting heart rate was recorded. Cardiac and lung auscultation was conducted. It was requested that participants refrain from consuming caffeinated products and alcohol.

Electrocardiography: Using a Philips PageWriter TC20, the 12-lead ECG was recorded at a speed of 50 mm/sn. The Tp-e/QT and Tp-e/QTc ratios, frontal QRS-T angle, PR, QT, QTc, and Tp-e intervals were among the ECG parameters that were assessed. From the beginning of the QRS complex to the T wave's termination, which is defined as the return to the T-P isoelectric line, the QT interval was measured. Bazett's formula was used to calculate the corrected QT (QTc) interval. The time interval between the peak of a positive T wave or the nadir of a negative T wave and the T wave's end was referred to as the Tp-e interval. Lead V5 was used to measure the Tp-e interval and heart rate was taken into account. Subsequently, the Tp-e/QT, Tp-e/Qtc, and corrected Tp-e/Qtc ratios were calculated as the ratio of Tp-e that led to the corresponding QT interval. The frontal QRS-T angle was calculated by subtracting the frontal plane QRS axis from the frontal plane T axis.

This study was approved by the ethics committee of a training and research hospital in Istanbul, Türkiye (approval code:14-2023, approval date: 01 March 2023).

Statistical analysis: Continuous variables are presented as mean ± standard deviation. The Kolmogorov–Smirnov test was used to assess the normality of their distribution. To compare continuous variables before and after the night shift, a paired t-test was applied for parametric data, while the Wilcoxon test was used for nonparametric data. A two-tailed P-value < 0.05 was considered statistically significant. Statistical analyses were performed using SPSS, version 23.0.

Results

Fifty-nine nurses working both on the day and night shifts in different departments of our hospital were enrolled in the study. The average age of the participants was 26.9±5.9 years, and 74.5% of them were female. The average body mass index was 23±4.2 kg/m^2. The physical examination of the patients revealed normal. Table 1 displays ECG parameters. No significant difference was detected in heart rate, PR duration, or P wave between after normal sleep and after acute sleep deprivation (74.8 ± 10.8 and 74.5±10.4beats/min., p=0.836; 143.6 ± 18ms and 145.1±17.9ms, p=0.298; 71±14.2ms and 70.8±13.7ms, p=0.907; respectively). Qt ve QTc duration was longer after acute sleep deprivation (373.2±23.7ms and 377.9±21.4ms, p=0.131; 414.4±24.1ms and 421±24.8ms, p=0.009; respectively). The mean Tp-e and corrected Tp-e intervals were significantly prolonged after acute sleep deprivation (62.3±12.6ms and 66.4±13.4ms, p=0.038; 69.2±14.5ms and 74.6±15.5ms; p=0.016; respectively). In addition, the Tp-e/Qt ratio and Tp-e /QTc were higher after acute sleep deprivation compared to after normal sleep (0.17±0.03 and 0.18±0.04, p=0.036; 0.15±0.03 and 0.16±0.03, p=0.091; respectively).

Table 1: Evaluation of the Electrocardiographic Parameters In Healthy Individuals After Normal Sleep and After Acute Sleep Deprivation

Parameters	After normal sleep	After acute sleep deprivation	p value
Heart rate (beats/min)	74.8 ± 10.8	74.5 ± 10.4	0.836
PR duration (ms)	143.6 ± 18	145.1 ± 17.9	0.298
P wave (ms)	71± 14.2	70.8 ± 13.7	0.907
QRS duration (ms)	92.1 ± 9.9	92.6 ± 10	0.581
QT duration (ms)	373.2±23.7	377.9±21.4	0.131
QTc duration (ms)	414.4±24.1	421±24.8	0.009
Frontal QRS-T angle	17(5-38)	17(3-30)	0.050
QRS axis	53±25.6	52.8±26.3	0.887
T axis	33.8±19.2	38.6±15	0.019
Tp-e (ms)	62.3±12.6	66.4±13.4	0.038
Corrected Tp-e (ms)	69.2±14.5	74.6±15.5	0.016
Tp-e /QT	0.17±0.03	0.18±0.04	0.036
Tp-e /QTc	0.15±0.03	0.16±0.03	0.091

QTc: corrected QT interval, Tp-e: Interval from the peak to the termination of the T-wave ,Tp-e/QT: Ratio of Tp-e to QT interval, Tp-e/QTc: Ratio of Tp-e to corrected QT interval

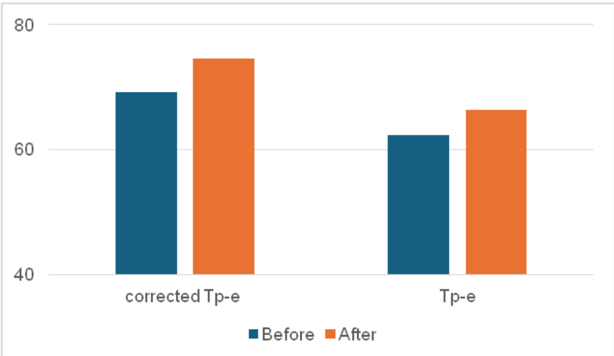

Fig. 1. Corrected Tp-e and Tp-e intervals in healthy individuals before (blue bars) and after (orange bars) acute sleep deprivation. Data are presented as mean ± standard deviation. Significant prolongation in both corrected Tp-e and Tp-e intervals was observed after sleep deprivation

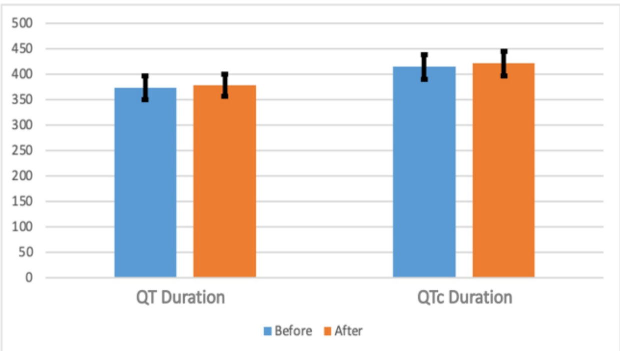

Fig. 2. QT and QTc durations in healthy individuals before (blue bars) and after (orange bars) acute sleep deprivation. Data are presented as mean ± standard deviation. QTc duration showed a significant increase after sleep deprivation, while QT duration did not change significantly

Discussion

In this study, we investigated the effects of acute SD on ECG parameters. Our findings indicate that acute SD causes prolongation in QTc duration, T axis, Tp-e interval as well as an increase in Tp-e/Qt ratio. Although sleep is a fundamental necessity for human rest and recuperation, it is widely acknowledged that its effects extend beyond this function. Sleep is a fundamental biological need that plays a crucial role in the maintenance of high-level cognitive functions and the restoration of the body's physiological processes (10, 11). Previous studies have suggested that SD can have an impact on the function of the central nervous system, autonomic nervous system, and cardiovascular system (5). SD is a condition associated with increased sympathetic and decreased parasympathetic activity and has many effects on the cardiovascular system through these mechanisms (1). The current findings indicate that even a single night of acute SD can result in alterations to several key biological markers, including those associated with inflammation, cortisol hormone levels, emotional state, and cognitive performance (12). As a result of increased sympathetic activity, an increase in blood pressure and heart rate is observed in SD. In our study, there was no significant difference in heart rate before and after acute SD. Similar to our study, Holmes et al. investigated the effect of SD on autonomic cardiac functions and found that SD had no significant effect on mean heart rate (13). Previous studies have demonstrated a strong correlation between one acute SD exposure and the development of atrial dysfunction, and increased arterial stiffness, preceding altered contractile function or overt left atrial enlargement (14, 15). In addition, another recent study demonstrated that acute SD results in a decline in the functionality of both ventricles and the left atrium (16). Consequently, epidemiological studies have established a correlation between acute SD and cardiovascular diseases which represent the leading cause of mortality and sudden cardiac death worldwide. However, the precise nature of this association remains uncertain (17).

In addition to the structural effects observed in the heart, there is evidence that acute SD also affects electrical activity (7, 8). Ventricular repolarisation is influenced by the autonomic nervous system. The length of the QT interval reflects the relationship between ventricular repolarization and the autonomic nervous system. (18). In a study, Toivonen et al. demonstrated that acute sympathetic activation alters ventricular repolarisation parameters. The sequence of events for PKA activation is as that which follows catecholamine stimulation. The activation voltage shifts to more negative potentials and the peak ICa increases significantly as a result of the fast phosphorylation of the L-type Ca^{+2} channel. This increases excitability and elevates the amount of Ca^{+2} in the cells (18). The QT interval is a marker that includes ventricular depolarisation and repolarisation, while the Tp-e duration is a marker that is more related to ventricular repolarisation (9). Various studies have demonstrated a correlation between prolonged QT interval and Tp-e duration and an increased susceptibility to ventricular arrhythmia. Ting-Yan Zhu et al. demonstrated that elevated Tp-e and Tp-e/QT ratios are associated with an increased risks of ventricular arrhythmias in patients with ICD for the purpose of primary prevention (19). In the study conducted by Mert et al. with bodybuilders, they demonstrated that Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratios were significantly greater in bodybuilders than in the control group and positively correlated with arrhythmias (20).

It was previously demonstrated that SD may increase the risk of ventricular arrhythmias. In a study, Fang et al. investigated the effect of acute SD on rat heart muscle cells. They found that action potential duration and repolarisation time were prolonged and the transient outward current (Ito) channel was inhibited (21). In a study on rats, Joukar et al. showed that 72 hours of REM SD prolonged the QT interval. It has been suggested that this prolongation may be caused by the imbalance between sympathetic and parasympathetic tonus, especially during prolonged SD. It has also been suggested that it may be secondary to electrolyte imbalance due to the diuresis that develops as a result of SD (22). In a study of 37 healthy subjects, Ozer et al. showed that acute SD caused a significant increase in QT interval and QT dispersion (8). In a study by Çakıcı et al., 40 healthy healthcare workers were observed to exhibit an increase in the QT interval, Tp-e corrected Tp-e interval, and Tp-e/Qt ratio following acute SD (9). Furthermore, in our study with a higher number of participants, an increase in the Tp-e/Qt ratio was observed in individuals experiencing acute SD. However, this increase was not statistically significant.

The QTc values of all subjects were within normal limits before and after SD in the current study. However, studies have shown that an increase in QTc, even if within normal limits, increases the risk of arrhythmia (23). Similarly, all subjects in the study had Tp-e values below 85 ms before and after SD, which we set as the upper limit. However, as there is no study in the literature showing the clinical effect of an increase in Tp-e on arrhythmia in healthy subjects, we cannot make the same interpretation in this case as we did for the QTc interval. The combined depolarisation and repolarisation marker frontal QRS-T angle that is another parameter associated with arrhythmia risk was not found to be significantly different between the two groups.

Our findings are partially consistent with previous studies. We have demonstrated that acute SD due to various factors, including working conditions, has cardiac effects in the present which can be detected from a 12 lead surface ECG. This study with healthy volunteers suggests that clinically significant results may be obtained if similar studies are conducted in patients with various rhythm disorders or different chronic heart diseases in the future. This will further highlight the protective physiological effects of sleep, which is an important part of a healthy life, against adverse events. Although our population is larger than that of similar studies in the literature, it is nevertheless relatively small. Furthermore, the self-reporting of the sleep hours of the study participants represents another limitation.

Acute SD induces significant changes in ECG parameters, especially those that indicate ventricular repolarisation. To the best of our knowledge, this is the first study to examine the impact of acute SD on novel repolarisation parameters including the frontal QRS T angle.

Our findings indicate that there was no significant difference between the two groups. Consequently, the results of our study may indicate that acute SD has a significant impact on ECG parameters especially related to repolarisation, while the effect on depolarisation parameters remains uncertain.

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