

Electrocardiographic changes during acute mental stress

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Abstract

Background: Electrocardiogram (ECG) waveforms are influenced by physiological, pathological, and psychological factors. Acute mental stress affects the hypothalamic–pituitary–adrenal axis resulting in alterations in various physiological functions of the body particularly the cardiovascular system.

Objective: To study the ECG changes during acute mental stress and also to understand the reasons for the changes in ECG caused by acute mental stress.

Materials and Methods: ECG was recorded in relaxed state and in acute stress state in healthy male subjects in the age group of 18–24 years. ECG recordings in relaxed state were compared with those in acute stress state.

Result: Increased heart rate, decreased PR interval, decreased QT interval, and prolonged QTc interval were observed in ECGs in acute stress state which were statistically significant. Also an increase in QRS duration and change in QRS axis were observed during acute stress which were statistically not significant.

Conclusion: Increased sympathetic activity caused by acute mental stress may be the cause for this altered electrical activity of the heart.

KEY WORDS: ECG; acute stress; sympathetic activity; vagus nerve.

Introduction

Electrocardiogram (ECG) waveforms are influenced by physiological, pathological, and psychological factors. Physiological factors that affect the ECG waveforms are body mass index, pregnancy, sex, race, and age. The pathological factors are bundle branch block, coronary artery disease, electrolyte imbalances, ventricular hypertrophy, acute pericarditis, etc.^[1]

Along with physiological and pathological factors even psychological factors, such as acute stress and chronic stress, affect the ECG waveforms.

Stress is common in day-to-day life and it affects many physiological functions of the body, mainly the cardiovascular system.^[2] Mental and physical stress is widely renowned as playing an important role in ventricular arrhythmias and sudden cardiac death. In fact, mental and physical stress can cause ischemia, and ischemia may precipitate ventricular tachycardia and ventricular fibrillation.^[3,4]

The neural pathways from the brain to the heart are via the right and left sympathetic and parasympathetic nerves (autonomic nerves) that are distributed asymmetrically in the ventricular myocardium.^[5,6] On the basis of the fact that the sinus node, which governs heart rate, is influenced predominantly by the right sympathetic nerves,^[7] several studies have shown that stimulation of the right-sided brain structures, but not the left, induces an increase in heart rate.^[8]

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ECG is a commonly used test for cardiovascular studies.^[2] Acute stress is now one of the major causes leading to fatal ventricular arrhythmia. The mechanisms by which acute stress causes arrhythmias are not completely understood.^[9]

In this study, an attempt has been made to study the ECG changes during acute mental stress and also to understand the reasons for the changes in ECG caused by acute mental stress.

Materials and Methods

This study was carried out in Department of Physiology, SVIMS, Tirupati, Andhra Pradesh, India. The study group included 33 healthy men in the age group of 18–24 years pursuing paramedical and physiotherapy courses in SVIMS University. This study was approved by the Institutional ethics committee of Sri Venkateswara Institute of Medical Sciences. Informed consent was obtained from all the study subjects. Clinical examination was carried out to rule out any underlying disease. Subjects with smoking history, anemia, and cardiovascular disease were excluded.

All the ECGs were recorded at a speed of 25 mm/s with Cardiouch 3000 ECG machine (Bionet America, Inc.). ECG was recorded in relaxed state on first day. The following day ECG was recorded in acute stress state on the same subjects. Acute stress is defined as stress in which the subject is required to actively cope (do something) or perform in a challenging situation. In this study, participants were asked to perform a 1-min mental arithmetic task, which has been shown to induce psychological stress.^[10] The stressed subjects were evaluated with Spielberger's State and Trait Anxiety Inventory (STAI) score.^[11] The ECGs were analyzed for heart rate, PR interval, QRS duration, QT interval, QTc interval, and QRS axis.

Statistical Analysis

Data was presented as mean \pm SD. Stress-induced responses were examined by paired Student's *t*-test. The data was compiled in Microsoft Excel spread sheets. Differences were considered as significant if *p*-value < 0.05 .

Result

ECGs were collected from each subject at two stages, first in relaxed state, second in acute stress state during arithmetic task performance. The findings observed in ECGs were tabulated in [Table 1]. The mean age of the study population was 19.27 ± 1.21 years, mean height was 164.7 ± 7.23 cm, and mean weight was 54.76 ± 9.4 kg.

Increased heart rate (75.72 ± 9.99 vs 99.06 ± 19.05 ; $p < 0.0001$), decreased PR interval (128.48 ± 17.76 vs 120.72 ± 22.58 ; $p = 0.0126$), decreased QT interval (354.30 ± 26.50 vs 336.72 ± 27.10 ; $p = 0.0001$) and prolonged QTc interval (393.06 ± 21.76 vs 426.69 ± 32.73 ; $p < 0.0001$) were observed in ECGs in acute stress state that were

statistically significant [Table 1]. An increase in QRS duration (97.69 ± 16.67 vs 99.39 ± 17.34 ; $p = 0.190$), and change in QRS axis (64.57 ± 29.91 vs 57.39 ± 37.80 ; $p = 0.212$) were also observed during acute stress which were statistically insignificant [Table 1].

Discussion

During acute stress state an increase in heart rate was observed. This is due to increased sympathetic activity that is elicited by acute stress. There was also decrease in PR and QT intervals. Since the heart rate is the major determinant of QT interval, the QT interval was reduced due to increased heart rate.^[12] Similar findings were reported by Magri *et al.*,^[13] Lampert *et al.*,^[14] Nagaraja *et al.*^[15] We also found a prolonged QTc interval during acute stress condition. This may be due to autonomically induced repolarization changes. During acute stress there is inhomogeneity of repolarization that may be due to autonomic imbalance.^[16] Similar findings were reported by Andr assy *et al.*^[17]

Acute emotional stress can have significant adverse effects on heart. Abnormalities such as left ventricular contractile dysfunction, myocardial ischemia, and disturbances in cardiac rhythm can be produced by acute emotional stress, which is only transient. But their consequences can be sometimes fatal. Physiologic pathways by which emotional stress triggers cardiovascular events are now being studied with the help of functional neuroimaging. Recent evidences suggest that asymmetric brain activity during acute emotional stress causes asymmetric activation of heart leading to production of inhomogeneous areas of repolarization and production of electrical instability of heart.^[18] The right and the left autonomic nerves are asymmetrically distributed over the ventricles. Therefore functional autonomic asymmetry exists in response to selective autonomic nerve stimulation.^[9]

The neural input to the heart consists of vagal efferents that innervate the sinoatrial node and the atrioventricular node. Atrial muscle is affluently innervated by vagal efferents as well, but the ventricular myocardium is only meagerly innervated by the vagus nerve. By contrast, sympathetic stimulation is widespread throughout the heart. Sympathetic efferents are present throughout the atria, especially in the sinoatrial node, and throughout the ventricular myocardium and cardiac conduction system. The increased heart rate observed in this study is due to the increased sympathetic activity on the myocardium. It has been known for decades that central sympathetic outflow to the heart can trigger ventricular arrhythmias.^[19]

More recent evidence indicates that lateralization of cerebral activity during emotional stress may stimulate the heart asymmetrically and produce areas of inhomogeneous repolarization that create electrical instability and facilitate cardiac arrhythmias.^[20] The contribution of central neurogenic factors to the generation of arrhythmia is highlighted further in studies demonstrating pathological ECG changes elicited by stimulation of specific brain regions.^[21,22] Anatomically, there is evidence

Table 1: ECG findings in relaxed state and during acute stress

| ECG parameters | Mean±SD | | p-Value |
|------------------------|----------------|--------------------|----------|
| | Relaxed state | Acute stress state | |
| Heart rate (beats/min) | 75.72 ± 9.99 | 99.06 ± 19.05 | <0.0001* |
| PR interval (ms) | 128.48 ± 17.76 | 120.72 ± 22.58 | 0.0126* |
| QRS duration (ms) | 97.69 ± 16.67 | 99.39 ± 17.34 | 0.190 |
| QT interval (ms) | 354.30 ± 26.50 | 336.72 ± 27.10 | 0.0001* |
| QTc interval (ms) | 393.06 ± 21.76 | 426.69 ± 32.73 | <0.0001* |
| QRS axis (degrees) | 64.57 ± 29.91 | 57.39 ± 37.80 | 0.212 |

*Indicates a significant *p*-value (*p*<0.05).

for subcortical lateralization of efferent sympathetic pathways, with segregation of left and right responses maintained at the level of brain stem and spinal cord.^[23] The left- and right-sided autonomic (sympathetic) nerves are distributed asymmetrically over the ventricles, and unilateral stimulation of either side may alter repolarization inhomogeneity.^[5] Thus, repolarization inhomogeneity may reflect 'upstream' influences on the right-left symmetry of sympathetic cardiac drive, for example asymmetric brain activation in response to stress. When midbrain activation in response to stress was bilaterally symmetrical, the repolarization inhomogeneity in the heart was unchanged. However, when stress-induced midbrain activation was lateralized to the right, repolarization inhomogeneity (i.e., the arrhythmogenic substrate) was enhanced.

Stress may precipitate cardiac arrhythmia and sudden death in vulnerable patients, presumably via centrally driven autonomic nervous system responses. From a cardiological perspective, the likelihood of arrhythmia is strongly associated with abnormalities in electrical repolarization (recovery) of the heart muscle after each contraction. Inhomogeneous and asymmetric repolarization, reflected in ECG T-wave abnormalities, is associated with a greatly increased risk of arrhythmia, that is, a proarrhythmic state.^[24]

Various stress effector systems serve as neural conduits that integrate cognitive cues and mediate the stress response. These stress effector systems include the sympathoneural system; the adrenomedullary hormonal system; the parasympathetic nervous system; and the hypothalamic-pituitary-adrenal axis, renin-angiotensin-aldosterone, and vasopressin systems.^[25] Sympathetic hyperactivation in response to both mental challenge and affective distress increases circulating levels of epinephrine and norepinephrine.^[4,26] The prolonged QT interval is regarded as a marker of imbalanced distribution of sympathetic nervous system activity on the heart; also QT interval prolongation has been associated with a lowered ventricular fibrillation threshold and with the occurrence of sudden cardiac death.^[27]

Now there is substantial evidence that an interactive system formed by the higher brain centers (cerebral cortex), the brain stem, and autonomic nervous system can affect cardiac electrophysiology. These heart-brain interactions help explain sudden cardiac events related to acute stress and provide

insights into newer therapies in future to prevent sudden death.^[28]

Limitation

Sample size is relatively small to generalize the study findings.

Conclusion

Acute mental stress causes an increase in heart rate, decrease in PR interval, decrease in QT interval, and prolongation of QTc interval. Increased sympathetic activity caused by acute mental stress may be the cause for this altered electrical activity of the heart.

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