Effect of mental stress on autonomic nervous function in young adults

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Abstract

Introduction: Autonomic imbalance is one of the important pathways through which psychological stress contributes to cardiovascular diseases/sudden death. Stress is a normal physical response to events that make us feel threatened or upset our balance in some way. There is a major role of mental stress in provoking silent myocardial ischemia, cardiac arrhythmia, catecholamine induced increase in heart rate, increase in blood pressure that result in enhanced myocardial oxygen demand and sometimes sudden death. In this study an attempt is made to study the variations in electro cardiogram (ECG) after inducing mental stress in normal subjects.

Materials and Methods: The study included 50 healthy medical students in the age group of 18-25 studying in VIMS, Bellary. Following an explanation about the nature and purpose of the study, those subjects who are willing to participate were included after obtaining their consent. Blood pressure and ECG was recorded at rest in supine position. Then mental stress was induced on each by a no. of mental tasks and ECG was recorded in them immediately.

The stressed subjects were evaluated with Speilberger's State and Trait Anxiety Inventory (STAI) score. Blood pressure and ECG results were evaluated for different parameters and the data collected was tabulated and subjected to statistical analysis.

Results: There was statistically significant increase in heart rate, systolic BP, diastolic BP, decrease in PR interval and QRS interval, increase in QTc interval, T wave pattern changes, ST segment depression found after inducing mental stress.

Conclusion: The study shows that there were varieties of ECG changes in mental stress that may affect the health of human beings.

Keywords: Blood pressure (BP), Electro cardiogram (ECG)**,** Mental stress.

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Introduction

Stress is common in day-to-day life and it affects many physiological functions of the body, mainly the cardiovascular system.[1] 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.[2]

The association between autonomic imbalance and increased cardiovascular disease (CVD) risks has been emphasized by earlier studies.[3] A relative or absolute decrease in vagal activity or an increase in sympathetic activity influences heart rate and beat-to-beat rhythm.[4] Autonomic imbalance increases arrhythmias involved in coronary occlusion and lowers ventricular fibrillation threshold resulting in sudden death.[5] Electrocardiographic (ECG) QT intervals, as well as heart rate variability (HRV), blood pressure and heart rate, have been considered as a biomarker of the development of ventricular arrhythmia or of the susceptibility to sudden death, because QT intervals represent the duration between ventricular depolarization and subsequent repolarization.[6]

ECG is a commonly used test for cardiovascular studies. Acute stress is now one of the major causes leading to fatal ventricular arrhythmia.[7-9] The mechanisms by which acute stress causes arrhythmias are not completely understood. 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

Study was conducted in Department of Physiology, VIMS, Bellary during the period from 2011-2012 which included 50 healthy medical students in the age group of 18 to 25 years studying in VIMS, Bellary. Individuals with h/o heart diseases like myocardial infarction, congenital heart disease, arrhythmias, and hypertension, respiratory diseases like chronic obstructive pulmonary disease, cor

pulmonale, anemia, renal disease or thyroid disease, chronic medication, psychiatric illness, abuse of psychoactive substances and alcohol were excluded.

After taking consent and a detailed history from the subjects, electrocardiogram was recorded at rest in supine position, then mental stress was induced on each by a no. of mental tasks (eg. public speaking, type A interviews, seminars, mental arithmetic problems) and ECG was recorded in them immediately. The stressed subjects were evaluated with Speilberger's State and Trait Anxiety Inventory (STAI) score.[10] The study was approved by the institutional ethical committee overseeing human studies.

The ECG results were evaluated for different parameters like heart rate, P wave, PR interval, QRS complex, QT interval, QT_c interval, ST segment and T wave.

Statistical Analysis

The statistical analysis was carried out by using the SPSS (Statistical Package for Social Sciences) software version15. The Paired't' test was applied for the statistical analysis and the results were expressed in mean ± SD, P values ($p < 0.001$) were considered as highly significant. Wilcox sign rank test is used to compare ordinal data before and after the intervention. Mc.nemar test is used for comparing qualitative variable before and after the intervention.

Results

Table 1:**Age-wise distribution of study subjects**

Age group	Frequency	Percentage
19 yrs	10	20%
20 yrs	08	16%
21 yrs	12	24%
22 yrs	12	24%
23 yrs	08	16%
Total	50	100%

Total study subjects participated in the study was 50. Among them, highest percentages of study subjects were in between 20-22 years. (Table 1)

Table.2: **Mean ± SD of HR, SBP, DBP, PR, QRS, QT and QTc intervals at rest and after mental stress**

Measurements	Resting value	After mental stress	P value*
Heart rate (bpm)	75.6 ± 3.9	81.12 ± 3.8	0.001
Systolic BP (mmHg)	118.04 ± 3.7	122.88 ± 2.3	0.001
Diastolic BP(mmHg)	78.04 ± 3.4	82.32 ± 2.1	0.01
PR interval(sec)	0.148 ± 0.015	0.138 ± 0.011	0.001
QRS interval(sec)	0.080 ± 0.000	0.07 ± 0.004	0.001
QT(sec)	0.344 ± 0.014	0.341 ± 0.016	0.09
QTc(sec)	0.385 ± 0.009	0.394 ± 0.011	0.01*

* paired 't' test, (n=50)

Heart rate (81.12 ± 3.8), Systolic BP (122.88 ± 2.3) and diastolic BP (82.32 ± 2.1) was more after mental stress, when compared to that of rest (75.6 ± 3.9), (118.04 ± 3.7) and (78.04 ± 3.4) respectively and it was statistically significant. PR interval (0.138 ± 0.011) was shortened after mental stress when compared to that of rest (0.148 ± 0.015) with a difference of 0.010 seconds. QRS interval (0.07 ± 0.004) was shortened after mental stress when compared to that of rest (0.080 ± 0.000) with a difference of 0.01sec (Table 2)

There was no much difference in duration of QT interval (0.341 ± 0.016) after mental stress when compared to that of rest (0.344 ± 0.014), and it was statistically not significant. But QTc interval (0.394 ± 0.011) was slightly high after mental stress when compared to that of rest (0.385 ± 0.009) (Table 2).

Table 3: **Mean ± SD of P wave at rest & after mental stress (n=50)**

Measurements	Resting value	After mental stress	P value*
Duration(sec)	0.065 ± 0.009	0.065 ±0.009	Not applicable
Amplitude(mm)	1.00 ± 0.00	1.00 ± 0.00	Not applicable

* paired 't' test

There was no difference in P wave duration (0.065 ± 0.009) & amplitude (1.00 ± 0.00) after active mental stress when compared to that of rest condition and it was statistically not significant (Table 3)

Table 4: Mean ± SD of T wave at rest & after mental stress (n=50)

T wave	Under rest	After mental stress*
Normal	50 (100%)	33 (66%)
Flat	00	08 (16%)
Inverted	00	09 (18%)
Total	50 (100%)	50 (100%)

* Wilcox sign rank test p value – 0.001(significant)

66% of the subjects had normal T wave, 16% had flattened T wave, 18% had inverted T wave pattern after active mental stress when compared to that of rest and it was statistically significant (Table 4).

Table 5: Mean ± SD of ST segment at rest &after mental stress (n=50)

T wave	Under rest	After mental stress*
Isoelectric	50 (100%)	38 (76%)
Depressed	00	12 (24%)
Total	50 (100%)	50 (100%)

* Mcnemar test p value – 0.001 (significant)

76% of the subjects had isoelectric ST segment, 24% had depressed ST segment after active mental stress when compared to that of rest and it was statistically significant (Table 5)

Discussion

Heart rate (81.12 ± 3.8), Systolic BP (122.88 ± 2.3) and diastolic BP (82.32 ± 2.1) was more after mental stress, when compared to that of rest (75.6 ± 3.9), (118.04 ± 3.7) and (78.04 ± 3.4) respectively and it was statistically significant (Table 2). Similar findings were reported by Nis Hjortskov et al,[11] C Noel bairey merz et al,[12] Lampert R et al.[13] The increase in heart rate could be due to activation of sympathetic nervous system (sympathetic predominance), vagal withdrawal because of mental stress. Increased heart rate is associated with increased mortality rates.

PR interval (0.138 ± 0.011) was shortened after mental stress when compared to that of rest (0.148 ± 0.015) with a difference of 0.010 seconds (Table 2), similar findings was reported by Arpana Bhide et al.[14] PR interval was shortened after inducing mental stress and this was statistically significant. Mental stress could lead to augmentation of sympathetic system, catecholamine release which can lead to increase in the velocity of conduction in atria.

QRS interval (0.07 ± 0.004) was shortened after mental stress when compared to that of rest (0.080 ± 0.000) with a difference of 0.01sec, and this differences was statistically significant. (Table 2) A similar finding was reported by Arpana Bhide et al.[14] Mental stress influences on intra ventricular conduction time by changing autonomic balance, which can lead to reduced QRS duration.

There was no much difference in duration of QT interval (0.341 ± 0.016) after mental stress when compared to that of rest (0.344 ± 0.014), and it was statistically not significant. But QTc interval (0.394 ± 0.011) was slightly high after mental stress when compared to that of rest (0.385 ± 0.009) and it was statistically significant. (Table 2**)** Similar findings were reported by Gabor Andrassy et al.[15] Mental stress could lead to ANS imbalance, adrenergic release, increased sympathetic tone, which can lead to ventricular depolarization heterogeneity leading to prolonged QTc interval.

There was no difference in duration and amplitude of P wave after mental stress (0.065 ± 0.009) when compared to that of rest (0.065 ± 0.009) (Table 3). P wave duration and amplitude

measurements did not show any statistically significant difference after inducing mental stress when compared to ECG under rest.

66% of the subjects had normal, 16% had flattened, 18% had inverted T wave pattern after mental stress when compared to that of rest and it was statistically significant. (Table 4) Similar findings was reported by Gabor Andrassy et al,[15] Jan henrik atterhog et al[16], Mental stress may lead to changes in ventricular repolarisation by augmentation of ANS, discharge of catecholamines, glucocorticoids and this could be the cause for T wave pattern changes.

ST segment observed 76% of the subjects had isoelectric, 24% had depressed after mental stress when compared to that of rest and it was statistically significant (Table 5). A similar finding was reported by L Toivonen et al,[17] Peter H Stone et al.[18] Mental stress may lead to changes in ventricular repolarisation by augmentation of ANS, which may lead to ischemic related ECG changes.

Conclusion

There is a major role of mental stress in provoking catecholamine induced increase in heart rate, BP, cardiac arrhythmia, myocardial infarction that may affect the health of human beings. There was significant increase in heart rate, systolic BP, diastolic BP. Decrease in PR interval and QRS interval and increase in QTc interval, no changes in P wave, T wave pattern changes and ST segment depression found after inducing mental stress in this study.

This study provide a glimpse into the variety of ECG changes in people who are in mental stress and thereby helps in creating awareness so that, they can alter their lifestyle in order to prevent its ill effects. Additional research is needed to identify different types of stress that are strongly associated with autonomic imbalance.

References

1. Kumar R, Kela A, Tayal G. Effect of acute stress on rat ECG. Internet J Pharmacol 2009;8(1).
2. Lampert R, Jain D, Burg MM, Batsford WP, McPherson CA. Destabilizing effects of mental stress on ventricular arrhythmias in patients with implantable cardioverter-defibrillators. Circulation 2000;101:158–64.
3. Thayer JF, Yamamoto SS, Brosschot JF. The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. Int J Cardiol 2010;141:122–31.
4. Collins SM, Karasek RA, Costas K. Job strain and autonomic indices of cardio vascular disease risk. Am J Ind Med 2005;48:182–93.
5. Jouven X, Empana JP, Schwartz PJ, Desnos M, Courbon D, Ducimetière P. Heart-rate profile during exercise as a predictor of sudden death. N Engl J Med 2005;352:1951–8.
6. Straus SM, Kors JA, De Bruin ML, van der Hooft CS, Hofman A, Heeringa J, Deckers JW, Kingma JH, Sturkenboom MC, Stricker BH, Witteman JC. Prolonged QTc interval and risk of sudden cardiac death in a population of older adults. J Am Coll Cardiol 2006;47:362-7.
7. Yun J, Hwangbo E, Lee J, Chon CR, Kim PA, Jeong IH, Park M, Park R, Kang SJ, Choi D. Analysis of an ECG record database reveals QT interval prolongation potential of famotidine in a large Korean population. Cardiovasc Toxicol (in press) 2014.
8. Taggart P, Boyett MR, Logantha SJ, Lambiase PD. Anger, emotion, and arrhythmias: from brain to heart. Front Physiol 2011;2(67):1–11.
9. http:// help guide.org/mental/stress-signs.htm. 2012;30:13.
10. C D Speilberger, R L Gorsucch, R Lushene et al. Manual for the State and trait anxiety inventory STAI score; Stress assessment by STAI Questionnaire. Consulting psychologists press, Inc 1983; Annals of non invasive electro cardiology 2007;12(3):251-59.
11. Nis Hjortskov, Dag Rissen, Anne Katrine Blangsted et al. The effect of mental stress on heart rate variability and BP during computer work; European journal of applied physiology 2004;92(2):84-9.
12. C Noel, Bairey Merz, Yosef Pardo et al. Mental vs physical stress, QT prolongation and autonomic nervous system; division of cardiology, university of California, los angeles 2000;101 e :213-14.
13. Lampert R, Shusterman V, Burg M M, Lee F A, et al. Effects of psychological stress on repolarisation and relationship to autonomic and hemodynamic factors; Yale university, Journal of cardiovascular electrophysiology 2005;16(4):372-7.
14. Arpana Bhide, Rajasekhar Durgaprasad, Latheef Kasala, Vanajakshamma Velam, Narendra Hulikal. Electrocardiographic changes during acute mental stress. International Journal of Medical Science and Public Health 2016;5(5):835-38.
15. Gabor Andrassy, Attila Szabo, Gyongyver Ferencz et al. Mental stress may induce QT interval prolongation and T wave notching, Annals of non invasive electro cardiology 2007;12(3):251-9.
16. Jan henrik Atterhog, Keith Elliasson, Paul Hjemdahl et al. Sympathoadrenal and cardiovascular responses to mental stress, isometric hand grip, cold presser in asymptomatic young men with primary T wave abnormalities in ECG; Br heart F 1981;46:311-9.
17. L Toivonen, K Helenius and M Viitasalo, Electro cardio graphic repolarisation during stress from awakening on alarm call; Journal of The American College of Cardiology 1997;30(3):774 -9.
18. Peter H Stone, David S Krantz, Robert P Mc Mahon et al. Relationship among mental stress-induced ischemia and ischemia during daily life

and during exercise: the psychophysiologic investigations of myocardial ischemia study; Journal of The American College of Cardiology1999;33(6):1476-84.