Effect of Mental Stress on R Wave Amplitude of the Electrocardiogram in Young Healthy Male Subjects

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SUMMARY

The electrocardiographic effects of mental arithmetic stress were studied in 19 young, healthy male subjects. Blood pressure was measured by the cuff method before and during mental stress and electrocardiogram (V_5) was monitored and recorded continuously throughout the study using a computerized ECG monitoring system.

Heart rate increased from 65.8 ± 7.0 (mean $\pm$ standard deviation) to 76.7 ± 9.9 beats/min ($p < 0.001$). Systolic blood pressure rose from 122.7 ± 9.6 to 129.1 ± 11.7 mmHg ($p < 0.05$), diastolic blood pressure from 76.2 ± 8.8 to 83.0 ± 11.0 mmHg ($p < 0.001$), and mean blood pressure from 91.7 ± 7.9 to 98.5 ± 10.1 mmHg ($p < 0.001$). These changes were all statistically significant. R wave amplitude decreased significantly from 18.6 ± 6.9 to 17.0 ± 6.5 mm for the group ($p < 0.001$). No changes in the ST segment were observed; ST depression decreased from 0.56 ± 0.47 to 0.54 ± 0.43 mm (N.S.) and ST slope from 0.87 ± 0.59 to 0.77 ± 0.63 mm/sec (N.S.).

Thus it is apparent that mental stress reduced R wave amplitude without causing ST segment changes in young, healthy subjects. The mechanism and clinical implications are discussed.

Additional Indexing Words:

Mental arithmetic stress R wave amplitude

RECENTLY, there has been increasing interest in the evaluation of not only the ST segment, but also R wave amplitude during physical exercise testing as an indicator of cardiac status,¹⁾⁻⁵⁾ and many hypotheses have been proposed for the mechanism causing changes in this parameter.⁶⁾⁻¹⁰⁾ Although it has been reported that mental stress affects the ST-T wave¹¹⁾ probably through an adrenergic mechanism,¹²⁾ changes in the R wave during

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mental stress are not well described. We have attempted in this study to evaluate the effect of mental stress (mental arithmetic) on R wave amplitude in young, healthy male subjects.

METHODS

Nineteen young healthy men, 18 physicians, and 1 technician in our hospital, aged 22–34 (mean 28.9) years were studied. A computer assisted system for exercise (CASE, Marquette Electronics, Inc, Milwaukee, Wisconsin) was used to monitor and record the ECG throughout the study. Analog-to-digital conversion at 4-msec sampling intervals with 12 bit precision, wave recognition, and measurements of ST depression and its slope were performed using a Digital Equipment Corporation LSI-11 computer with an accuracy within 0.1 mm. Three automatically averaged QRS complexes, each of which represented an average of 25 consecutive QRS complexes, were measured manually and averaged to eliminate any respiratory variation of R wave amplitude, such that a change of more than 0.5 mm in amplitude was significant. After stabilization of blood pressure in the supine position, the standard 12 leads were recorded and continuous monitoring of lead V₅ was started. The monitored ECG was recorded at every 25 beats thereafter. Both heart rate, calculated from the R-R interval, and depression and slope of the ST segment were printed at 1 min intervals. After at least 3 averaged QRS complexes were recorded, each subject was challenged to solve as many mental arithmetic problems as possible within 3 min while in the supine position. During this period he was stimulated with a clicking metronome and by one of our staff members telling him the time remaining. Immediately after 3 min of mental arithmetic, blood pressure was measured by the auscultatory method and mean blood pressure calculated by the conventional formula: (systolic blood pressure—diastolic blood pressure)/3 + diastolic blood pressure. All variables such as heart rate, blood pressure, R wave amplitude, and depression and slope of ST segment, both at rest and during mental arithmetic, were compared. A paired t test was used for the statistical analysis.

RESULTS

All hemodynamic and ECG data at rest and during mental arithmetic are given in Tables I and II. A positive correlation was found between changes in blood pressure and those in heart rate as seen in Fig. 1, indicating that they increased proportionately during the stress. Heart rate increased

Table I. Effects of Mental Stress on Hemodynamics

Case #	Age	Heart rate (beats/min)				Blood pressure (systolic/diastolic) (mmHg)				Blood pressure (mean) (mmHg)			
		C	S	S-C	%	C	S	S-C	%	C	S	S-C	%
1	27	60	63	3	5.0	136/80	130/80	-6/0	-4.4/0	98.6	96.6	-2.0	-2.0
2	33	64	82	18	28.1	130/80	154/100	24/20	18.4/25	96.6	118.0	21.4	22.1
3	28	57	61	4	7.0	130/80	112/88	-18/8	-13.8/10	96.6	96.0	-0.6	-0.6
4	30	72	81	9	12.5	124/90	130/100	6/10	4.8/11	101.3	110.0	8.7	8.5
5	26	69	91	22	31.8	120/80	134/88	14/8	11.6/10	93.3	103.3	10.0	10.7
6	33	59	66	7	11.8	114/70	120/78	6/8	5.2/11	84.6	92.0	7.4	8.7
7	27	68	93	25	36.7	128/92	142/98	14/6	10.9/6.5	104.0	112.6	8.6	8.2
8	22	74	71	-3	-4.0	110/60	126/60	16/0	14.5/0	76.6	82.0	5.4	7.0
9	28	74	87	13	17.5	112/78	120/80	8/2	7.1/2.5	89.3	96.6	7.3	8.1
10	33	61	67	6	9.8	116/80	128/88	12/8	10.3/10	92.0	101.3	9.3	10.1
11	28	73	81	8	10.9	120/70	122/70	2/0	1.6/0	86.6	87.3	0.7	0.8
12	31	62	79	17	27.4	148/90	150/98	2/8	1.3/8.8	109.3	115.3	6.0	5.4
13	25	54	73	19	35.1	116/80	138/80	22/0	18.9/0	92.0	99.3	7.3	7.9
14	26	69	89	20	28.9	130/70	140/78	10/8	7.6/11.4	90.0	98.6	8.6	9.5
15	34	57	73	16	28.0	124/70	126/78	2/8	1.6/11.4	88.0	94.0	6.0	6.8
16	30	67	74	7	10.4	128/66	130/88	2/22	1.5/33.3	86.6	102.0	15.4	17.7
17	31	64	69	5	7.8	116/70	118/74	2/4	1.7/5.7	85.3	88.6	3.3	3.8
18	28	66	69	3	4.5	118/66	114/70	-4/4	-3.3/6	83.3	84.6	1.3	1.5
19	29	80	88	8	10.0	112/76	118/80	6/4	5.3/5.2	88.0	92.6	4.6	5.2
mean	28.9	65.8	76.7	10.9	16.8	122.7/76.2	129.1/83.0	6.3/6.7	5.3/8.8	91.7	98.5	6.8	7.3
S.D. (±)	3.1	7.0	9.9	7.7	12.0	9.6/8.8	11.7/11.0	9.9/6.0	8.0/8.4	7.9	10.1	5.5	5.8
					p<.05/p<.001					p<.001			

p<.001

p<.05/p<.001

p<.001

Abbreviations : C=control; S=during mental arithmetic; S-C= difference between values during stress and those at control; %=the percentile difference between S and C.

Table II. Effects of Mental Stress on R Wave and ST Segment in Lead V₅

Case #	R wave amplitude (mm)				Depression of ST segment (mm)				Slope of ST segment (mm/sec)			
	C	S	S-C	%	C	S	S-C	%	C	S	S-C	%
1	29.1	23.1	-6.0	-20.6	1.9	1.5	-0.4	-21.0	1.1	1.1	0	0
2	22.5	21.6	-0.9	-4.0	0.4	0.3	-0.1	-25.0	0.3	0.3	0	0
3	19.5	19.6	0.1	0.5	0.6	0.9	0.3	50.0	0.8	0.3	-0.5	-62.5
4	11.6	11.0	-0.6	-5.1	0.6	0.7	0.1	16.6	0.9	0.8	-0.1	-11.1
5	21.6	20.4	-1.2	-5.5	0.5	0.4	-0.1	-20.0	0.6	1.1	0.5	83.3
6	19.5	17.8	-1.7	-8.7	0.3	0.5	0.2	66.6	0.8	0.8	0	0
7	18.7	17.0	-1.7	-9.0	0.8	0.6	-0.2	-25.0	0.4	0.5	0.1	25.0
8	25.0	21.5	-3.5	-14.0	0.6	0.2	-0.4	-66.6	0.7	0.4	-0.3	-42.8
9	14.0	11.6	-2.4	-17.1	1.1	1.0	-0.1	-9.1	2.4	2.3	-0.1	-4.1
10	27.3	27.8	0.5	1.8	0.1	0	-0.1	-100.0	0.4	0.1	-0.3	-75.0
11	8.0	7.5	-0.5	-6.2	-0.1	-0.2	-0.1	-100.0	0.3	0.1	-0.2	-66.6
12	28.6	26.1	-2.5	-8.7	-0.1	-0.2	-0.1	-100.0	0.2	0.2	0	0
13	8.7	7.5	-1.2	-13.8	0.5	0.6	0.1	20.0	0.7	0.4	-0.3	-42.8
14	14.0	13.0	-1.0	-7.1	0.4	0.2	-0.2	-50.0	0.7	0.5	-0.2	-28.5
15	23.3	20.5	-2.8	-12.0	0.4	0.7	0.3	75.0	1.4	1.4	0	0
16	21.0	20.1	-0.9	-4.3	0.1	0.5	0.4	400.0	0.2	0	-0.2	-100.0
17	15.8	13.7	-2.1	-13.3	0.9	1.0	0.1	11.1	1.6	1.6	0	0
18	19.0	17.8	-1.2	-6.3	1.1	0.9	-0.2	-18.1	1.8	1.7	-0.1	-5.5
19	6.1	4.5	-1.6	-26.2	0.6	0.7	0.1	16.6	1.2	1.0	-0.2	-16.6
Mean	18.59	16.95	-1.64	-9.45	0.56	0.54	-0.02	6.37	0.87	0.77	-0.10	-18.27
S.D. (±)	6.86	6.45	1.44	6.94	0.47	0.43	0.23	108.60	0.59	0.63	0.21	40.90
	p<.001				N.S.				N.S.			

Abbreviations: C=control; S=during mental arithmetic; S-C=difference between values during stress and those at control; %=the percentile difference between S and C.

Fig. 1. Mental arithmetic induced changes in heart rate are plotted against changes in systolic blood pressure. There is a positive correlation between the two ($r=0.47$, $p<0.05$), indicating that both of them increased proportionately during the stress.

Fig. 2. Changes in hemodynamics induced by mental arithmetic. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean blood pressure (MBP) are plotted. Values are presented as the mean $\pm$ SD. All of these variables demonstrate a statistically significant increase with mental stress.

by 10.9 ± 7.7 beats/min, from a mean of 65.8 to 76.7 beats/min for the group ($p<0.001$), systolic blood pressure by 6.3 ± 9.9 mmHg from 122.7 to 129.1 mmHg ($p<0.05$), diastolic blood pressure by 6.7 ± 6.0 mmHg from 76.2 to 83.0 mmHg ($p<0.001$), and mean blood pressure by 6.8 ± 5.5 mmHg from 91.7 to 98.5 mmHg ($p<0.001$). Fig. 2 demonstrates these changes.

Changes in R wave amplitude and ST segment from the control are shown in Fig. 3. R wave amplitude decreased by 1.6 ± 1.4 mm from a mean of 18.59 to 16.95 mm ($p<0.001$). ST segment did not show any consistent changes in depth or slope, depression changing from 0.56 to 0.54 (N. S.) and slope from 0.87 to 0.77 mm/sec (N. S.). The change in R wave amplitude

Fig. 3. Changes in R wave amplitude in lead V₅ (RV₅), ST segment depression (STV₅ depression), and its slope (STV₅ slope) from the control are shown. Values are presented as the mean $\pm$ SD. R wave amplitude decreased significantly during mental stress ($p < 0.001$). ST segment did not reveal any consistent change in depression or slope.

versus the change in heart rate, and the change in R wave amplitude versus the change in systolic blood pressure are illustrated in the scattergrams of Figs. 4 and 5, respectively. There were no correlations in either case.

Fig. 4. Changes in R wave amplitude are plotted against changes in heart rate. There is no correlation between the two, suggesting that heart rate is not a major factor in reduction of R wave amplitude during mental stress.

Fig. 5. The changes in R wave amplitude versus the changes in systolic blood pressure are illustrated. There is no correlation between the two, suggesting blood pressure is not a major determinant in reduction of R wave amplitude during mental stress.

DISCUSSION

Psychological or mental stress is known to play a role in the pathogenesis of cardiovascular disease and hypertension.^{13),14)} Stressful situations have been said to cause acute changes in hemodynamics, and may precipitate angina pectoris if there is underlying coronary heart disease.¹⁴⁾ Robinson reported that the hemodynamic changes during mental stress were comparable to those caused by light to moderate exercise, and indeed, an anginal episode was provoked in 1 patient with mental stress.¹⁵⁾ Hemodynamic changes caused by mental stress are reportedly characterized by increase in cardiac output, heart rate, blood pressure and myocardial contractility, and by a decrease in total peripheral resistance.¹²⁾ This is probably through an adrenergic mechanism, since plasma catecholamine levels increase during mental stress,^{16),17)} and the effects are preventable by β -adrenergic blockade.¹⁸⁾

In the present study, mental stress was induced by 3 min of mental arithmetic. The hemodynamic changes were quite similar to those reported by Falkner¹⁹⁾ and Bassan.²⁰⁾ Falkner et al reported increases in systolic blood pressure (5.9 mmHg), diastolic pressure (3.5 mmHg), and heart rate (7.1 beats/min) following 10 min of mental stress. Blood pressure values, obtained indirectly, were almost the same as those obtained 3 min after the initiation of mental stress in their study. Bassan et al had their subjects perform mental arithmetic for 2 to 2.5 min, and measured arterial blood pressure directly from a radial or femoral artery. They found that systolic blood pressure increased by 19.3 mmHg, diastolic blood pressure by 7.1 mmHg, and heart rate by 11.6 beats/min. The higher baseline systolic blood pressure and the greater increase seen during mental stress compared with our findings as well

as Falkner's were probably due to the differences in age of subjects and method of determining blood pressure. Bassan's subjects were older (mean age of 53.1 years) than ours (28.9 years), who were older than Falkner's (14.8 years). Based on these findings, 3 min seems to be a reasonable time period for evaluating the effects of mental stress on the cardiovascular system.

Physical exercise testing, such as Master's two-step testing and treadmill exercise testing, has been commonly performed to evaluate coronary heart disease in clinical cardiology,²¹⁾ and ST segment depression has been an important criterion in detecting myocardial ischemia.²²⁾ Recently, it has been noticed that either augmentation or no change in R wave amplitude during physical exercise testing correlates with left ventricular dysfunction in ischemic heart disease.^{1),5)} It has also been reported that the sensitivity and specificity of physical exercise testing in the diagnosis of coronary heart disease are increased if both ST depression and R wave changes are taken into account at the same time.²⁾ Simoons et al demonstrated a decrease in R wave amplitude during exercise in healthy men and speculated that the mechanism was a reduction in the blood conductivity and intracardiac volume (Brody effect).^{6),23)} Other possible mechanisms are the effect of heart rate, hyperventilation, shift in position of body or electrode, displacement of the heart from chest wall, myocardial ischemia, and increased myocardial contractility itself.^{7)-10),24),25)} In our study, there was poor correlation between changes in R wave amplitude and heart rate, and also between changes in R wave amplitude and blood pressure suggesting that neither heart rate nor blood pressure were major determinants of R wave amplitude reduction. It is unlikely that there was any effect due to a shift in body or electrode position, because the subjects were kept in the supine position throughout the study. Hyperventilation was not observed in any subjects. The most likely mechanism to account for the reduction in R wave amplitude seen in the present study seems to be enhanced myocardial contractility and increased cardiac output, which have been reported to occur during mental arithmetic,¹²⁾ and which would reduce cardiac volume and decrease R wave amplitude through the Brody effect or Battler's hypothesis, i.e., increased distance between the heart and anterior chest wall. Echocardiography, which would provide information about cardiac dimensions or volume, might be helpful in confirming these mechanisms.

Neither degree of depression nor slope of the ST segment changed in any consistent manner. This is in agreement with Guazzi's study, which showed no ST segment depression during mental stress in healthy subjects.¹²⁾ However, they did observe inverted T waves in hyperkinetic patients and enhanced T wave inversion and ST segment depression in ischemic and hy-

pertensive patients.

R wave amplitude in V_5 is also important as a voltage criterion for left ventricular hypertrophy.²⁶⁾ Since it may be greatly influenced by the patient's psychological condition, as seen in this study (maximum 6 mm decrease in case #1), it is important to bear in mind that there is always the possibility that left ventricular hypertrophy may be overlooked in borderline cases on the first visit to the clinic, and to keep patients relaxed during ECG recording.

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