Original Article

Hemodynamic Effects of Repeated Bouts of Mental Stress

Muhammad Qasim Memon

ABSTRACT

OBJECTIVES: To determine whether repeated stints of mental stress had similar cardiovascular effects (CV) and also to establish that mental arithmetic tasks affect CV parameters and have reproducibility of the effects.
DESIGN: Cross Sectional, observation study.
SETTING: Medical School, University of Nottingham, UK.
METHODS: Ten healthy, white European males participated in this study. Employing a Finometer, beat-to-beat CV parameters were recorded for 30 min baseline. Volunteers undertook mental arithmetic task (MAT) thrice, with different sets of questions each time, displayed as Power Point slides. Fifteen min interval lapsed between the tests; post MAT-3 recordings then continued for 10 min before the experiment ended.
RESULTS: No significant difference between the three baseline values was noted. MAT resulted in significant elevation of SBP, 20 mmHg (15%), DBP, 14 mmHg (18%), MAP, 18 mmHg (19%), HR 26 beats/min, i.e., 42% and CI by 4.35l (50%) from the baseline ($P < 0.001$) whereas TPR decreased by 0.213 units (-21%; $P < 0.001$). CV variables returned to baseline as the task ended. Effects of MAT-1 -2 and -3 on the CV variables were similar ($P > 0.05$).
CONCLUSION: Repeated exposure to mental stress results in identical CV reactivity response, effects are attributable to adrenaline gush and that quick adaptation to stressor does not occur; recovery may take place with CV parameters returning to the baseline as soon as stress was over.

KEY WORDS: Stress, Psychological, reproducibility, sympathetic nervous system.

INTRODUCTION

Mental stress has been shown to produce deleterious cardiovascular (CV) effects[1]. Such effects primarily result from stimulation of the sympathetic nervous system (SNS) and are mediated, inter alia, by release of catecholamines[2]. Frequent spells of mental stress may produce damaging effects on the vascular endothelium leading to endothelial dysfunction resulting in defective vasodilatation as a consequence of diminished release of nitric oxide (NO)[3]. Endothelial dysfunction and defective release of NO predispose to atherosclerosis and future adverse CV events[4]. Evidence suggests that mental stress may lead to myocardial ischemia in patients with coronary artery disease (CAD)[5]. It has been determines using Stroop test, that that mental stress when induced experimentally in healthy subject resulted in hyper coagulability in healthy human subjects[6]. It was however found, that the reversible increased aggregability and sensitivity of platelets to thrombin seen after heavy exercise, in contrast only weakly affected after mental stress[7]. Inducing mental stress by non pharmacological approaches to stimulate SNS and its consequent effects on CV system can provide useful information. The purpose of present study is to document "whether repeated stints of mental stress had similar CV effects"? As it was hypothesized that such effects may get attenuated on recurring stimulation. Secondary objective was to establish that mental arithmetic tasks (MAT) affect CV parameters and has reproducibility of the effects.

MATERIALS AND METHODS

Ten healthy, non-smoker white European males were recruited for this study through recruitment posters in the Medical School, University of Nottingham and the Queen's Medical Centre, Nottingham. Subjects were aged 25 ± 5 years, weighed 76 ± 11 kg, and were 182 ± 6 cm tall. They were not on any regular medication. Before coming for a medical screening session, volunteers were requested to avoid eating or drinking anything for at least 2h. The screening involved recording resting BP, height and weight and a 6 lead electrocardiogram, and completing a medical screening questionnaire and consent form.
The study itself involved single visit. Volunteers were contacted via e-mail a day before their visit and requested to fast for 2 hr before coming for the experiment. However, if they preferred to have breakfast, then it should be a light one, preferably corn flakes or toast with milk. In that case, their last meal should be 2 hr before the experiment. They were also advised to avoid strenuous exercise for 24 hr before the visit and to use the lift to come to the laboratory.

Upon arrival, volunteers were requested to void their bladder before the experiment began. They rested on a bed, semi-recumbent, in a thermo-regulated room and their age, gender, height and weight was entered in the Finometer (FMS, Finapres Medical Systems BV, The Netherlands), which was switched to Research Mode. The Finometer's arm cuff was attached to the left upper arm with the smaller finger cuff wrapped around the ipsilateral middle finger's middle phalanx. A slide projector (In-focus, LP-530, USA) was setup to display Power Point slides on the wall in front of the volunteer. Room lights were dimmed to ensure good visibility of the projected slides.

Protocol

Baseline recordings were made for 30 min. At the end of this period, a MAT was presented, displayed as Power Point slides. During this mental arithmetic task (MAT-1), volunteers were asked to solve 20 simple arithmetic questions in 6 sec each (the slide transition time). The questions, for example 97-8+13=, 68+38-27=, 52+5-18=, were formulated in such a way that the volunteer had to concentrate to give a correct answer. They were requested to say their answers loudly and were expected to answer correctly at least 17 (85%) question. The investigator had the answers on a separate sheet; responses were marked by the investigator and conveyed to the subject whether or not an answer was correct. This task lasted for 2 min. During the task and for the subsequent 15 min, Finometer data collection continued. A second mental arithmetic task (MAT-2) was then administered with a different set of questions. This task also lasted for 2 min followed by post MAT-2 recordings for 15 min. A third 2 min task (MAT-3) was then applied using different set of questions with subsequent 15 min post-test recordings.

Thus, the study lasted a total of approximately 80 min. At the end of the experiment all equipment was removed and volunteers were free to go.

Data analysis

Collected data were down-loaded from the Finometer onto a remote PC using the 'Beatscope' software program. Data were averaged over 2 min intervals for the pre and post-MAT-3 periods and at 20 sec intervals for the MAT duration. Data were transferred to 'Biomed' (software program) data sheets. The pre-MAT period time points (0, 1, 2); (5, 6, 7) and (10, 11, 12) for each variable, for each subject were used as the baseline, whereas post-MAT-3 time points (15, 16, 17) were used as the recovery period, i.e., up to 6 min post-MAT-3. Intervening time points, e.g., from 2.33 to 4 were the MAT-1 duration; similarly, values were entered for the time periods 7 to 9 (MAT-2) and 13 to 14 (MAT-3) as the duration of MAT. Thus, 17 time points represented 30 min. Statistical analysis was done using Quade, Friedman and Wilcoxon tests. SV, CO and TPR and were factored by the weight of the volunteer. Statistical significance was laid down as $P < 0.05$.

RESULTS

Volunteers found MAT to be stressful and looked visibly under stress while they performed the task. However, they seemed relaxed as soon as the task was over. On average volunteers gave 7 ± 4 correct answers of MAT-1, 8 ± 5 of MAT-2 and 9 ± 4 of MAT-3. Data before each MAT are shown in **Table I**. Statistically no significant differences between values were noted.

Effects of MAT

The MAT resulted in significant elevation of SBP, DBP, MAP, HR and CI from the baseline. During MAT -1, peak increases of 20 mmHg (15%) in SBP; 14 mmHg (18%) in DBP and 18 mmHg (19%) in MAP were noted. HR increased by 26 beats/min, i.e., 42% and CI by 4.35l (50%). P value for all the changes mentioned above was < 0.001. TPR decreased as soon as the task began, with a significant decrease being noted in the first min of the task. The greatest fall was a decrease of 0.213 units (-21%; $P < 0.001$). TPR remained below baseline for the remaining 1 min duration of the task but values were not significant.

The second task resulted in a peak increase of 22 mmHg in SBP, i.e., a 17% rise. Peak elevations in the DBP and MAP were similar to the values noted during MAT-1, i.e., (14 mmHg; 18%) and (18 mmHg; 19%), respectively. An increase in HR of 29 beats/min (47%) and 4.74l (54%) in CI was noted. TPR maximally decreased by 0.245 units (-24%) during the first min of the task then returned to non-significant values. P value for all the changes mentioned above was < 0.001.

During MAT-3, peak increases in SBP, DBP and MAP were (18 mmHg; 13%; $P < 0.001$); (13 mmHg; 18%; $P < 0.001$) and (17 mmHg; 18%; $P < 0.001$) respectively. An increase in HR (26 beats/min; 41%; $P < 0.001$) and in CI (3.3l; 37.5%; $P < 0.001$) was noted. The increase in CI was during the first min of the task; it then returned to baseline values. TPR decreased (for 40 sec) as the task begun; the greatest fall was of 0.158 units (-16%; $P < 0.01$). TPR subsequently increased in the recovery period; peak increase of 0.083 units (8%; $P < 0.01$) above baseline was noted.

No significant change from baseline in SV was noted during any of the three tasks (**Figure I; Table II**).

As may be seen in Fig. 1, SBP, DBP and MAP increased gradually as the MAT progressed, returning to baseline values with the completion of the task. HR and CI increased abruptly as the task begun but returned to the baseline as the task progressed to an

end. Changes in TPR were almost a mirror image of the changes in CI; TPR decreased with the initiation of the MAT but gradually increased as the task progressed to completion.

Comparison of effect of MAT 1, 2 and 3 on the CV variables:

When effects of MAT-1 -2 and -3 on the CV variables were compared no significant difference was noted between the effects of the three stressors (P > 0.05).

TABLE I: BASELINE VALUES OF CV VARIABLES BEFORE MAT-1, MAT-2 AND MAT-3 SHOWN AS MEAN±SEM

Mental arithmetic Baseline	MAT-1	MAT-2	MAT-3
SBP mmHg	132 ± 5	133 ± 4	135 ± 4
DBP mmHg	75 ± 2	76 ± 2	78 ± 2
MAP mmHg	96 ± 3	97 ± 3	98 ± 3
HR Beats/min	68 ± 3	65 ± 3	67 ± 3
CO l/min	7.1 ± 0.3	7.1 ± 0.2	7.0 ± 0.2
TPR resistance units	0.790 ± 0.03	0.807 ± 0.03	0.847 ± 0.04
SV ml	113 ± 5	113 ± 5	109 ± 6

Key for abbreviation is missing

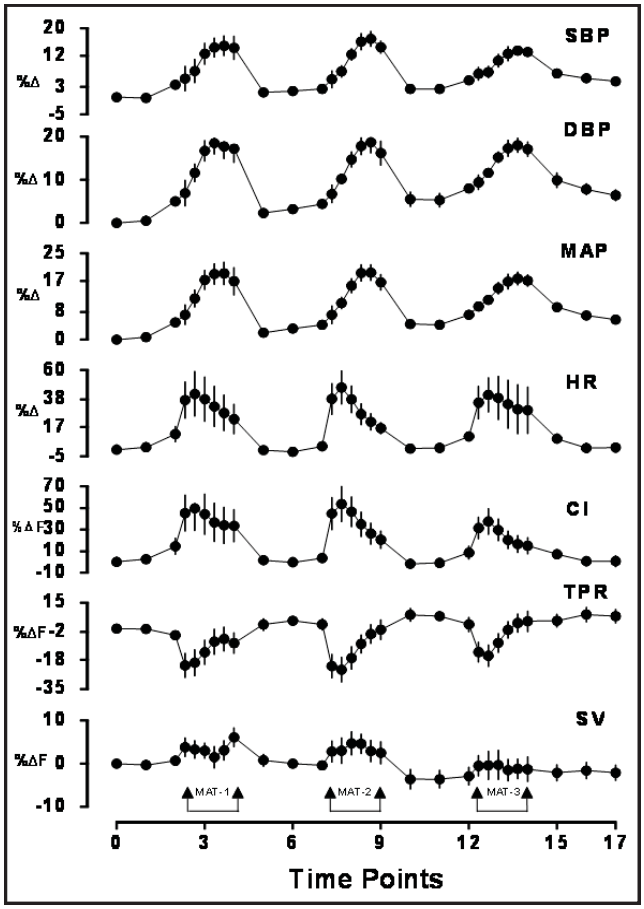
TABLE II: EFFECTS OF MAT-1 -2 -3 ON CV VARIABLES AS PEAK AND PERCENT CHANGE FROM THE BASELINE PEAK CHANGE/PERCENT CHANGE

Variable	MAT-1	MAT-2	MAT-3
Variable	MAT-1	MAT-2	MAT-3
	Peak /%	Peak /%	Peak %Δ
SBP mmHg	20/15	22/17	18/13
DBP mmHg	14/18	14/18	13/18
MAP mmHg	18/19	18/19	17/18
HR bpm	26/42	29/47	26/41
CI l/min	4.35/50	4.74/54	3.3/38
TPR r units	-0.213/21	-0.245/24	-0.158/16

DISCUSSION

This study employed non-pharmacological approach to stimulate the SNS and non-invasive method to record the CV parameters beat-by-beat. The results depict a classical picture of adrenaline gush in response to MAT stimulating the SNS. It was hypothesized that the effects of metal stress may get attenuated on repeated bouts but it was determined that the intensity of effect on the CV system may not lessen. Evidence suggests that repeated stress episodes may not adapt responses to stressor but continue to produce damage through various reactionary mechanisms such as release of catecholamine, activation of renin-angiotensin system, release of homocysteine leading to increased CV reactivity and endothelial damage[8] culminating in atherosclerosis[9]. Such events multiply and increase risk for CAD and subsequent CV events if the initial stimulus, i.e., mental stress continues and is not managed[10]. It has been reported that real-life mental stress episodes increase arterial pressure and may consequently augment risk for hypertension[11]. Present study generated results similar to the ones previously conducted[12]; however, the reproducibility and similarity in magnitude of response in the present study indicate that the SNS may get stimulated in an identical manner with every episode of mental stress. It has been suggested that trait rumination influence

FIGURE I: CHANGES IN CV PARAMETERS WITH REPEATED INDUCTION OF MATS. DATA ARE MEAN ± SEM. %Δ = PERCENT CHANGE; %ΔF = PERCENT CHANGE FACTORED (BY WEIGHT OF THE VOLUNTEER)



CV responses to mental stress by delaying recovery and by preventing adaptation[13] on repeated exposure to the stressor[14]. Research has determined that the period of CV reactivity, for example, increases in BP that remained in the elevated state for longer period of time than an acute elevator response to a stressor are more damaging for the CV health[15] and that delayed recovery even in normotensives but with a family history of hypertension denote future potential for development of high blood pressure[16] however, this study determined that the CV reactivity ended as soon as the stress was over and values returned to baseline. Present study also demonstrated that the increase in CO is principally due to increase in HR as SV did not change from the baseline. These results are consistent with existing evidence[16].

It is concluded that repeated exposure to mental stress result in identical CV reactivity response, effects may be attributed to adrenaline gush and that quick adaptation to stressor do not occur. However, fast recovery may take place with CV parameters returning to the baseline as soon as stress was over.

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AUTHOR AFFILIATION:

Dr. Muhammad Qasim Memon
Department of Physiology
Liaquat University of Medical and Health Sciences
Jamshoro, Sindh-Pakistan.
E-mail: memon.qasim@lumhs.edu.pk