ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Mental stress reactivity and post stress recovery of cardiovascular parameters in an Indian late adolescent population

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ABSTRACT

The complete characteristics of essential hypertension have not been unraveled. Stress reactivity and post-stress recovery perturbations have been suggested as possible identifiers for future development of hypertension in susceptible individuals, but these aspects are not uniformly seen across all the races. The aim of this study was to observe stress reactivity and post-stress recovery status of various cardiovascular parameters to a mental stress task in an Indian subpopulation. Twenty nine normotensive students with positive family history of hypertension and equal number of age, height, weight and BMI (Body Mass Index) matched controls were asked to perform a serial subtraction task. Blood pressure (Systolic and Diastolic) and heart rate were measured at rest, during cognitive task, one minute and five minute post recovery. The parameters were analyzed by using unpaired student's't' test. There was a slight increase in blood pressure (Systolic and Diastolic) and heart rate in normotensive subjects with a positive family history of hypertension (but were statistically insignificant) in response to mental arithmetic (MA) task. Both subjects and controls also showed good recovery of all cardiovascular parameters. Our study found neither an exaggerated stress response to mental stress nor an impaired recovery after mental arithmetic task in normotensive offspring's with parental history of hypertension. Because stress reactivity varies in different populations, hence stress reactivity may not be a good measure of predicting future risk of hypertension.

Keywords: Stress reactivity, blood pressure, family history, mental arithmetic, post-stress recovery

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ISSN: 0975-8585

INTRODUCTION

Hypertension is a disease which afflicts a large percentage of people in any population, but the full knowledge regarding the etiologies, pathophysiology and effective treatment/preventive interventions for the most common variant of hypertension plaguing us i.e. “Essential hypertension” is still a mirage. The knowledge bank of these aspects of essential hypertension is being updated regularly by numerous studies, thereby enabling the essential hypertension “picture puzzle” to be solved. Essential hypertension is not a single entity but rather is considered to be a spectrum with different underlying pathophysiologic processes and is the culmination of a complex interaction between various environmental and genetic factors. The heritable component contribution to essential hypertension is estimated to be about 15-35 % [1].

One of the tools employed and which is considered to be useful in unraveling the possibility of anyone developing hypertension, is to subject a normotensive person albeit with a genetic burden of hypertensive preponderance to various stressors (physical or mental) and observe their cardiovascular responses. Cardiovascular reactivity to mental stress and basal blood pressure are influenced by independent mechanisms [2]. A rise in the Heart rate during mental stress has been attributed to increased sympathetic tone as well as a decreased parasympathetic tone [3]. While increased SBP reactivity has been found to predict future development of hypertension, the same was not attributable to increased DBP reactivity in a 12 year follow up study [4]. A prospective study done in Norway after a 18 year follow-up has found that the levels of catecholamine’s during mental stress was able to explain about 12.7% of the variation of future systolic blood pressure [5]. Similarly an impaired cardiovascular recovery, especially of heart rate and DBP after mental stress has been found to occur earlier than increased reactivity in various cardiovascular parameters becomes obvious in normotensive individuals with a positive family history of hypertension [3].

Though increased cardiovascular reactivity to mental stress has been found by many studies, a need to observe such changes if any in different cultural settings is stressed [6]. Hence this study was envisaged to observe cardiovascular reactivity to mental stress and post-stress recovery in a young Indian normotensive subpopulation with positive family history of hypertension in order to detect any cultural variations in stress reactivity and recovery.

MATERIALS AND METHODS

The present study was conducted after the proposal was cleared by the institutional ethics committee. The subjects and controls were drawn from the first year medical students of the institute. A detailed questionnaire collected all the relevant details including family history of hypertension. Informed consent was obtained from all the participants in this study. Apparently healthy, male first year medical students with a family history of hypertension and who were not themselves hypertensive, not indulging in excessive physical activity and not on any drugs that could alter the cardiovascular autonomic functions were included in the study as

subjects. The above inclusion criteria were met by 29 students. Persons who were hypertensive, smokers and who consumed alcohol were excluded from the purview of the study. Twenty nine age, height, weight and BMI matched students without family history of hypertension constituted the control group. All the recordings were done in the morning hours after a light breakfast and at least a 12 hour abstinence from consumption of caffeinated drinks. Heart rate (HR) calculations were done from a lead II rhythm strip obtained from ECG machine BPL Cardiart 108/MK-VI. Whereas Systolic (SBP) and diastolic (DBP) blood pressures were measured by using ISO 9002 approved BP instrument (CH-403C) manufactured by Citizen Corp, Japan. The instrument was calibrated and checked for linearity by using a mercury sphygmomanometer for each and every subject during basal BP measurement. Measurement of BP by automated oscillometric method has been found to remove any observer bias associated with auscultatory methods [3].

The HR and BP recordings were obtained under four experimental situations namely; Baseline/resting, test (mental stress), one minute recovery and five minute recovery. Mental stress tests are usually categorized as problem solving, information processing, psychomotor, affective and aversive/painful tests [7]. We used a form of Mental arithmetic(MA) test called as Serial subtraction task, wherein the subject begins with a large number (e.g. 723) and then serially subtracts the number seven until told to stop [8]. The MA task was performed by the subjects for a period of two minutes with vocalization of the results of subtraction. The recovery values for all the three cardiovascular parameters were defined as (Parameter value at a particular point of time after termination of stress – Parameter value at baseline) [9]. The results of the study were analyzed by student's unpaired 't' test using Microsoft Excel 2007 software, with a P value of <0.05 being considered as significant.

RESULTS

The controls and subjects were well matched regarding age, height, weight and BMI and there were no significant differences in these parameters between the groups studied. (Table-1)

Table – 1 : Anthropometric data (Mean ± SD)

Group	Age (yrs)	Height (cms)	Weight (Kgs)	BMI
Controls (n = 29)	18.62 ± 0.72*	166.79 ± 8.60*	64.51 ± 11.37*	23.33 ± 4.44*
Subjects (n = 29)	19.17 ± 1.31*	169.00 ± 7.62*	66.93 ± 10.81*	23.54 ± 4.31*
*P>0.05 = Not significant				

The basal, test and the difference between test-basal (i.e. the rise) of Systolic blood pressure (Table-2) was higher in subjects than in controls but were not statistically significant.

ISSN: 0975-8585

Table – 2 : Systolic Blood pressure (mm of Hg) Mean ± SD			
Group	**Basal**	**Test**	**Difference**
Controls (n = 29)	116.89 ± 10.05*	126.00 ± 9.62*	9.10 ± 7.22*
Subjects (n = 29)	119.03 ± 10.83*	128.27 ± 11.73*	9.24 ± 7.57*
*P>0.05 = Not significant			

The basal DBP values were slightly lower in subjects but test and the difference between test-basal (i.e. the rise) values of Diastolic blood pressure (Table-3) was higher in subjects than in controls but all were statistically not significant.

Table – 3 : Diastolic Blood pressure (mm of Hg) Mean ± SD			
Group	**Basal**	**Test**	**Difference**
Controls (n = 29)	72.10 ± 7.96*	81.89 ± 7.84*	9.79 ± 5.25*
Subjects (n = 29)	71.72 ± 6.51*	82.83 ± 7.85*	11.10 ± 5.72*
*P>0.05 = Not significant			

Whereas the basal and test values for heart rate were higher in subjects than in controls, but the difference was lower. Here again the results were statistically insignificant. (Table-4)

Table – 4 : Heart rate (bpm) Mean ± SD			
Group	**Basal**	**Test**	**Difference**
Controls (n = 29)	76.41 ± 11.55*	93.00 ± 14.53*	16.24 ± 9.68*
Subjects (n = 29)	78.10 ± 12.83*	93.24 ± 14.60*	15.14 ± 8.72*
*P>0.05 = Not significant			

The recovery rates of SBP, DBP and heart rate did not show any statistical differences between the subjects and controls**.** (Table-5, Table-6 and Table-7) In fact both the groups showed good recovery i.e. reaching baseline values for SBP and DBP at one min. HR recovery though was not complete at one minute in subjects but had recovered by five minutes.

Table – 5 : Systolic BP recovery (Mean ± SD)		
Group	**One minute recovery**	**Five minute recovery**
Controls (n = 29)	-2.27 ± 5.12*	-1.93 ± 5.22*
Subjects (n = 29)	-2.86 ± 7.15*	-3.93 ± 7.21*
*P>0.05 = Not significant		

ISSN: 0975-8585

Table – 6 : Diastolic BP recovery (Mean ± SD)		
Group	**One minute recovery**	**Five minute recovery**
Controls (n = 29)	-1.55 ± 7.08*	-0.76 ± 6.04*
Subjects (n = 29)	-1.34 ± 5.82*	-0.90 ± 5.52*
*P>0.05 = Not significant		

Table – 7 : Heart rate recovery (Mean ± SD)		
Group	**One minute recovery**	**Five minute recovery**
Controls (n = 29)	-1.07 ± 8.40*	-1.21 ± 8.21*
Subjects (n = 29)	0.55 ± 5.86*	-0.79 ± 8.93*
*P>0.05 = Not significant		

DISCUSSION

One of the main findings of our study was a slightly enhanced but statistically insignificant reactivity score of SBP, DBP and HR in normotensive subjects with positive family history of hypertension. Several factors tend to influence the cardiovascular reactivity to stress – the type and performance of mental stress task, behavioral characteristics of the concerned individual and of utmost importance being the genetic makeup of the individuals studied.

The nature of performance of the mental stress task also seems to affect the cardiovascular reactivity. Additional pressure on the subject while performing the MA task like offering monetary incentives for better performance [6], coaxing the subjects to perform the task quickly [10], exposing to affective noise during the task [11] or by using critical comments about the performance [3] tends to produce an exaggerated reactivity. Similarly vocalization of the results while performing the MA task produces greater cardiovascular reactivity compared to silent performance [3]. In our study no attempt was made to potentiate the stress during the MA task.

Behavioral patterns of subjects also tend to influence the reactivity scores and suppression of anger has been shown to produce high reactivity scores than which is observed in people who express anger [12]. Addition of a social support intervention (like pet ownership) lowers cardiovascular responses to psychological stress. In fact improved task performance was observed in persons who owned pets [2].

A lesser magnitude of stress reactivity to mental stress observed in our study is also echoed by a study done on young black females, which has demonstrated that hyper-responsiveness to mental stress was less compared to those that were seen in whites [13]. This points to the possibility of genetic attributes of a subpopulation influencing the amount of stress reactivity. The reasons for these variations could be multiple, a study done on psychologically normal young adults has shown that individuals with short/short form of the

promoter region of serotonin transporter gene (SLC6A4) exhibited exaggerated SBP, DBP and HR reactivity to mental stress and these were more pronounced when there was a negative audience, although this was limited to only females [14]. Similarly the GenSalt (Genetic Epidemiology Network of Salt sensitivity) study done on Han Chinese has shown that common variants of ENaC subunits produce varied BP responses to dietary salt intake [15]. Such variations in genetic composition may be quite recent and they may not occur uniformly in all populations [16]. Hence findings in one specific population cannot be safely extrapolated to another genetically different one. Therefore it becomes pertinent to observe every genetically distinct population as a separate entity. Finally the environmental milieu to which a person is exposed to in the very early part of life may also shape the stress reactivity, as shown in a study on an animal model (wild type rats) wherein early life stress produces an exaggerated stress mediated cardiovascular response to acute stress and which is mediated via the endothelin pathway [17]. The Cardiovascular reactivity to stress (mental/physical) is therefore affected by many genetic and environmental factors and hence while an enhanced cardiovascular reactivity in normotensives with a positive family history may signal future development of hypertension, an insignificant reactivity may not necessarily mean “all is well”.

Another finding of our study was that the recovery rates of SBP, DBP and HR after mental stress was not statistically significant between subjects and controls. Good recovery was noted at one minute in both the groups under study. This good recovery could be attributed to younger age of the study population (Average age in controls =18.62 + 0.72 and in subjects =19.17 + 1.31 years), as post stress recovery is found to be quick and efficient in young individuals [18]. Also prolonged recovery in CV parameters has been noted only in mental stress protocols which involved some type of post stress reflection on the stress itself [19].

CONCLUSION

In conclusion we found increased but insignificant stress reactivity as well as good recovery in blood pressure and heart rate responses to mental stress in normotensive individuals with a positive family history of hypertension, hence stress reactivity per se would not be a good indicator of future development of hypertension and therefore other factors may have to be factored in to provide a better means of prediction.

The drawback of the present study was that no measurement of cortisol and catecholamine levels at rest and during cognitive task was done which could have provided a correlative support to the changes in cardiovascular parameters to mental stress. Further in order to clearly delineate the role of genetic contribution to stress reactivity in response to mental stress in this subpopulation, stress reactivity patterns need to be studied under different mental stress protocols and over varied behavioral settings.

ISSN: 0975-8585

ACKNOWLEDGEMENT

We hereby acknowledge the support offered by the Indian Council of Medical Research with gratitude.

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