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Original Research Article

Influence of Stress on Cardiorespiratory Profile in an Urban Female Population of Reproductive Age Group

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ABSTRACT

Background: The postmodern over-industrialized and highly competitive metropolitan culture has added up to stresses in human lives at many levels.
Aims: To study the influence of stress on cardiovascular risk factors in an urban female population of reproductive age group.
Materials and methods: The present longitudinal interventional double blinded randomized control trial was conducted on females of reproductive age group in a time span of 5years after taking institutional ethical clearance and informed consent of the subjects. Parameters recorded were Waist/ hip ratio, Body mass index (BMI), Pulse rate, Blood pressure (BP), Electrocardiogram (ECG), fasting blood sugar (FBS), Lipid profile, Pulmonary Function test (PFT), Perceived Stress levels, Presumptive Stressful Life Event Scale (PSLES) score. The subjects were grouped into two: Subjects with PSLES SCORES above 200(G1) and Subjects with PSLES Scores less than 200 but more than 40 (G2) with 1006 subjects in each group. All subjects were given a training of progressive muscle relaxation. Training involved tensing the specific muscle groups of body for 7-10 sec., followed by releasing them for 15-20sec. The subjects were asked to practice this technique at home for 20 minutes every day for 3 months and come for follow up at regular intervals. All parameters were reevaluated. T test was used to analyze the data.
Results: Subjects with higher stress scores were found to have more BMI, W/H ratio, dyslipidemia and altered autonomic functions and practice of PMR for 3 months caused significant improvement in nearly all health parameters assessed.
Conclusions: Perceived stress may adversely affect cardiovascular respiratory profile and anthropometric parameters of healthy adult females of reproductive age group and regular practice of PMR may decrease perceived stress levels and help in modulating cardiorespiratory profile with decreasing future complications like diabetes, dyslipidemia, coronary artery disease, metabolic syndrome.
Keywords: Stress, Progressive muscle relaxation, Women of reproductive age group.

INTRODUCTION

The globalized postmodern world has given us many boons, no doubt, but with a few boons sometimes there comes a curse too. Our over-industrialized and highly competitive metropolitan culture has added up to our stresses at many levels. Sometimes violence (gendered or otherwise) along with repression, neurosis, loneliness and other psychological factors lessen the wellbeing of an individual, both physically and psychologically. Stress is body's way of responding to the demand which is caused by both good and bad events/experiences. The body reacts by releasing chemicals in the blood to combat this demand by a complex repertoire of behavioral and physiologic adaptive responses. [1-2] Stress experiences often lead to various chronic health conditions such as hypertension, coronary heart disease. [1]

Women have major roles to play in the collective social wellbeing. Stress may lead to early aging and death or sometimes

in reduced levels of performances in females. There are various risk factors for development of coronary heart disease (CHD). Some are modifiable (cigarette smoking, high blood pressure, elevated serum cholesterol, diabetes, obesity, sedentary habits, stress) and others non-modifiable (age, sex, family history, genetic factors). Stress is a modifiable risk factor for development of CHD. Recent researches have proved that most women (across cultures and borders) are too much stressed. As has been proved, twenty-five percent of females in United States die of heart diseases, [1-3] while eighty-seven percent of Indian women feel stressed most of the times, with eighty-two percent having insufficient time to relax. [4] As is evident from Neilson's reports women in emerging economic and social markets are more stressed than those in developed countries. [4]

The cardiovascular death rate has declined steadily only in men. [5-6] Women who have angioplasties and bypasses do not do as well as their male counterparts. Most of them suffer from heart attacks or congestive heart failure after treatment. [5-6] The discovery of widespread microvessel disease helps to explain why so many women with Ischemic Heart Disease are misdiagnosed and undertreated. [5-6] In Coronary artery disease (CAD) patients, mental stress is a well-described provocateur of ischemia. Mental stress increases myocardial oxygen demand. The physiologic response to mental stress encompasses an increased heart rate and often a peripheral vasoconstrictor response that increases left ventricular (LV) after load. In health, mental stress may have variable effects on cardiac output and is occasionally associated with a decline in LV ejection fraction. In CAD patients, a subset (20% to 70%) demonstrate a pronounced response, termed mental stress–induced myocardial ischemia (MSIMI), defined by left ventricular ejection fraction reduction ≥5%, new or worsening regional wall motion abnormalities, or new myocardial perfusion defects. [5-9]

Stress is thought to influence human eating behavior. Stress appears to alter overall food intake in two ways, resulting in under or overeating. Chronic life stress seems to be associated with a greater preference for energy and nutrient dense foods. [8-10] Stress-induced eating may be one factor contributing to the development of obesity which increases the risk of cardiovascular diseases (CVD). [6-11]

The autonomic nervous system plays a central role in governing the response to stress and how the body recovers following a stressor. Functional magnetic resonance imaging (MRI) have been used in studies to examine the hypothesized heart–brain connection and has found concurrent associations between vagal influenced heart rate variability and changes in blood flow through areas of the brain known to be involved in emotional responses, attention, and working memory. [12]

Yoga, meditation, hypnosis may help to reduce stress levels and these techniques need professional supervision and training, but progressive muscle relaxation (a technique developed by Edmund Jacobson) [2] though a recognized method for reduction of stress and anxiety is easy to learn and requires no constant guidance.

Hasim HA in 2011 [13] compared the effects of two different relaxation techniques: progressive muscle relaxation (PMR) and autogenic relaxation (AGR), on moods of young soccer players. They concluded that these two relaxation techniques bring about equivalent mood responses and may be used to regulate mood states.

The present study was conducted to detect stress levels among females in reproductive age group and the impact of PMR in these subjects, so that early lifestyle modifications may improve the quality of their lives and increase longevity. Women have a life-expectancy advantage over men, but a marked disadvantage with regards to

morbidity. Individual differences in physical and mental health are further notably explained by the degree of stress individuals endure, with women being more affected by stressors than men. [4] Female subjects in reproductive age group were included because of a specific reason. As has been already stated most Indian women are over stressed. In a survey conducted in 2011,[4] it was found the highest stress is perceived by women between 25-55 years who have to manage multiple roles in various fields. It is seen that the average lifespan of Indian women is 65years, while in developed countries it is 80 years. [14] Hence women of our country specifically of this age group deserve special attention.

MATERIALS AND METHODS

The present longitudinal interventional double blinded randomized control trial was conducted on females of reproductive age group in a time span of 5 years.

Sample size was calculated using online sample size calculator at Calculator.net (Confidence level 99.99%; Confidence interval 3%; population proportion 87% as per global research firm Nielson survey; [4] Population size 29437212 as per census report of India 2011) and calculated sample size was 1902 or more. Two thousand and twelve subjects were finally included in the study for final analysis of data. Though the calculated sample size was 1902 we selected two thousand and twelve subjects considering drop out cases as the study required three months of PMR practice and follow up.

Sampling procedure: Randomization was done using this online randomizer to select the subjects for allocation to different groups according to (Presumptive life event stress scale) PSLES [15] scores. Following inclusion and exclusion criteria seven thousand and five hundred subjects were initially screened and divided into three groups A, B, C using the above online randomizer (as according to PSLES scores subjects can be grouped into 3 categories) with 2500 subjects in each group. All were given a unique identification number. They were then asked to fill up PSLES scale. No subject had PSLES score less than 40. So the third group i.e. Group C had to be excluded. In group A 1425(57%) subjects had PSLES scores more than 200 and 1075(43%) had PSLES scores less than 200; In group B 1410(56.4%) subjects had PSLES scores more than 200 and 1090(43.6%) had PSLES scores less than 200; In group C 1420(56.8%) subjects had PSLES scores more than 200 and 1080(43.2%) had PSLES scores less than 200; Among all subjects 4255 (56.73%) subjects had PSLES scores more than 200 and 3245(43.27%) had PSLES scores less than 200. Using Chi square no significant difference was found between these three groups. From Group A 1006 subjects with PSLES scores more than 200 were chosen serially and were taken as G1and from Group B same number of subjects G2 with PSLES scores less than 200 but more than 40 were included according to the numbers allotted to them using the randomizer (G1 = subjects with PSLES more than 200; G2= subjects with PSLES less than 200 but more than 40).

• **Inclusion criteria:** Women under stress without any gross systemic, metabolic and infective disease in the reproductive age group were selected.

• **Exclusion criteria:**
Subjects suffering from chronic debilitating diseases such as:
1. Cardiac arrhythmias.
2. Hypertension.
3. Diabetes mellitus.
4. Ischemic heart disease.
5. Retinopathy.
6. Nephropathy.
7. Respiratory diseases.
8. Neuropathy.
9. Smokers and alcoholics.
10. Females receiving any drugs that may affect the autonomic reflexes.

11. Postmenopausal females, subjects on treatment from psychiatry problem or with past history of treatment were excluded.
12. Pregnant women, puerperal mothers, adolescent girls, perimenopausal women, sportswomen, yogis, subjects on regular meditation and exercise regime were not included.

The subjects were grouped into two: Subjects with PSLES SCORES[398] above 200(G1) and Subjects with PSLES SCORES less than 200 but more than 40 (G2) with 1006 subjects in each group. There was no significant difference in age and dietary habits between the two groups.

Methods:

1. Approval from the institutional ethics committee of Burdwan Medical College was taken before conduction of the study
2. Participants were recruited by random sampling.
3. Informed consent was taken from participating subjects
4. PSLES scores were assessed and subjects were divided into two groups.
5. Detailed history was taken regarding any past or current illness, hospitalization, medication, smoking and alcoholism and daily habits.
6. Participants were further screened based on the inclusion and exclusion criteria for final selection.
7. Pre-test instructions were given to avoid consumption of any drugs that may alter the Cardio respiratory parameters 48 hours prior to the test. The subjects were advised for a good restful sleep and to fast at least for 12 hours after a light dinner at the night before the test day. On the day of the test, no cigarette, nicotine, coffee, or drugs were permitted.
8. Life event stress and perceived stress of the subjects were measured by using Presumptive life event stress scale and Perceived stress scale[17] respectively.
9. Anthropometric measurements, resting pulse rate and blood pressure and ECG were recorded.
10. PFT was analyzed by Computerized spirometry.
11. Blood samples were drawn from subjects by sterile needle and syringes and sent to biochemical laboratory in sterile vials for analysis of FBS and Lipid profile.

Increased sympathetic activity has been observed during the premenstrual phase and this was positively correlated with the stress levels in previous studies. To avoid stress effects of the premenstrual phase, we examined our subjects during the postmenstrual phase.

All subjects were given a training of progressive muscle relaxation. Training involved tensing the specific muscle groups of body for 7-10 sec., followed by releasing them for 15-20sec. [2]

The subjects were asked to practice this technique at home for 20 minutes every day for 3 months and come for follow up at regular intervals. All parameters were reevaluated. 3 subjects in each group left the study during the training programme. Trainers were not aware about the stress scores and other parameters of the subjects. Subjects were only provided with the study results after completion of 3 months of PMR training and analysis of data to prevent any bias. Blinding was imposed on researchers, technicians, and subjects in this manner. This comparative interventional study was typically conducted as double-blind trials, where neither the physician nor the patient knew which group they belonged to. Double-blind trials help to eliminate any biased results.

Statistical analysis:

The computer software "Statistical Package for the Social Sciences (SPSS) version 16 (SPSS Inc. Released 2007. SPSS for Windows, Version 16.0. Chicago, SPSS Inc.)" was used to analyze the data, $P < 0.05^{*}$ was considered as significant and $P < 0.01^{**}$ was considered as highly significant.

RESULTS

Mean±SD values of PSLES score of two thousand and twelve subjects recruited

for the study was 237.99±79.44 and PSS score was 23.76±5.24; there was a positive correlation between PSLES scores and PSS scores with r value of 0.8.

PSLES scores: Group1 312.93±27.19; Group2 162.99±25.63 and there was significant difference between the two groups with P value<0.0001. PSS scores before PMR: Group128.15±3.09; Group 2 19.37±2.59 and there was significant difference between the two groups with P value<0.0001. PSS scores: After PMR Group1 24.2±3.43; Group 2 14.11±2.04 and there was significant difference between the two groups with P value<0.0001. PSS scores were positively correlated with PSLES score with r values of 0.0865 and 0.019186 respectively. PSS score were significantly decreased following PMR training in both groups. Age (years) G1 22.8±3.22 vs G2 22.64±3.15 and P value 0.26. 93% subjects in G1 were non-vegetarian and 92% in G2 and using chi square test we found no significant difference (P value 0.968 in dietary habits between the two groups.

TABLE 1: Shows Comparison of Different Parameters of Subjects with PSLES Scores More Than 200 and Less Than 200 before PMR Training

PARAMETERS	BEFORE PMR PSLES SCORES MORE THAN 200 N=1006 MEAN±SD	BEFORE PMR PSLES SCORES LESS THAN 200 N=1006 MEAN±SD	P VALUE
BMI(Kg/m^2)	25.14±2.83	24.29±2.63	<0.0001**
WAIST/HIP ratio	0.89±0.049	0.88±0.48	0.003**
PULSE(beats/min)	85.67±8.46	84.82±7.55	0.019*
SBP(mm of Hg)	122.8±9.54	122.24±8.59	0.168
DBP(mm of Hg)	80.61±4.89	80.22±4.24	0.057
FBS(mg/dl)	86.65±12.52	84.77±11.6	0.00045**
CHOLESTEROL(mg/dl)	163.68±12.79	156.77±12.12	<0.0001**
TRIGLYCERIDE(mg/dl)	136.09±24.53	129.08±20.22	<0.0001**
LDL(mg/dl)	92.98±18.74	93.2±15.8	0.811
HDL(mg/dl)	40.46±5.5	40.78±6.46	0.22
VLDL(mg/dl)	24.7±5.3	24.52±4.11	0.34
P WAVE (mv)	0.114±0.0269	0.112±0.0289	0.203
QRS(sec)	0.0497±0.0188	0.053±0.019	0.0002**
T(mv)	0.277±0.0865	0.27±0.077	0.075
PR(sec)	0.1577±0.05	0.169±0.015	<0.0001**
ST(mm)	0.1122±0.315	0.1698±0.375	0.0002**
QT(sec)	0.328±0.32	0.32±0.029	0.00015**
RR(sec)	0.71±0.08	0.71±0.07	0.1106
FEV_1(L/sec)	2.62±0.877	2.87±0.65	<0.0001**
FVC(L)	2.8 ±0.89	3.07±0.64	<0.0001**
FEV_1/FVC%	93.16±6.77	93.12±7.37	0.89
PSS	28.15±3.09	19.37±2.59	<0.0001**

Results show significant difference in values of BMI, waist/hip ratio, pulse, fbs, cholesterol, triglyceride, qrs complex, pr interval, st segment, qt interval, fev_1, fvc and pss scores between the two groups.

P < 0.05* was considered as significant and P < 0.01** was considered as highly significant.

BMI and WAIST/HIP were significantly more in G1 as compared to G2 before and after PMR training. BMI and WAIST/HIP were significantly decreased in both groups following PMR training. PSS was more positively correlated with BMI and WAIST/HIP in G1both before and after relaxation training. Pulse rate was significantly more in group with higher stress level and there was significant decrease in pulse rate following PMR training. PSS scores were positively correlated with pulse rate. There was no significant difference in SBP both before and after PMR session. SBP was significantly decreased in both groups following PMR training. PSS scores were positively correlated with SBP.DBP was significantly more in group with higher stress level after practicing PMR but there was no significant difference between the two groups before practicing PMR and there was significant decrease in both groups in DBP following PMR training. PSS scores were positively correlated with DBP (Table: 1-6).

Table 2: Shows comparison of different parameters of subjects with PSLES scores more than 200 and less than 200 after PMR training

PARAMETERS (AFTER PMR)	PSLES SCORES MORE THAN 200 N=1003 MEAN±SD	PSLES SCORES LESS THAN 200 N=1003 MEAN±SD	P VALUE
BMI(Kg/m^2)	23.8±1.99	23.35±2.88	< 0.0001**
WAIST/HIP	0.869±0.067	0.85±0.69	0.00024**
PULSE(beats/min)	82.26±7.07	83.16±7.95	0.007**
SBP(mm of Hg)	117.88±8.6	118.11±7.11	0.495
DBP(mm of Hg)	77.21±7.68	76.36±8.13	0.015*
FBS(mg/dl)	84.22±5.87	81.5±6.64	< 0.0001**
CHOLESTEROL(mg/dl)	159.9±15.14	151.42±4.2	< 0.0001**
TRIGLYCERIDE(mg/dl)	119.6±27.47	114.56±24.2	< 0.0001**
LDL(mg/dl)	90.63±10.43	79.28±13.84	< 0.0001**
HDL(mg/dl)	45.44±4.81	47.13±2.07	< 0.0001**
VLDL(mg/dl)	24.16±6.23	22.9±4.94	< 0.0001**
P WAVE (mv)	1.125±0.25	1.093±0.24	0.0045**
QRS(sec)	0.068±0.02	0.0775±0.023	< 0.0001**
T(mv)	0.1727±0.088	0.178±0.081	0.143
PR(sec)	0.195±0.031	0.18±0.033	< 0.0001**
ST(mm)	0.044±0.017	0.073±0.02	0.003**
QT(sec)	0.345±0.038	0.33±0.035	<0.0001**
RR(sec)	0.73±0.68	0.728±0.073	0.03*
FEV_1(L/sec)	2.76±0.76	3.1±0.74	< 0.0001**
FVC(L)	2.94±0.82	3.28±0.8	< 0.0001**
FEV_1/FVC%	93.98±5.77	94.7±4.14	0.0014**
PSS	24.2±3.43	14.11±2.04	< 0.0001**

There was significant difference in all parameters excepting SBP and t wave between the two groups after PMR training.
P < 0.05* was considered as significant and P < 0.01** was considered as highly significant.

Table 3: Shows Comparison of Different Parameters of Subjects with PSLES Scores More than 200 Before And After PMR Training

PARAMETERS	BEFORE PMR IN SUBJECTS WITH MORE THAN 200 PSLES SCORES MEAN±SD	AFTER PMR IN SUBJECTS WITH MORE THAN 200 PSLES SCORES MEAN±SD	P VALUE
BMI(Kg/m^2)	25.14±2.83	23.8±1.99	< 0.0001**
WAIST/HIP	0.89±0.049	0.869±0.067	< 0.0001**
PULSE(beats/min)	85.67±8.46	82.26±7.07	< 0.0001**
SBP(mm of Hg)	122.8±9.54	117.88±8.6	< 0.0001**
DBP(mm of Hg)	80.61±4.89	77.21±7.68	< 0.0001**
FBS(mg/dl)	86.65±12.52	84.22±5.87	< 0.0001**
CHOLESTEROL(mg/dl)	163.68±12.79	159.9±15.14	< 0.0001**
TRIGLYCERIDE(mg/dl)	136.09±24.53	119.6±27.47	< 0.0001**
LDL(mg/dl)	92.98±18.74	90.63±10.43	0.000552**
HDL(mg/dl)	40.46±5.5	45.44±4.81	<0.0001**
VLDL(mg/dl)	24.7±5.3	24.16±6.23	0.024*
P WAVE (mv)	0.114±0.0269	0.1125±0.025	0.188
QRS(sec)	0.0497±0.0188	0.068±0.02	< 0.0001**
T(mv)	0.277±0.086	0.17±0.088	< 0.0001**
PR(sec)	0.1577±0.05	0.195±0.031	< 0.0001**
ST(mv)	0.1122±0.315	0.044±0.17	< 0.0001**
QT(sec)	0.328±0.32	0.345±0.038	< 0.0001**
RR(sec)	0.71±0.08	0.73±0.68	< 0.0001**
FEV_1(L/sec)	2.62±0.877	2.76±0.76	< 0.0001**
FVC(L)	2.8 ±0.89	2.94±0.82	< 0.0001**
FEV_1/FVC%	93.16±6.77	93.98±5.77	< 0.0001**
PSS	28.15±3.09	24.2±3.43	< 0.0001**

ALL PARAMETERS EXCEPTING P WAVE WERE SIGNIFICANTLY DIFFERENT BEFORE AND AFTER PMR SESSION IN SUBJECTS WITH PSLES SCORES MORE THAN 200.
P < 0.05* was considered as significant and P < 0.01** was considered as highly significant.

FBS, Cholesterol, Triglyceride levels, were significantly more in group with higher stress level and there was significant decrease in FBS following PMR training. PSS scores were positively correlated with FBS, Cholesterol, Triglyceride levels. Subjects with higher stress scores had significantly higher LDL Cholesterol levels after PMR session. LDL Cholesterol was significantly decreased in both groups following PMR training. PSS scores were positively correlated with LDL Cholesterol. There was significant difference in HDL levels after PMR session

and subjects having less PSS scores had higher HDL levels. HDL was significantly increased in both groups following PMR training. PSS scores were negatively correlated with HDL. Subjects with higher stress scores had significantly higher VLDL Cholesterol levels after PMR session. VLDL Cholesterol was significantly decreased in both groups following PMR training. PSS scores were positively correlated with VLDL Cholesterol (Table: 1-6).

TABLE 4: SHOWS COMPARISON OF DIFFERENT PARAMETERS OF SUBJECTS WITH PSLES SCORES LESS THAN 200 BEFORE AND AFTER PMR TRAINING

PARAMETERS	BEFORE PMR IN SUBJECTS WITH PSLES SCORES LESS THAN 200 MEAN±SD	AFTER PMR IN SUBJECTS WITH PSLES SCORES LESS THAN 200 MEAN±SD	P VALUE
BMI(Kg/m^2)	24.29±2.63	23.35±2.88	< 0.0001**
WAIST/HIP	0.88±0.48	0.85±0.69	< 0.0001**
PULSE(beats/min)	84.82±7.55	83.16±7.95	< 0.0001**
SBP(mm of Hg)	122.24±8.59	118.11±7.11	< 0.0001**
DBP(mm of Hg)	80.22±4.24	76.36±8.13	< 0.0001**
FBS(mg/dl)	84.77±11.6	81.5±6.64	< 0.0001**
CHOLESTEROL(mg/dl)	156.77±12.12	151.42±4.2	< 0.0001**
TRIGLYCERIDE(mg/dl)	129.08±20.22	114.56±24.2	< 0.0001**
LDL(mg/dl)	93.2±15.8	79.28±13.84	< 0.0001**
HDL(mg/dl)	40.78±6.46	47.13±2.07	< 0.0001**
VLDL(mg/dl)	24.52±4.11	22.9±4.94	< 0.0001**
P WAVE (mv)	0.11±0.0289	0.1093±0.024	0.009**
QRS(sec)	0.053±0.019	0.0775±0.023	<0.0001**
T(mv)	0.27±0.077	0.178±0.081	< 0.0001**
PR(sec)	0.169±0.015	0.18±0.033	< 0.0001**
ST(mv)	0.1698±0.375	0.073±0.2	< 0.0001**
QT(sec)	0.32±0.029	0.33±0.035	< 0.0001**
RR(sec)	0.71±0.07	0.728±0.073	< 0.0001**
FEV_1(L/sec)	2.87±0.65	3.1±0.74	< 0.0001**
FVC(L)	3.07±0.64	3.28±0.8	< 0.0001**
FEV_1/FVC%	93.12±7.37	94.7±4.14	< 0.0001**
PSS	19.37±2.59	14.11±2.04	< 0.0001**

ALL PARAMETERS EXCEPTING WERE SIGNIFICANTLY DIFFERENT BEFORE AND AFTER PMR SESSION IN SUBJECTS WITH PSLES SCORES LESS THAN 200.

P < 0.05* was considered as significant and P < 0.01** was considered as highly significant.

TABLE 5: SHOWS CORRELATION OF PERCEIVED STESS SCORES WITH DIFFERENT PATAMETERS OF THE TWO GROUPS BEFORE PMR TRAINING

PARAMETERS (BEFORE PMR)	PSLES MORE THAN 200 (CORRELATION WITH PSS) r value	PSLES LESS THAN 200 (CORRELATION WITH PSS) r value	P VALUE
BMI(Kg/m^2)	0.533002223	0.088462724	<0.0001**
WAIST/HIP ratio	0.010324579	0.039082052	0.003**
PULSE(beats/min)	0.150156019	0.061862305	0.019*
SBP(mm of Hg)	0.025355677	0.035302703	0.168
DBP(mm of Hg)	0.070227538	0.024930742	0.057
FBS(mg/dl)	0.18381459	0.192815326	0.00045**
CHOLESTEROL(mg/dl)	0.244300091	0.108917674	<0.0001**
TRIGLYCERIDE(mg/dl)	0.141453678	0.136265093	<0.0001**
LDL(mg/dl)	0.082076826	0.036698413	0.811
HDL(mg/dl)	-0.139638013	-0.053918273	0.22
VLDL(mg/dl)	0.002965216	0.047787982	0.34
P WAVE (mv)	0.129727928	0.130740356	0.203
QRS(sec)	0.312078791	-0.067569232	0.0002**
T(mv)	0.109313459	0.048341261	0.075
PR(sec)	-0.106119462	-0.038889121	<0.0001**
ST(mv)	0.295646324	-0.191989556	0.0002**
QT(sec)	-0.023538285	-0.037082298	0.00015**
RR(sec)	-0.139056064	-0.080790883	0.1106
FEV_1(L/sec)	-0.120908738	-0.348523923	<0.0001**
FVC(L)	-0.1104796	-0.361021419	<0.0001**
FEV_1/FVC%	-0.131367211	-0.047117993	0.89

RESULTS SHOW SIGNIFICANT DIFFERENCE IN VALUES OF BMI, WAIST/HIP RATIO, PULSE, FBS, CHOLESTEROL, TRIGLYCERIDE, QRS COMPLEX, PR INTERVAL, ST SEGMENT, QT INTERVAL, FEV_1, FVC AND PSS SCORES BETWEEN THE TWO GROUPS.

"r" correlation coefficient showing correlation of PSS with all other parameters.

P < 0.05* was considered as significant and P < 0.01** was considered as highly significant.

TABLE 6: SHOWS CORRELATION OF PERCEIVED STESS SCORES WITH DIFFERENT PATAMETERS OF THE TWO GROUPS AFTER PMR TRAINING

PARAMETERS (AFTER PMR)	PSLES MORE THAN 200 (CORRELATION WITH PSS) r value	PSLES LESS THAN 200 (CORRELATION WITH PSS) r value	P VALUE
BMI(Kg/m^2)	0.09358553	0.072	<0.0001**
WAIST/HIP	0.104253812	0.089068798	<0.0001**
PULSE(beats/min)	0.225621261	0.118971463	0.00024**
SBP(mm of Hg)	0.132141491	0.135553791	0.007**
DBP(mm of Hg)	0.059753607	0.272619481	0.495
FBS(mg/dl)	0.069729642	0.07	0.015*
CHOLESTEROL(mg/dl)	0.033744967	0.098	<0.0001**
TRIGLYCERIDE(mg/dl)	0.237012126	0.06	<0.0001**
LDL(mg/dl)	0.107977754	0.300277052	<0.0001**
HDL(mg/dl)	-0.043743097	-0.021675526	<0.0001**
VLDL(mg/dl)	0.273675892	0.284185517	<0.0001**
P WAVE (mv)	-0.274318331	0.006797274	<0.0001**
QRS(sec)	-0.238126043	0.038125555	0.0045**
T(mv)	0.106288377	0.040262826	<0.0001**
PR(sec)	-0.274318331	0.15990501	0.143
ST(mv)	0.001351246	-0.062978425	0.000**
QT(sec)	-0.07340344	-0.228768649	0.003**
RR(sec)	-0.206450862	-0.139705444	<0.0001**
FEV_1(L/sec)	0.102518908	-0.134495741	0.03*
FVC(L)	0.113259627	-0.158265158	<0.0001**
FEV_1/FVC%	-0.091186813	-0.106298185	<0.0001**

THERE WAS SIGNIFICANT DIFFERENCE IN ALL PARAMETERS EXCEPTING SBP AND T WAVE BETWEEN THE TWO GROUPS AFTER PMR TRAINING.

"r" correlation coefficient showing correlation of PSS with all other parameters.

P < 0.05* was considered as significant and P < 0.01** was considered as highly significant.

Subjects with higher stress scores had significantly higher P-wave after PMR session. P-wave was significantly decreased in G2 following PMR training. Subjects with higher stress scores had significantly lower QRS complex before and after PMR session. QRS complex was significantly increased in both groups following PMR training. Subjects with higher stress scores had significantly higher T-wave before PMR session. T-wave was significantly decreased in both groups following PMR training. PSS scores were positively correlated with T-wave. P-R interval was significantly increased in both groups following PMR training. S-T-segment was significantly decreased in both groups following PMR training. QT interval increased significantly following PMR. PSS scores were negatively correlated with Q-T interval. There was no significant difference between the two groups in respect of R-R interval before PMR but the difference became significant after PMR session. R-R interval was significantly increased in both groups following PMR training. PSS scores were negatively correlated with R-R interval (Table: 1-6).

Subjects with higher stress scores had significantly lower FEV1, FVC before and after PMR session. FEV1, FVC were significantly increased in both groups following PMR training. Subjects with higher stress scores had significantly lower FEV1/FVC% levels after PMR session. FEV1/FVC% was significantly increased in both groups following PMR training. PSS scores were negatively correlated with FEV1/FVC% (Table: 1-6).

DISCUSSION

The rapid rise of non-communicable diseases (NCDs) is a formidable challenge in the twenty-first century that threatens economic and social development of the world. NCDs are currently responsible for 68% deaths globally i.e. more than all other causes combined. In India, NCDs are estimated to account for 60% of total deaths and also 26% of premature deaths between the age group of 30 and 70 years. [17]

The highest coronary mortality at present is seen in European Region followed by South-East Asia Region and India being a part of this region also faces the challenge of coronary mortality. There has been

considerable increase in prevalence of CHD in urban areas in India in the last decade. [18]

INCIDENCE OF BURDEN OF CHD

INDICES	URBAN	RURAL
PREVALANCE RATE / 1000	64.37	25.27
DEATH RATE/1000	0.8	0.4
DALY PER 100000	2703.4	986.2

AGE SPECIFIC PREVALANE RATE PER 1000 FOR CHD

AGE GROUP IN YEARS	URBAN MALE	URBAN FEMALE
20-24	8	6.8
25-29	19.65	26.24
30-34	17.05	22.96
35-39	43.18	48.44
40-44	47.25	65.85
45-49	83.26	105.35
50-54	93.07	111.88

The above two tables demonstrate that urban populations are at higher risk of developing CHD and prevalence rate per 1000 for CHD is higher in females as compared to males in the age group of 25-54 years. There are various risk factors for development of CHD. Some are modifiable (cigarette smoking, high blood pressure, elevated serum cholesterol, diabetes, obesity, sedentary habits, stress) and others non-modifiable (age, sex, family history, genetic factors). Stress is a modifiable risk factor for development of CHD. [18]

It is in the above context that the present study was aimed to study the effect of stress on BMI, Waist/Hip ratio, cardio respiratory parameters, fasting blood sugar, lipid profile, ECG and PFT of urban females in reproductive age group and to demonstrate the effect of PMR on these parameters in these individuals.

Stress in a known factor responsible for the development of dyslipidemia, and dyslipidemia is known to alter autonomic functions by decreasing heart rate variability and baroreceptor sensitivity. [19-23] This effect of stress is also evident in the present study. Stress also alters distribution of body fat and significant positive correlation of PSS with BMI and WAIST/ HIP was also found in the present study.

A study in 2013, explored the concept that whether viewing pictures of nature prior to a stressor altered the autonomic function during recovery from the stressor. Natural scenes are thought to produce relaxation effects on the mind. [20] Parasympathetic activities were significantly higher during recovery following the stressor when viewing scenes of nature compared to viewing scenes depicting built environments. It was concluded that viewing nature scenes prior to a stressor alters the autonomic activity in the recovery period. Our study also demonstrates an improvement in autonomic functions following mental relaxation after PMR exercises.

Torres SJ et al in 2007 [24] implied that, stress is thought to influence and change human eating behavior. Stress appears to alter overall food intake resulting in under or overeating. Chronic stressful life is known to be associated with a greater preference for energy and nutrient dense foods. [24] Longitudinal studies suggest that chronic life stress may be causally linked to weight gain. Stress-induced over-eating may be one reason contributing to the development of obesity which increases the risk of CVD[25]. Perceived stress was positively correlated with BMI and Waist hip ratio and these findings are similar to the present study.

In 2010, Shahnam M et al. [25] demonstrated higher levels of total cholesterol and LDL-C and lower levels of HDL-C among stressed individuals. LDL cholesterol is known to decrease baroreceptor sensitivity and total cholesterol decreases HRV and reduction of LDL cholesterol and total cholesterol following relaxation exercises may contribute to increases in HRV. Thus, stress produces significant cardiovascular hazards by altering biochemical parameters. Practicing PMR may alter the lipid profile and help in prevention of cardiovascular diseases.

Matousek RH et al [26] observed that PMR not only improves the clinical parameters but also decreases cortisol levels and results in decrease of total cholesterol, triglyceride, and LDL-C levels. In the present series also, we found significant

decrease in total cholesterol, triglyceride, LDL, VLDL levels and increase in HDL levels in subjects after practicing PMR. We also observed decrease in blood pressure, lipid profile, fasting sugar, and HR in our subjects after practicing PMR for 3 months.

The electrocardiographic response patterns during exercise at low and high heart rate were compared with the response pattern during emotional stress in a study by Hijzen TH et al [27] in 1995. Significant differences in ECG during exercise and emotional stress were observed:

1. During exercise the ST segment was more depressed as compared to emotional stress
2. T-wave amplitudes were larger and QT and PQ were significantly shorter than during emotional stress.

Emotional stress did not evoke an exercise-like cardiovascular response pattern, which may lead to a metabolically maladaptive situation. ECG changes during emotional stress were similar to the ECG changes during right stellate ganglion stimulation, while the ECG changes during exercise were similar to the ECG changes obtained during left stellate ganglion stimulation.

The left and right stellate ganglia provide the majority of the sympathetic supply to the heart via post-ganglionic fibers, although the left stellate ganglion is quantitatively dominant at the ventricular level. Myocardial Infarction (MI) may lead to partial denervation of these fibers at the level of the myocardium and paradoxically induce a super sensitivity to catecholamines, making the heart more vulnerable to the electrical induction of ventricular arrhythmias. Denervation of sympathetic fibers counteracts this pathologic process by reducing the amount of norepinephrine released at the ventricular level and increasing the ventricular fibrillatory threshold. Coronary vasodilation and vagal stimulation to the heart are also increased. [28-30]

The QT interval is regulated by cardiac sympathetic innervation; shortening of the QT interval occurs with left stellate ganglion block (LSGB) whereas lengthening of the QT interval occurs with right stellate ganglion block. In the present study there was significant lengthening of QT interval following PMR training. This is similar to findings of Hijzen TH et al. [27] Their study demonstrated that the ECG changes during emotional stress were similar to the ECG changes during right stellate stimulation, and significant lengthening of QT interval following PMR training in the present study may be due to modulation of cardiovascular responses following PMR training.

In experimental models, stellate ganglion block (SGB) reduces the induction of atrial fibrillation (AF), but these findings are not well studied in humans till date. The aim of a study by Leftheriotis D et al in 2016 [31] was to assess the effect of unilateral SGB on atrial electrophysiological properties and AF induction in patients with paroxysmal AF. Thirty-six patients with paroxysmal AF were included in the study. They were randomized in a 2:1 order to temporary, transcutaneous, pharmaceutical SGB with lidocaine or placebo before pulmonary vein isolation. Lidocaine was randomly infused to the right or left ganglion. Before and after randomization, atrial effective refractory period (ERP) of each atrium, difference between right and left atrial ERP, intra- and interatrial conduction time, AF inducibility, and AF duration were assessed.

After SGB, right and left atrial ERP was prolonged. AF was induced by atrial pacing in all 24 patients before SGB, but only in 13 patients after intervention. AF duration was shorter after SGB. It was demonstrated in the study that unilateral temporary SGB decreased AF duration, prolonged atrial ERP, reduced AF inducibility. An equivalent effect of right and left SGB on both atria was observed. [31]

Short atrial ERP has been recognized as a factor that predisposes to AF. Sinus rhythm restorations as well as the reversal in AF-induced atrial remodeling are

associated with ERP prolgation. In canine models unilateral stimulation of the stellate ganglion is associated with significant increase in ERP dispersion (dERP), shortening of atrial ERP, and facilitation of AF. Unilateral ganglionectomy reverses these effects. [31]

The study observed a beneficial effect of unilateral SGB, expressed by the prolongation of ERP without dERP impairment and the protection against AF induction. The findings may imply that there is not a discrete ipsilateral distribution of cardiac projections of the stellate ganglia in humans. The equivalent effect of right and left SGB may be explained by common neural autonomic pathways via right vagal projections and superior left ganglionated plexi. [31]

The presence of regions with slowed conduction, facilitating the functional substrate for the occurrence of reentry circuits within the atria has been described as a mechanism of AF establishment. Abnormalities in atrial conduction, predisposing to AF, have been found to be associated with atrial dilation, stretch, as well as fibrosis. The influence of autonomic modulation on atrial conduction properties in patients with paroxysmal AF till date remains an unexplored field of study. [31]

Unilateral right or left stellate ganglion stimulation in vivo is associated with increased levels of transcardiac norepinephrine and arising of ectopic arrhythmias from the pulmonary veins, the atria, and the vein of Marshal. Continuous low level vagus nerve stimulation suppresses AF inducibility by directly suppressing the neural activity of major ganglionatedplexi within intrinsic cardiac autonomic nervous system and sympathetic nerve activity. Bilateral stellectomy significantly decreases the incidence of electrically induced AF and facilitates sinus rhythm maintenance. It has been observed that unilateral stellate ganglionectomy can reduce AF initiation and prevent electrical remodeling in the atrium and pulmonary veins. [31]

The suppressed sympathetic activity and down regulated β1-adrenergic receptors have been proposed as mechanisms contributing to this beneficial effect. Vagal stimulation associated with an altered release of neuromodulators (such as serotonin and norepinephrine), as well as nitric oxide, which is coupled to the signaling pathways of multiple neuropeptides and ion channels, may also play a role. Relaxation exercises may be a significant contributor in this aspect. [1-2]

In humans, unilateral right stellate ganglion block (SGB) has a combined sympathetic and parasympathetic influence on sinus node, resulting in a decrease in sinus rate. Mental stress has been shown to be an important factor in stimulation of right stellate ganglion in previous studies. After SGB, a decrease in low frequency and low/high frequency ratio of heart rate variability has been observed, suggesting a shift in cardiac autonomic balance toward the predominance of parasympathetic over sympathetic activity. [28-31] Similar effect in autonomic balance was noticed in the present study following PMR training. QT and RR interval were significantly increased following PMR training.

Rachel Lampert in 2015 [32] looked directly at effects of stress on heterogeneity of repolarization. Heterogeneity of repolarization has long-recognized to be an important factor in arrhythmogenesis. This was studied by measuring T-wave alternans (TWA) during a laboratory mental stress protocol. Similar to a creating physical stress on a treadmill exercise test, they created mental stress in the laboratory which included the following protocols: these included asking the subject to do arithmetic mentally without any other support e.g. serial subtraction of 7 from a 3-digit number, or involve in a speaking task with emotional content. They also did “anger recall” test. The subjects were asked to recapitulate and speak out about a recent incident in which they were irritated or angry. They evaluated effects of mental stress on three surface measures of

heterogeneity which can be determined from Holter monitoring:

1. T-wave alternans (TWA,)
2. T-wave amplitude (Tamp,)
3. T-wave area (T area,) calculating TWA in the time domain using the inter beat average technique.

In this study, 33 patients with ICDs and a history of ventricular arrhythmia were included. TWA was seen to increase from 22 at baseline to 29 during mental stress ($p<0.001$.) All other measures of heterogeneity also increased following mental stress. Broad-range repolarization instability was also increased during stress.

The effect of anger on TWA in the laboratory may be predictive of arrhythmias in real life. In a long time, follow-up of the study of anger and TWA it was found that anger-induced TWA was a significant predictor of arrhythmia. Patients with gross abnormal results in this test had 10 times more common incidences of ventricular arrhythmias as compared to that of other patients (CI 1.6-113, $p<0.05$.) Anger-induced TWA remained predictive factor after controlling for standard predictors of arrhythmia such as ejection fraction, prior clinical arrhythmia, and wide QRS. The findings of this study are consistent with the impact of autonomic factors on TWA which previous studies have observed.

Conduction in the atria can be measured noninvasively using the signal-averaged P wave. A prolonged SA-P duration has been associated with recurrent AF. [32] In the normal atrium, sympathetic stimulation with isoproterenol shortens SA-P duration; while, beta-blockers slow conduction. Atropine given after beta-blockade shortens SA-P duration. P-wave duration measured from 24-hour Holter monitoring have been observed to be shorter in daytime. This further suggests that changes in sympathovagal balance alter conduction through the atria.

Potential mechanisms of the arrhythomogenic effects of stress on AF are less well documented. Repolarization as measured in invasive experiments by the atrial effective refractory period shortens with sympathetic stimulation in most although not all studies. Also, sympathetic stimulation acts synergistically when combined with vagal stimuli. [32]

From the above discussions it is evident that mental stress may alter autonomic functions, induce arrhythmias, ischemia. Stress management strategies like PMR, may help to modulate these effects.

The present study is in variance to studies conducted by Putt MT et al [33] but concurs with the findings of Lahmann et al. in 2009. [34] Putt MT et al. in 2008 conducted a study to determine if a specific hold and relax stretching technique was capable of (1) reversing the effect of tight chest wall muscles by increasing chest expansion, vital capacity, and shoulder range of motion and (2) decreasing perceived dyspnea and Respiratory Rate(RR) in persons with chronic obstructive pulmonary disease. No significant effect on axillary and xiphisternal chest expansion, perceived dyspnea, or RR was observed. We had included only healthy individuals and this may be the cause in variance of results.

Ritz T et al in 2005 [35] reviewed studies from 15 years that have used experimental emotions and stress induction techniques or longitudinal diary observations to explore these influences of stress on lung function tests. Findings suggested that unpleasant emotional states are associated with a decline in lung function in health and asthma. Pleasant emotional states were also sometimes associated with a lung function decline, suggesting a susceptibility of the airways to arousal in general.

FEV1 AND FVC were positively correlated with PSS in G1 after PMR. Pleasant emotional states are sometimes associated with a lung function decline. This suggests a susceptibility of the airways to arousal in general and parasympathetic stimulation. This may be the cause of the above finding which is in concordance with findings of Ritz T et al. [35]

Lolak S et al in 2008 [36] examined the effect of progressive muscle relaxation (PMR) training in patients with chronic breathing disorders. These subjects were receiving pulmonary rehabilitation (PR). For depression, there was a significant improvement within each group over time. Depression scores were also lower as a result of PMR practice throughout the period of study. Our study demonstrated similar effects of PMR on PSS and PFT.

CONCLUSIONS

Perceived stress may adversely affect cardiovascular respiratory profile and anthropometric parameters of healthy adult females of reproductive age group and regular practice of PMR may decrease perceived stress levels and help in modulating cardiorespiratory profile with decreasing future complications like diabetes, dyslipidemia, coronary artery disease, metabolic syndrome. Evidence-based techniques like PMR are easy to learn and practice, with good results in individuals with good health or with a disease.

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