DOI: https://doi.org/10.53350/pjmhs22166135

ORIGINAL ARTICLE

Exercise Induces Autonomic and Neuro-endocrine Response among Psychologically Stressed Medical Students

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ABSTRACT

Background: Psychological stress activates Hypothalamic- Pituitary- Adrenal axis (HPA) and Sympathoadrenal (SMA) axis". Exercise has stress buffering action and it improves HRV and normalizes the cortisol response to stress.
Aim: To evaluate the effect of moderate exercise in stabilizing autonomic and neuro-endocrine response to Psychological stress". **Study design**: Randomized experimental study.
Methodology: 100 MBBS students were engaged through DASS (Depression anxiety stress scale) proforma. They were divided into two groups; stress and control. HRV and serum cortisol levels were obtained. Stressed group did regular moderate exercise for 06 weeks. Independent t test was employed to see the difference in two parameters among the two groups. A p value of ≤ 0.05 was taken as significant.
Results: After the intervention stressed group exhibited prominent decrease in LF/HF, LFnu, LFms$^{2"}$ and serum cortisol (0.009,0.033,.027 and 0.007 respectively. HFnu was significantly improved post exercise (.004). Significant reduction in serum cortisol was observed in stressed group after exercise (.007).
Conclusion: Exercise can be employed to buffer the effect of psychological stress as it normalizes HPA axis" and autonomic response.
Keywords: Heart rate variability, Psychological Stress, Exercise and Hypothalamic Pituitary Adrenal Axis'.

INTRODUCTION

Psychological stress is a big health issue throughout the world that reduces the efficiency of its victims[1]. It activates both Hypothalamic- Pituitary- Adrenal axis (HPA) & Sympathoadrenal (SMA) axis. It is processed in cerebral cortex causes stimulations of paraventricular neurons (PVN) of hypothalamus leading release of corticotrophin releasing hormone (CRH) into hypothalamic pituitary portal system. CRH goes to pituitary thus releases adrenocorticotropic hormone (ACTH) hormone. ACTH arouses the adrenal gland to produce cortisol. It regulates HPA axis via negative feedback mechanism halts the release of CRH and ACTH from the hypothalamus and pituitary however repeated stimulation of HPA axis causes a state of persistent hypercortisolemia[2].

It stimulates sympathetic branch of autonomic nervous system (ANS). ANS has sympathetic & parasympathetic systems, the two being operating in balance. SNS works in circumstances of stress and PNS is works in resting conditions. Sympathetic stimulation raises heart rate and lowers Heart rate variability. ANS is a system which errs on the side of caution and when in doubt prepares for the worst thus maximizing survival and adaptation response[3].

Heart rate variability (HRV) is a quantitative way to judge cardiac autonomic function[4]. It estimates the collaborative working of various factors which are involved in modulation of heart rate, thus showing continuous communication among CNS and sinoatrial node working[5]. Decrease heart rate variability is linked with many medical and psychosomatic issues[6].

HRV is quantified by means of time and frequency domain methods. Frequency domain method depends upon spectral analysis. Three frequency components are used as a standard in spectral band; High frequency HRV, extents 0.15–0.4 Hz", is due heart rate variation due to respiration & vagal outflow, lower-frequency (LF) HRV (0.05–0.15 Hz), is hypothesized to be intermediated by sympathetic & parasympathetic system and very low frequency (VLF) <0.04 Hz.[7]

Exercise normalizes HRV and protects cardiovascular system by decreasing heart rate by raised cardiac vagal tone[8]. It buffers by regulating HPA axis. Regular exercise has a documented role in evolving changes brain that leads to better cognitive performance[9]. Medical education is very stressful and tiring and high level of stress negatively effects learners' performance[10]. The students should thus be engaged into different exercises.

The objective of the study was to evaluate the effective of moderate exercise in stabilizing autonomic and neuroendocrine response to Psychological stress.

Received on 07-11-2021
Accepted on 17-05-2022

METHODOLOGY

"Randomized experimental study was directed in Physiology lab at Islamic International Medical College and Yusra Medical and Dental College from June 2014 to December 2017 following approval from research ethical committee of Islamic International Medical College, Riphah University Rawalpindi. 100" MBBS students were included. The participants did not had any known acute or chronic disease. They were grouped into stress (score: 19-33) and control (score:0-14) groups after filling DASS (Depression anxiety stress scale) profoma[11]. Consent was taken.
Exercise Protocol: They underwent exercise for five days a week for six consecutive weeks using exercise cycle model HF-700. They exercised for half an hour at the speed of thirteen miles per hour and resistance was set at moderate level.
HRV recording: Fifteen minutes ECG was documented & studied for HRV in agreement with Task Force of European Society of Cardiology and the North American Society of Pacing Electrophysiology[7]. Participants were asked to refrain from taking "tea" or coffee at least 12 hours before HRV recording. Female students during their "follicular phase" of "menstrual cycle" to report as ovarian hormones have effect on HRV. Post intervention, students' blood samples for cortisol were collected. Their bodyweights were recorded for BMI and HRV calculation.
Statistical Analysis: SPSS 21 analyzed the data. Mean±SD of the parameters was calculated. Independent t test was applied for quantitative parameters. Paired t test was also applied for post-exercise effect on HRV indices and serum cortisol.

RESULTS

General parameters of enrolled subjects were shown in Table-1. Mean age of the stress group was 21.10 ± 5.68 for female was and 22.20 ± 4.69 years.

Table-1: Descriptive Characteristics of Subjects (n=100)

Parameters	Group-A (x̄ ± SD)	Controls (x̄ ± SD)
Age (years)	22.11± 4.38	22.30± 3.69
Weight (kg)	69.10 ±12.32	65.60±12.12
BMI (kg/m^2)'	23.00± 2.3	22.6±1.3
Heart rate /min	78.31±1.83	80.30±6.56

Table-2 depicted mean LFms^2 was 804.04± 540.25ms^2 in stressed students and 546.06±798.58ms^2 with difference being very significant ($p \leq 0.05$).

Table-2: Comparison of HRV and Serum Cortisol among Groups

Parameters	Stressed students x̄ ± SD	Controls students x̄ ± SD	*P* value
LFms^2	804.04 ± 540.25	546.06.36 ± 798.58	0.042*
HFms^2	349.07 ± 211.88	326.60 ± 171.68	0.868
LFnu	63.05 ± 12.44	49.22 ± 12.24	0.001*
HF nu	25.472 ± 9.58	40.29 ± 11.02	0.000**
LF/HF	3.514 ± 3.172	1.39 ± 0.915	0.000**
Serum Cortisol (ng/dl)	139.94 ± 56.16	98.49 ± 41.92	0.011*

*Statistically significant

Table-3 showed values of HRV indices and serum cortisol before and after exercise. Post exercise HFnu was 38.71±16.01. Post exercise cortisol 99.30+40.01ng/dl, considerably less than pre-exercise values in stressed group.

Table-3: HRV and "serum cortisol" in stressed group pre and post exercise

Parameters	Mean ± SD		*p* value
	Pre exercise	Post exercise	
LFms^2	804.04 ± 540.25	445.82 ± 283.34	0.027*
HFms^2	349.35 ± 211.87	382.15 ± 30.0	0.900
LFnu	63.05± 12.44	52.19 ± 18.76	0.033*
HFnu	24.472± 8.58	38.71 ± 16.01	0.004*
LF/HF	3.514 ± 3.172	1.87 ± 1.2	0.009*
Serum Cortisol (ng/dl)	139.94 ± 56.16	99.30±40.01	0.007*

*Statistically significant

DISCUSSION

Medical education is extremely tiring, time demanding and full of stresses. It demands professionalism and commitment. In medical students and doctors, psychological stress is an important risk factors for the development and progression of metabolic syndromes through various mechanisms.[10] It is considered as major health issue that leads to alteration in "cardiovascular" and "biochemical" parameters. Various researches have clearly the mechanisms through which stress causes derangements in "human Physiolog"[11].

Current study investigated the affect of stress on cardiovascular & biochemical factors of enrolled subjects. Mean LFnu, LF/HF and LFms$^{2"}$ were markedly high in stressed group in comparison to control group thus depicting enhanced sympathetic activity. Comparable findings have been reported by various studies. Work of Punita P highlighted decrease in HRV indices in "females[12]. Other studies showed decline in HRV" among males following mental stress[13,14]. A research by Föhr et al also depicted decrease in HRV after subjects were exposed to stress.[15] The meta-analysis by Kim HG et al same finding[10]. Decreased RMSSD component of HRV in response to stressful events of brief period was reported Verkuil B et al[16]. Exercise has protecting effect on cardiovascular system due to increased vagal tone and reduction in heart rate at rest. Exercise is suggested as a therapeutic management for re-establishing disturbed autonomic balance among stressed individuals. Numerous studies demonstrated that training improves the working of cardiac autonomic pathways[8]. Gurupriya R, exposed that yoga exercise decreases HRV and releases stress in undergraduate medical students[17]. May RW estimated that various biofeedback interventional pragrammes improve cognitive performance with increased HRV in undergraduate college students with burnout[18-20]. Grässler B also testified improvement in HRV after various execises[4]. Lais onello evidently illustrated that physical activity significantly reduces job related stress in terms of enhancement in parasympathetic indices of HRV[21].

Stress leads to increase in cortisol level as a body adaption to stress, but stress for prolong period can have a detrimental effect on the body[2]. Psychological stress dysregulates HPA axis leading to high levels of awakening cortisol[22]. Donovan AO et al showed reduced morning cortisol in nervous patients, an observation contrary to the present study's results[23]. A work of Wood **e**t al reported that reduction in cortisol level occur after walk of 3 km for 30 minutes among stressed individuals[24]**.**

Limitations: Single centre study with limited resources and time frame-work.

CONCLUSION

It was concluded that exercise can be employed to buffer the effect of psychological stress as it normalizes HPA axis and autonomic response.

Conflict of Interest: None to declare

Financial Disclosure: None

Authors' Contribution: GJ&HFK: Conceptualized the study, analyzed the data, and formulated the initial draft, **MI&AH:** Contributed to data collection, **SA&LS:** Proof read the final draft and analyzed data.

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