Original Article

Gender Differences in Autonomic Cardiovascular Functions During Cold Pressor Test Beforeand After Whole Body Heating

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Abstract

Introduction: Delicate balance of sympathetic and parasympathetic tone of the autonomic nervous system is important in aviation scenario. The military aviators are often encountered with various aviation stressors like +Gz forces, thermal stress, vibration stress, hypoxic stress and so on. These stressors try to jeopardise the autonomic balance of the aviators and make them vulnerable for autonomic cardiovascular instability. Systematic studies are not available where autonomic cardiovascular function of males and females have been compared under an autonomic neural function challenging task. Cold Pressor Test (CPT) is a recognized physiological technique to evaluate autonomic cardiovascular functions. It is important to comprehensively assess autonomic cardiovascular reactivity of males and females in an autonomic neural function challenging task to assess the autonomic balance of cardiovascular functions.

Methodology: Twenty healthy males and eighteen females participated in the study. The heart rate (HR) and blood pressure (BP) of the participants were measured during CPT before and after whole body heating. HR was measured by single lead electrocardiography (ECG) and BP was measured using an arterial tonometer on a beat to beat basis. Heart rate variability indices were computed from the ECG signal.

Results: Results showed that males had a higher sympathetic drive than females during pre-heat CPT (LF n.u. 62.5 in males vs. 50.5 in females and LF/HF ratio 2.54 in males vs. 1.27 in females). After heat stress, females had an augmented sympathetic drive as compared to pre-heat stress (LF n.u. 63.8 in males vs. 57.9 in females and LF/HF ratio 2.60 in males vs. 2.10 in females). Males and females had non-significant attenuation in blood pressure during post-heat CPT as compared to pre-heat CPT.

Conclusion: The study concluded that autonomic cardiovascular reactivity in females was mediated via sympathetic neural system after whole body heating.

Key words: Cold pressor test, gender differences, autonomic neural functioning, heart rate variability, baroreflex sensitivity, heat stress

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Introduction

Military aviation is highly challenging task and military aviators are often exposed to many stressors like hypoxia, +Gz acceleration, vibration and thermal stress. Maintenance of cardiovascular homeostasis is of prime importance in extremely challenging situation of aviation. Cardiovascular homeostasis is the net result of sympathetic and vagal tone of the autonomic neural system that ultimately play a critical role in the regulation of blood pressure (BP) and heart rate (HR) in the Military aircrew under stressful situation of aviation.

Women have made an entry to aviation arena and IAF is planning to induct women in combat role as fighter aircraft pilots.

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Scientific studies have indicated that there is a great deal of differences in physical and physiological features of women from men. The literature suggests that physiologic differences with regard to adaptation to +Gz stress among women and men exist. Women tend to lack adaptation with no increases in cardiac contractility or baroreceptor sensitivity resulting in greater effort with more strain versus men to sustain Gz loads [1]. Studies have also reported that autonomic cardiovascular function varies among males and females. Ramaekers et al in a study reported that sympathetic functions, as assessed from 24 hours of ECG, were significantly higher in males than females [2]. Study by Huikuri et al suggested that baroreflex sensitivity, an important determinant of autonomic cardiovascular activity, was higher in men than women [3]. No scientific study is available in the Indian perspective which has compared and comprehensively assessed the autonomic cardiovascular functions in males and females in an autonomic neural function challenging task. The knowledge of this specific response could improve the comprehension and help to strategize the combat role of two genders.

Cold Pressor Test (CPT) is one of the recognized physiological evaluation techniques to assess non-baroreflex mediated autonomic cardiovascular functions [4]. In this test, the individual immerses one of his hands into ice cold water (4-6°C) up to wrist for 1-6 min. Blood pressure (BP) and heart rate (HR) response to CPT is recorded continuously. In normal subjects, a vascular sympathetic response is increased during CPT resulting in increased peripheral resistance [5]. The HR response to CPT is variable and not homogenous for entire CPT period [6,7].

With the above background, we planned to conduct a study to assess and compare autonomic cardiovascular function in males and females during CPT. The study also wanted to investigate how exposure to whole body heating would have an impact on autonomic cardiovascular reactivity to CPT in both genders, as scientific study has suggested that heat stress attenuates the increase in arterial blood pressure during CPT [8]. The aim of the present study was to assess and compare autonomic cardiovascular reactivity to CPT in males and females before and after whole body heating.

Material and Methods

20 healthy males and 18 healthy females in the age range of 21-35 years were selected among post graduate trainees of the Institute as volunteers. They were examined clinically and ruled out that they were not suffering from any disease. They were explained about the protocol of the study in detail and possible consequences of CPT. Voluntary informed consent was obtained from each participant. The Ethics Committee of the Institute approved the study protocol.

Each participant reported to the laboratory at 0900 hrs. The HR of the participant was measured by single lead electrocardiography in physiological data recorder, ProcompInfiniti 5.0 (Thought Technology, Montreal, Canada). The BP of the participant was measured by arterial tonometer, Finometer Midi (Finapres Medical System, Netherland) on a beat to beat basis. The parameters were recorded for 5 min before CPT and for 3 min during CPT. All the measurements were carried out in sitting position. Recording of physiological parameters for 5 min before CPT served as pre-heat baseline reading.

The participant performed CPT under the controlled environment of the laboratory in sitting position. They dipped their left hand up to wrist into ice cold water at 4°C. CPT was carried out as per the protocol described by Mourot et al and Cui et al [5,7]. The temperature of the water was maintained at 4-5°C throughout the test.

After the CPT, the participant was exposed to simulated thermal environment of 40°C temperature and relative humidity of 40% in multi-seater environmental chamber. The thermal chamber was developed by Defsys Solutions Pvt Ltd, India. The exposure duration to simulated thermal condition was continued for 40 min. After exposure to heat, the physiological parameters of the participant were recorded for 3 min which served as post-heat baseline reading. The participant performed the CPT again for 3 min after whole body heating.

For recording of ECG, three electrodes were placed on skin of anterior surface of the chest, one at right shoulder, second at left shoulder and the third was placed at left iliac fossa. ECG was recorded at baseline and during CPT both before and after heat stress. During exposure to heat stress, ECG could not be captured due to technical limitation of the equipment. Recorded R-R interval of the ECG signal of baseline and CPT was subjected to analysis for calculation of time domain and frequency domain descriptors of heart rate variability (HRV). Kubios HRV analysis software, version 2.1, Finland, was used for analyzing the HRV data. The details of analysis of HRV have been described elsewhere [9].

Various time domain indices of HRV computed were mean HR (beats per minute), standard deviation of N-to-N intervals or RR intervals (SDNN in ms), root mean square of successive differences between adjacent NNs (RMSSD in ms), number of successive RR interval pairs that differ by more than 50 ms (NN50 count), NN50 divided by the total number of RR intervals (%) (pNN50). Various frequency domain indices of HRV computed were LF power (0.04-0.15 Hz-ms^2), LF power n.u. (Power of low frequency in normalized unit), HF power (0.15-0.4 Hz- ms^2), HF power n.u. (Power of high frequency expressed in normalized unit), total power (ms^2) (VLF+ LF+ HF) and LF/HF ratio.

Arterial blood pressure was measured on a beat to beat basis by arterial tonometer, Finometer Midi (Finapres Medical System, Amsterdam, Netherland). Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and pulse pressure (PP) were recorded at baseline and CPT. The principle of recording of continuous non-invasive BP from finger was based on volume clamp technology discovered by the Czech physiologist Jan Peňáz and Wessling et al [10]. In this technique, an appropriate sized finger cuff is wrapped around 2^{nd} phalanx of the middle finger of left hand of the participant. Beatscope software of Finapres arterial tonometer extrapolates the brachial arterial blood pressure from finger arterial pressure. Scientific studies have revealed that the finger arterial pressure measured by Finapres correlated well with the brachial arterial pressure in clinical as well as experimental settings [11].

Baroreflex sensitivity (BRS) is defined as change in inter-beat interval in milliseconds per unit change in SBP. BRS was computed by sequence method discovered by Bahjaoui-Bouhaddi et al [12]. The BRS calculation was carried out using NEVROKARD 9.0.0 BRS analysis software, Slovenia.

Other cardiovascular parameters recorded were stroke volume (SV), cardiac output (CO), left ventricular ejection time (LVET), total peripheral resistance (TPR), skin conductance (SC), skin temperature (Skin temp) and an indirect estimate of myocardial contractility (dp/dt in mmHg/s).

Data Reduction and Statistical Analysis

Statistical software, Statistica 6.0 was used to analyze the data. Data was first checked for normality by Shapiro Wilks 'W' statistic. Two factors repeated measure ANOVA was carried out for analysis. The First factor was 'heat stress' that had two levels i.e. before and after exposure to heat stress. Second factor was 'condition of testing' that again had four levels i.e. baseline before heat stress, pre-heat CPT, baseline after heat stress and post-heat CPT. The categorical factor was 'Gender'. After significant outcome of the ANOVA, Tukey HSD test was carried out for individual comparisons. Level of significance was kept at $p<0.05$.

Results

The age, height and weight of male and female participants in the present study were 27.6±4.35 years, 171.2±7.47 cm, 68.3±9.65 kg and 29.4±4.96 years, 158.8±5.84 cm, 58.5±8.53 kg respectively. The males were having significantly higher body height than females ($p<0.001$) and females were having significantly lower body weight than males ($p<0.01$).

Table 1 shows heart rate and blood pressure responses to CPT in males and females before and after exposure to whole body heating. HR did not show any significant difference between males and females during CPT either before or after whole body heating. SBP increased significantly during CPT from baseline in males and females before and after whole body heating ($p<0.001$; both before and after heating). DBP

increased significantly during CPT from baseline in males and females before and after heating ($p<0.001$, both before and after heating). Females had a significantly lower DBP than males during CPT before heating ($p<0.01$) and after heating ($p<0.05$). PP increased significantly in males ($p<0.001$) and in females ($p<0.001$) during CPT before heating and only in females ($p<0.001$) after heating. Females had a significantly higher PP during CPT than males before heating ($p<0.05$). MAP increased significantly during CPT in both males and females before and after heating ($p<0.001$; both before and after heating).

Table 2 shows central and peripheral hemodynamic responses to CPT in males and females before and after whole body heating. SV increased in females during CPT from baseline before heating ($p<0.001$) and in males and females during CPT from baseline after heating ($p<0.001$ in both cases). CO increased significantly during CPT from baseline in males and females before and after heating ($p<0.01$ in both the case). LVET increased significantly in males ($p<0.01$) and females ($p<0.001$) during pre-heat CPT from baseline and in males ($p<0.01$) and in females ($p<0.001$) during post-heat CPT from baseline. TPR did not show any significant changesin males and females during CPT from baseline either before or after heat or between males and females. SC increased significantly in females during pre-heat CPT from baseline ($p<0.05$). SC was significantly higher in males ($p<0.001$) and females ($p<0.001$) after heat during CPT. Sk temp decreased significantly in female during pre-heat CPT from baseline ($p<0.01$) and in males and females during post-heat CPT from baseline ($p<0.001$ in both cases). Sk temp was significantly higher in males ($p<0.001$) and females ($p<0.001$) during post-heat CPT as compared to pre-heat CPT. dp/dt increased significantly in males ($p<0.05$) and females ($p<0.01$) during pre-heat CPT and in males ($p<0.001$) and females ($p<0.001$) during post-heat CPT. Males had a significantly lower dp/dt during post-heat CPT than pre-heat CPT ($p<0.05$) and females also had a significantly lower dp/dt during post-heat CPT than pre-heat CPT ($p<0.05$).

Table 3 shows frequency domain indices of HRV in males and females at baseline and during CPT before and after heat stress. LF power reduced significantly in males during post-heat CPT from post-heat baseline ($p<0.05$). LF n.u. and HF n.u. were significantly higher and lower respectively in males than females during pre-heat CPT. HF power reduced significantly in males during post-heat CPT as compared to post-heat baseline ($p<0.05$). Total power reduced significantly in males ($p<0.01$) and increased non-significantly in females during post-heat CPT. LF/HF ratio was significantly higher in males than females ($p<0.01$) during CPT before heat stress. The ratio increased non-significantly in males and females during post-heat CPT as compared to pre-heat CPT. The increase was to a larger magnitude in females than males.

BL BH- Baseline value before heat; BL AH-Baseline value after heat. CPT BH- Cold pressor test before whole body heating; CPT AH-Cold pressor test after whole body heating. Table 4 shows time domain indices of HRV in males and females at baseline and during CPT before and after heat stress. RMSSD was significantly higher in females than males during pre-heat CPT ($p<0.01$). Fig 1 shows BRS of males and females during cold pressor test before and after heat stress.

Before heat, BRS was significantly lower in females than males during pre-heat CPT Male: 9.78 ms/ mm Hg vs. Females: 6.59 ms/ mm Hg) ($p<0.01$). BRS also reduced significantly in females during CPT from baseline (9.04 ms/mm Hg to 6.59 ms/mm Hg) ($p<0.05$).

After heat, BRS was also foundto be significantly lower in females than males at baseline ($p<0.01$) and during post-heat CPT (9.58 ms/mm Hg in male vs. 7.04 ms/ mm Hg in female) ($p<0.05$).

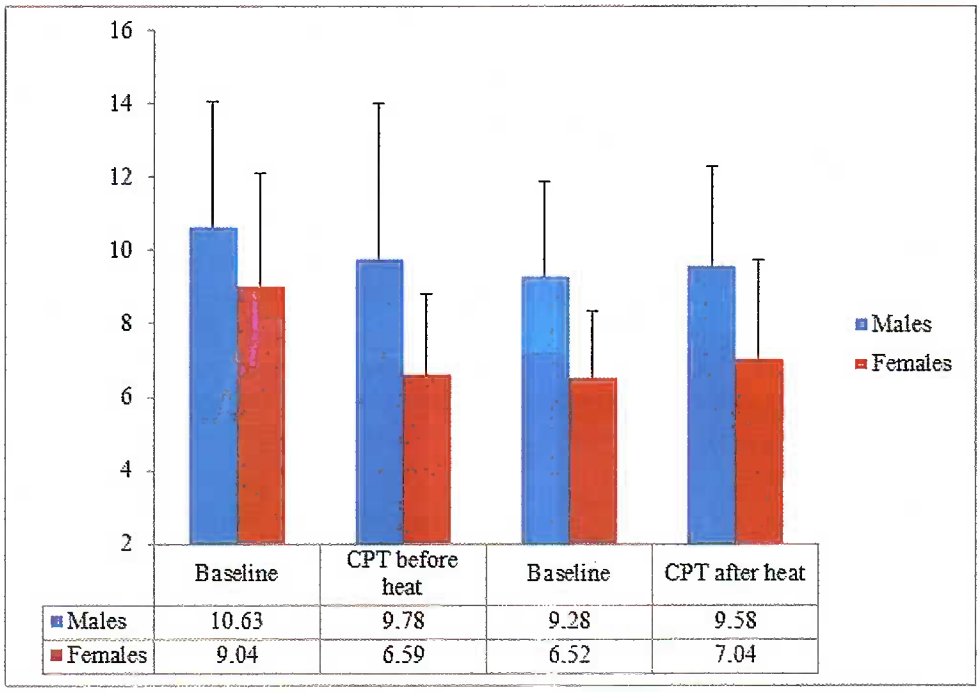

Fig 1. Baroreflex sensitivity (ms/mm Hg) to cold pressor test in males and females before and after heat stress. Values are mean± SD.

Table 1 – Cardiovascular responses to CPT in males (n=20) and females (n=18) before and after heat stress. Values are given in mean± SD.

Parameters	Before Heat Stress				After Heat Stress			
	Male		Female		Male		Female	
	Baseline	CPT	Baseline	CPT	Baseline	CPT	Baseline	CPT
Heart rate (bpm)	78.6 ± 10.11	83.6 ± 11.28	87.9 ±5.87	83.6 ± 10.35	83.1 ± 13.32	81.6 ± 9.41	85.3 ± 6.85	85.0 ± 10.89
Systolic blood pressure	110.6 ± 14.67	133.7 ± 19.61***	104.4 ± 13.66	129.3 ± 19.75***	114.1 ± 23.84	128.8 ± 20.2***	102.4 ± 9.00	125.6 ± 16.57***
Diastolic blood pressure	63.6 ± 13.82	80.6 ± 15.16***	54.8 ± 8.54	67.5 ± 10.50*** $$	65.7 ± 16.99	77.4 ± 12.96***	55.1 ± 6.64	67.8 ± 7.72***$
Pulse pressure	47.0 ± 6.95	53.1 ± 8.91***	49.6 ± 8.75	61.8 ± 13.11***$	48.5 ± 11.02	51.4 ± 10.84	47.3 ± 8.36	57.8 ± 10.68***
Mean arterial pressure	81.9 ± 17.43	102.4± 16.00***	75.6 ± 11.17	94.0 ± 12.89***	83.1± 18.60	98.9 ± 14.28***	75.6 ± 6.37	92.4 ± 10.35***

*** denotes significant difference from baseline;** p<0.01; *** p<0.001**
$ denotes significantly different value between males and females; $ p<0.05; $$ p<0.01.

Table 2. Hemodynamic responses to CPT in males (n=20) and females (n=18) before and after heat stress. Values are given in mean± SD.

	Before Heat Stress				After Heat Stress			
	Male		Female		Male		Female	
Parameters	Baseline	CPT	Baseline	CPT	Baseline	CPT	Baseline	CPT
Stroke volume (ml/beat)	89.0 ± 18.25	95.9 ± 26.23	81.7 ± 20.62	100.7 ± 24.42 ***	82.6 ± 19.01	94.5 ± 27.79 ***	74.7 ± 13.57	94.5 ± 17.82 ***
Cardiac output (L/min)	6.96 ± 1.11	7.77 ± 1.71 **	7.22 ± 1.88	8.20 ± 1.41 **	6.62 ± 1.52	7.51 ± 1.74 **	6.51 ± 1.32	7.98 ± 1.44 ***
LVET (ms)	279.0 ± 13.73	289.5 ± 13.77 **	288.3 ± 15.7	305.5 ± 22.06 ***	277.1 ± 12.15	287.0 ± 15.20 **	290.0 ± 14.25	304.7 ± 21.82 **
Total peripheralresistance (dyn.s/cm$^{5)}$	998.0 ± 395.45	1116.9± 424.73	893.3 ± 188.05	950.3 ± 162.41	1064.4 ± 408.04	1136.0 ± 483.33	976.3 ± 163.94	972.2 ± 195.29
Skin conductance (µS-micro-siemens)	1.34 ± 0.88	1.53 ± 1.16	1.61 ± 1.38	2.12 ± 1.28 *	2.22 ± 1.56	2.28 ± 1.83ΔΔΔ	2.99 ± 1.89	3.15 ± 1.89ΔΔΔ
Skin temperature (degree Celsius)	33.0 ± 2.19	32.9 ± 2.06	33.5 ± 1.93	32.9 ± 1.97 **	36.1 ± 0.82	34.9 ± 0.76 *** ΔΔΔ	35.9 ± 0.70	34.9 ± 0.87 *** ΔΔΔ
dp/dt (mmHg/s)	1137.9 ± 237.99	1289.4 ± 305.33*	1093.8 ± 277.29	1300.8 ± 422.73**	1069.1 ± 321.42	1151.0 ± 279.35 Δ	1042.3 ± 338.68	1108.5± 319.54 Δ

*** denotes significant difference from baseline;** p<0.01; *** p<0.001**
$ denotes significantly different value between males and females; $ p<0.05; $$ p<0.01.

Table 3 – Frequency domain indices of heart rate variability to CPT in males (n=20) and females (n=18) before and after heat stress. Values are given in mean± SD.

Parameters	Conditions	Male Mean ± SD	Female Mean ± SD	Significance level (p-value) ^
Low Frequency Component (LF) (ms^2)	BL BH	1073.6 ± 967.5	856.6 ± 569.7	0.487 (NS)
	CPT BH	1516.7 ± 1887.7	833.0 ± 698.0	0.240 (NS)
	BL AH	**1815.6 ± 1439.3 ***	**889.3 ± 807.0**	**0.050 (S)**
	CPT AH	1046.8 ± 1030.8 +	1185.9 ± 1293.7	0.743 (NS)
Low Frequency Component (LF) (n.u.)	BL BH	62.0 ± 15.02	56.4 ± 19.14	0.358 (NS)
	CPT BH	62.5 ± 20.74	**50.5 ± 16.46**	**0.049 (S)**
	BL AH	58.0 ± 20.73	55.4 ± 15.96	0.711 (NS)
	CPT AH	63.8 ± 19.39	57.9 ± 22.62	0.443 (NS)
High Frequency Component (HF) (ms^2)	BL BH	690.6 ± 202.34	616.3 ± 254.6	0.797 (NS)
	CPT BH	721.7 ± 159.83	1006.3 ± 198.7	0.99 (NS)
	BL AH	1099.8 ± 354.25	701.6 ± 251.36	0.98 (NS)
	CPT AH	555.4 ± 205.46 +	847.8 ± 654.21	0.99 (NS)
High Frequency Component (HF) (n.u.)	BL BH	37.8 ± 14.92	42.0 ± 19.43	0.497 (NS)
	CPT BH	**36.9 ± 20.76**	**47.7 ± 16.48**	**0.05 (S)**
	BL AH	41.5 ± 20.47	42.3 ± 16.52 ·	0.920 (NS)
	CPT AH	37.8 ± 21.17	40.0 ± 22.69	0.785 (NS)
Total Power (ms^2)	BL BH	1912.8 ± 523.45	1611.2 ± 324.78	0.603 (NS)
	CPT BH	2363.6 ± 459.86	1939.7 ± 489.36	0.588 (NS)
	BL AH	**3168.6 ± 567.89 ** **	**1730.6 ± 958.45**	**0.05 (S)**
	CPT AH	1737.2 ± 499.21 ++	2173.1 ± 789.45	0.451 (NS)
LF/ HF ratio	BL BH	2.03 ± 0.958	1.83 ± 0.547	0.667 (NS)
	CPT BH	**2.54 ± 0.654**	**1.27 ± 0.568**	**0.010 (MS)**
	BL AH	2.36 ± 0.478	1.52 ± 0.847	0.199 (NS)
	CPT AH	2.60 ± 0.659	2.10 ± 0.458	0.442 (NS)

BL BH- Baseline value before heat; BL AH- Baseline value after heat. CPT BH- Cold pressor test before whole body heating; CPT AH- Cold pressor test after whole body heating

^ Intergroup comparison; Unpaired t-test; $p<0.05$: S (significant); $p<0.01$ MS (moderately significant); $p<0.001$: HS (highly significant) and $p>0.05$: NS (not significant)

*Intragroup comparison; significantly different form pre-heat baseline, * $p<0.05$; ** $p<0.01$;

+ Intragroup comparison; significantly different from post-heat baseline, + $p<0.05$; ++ $p<0.01$

Table 4 – Time domain indices of heart rate variability to CPT in males (n=20) and females (n=18) before and after heat stress. Values are given in mean± SD.

Parameters	Conditions	Male Mean ± SD	Female Mean ± SD	Significance level (p-value) ^
SDNN (ms)	BL BH	46.1 ± 14.28	42.9 ± 14.93	0.544 (NS)
	CPT BH	51.6 ± 20.19	58.8 ± 18.97	0.597 (NS)
	BL AH	58.9 ± 24.07	54.5 ± 22.78	0.612 (NS)
	CPT AH	43.5 ± 15.08	45.2 16.54	0.775 (NS)
RMSSD (ms)	BL BH	37.4 ± 15.13	42.2 ± 21.36	0.459 (NS)
	CPT BH	44.3 ± 17.93	66.2 ± 20.93 **	0.233 (NS)
	BL AH	52.7 ± 32.96 *	50.6 ± 31.40	0.863 (NS)
	CPT AH	37.9 ± 17.57	41.0 ± 13.94	0.607 (NS)
NN50 (count)	BL BH	12.3 ± 8.65	12.5 ± 8.12	0.949 (NS)
	CPT BH	17.9 ± 7.25	19.8 ± 7.58	0.786 (NS)
	BL AH	14.2 ± 8.67	14.6 ± 5.69	0.903 (NS)
	CPT AH	16.1 ± 5.89	14.3 ± 5.45	0.776 (NS)
pNN50 (%)	BL BH	15.5 ± 10.42	14.7 ± 9.68	0.833 (NS)
	CPT BH	20.0 ± 13.75	23.6 ± 7.42	0.541 (NS)
	BL AH	20.0 ± 10.35	18.8 ± 7.87	0.772 (NS)
	CPT AH	18.0 ± 7.89	19.1 ± 5.55	0.841 (NS)

BL BH- Baseline value before heat; BL AH- Baseline value after heat. CPT BH- Cold pressor test before whole body heating; CPT AH- Cold pressor test after whole body heating

^ Intergroup comparison; Unpaired t-test; $p \leq 0.05$: S (significant); $p < 0.01$ MS (moderately significant); $p < 0.001$: HS (highly significant) and $p > 0.05$: NS (not significant)

* Intragroup comparison; significantly different form pre-heat baseline, * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

+ Intragroup comparison; significantly different from post-heat baseline, + $p < 0.05$; ++ $p < 0.01$; +++ $p < 0.001$

Discussion

The present study examined the autonomic cardiovascular functions in males and females during CPT before and after exposure to whole body heating. The primary finding of the present study was that males showed a non-significant increase in HR during pre-heat CPT, contrasting to females who did not show an increase in HR. Increase in HR during hand immersion in ice cold water has been reported in a group of male students [13] and in a group of male and female participants [14]. Study has also documented that HR response to CPT varies among males and females [15]. A biphasic alteration in HR during CPT, with an initial increase followed by a slow decrease, has been observed by many researchers [16-18]. Studies have reported that autonomic modulation of HR during CPT is caused by decreased cardiac vagal outflow along with concomitant involvement of sympathetic activity [19-21]. It has also been reported in the literature, that an increase in vagal activity induced by baroreceptor activation may occur during CPT [22-23]. Heart rate variability indices also indicated that males had heightened sympathetic activity along withreduced vagal drive during pre-heat CPT. Higher sympathetic drive in males has been reflected by comparatively higher LF n.u. in them as compared to females. Lower vagal drive has been reflected by comparatively lower HF n.u. in males than females.

Non-significant increase in LF n.u. in females during post-heat CPT as compared pre-heat CPT is perhaps suggestive of sympathodominance in females after heat stress contrasting to vagal dominance before heat. Increase in LF/HF ratio, though non-significant, during post-heat CPT in females further supports this sympathodominance.

Root mean square of successive differences (RMSSD), a time domain indices of HRV, has been reported to be associated with vagal mediated control of heart rate [24]. A significant increase in RMSSD during pre-heat CPT in females suggests further that control of heart rate in females before heat stress is mediated via vagal system. A reduction in RMSSD in females during post-heat CPT is suggestive of reduced vagal input. SDNN, standard deviation of all NN intervals, reflects all the cyclic components responsible for variability of heart rate in the period of recording [9]. Increased SDNN is considered as hallmarks of parasympathetic prevalence [25]. Relatively higher and lower value of SDNN in females than males during pre-heat and post-heat CPT respectively suggests that autonomic control of heart function in female was mediated via vagal system before heat stress and was shifted towards sympathetic side after heat stress.

CPT causes an immediate localand generalized vasoconstriction of peripheral vasculature due to enhanced vascular sympathetic response. Increased sympathetic response during CPT is not only due to a direct effect of cold on skin blood vessels, but also due to stimulation of pain receptor which activates spinal and hypothalamic reflexes [5,7,13,26].Increased sympathetic activity during CPT is independent of baroreflex mechanism [7,8]. Lower sympathetic drive in females during pre-heat CPT is perhaps due to less intense stimulation of pain receptor before heat stress contrasting to greater stimulation of pain receptor in males.

Sympathetic innervation to peripheral vasculature is reflected in peripheral resistance and diastolic blood pressure is a surrogate measure of peripheral resistance. The comparatively lower DBP in females than males during pre-heat CPT is probably an indication of reduced sympathetic neural drive. After heat stress, the slightly attenuated rise in DBP in males and no change in DBP in females suggest that sympathetic neural drive in females remainedsimilar or rather became higher after heat stress. The blood pressure response to CPT after heat stress becomes slightly less elevated than before heat stress. Cui et al observed in a study that whole body heating causes an attenuation in MAP during CPT [27]. They observed that under normothermic condition, MAP increased by 28 mm Hg during CPT and after whole body heating the increase in MAP was

attenuated to 18 mm Hg during CPT. This is apparent in the present study. The average increase in MAP during pre-heat CPT was 18-20 mm Hg and that after heat stress the increase in MAP was attenuated to 14-16 mm Hg. Also, noted in the present study the increase in MAP in females was negligibly higher than males. The reason for attenuated rise in blood pressure during post-heat CPT may be explained by the fact that whole body heating causes an increase in the skin blood flow by about 5 times and 50% of the cardiac output is channelized to the skin vasculature during heat stress [28]. The decrease in cardiac output coupled with an attenuated sympathetic activity to the skin vasculature during heat stress was believed to be the reason for attenuated blood pressure response to CPT [8]. In the setting of blood being drawn into intensely dilated skin vasculature during heat stress, it is likely that when autonomic cardiovascular system is challenged with CPT, the sympathetic neural drive mediated vasoconstriction gets blunted. Despite the fact, sympathetic neural drive was lesser in females during pre-heat CPT,it was augmented during post-heat stress.

Electrodermal activity has been closely linked with sympathetic activity and is used in psychophysiological setting to examine sympathetic arousal. It is the only autonomic psychophysiological variable that purely depends on the sympathetic input and is independent of parasympathetic influence [29]. Skin conductance, a measure of electrodermal activity, increased significantly in females during post-heat CPT when compared to pre-heat CPT. The baseline skin conductance was also significantly higherin female after heat stress. This suggested that sympathetic neural drive was augmented in females post heat stress. Slightly increased TPR in females during post-heat CPT as compared to pre-heat CPT reflects that autonomic function in females is influenced by sympathetic neural input after heat stress. TPR indicates the vasoconstriction of peripheral vasculature and is an estimate of sympathetic neural activity.

BRS predominantly measures the efficacy of cardiac parasympathetic regulation [30] BRS did not show any significant difference between pre-heat and post-heat values either in males or femalesand between males and females either before or after heat stress during CPT. Potts et al reported that carotid-cardiac baroreflex responsiveness remains unaltered after heat stress due to increased arterial compliance [31]. This is in accord with the findings of the present study.

Conclusion

The present study examined the autonomic cardiovascular functionin a group of males and females during cold pressor test before and after heat stress. Results revealed that before heat stress, males had higher low frequency spectral power & higher LF/HF ratio and females had higher high frequency spectral power and lower LF/HF ratio. This suggests that autonomic function in males and females were mediated via sympathetic and vagal system respectively before heat stress. After heat stress, males had similar low frequency power and LF/HF ratio whereas females had higher LF power and LF/HF ratio. This suggests that autonomic function in females was switched over from vagal to sympathetic side after heat stress. Baroreflex sensitivity remained almost similar in males and females after heat stress. Females had higher skin conductance, total peripheral resistance and skin temperature during post-heat CPT as compared to pre-heat CPT. These hemodynamic responses to CPT were suggestive of sympathodominance in females after heat stress.

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