Original Article

Effect of obesity on cardiovascular responses to submaximal treadmill exercise in adult males

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Abstract

Context: Obesity is a major risk factor for chronic diseases. Abnormal changes in cardiovascular responses to exercise indicate the alteration in autonomic activity in obese. **Aims:** To assess and compare the cardiovascular parameters before and after exercise among obese and nonobese adult males. **Subjects and Methods:** Thirty each obese and normal-weight males between the age group 18-45 years were selected as cases and controls, respectively. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), corrected QT intervals were measured before and after submaximal treadmill exercise (QT and QTc) were done according to Bruce protocol. HR, SBP, DBP, MAP, QT, and QTc were also measured during passive recovery at 1 min and 5 min after exercise. **Statistical Analysis Used:** One-way analysis of variance and *t*-test were used to assess changes before and after exercise. **Results:** Resting HR was significantly higher in obese when compared to nonobese ($P < 0.05$). SBP, DBP, MAP, QT, and QTc were significantly higher in obese when compared to nonobese ($P < 0.001$). Immediately after exercise HR, SBP, DBP, MAP, QT, and QTc were significantly higher in obese when compared to nonobese ($P < 0.001$) 1 min after exercise. **Conclusions:** Obese individuals had elevated resting cardiovascular parameters and showed increased responses to steady exercise which could be due to alteration in autonomic functions with sympathetic hyperactivity. Delayed rate of decrease in HR and BP after exercise was also observed in obese, which indicates that they are at risk of developing cardiovascular diseases.

Keywords: Electrocardiogram, obesity, QT interval, treadmill exercise

Introduction

Obesity being a metabolic problem, its prevalence continues to increase and is reaching almost epidemic proportions. According to the World Health Organization (WHO) global estimates from 2018, more than 1.4 billion adults, 20 and older, are overweight. Among overweight adults, over 200 million men and nearly 300 million women are obese.[1] During the last 2 decades, obesity has become a severe epidemic in India, especially in the male population.[2]

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Being overweight and obese confer an increased risk of adverse consequences including cardiovascular disease (CVD) risk regardless of metabolic status. It has also been suggested that metabolically healthy obesity increases the risk of developing heart failure in individuals free of CVDs.[3] Previous studies have demonstrated that obesity contributes to lipid-storing tissues which constitute metabolically active and inflammatory tissues secreting cytokines and chemokines that affect cardiac morphology and function preliminarily resulting in decreased diastolic function in men with obesity.[4-6] Long-term obesity can induce structural and functional abnormalities in the left ventricle. Thus, it is vital to identify the effects of obesity on cardiac measures at earliest for predicting the risk of comorbidities.[7]

Obesity does not preclude fitness, and large cohort studies support the benefit of physical activity and fitness in lessening

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the risk of cardiovascular and all-cause mortality in obese adults.[8] Understanding the mechanisms that drive the altered hemodynamic responses during and after exercise in obese individuals is vitally important. A delayed decrease in the heart rate during the first minute after graded exercise is a powerful predictor of overall mortality.[9] An exaggerated blood pressure response during an acute dynamic exercise bout has been considered as an indicator of cardiovascular risk.[10] The QT interval is an element of the electrocardiograph (ECG) tracing that is representative of the ventricular function including ventricular depolarization and repolarization. Measurement of QT interval and QTc (Corrected QT) can be used as a simple indicator of CVDs.[11] However, it's unsure whether or not, similar relationships exist in Asian populations, as they generally have a lower body mass index (BMI) and a higher percentage of body fat at a given BMI compared to individuals from Western countries. Moreover, Asians have a higher risk of cardiovascular events than individuals in Western populations at the same BMI range.[12]

Investigations regarding cardiovascular responses to exercise have mainly been directed towards clinical application in diagnosing cardiovascular abnormalities. Hence, cardiovascular responses to exercise in obese have to be understood precisely.[13,14] Exercise is a kind of stress and to some degree, an important activity in day-to-day life. Depending on the type of physical exercise carried out, its influence on heart rate and blood pressure response varies. As per earlier studies, dynamic exercise testing provides much more clinical information about the sympathetic activity.[15-17] Yet it is not done frequently because of potential risk. If monitored properly, dynamic exercise testing poses a low risk to the test subjects. As the population is becoming progressively more overweight and obese, there is a need to evaluate cardiovascular health at rest and responses during and after exercise in normal and obese adults. This information may assist in the early identification of individuals at risk of disease, provide guide more accurately designed exercise prescriptions, and help evaluate the effectiveness of treatment interventions. Although the literature search has revealed a majority of studies have examined cardiovascular responses following exercise; however, the influence of submaximal work on cardiovascular among obese men is still inconsistent and not documented in the Indian population. Hence, this study was undertaken to assess and compare the cardiovascular responses to submaximal treadmill exercise and compare the changes during the recovery period after exercise among obese and nonobese adult males.

Subjects and Methods

This study was undertaken in among healthy adult men aged 18-45 years recruited into two groups based on their BMI, as nonobese (18.5< BMI <25 kg/m^2) and obese (BMI >30 kg/m^2). A thorough history of existing medical conditions and medications taken in the past 6 months was obtained followed by clinical and systemic examinations. Subjects with established cardiopulmonary diseases, unstable coronary syndromes, and on vigorous athletic training exercises were excluded. The sample required was estimated using the formula[18] $n = (Za/2)^2 s^2/d^2$, the pilot study conducted on five healthy young adults, yielded a standard deviation of 4.7 at a confidence interval of 95% and an error rate of 5% resulted in the minimum sample size required to be 27 subjects; however, after the allowance of additional 10%, the corrected final sample size was estimated to be 30 subjects. However, a total of 60 (30 obese and 30 nonobese) male participants were included in the study after explaining the purpose and procedures in detail and obtaining written informed consent from each participant. This study complied with the Declaration of Helsinki and protocol was approved by the institutional review board.

Anthropometrical measurements

Anthropometric measures were obtained in the physiology lab after 30 min of physical rest. Weight was measured in light clothing without footwear on a spring weighing balance to the nearest 0.5 kg. Height was measured in centimeters (cm) barefoot against a wall with the help of a measuring tape to the nearest 0.2 cm. Measurement was done with heels close to the wall and feet close together so that weight was equally distributed, and the head in Frankfort's plane. BMI was derived from Quetelet's index.[19]

Heart rate (HR) & QT interval measurements

Skin preparation, electrode placement, and related protocols for obese and non-obese participants were similar to the established guidelines. Volunteers were assuming the supine position. Vital parameters like resting heart rate (HR), blood pressure (BP) were recorded. ECG was acquired in a room with a comfortable temperature, (22 to 25°C) at the speed of 25 mm/sec with a gain of 10 mm/mV *before and after the treadmill exercise* using computerized ECG system with Physiograph- three channels and MLU 268/8 Lab chart software on a Microsoft window based computer. Parameters analyzed included the mean HR and QT variables *viz*. QT, QTc intervals, QTd. Resting 12 lead ECG. Uncorrected QT interval, Corrected QT interval, QT dispersion were calculated from 12 lead ECG. Uncorrected QT interval was calculated as the beginning of Q wave to the end of T wave i.e., reaching of T wave to the isoelectric line. Corrected QT interval (QTc) was calculated by Bezett's formula (for baseline HRs) and QTc = QT/√RR.

Treadmill exercise test

The treadmill was pre-programmed with the speed of first two stages of Bruce protocol for submaximal treadmill exercise, i.e., a total of 6 mins duration, initial 3 mins speed of the treadmill is 2.74 km/hr or 1.7 mph with 10% grade and next 3 mins speed will be 4.02 km/h or 2.5 mph with 12% grade. The exercise was performed as per the standard protocol in a well equipped and ventilated room in the presence of a trained physician. Both obese and nonobese participants were instructed not to consume beverages and not to eat a heavy meal or participate in any vigorous activity 24 hours before the test and were properly acquainted with the experimental protocol. *Post exercise*, the BP, HR, QT, QTc were measured at termination of

exercise (immediate), passive recovery at 1 min and 5 mins after exercise among obese and nonobese study participants.

Statistical analysis

Statistical Package for the Social Sciences (SPSS) software version 19.0 (SPSS Inc., IBM Corporation, Chicago, IL, USA) was used for analysis. T test and analysis of variance (ANOVA) were used to compare between any dependent variables for significant differences. $P < 0.05$ was considered significant.

Results

The results obtained were expressed as mean ± standard deviation. On analysis of anthropometric parameters of 30 nonobese subjects, the mean age was 30.5 ± 6.14 years; the mean weight was 64.1 ± 6.04 kg; mean height was 12.7 ± 4.23 m; mean BMI was 22.9 ± 1.57 kg/m^2 [Table 1, Figure 1]. The obese subjects were of the mean age of 33.6 ± 5.48 years; the mean weight was 84.7 ± 6.95 kg; mean height was 1.6 ± 0.06 m, and mean BMI was 32.5 ± 2.16 kg/m^2 [Table 1 and Figure 1].

Pre Exercise – Baseline Values

Heart rate

The mean HR (bpm) in nonobese was 75.5 ± 5.53 and in obese was 78.1 ± 4.15. The higher mean HR in obese as compared to nonobese was statistically significant ($P < 0.05$) [Table 1 and Figure 1].

Blood pressure

The mean systolic blood pressure (SBP) in nonobese was 121.4 ± 4.33 mm Hg and in obese was 126.6 ± 3.56 mm Hg. The higher mean SBP in obese was statistically significantly high ($P < 0.001$). The mean diastolic blood pressure (DBP) in nonobese was 79.2 ± 2.99 mm Hg and in obese was 82.6 ± 2.97 mm Hg. The higher mean DBP in obese as compared to nonobese was of high statistical significance ($P < 0.001$). The mean arterial pressure (MAP) in nonobese (93.2 ± 2.85 mm Hg) was significantly less when compared to obese (97.2 ± 2.87 mm Hg). ($P < 0.001$) [Table 1 and Figure 1].

QT interval and QTc

The mean QT interval (ms) in nonobese and obese was 388.5 ± 28.5 ms and 410.8 ± 6.95 ms, respectively. The mean QTc interval (ms) in obese was significantly higher as compared to nonobese ($P < 0.001$) as shown in Table 1.

After Exercise

Heart rate

The mean HR immediately after exercise in non-obese was 134.30 ± 4.95 and in obese was 142.38 ± 4.25, 1 min after exercise in nonobese was 97.65 ± 4.6 and in obese was 108.31 ± 4.56, the 5 min after exercise HR in obese and in nonobese were 77.23 ± 4.18 and 74.91 ± 5.43, respectively. The higher mean HR in obese as compared to nonobese immediately after exercise, 1 min after exercise, and 5 min after exercise was statistically highly significant ($P < 0.001$) [Table 2 and 3, Figure 1].

Blood pressure

The mean SBP, DBP and MAP immediately after exercise, 1 min after exercise and 5 min after exercise in obese and nonobese subjects were as shown in Table 2. The higher mean SBP in obese as compared to nonobese immediately after exercise and 1 min after exercise was statistically highly significant ($P < 0.001$). The higher mean DBP and mean MAP in obese as compared to nonobese immediately after exercise, 1 min after exercise, and 5 min after exercise were statistically significant ($P < 0.0.001$) [Table 2 and 3, Figure 1].

Table 1: Anthropometric measures and Baseline/ Pre-exercise cardiovascular measures in obese and nonobese participants

	OBESE	NON- OBESE	95% CI of the Difference		Sig.* (*P*)
	Mean±SD	Mean±SD	Lower	Upper	
Age	33.6±5.48	30.5±6.14	.1559	6.1774	0.040
Weight	84.7±6.95	64.1±6.04	17.233	23.966	0.000
Height	1.6±0.06	12.7±4.23	-26.646	4.2995	0.154
BMI	32.5±2.16	22.9±1.57	8.6246	10.582	0.000
HR	78.1±4.15	75.5±5.53	.0393	5.0939	0.047
SBP	126.6±3.56	121.4±4.33	3.1478	7.2521	0.000
DBP	82.6±2.97	79.2±2.99	1.8554	4.9445	0.000
MAP	97.2±2.87	93.2±2.85	2.5633	5.5246	0.000
QT	410.8±6.95	388.5±28.5	11.550	33.049	0.000
QTc	438.8±2.82	426.3±18.3	5.6489	19.217	0.001

Sig. =Significant at $P \leq 0.01$; * unpaired *t*-test HR: heart rate; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure; SD: Standard deviation

Table 2: Post-exercise cardiovascular measures in obese and nonobese participants

	OBESE	NONOBESE	95% CI of the Difference		Sig. * (*P*)
	Mean±SD	Mean±SD	Lower	Upper	
HR_immediate	144.0±4.2	135.7±5.0	5.9537	10.779	0.000
SBP_immediate	157.1±4.3	150.2±4.4	4.6537	9.2128	0.000
DBP_immediate	76.9±2.95	74.8±2.6	.6799	3.5867	0.005
MAP_immediate	103.6±2.8	99.9±2.5	2.2911	5.0841	0.000
QT_immediate	401.3±6.8	379.8±25.9	11.691	31.308	0.000
QTc_immediate	429.6±3.0	417.1±18.3	5.7185	19.281	0.000
HR_1 min	110.1±4.2	98.9±4.4	8.9483	13.451	0.000
SBP_1 min	151.2±4.5	144.3±4.5	4.5621	9.3042	0.000
DBP_1 min	75.8±3.3	72.8±2.7	1.3616	4.5049	0.000
MAP_1 min	100.9±3.0	96.6±2.6	2.7987	5.7331	0.000
QT_1 min	406.4±6.6	384.9±26.3	11.543	31.383	0.000
QTc_1 min	435.8±3.4	422.9±18.5	6.0508	19.876	0.000
HR_5 min	79.0±3.95	76.4±5.41	.1591	5.0490	0.038
SBP_5 min	126.5±3.4	121.4±4.5	2.9873	7.1506	0.000
DBP_5 min	82.5±2.6	79.1±2.9	1.9569	4.8443	0.000
MAP_5 min	97.1±2.5	93.2±2.8	2.5581	5.3458	0.000
QT_5 min	410.9±7.0	388.1±28.4	12.063	33.469	0.000
QTc_5 min	438.8±2.8	425.8±17.9	6.3553	19.644	0.000

Sig.=Significant at $P \leq 0.01$; *unpaired *t*-test HR: Heart rate; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure

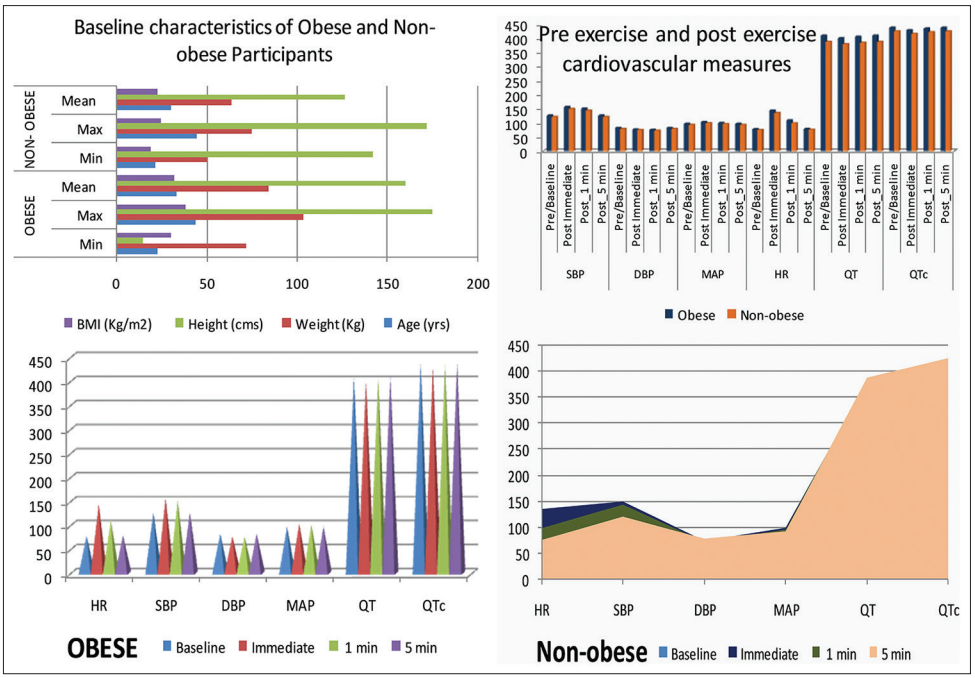

Figure 1: Cardiovascular measures before and after exercise among obese and nonobese adult males

QT interval and QTc

The mean QT (ms) immediately after exercise in nonobese was 379.8 ± 25.9 and in obese was 401.3 ± 6.84, 1 min after exercise in nonobese was 383.51 ± 25.8 and in obese was 401.68 ± 12.47; 5 min after exercise in nonobese was 384.9 ± 26.3 and in obese was 406.4 ± 6.61. The higher mean QT and QTc in obese as compared to nonobese immediately after exercise, 1 min after exercise, and 5 min after exercise were statistically highly significant ($P < 0.001$) [Table 2, Table 3 and Figure 1].

The distribution of HR, QT, and QTc dispersion immediately after termination of submaximal treadmill exercise among obese and non-obese participants is shown in Figure 2. However, in the linear regression analyses, cardiovascular responses such as HR, QT, and QTc dispersion showed a significant relationship with BMI in obese men [Table 4].

Discussion

Obesity is related to numerous cardiac complications like coronary heart disease, heart failure, and sudden death through its impact on the cardiovascular system. Deviations from the normal changes in cardiovascular parameters after exercise are indicators of cardiovascular risk.[9] In this study, the effects of obesity on cardiorespiratory responses to submaximal treadmill exercise in adults were assessed. A treadmill exercise test was performed among obese and nonobese subjects between the age group of 18 to 45 years. The comparison of the mean value of each parameter between nonobese subjects and obese subjects were analyzed and discussed.

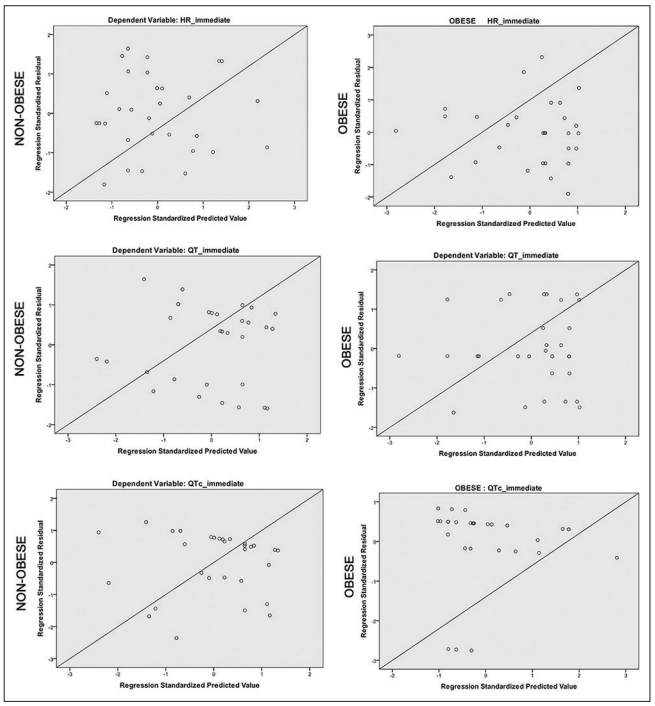

Figure 2: Linear regression analyses relationship between the BMI and HR, QT and QTc interval immediately after exercise in non-obese and obese men

Heart rate

In our study baseline HR in Obese subjects was in the normal range. However, it was significantly higher in obese when compared to that of nonobese. Immediately after exercise and 1 min after exercise, HR was significantly higher in obese than in nonobese. Similar findings were found in previous studies.[20-24] The HR recovery after exercise is a predictor of

Table 3: Comparison of cardiovascular responses before and after exercise among obese and nonobese adult males

OBESE	Pre exercise	After exercise			Sig (*t*- test) †	*P* (Anova)
	Baseline	Immediate	1 min	5 min		
Heart Rate	78.1±4.15	144.0±4.2	110.1±4.24	79.0±3.95	.000* A .000* B .000* C	0.000*
QT	410.8 ±6.95	401.3±6.84	406.4±6.61	410.9±7.07	.000* A .000* B .326‡ C	0.000*
QTc	438.8 ±2.82	429.6±3.06	435.8±3.48	438.8±2.82	.000* A .000* B .326‡ C	0.000*
NONOBESE						
Heart Rate	75.5±5.53	135.7±5.09	98.9±4.46	76.4±5.41	000* A .000* B .001* C	0.000*
QT	388.5±28.5	379.8±25.9	384.9±26.3	388.1±28.4	.000* A .000* B .136‡ C	0.588‡
QTc	426.3±18.3	417.1±18.3	422.9±18.5	425.8±17.9	000* A .000* B .036‡ C	0.192‡

*Highly significant; ‡Not significant. †Paired *t*- test: A- Baseline versus immediate after exercise; B - Baseline versus 1 min after exercise; C - Baseline versus 5 min after exercise.

Table 4: Linear regression analyses coefficients for HR, QT, QTC and BMI (Independent variable) for obese and non-obese men at immediate termination of submaximal treadmill exercise

BMI	Nonobese		Obese	
	β	*P*	β	*P*
HR _immediate	-0.062	0.745	0.921	<0.001
QT _immediate	0.305	0.101	0.693	<0.001
QTC _immediate	0.293	0.116	0.892	<0.001

BMI: Body mass index; HR: Heart rate

cardiovascular risk and cardiovascular mortality. The higher baseline HR and higher HR after exercise in obese is due to high sympathetic activity. 1 min after exercise HR was higher in obese when compared to nonobese suggests attenuated recovery of HR, which indicates that the obese individuals are at risk of cardiovascular mortality.[25] Reduced post-exercise baroreceptor sensitivity and impaired autonomic regulation have been associated with attenuated HR recovery in obese.[26,27] At 5 mins after exercise, the HR was almost recovered to baseline HR both in obese and nonobese.

Blood pressure

Sympathetic nervous and renin-angiotensin-aldosterone system activation appears to play a crucial role in sodium and water retention, rightward shift in the pressure-natriuresis, and BP elevation observed in obese individuals.[28] In this study, the mean resting SBP, DBP, MAP in obese was significantly higher as compared to that of nonobese, which was in line with the findings of Martin *et al.*[20], Roopa *et al.*[21] and Chrysohoou *et al.*[23] SBP, DBP, MAP immediately after exercise, 1 min after exercise was higher in obese as compared to that of nonobese; however, 5 mins after exercise SBP and DBP recovered to baseline. The higher baseline SBP and DBP is possibly due to high sympathetic nervous system activity. It is evident from the previous research that the higher SBP and DBP after exercise in obese, when compared to nonobese is due to the alteration in the arterial baroreflex, which can be linked to the sympathetic nervous system hyperactivity and blunted arterial distensibility leading to reduced post-exercise vasodilatation.[29] The exact mechanism for the reduced post-exercise vasodilatation and delayed decline in BP is not clear. Impaired potassium channel-mediated vasodilatation has been reported in skeletal muscle of obese Zucker rats and obese hamsters.[30,31] This could be partially involved in humans.

Significant higher BP responses 1 min after exercise in obese when compared to nonobese is suggestive of delayed SBP recovery. SBP recovery after exercise represents an important index of cardiovascular and autonomic nervous systems' response to physical stress, which is the clinical tool applied towards diagnosing cardiovascular abnormalities.[7] This suggests that obese individuals are at higher risk of cardiovascular mortality as compared to nonobese.

QT interval and QTc

In our study, the mean QT interval and QTc in obese were higher in obese when compared to that of non-obese which was statistically highly significant. These findings were similar to observations of Curione *et al.*,[32] Eposito *et al.*,[33] and Waheed S *et al.*[34] Higher QT interval and QTc in obese when compared to non-obese indicates an association of delayed cardiac repolarization and obesity. This is suggestive of autonomic nervous system dysfunction due to sympathetic overactivity leading to an alteration in cardiac repolarization times in obese individuals.[32,35] The prolongation of cardiac

repolarization times might contribute to their raised cardiovascular risk.

Obesity-related problems are common in family medicine practice. In clinical practice, it should be noted that obesity is an arrhythmogenic substrate as it prolongs the QT interval. The variation in QT interval is because of repolarization inhomogeneity and withdrawal of parasympathetic influence of HR. There is a consensus in primary care practice that to reduce the associated cardiovascular risk burden among patients and society efforts to *prevent and treat obesity* are most needed. In this regard, our study findings can serve as important implications for the primary care physicians to carry out a targeted prognostic ECG testing in obese individuals. The findings can be used in clinical practice to encourage the obese and their family to have a healthy lifestyle and also to improve motivation among otherwise healthy obese adults who are not ready to adopt lifestyle changes and necessary measure in control and treatment of obesity, thus preventing the associated long term adverse cardiovascular effects among them. The study findings might also be of significance in both making individual medical treatment and longitudinal follow-up after therapy in severe obesity.

The current study examined the effect of obesity on cardiovascular responses among adult obese men in comparison with nonobese adults. BMI is a global measurement and hence was taken as a parameter of choice for determining obesity. This study also tested the obese and nonobese for cardiovascular responses to physical activity, which usually is used as a prognostic factor in patients with coronary heart disease. A significant difference and association between pre-exercise and post-exercise cardiovascular measures were evident in obese adult men, which might be taken to have similar prognostic value in early determination of the cardiac morbidity risk in obese subjects.

This study has limitations of its own. First, we used BMI as a parameter of obesity for analyses which rather cannot differentiate body fat from muscle mass. In addition, our study did not assess the measures of central obesity, which would have provided additional insight into the association between obesity and myocardial function. Second, our study population was restricted to subjects from southern India, and hence, it is uncertain whether our findings could be extrapolated to other ethnicities. The generalization of our results to other populations may require caution. Although the sample sizes were moderate, they were adequate for comparisons within the site. However, the study findings can be used as the basis for planning longitudinal or cross-sectional studies with larger sample size and long term follow-up is required for further assessments in this regard.

Conclusion

Within the limits of this study, it can be concluded that obese subjects had higher resting HR, BP, QT interval, and increased response to steady dynamic exercise which can be due to alteration in autonomic activity. Delayed rate of decrease in HR and SBP after steady dynamic exercise was also observed, which are predictors of CVD. HR and BP recording during and after submaximal steady treadmill exercise may be useful in early detection of obese individuals at risk for the development of CVD and hence be of great significance in paving the way for future therapeutic implications of managing obesity.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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