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Impact of Exercise Stress Test on Cardiovascular Autonomic Functions among adults with different categories of Body Mass Index- A Cross Sectional Study

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Abstract

Background: Obesity is said to be due to the imbalance between calorie intake and energy expenditure and autonomic nervous system is involved in both energy metabolism and in cardiovascular regulation; it is believed that alterations in autonomic nervous system may be involved in the pathophysiology of obesity.

Objectives: The purpose of our study is to compare the cardiac autonomic functions between the individuals with normal, overweight and obese categories of body mass index (BMI) in a South Indian population.

Methods: A cross sectional study in department of Cardiology and Master Health Checkup in Chennai medical College and hospital, Trichy, a tertiary care teaching hospital during December 2015 to December 2016. Total participants included were 135. Data was entered and analyzed using Statistical Package for Social Sciences version 18. Descriptive statistics and analysis of variance tests were used, appropriately to test the statistical significance.

Results: Systolic blood pressure among obese participants is significantly high even after 3 minutes of exercise whereas diastolic blood pressure showed significant difference between the all BMI categories during and after the exercise. Heart rate recovery after 1 and 3 minutes of exercise, duration of exercise, metabolic equalent value and chronotropic incompetence were found to show no significant difference among normal, overweight and obese participants.

Conclusion: Obesity and overweight affects the cardiovascular system in many ways and it predicts all- cause mortality and cardiovascular mortality. To reduce the burden of cardiovascular disease in the population, we must overcome this by promoting weight reduction.

Keywords: Obesity, body mass index, exercise stress test, autonomic function

Introduction

Autonomic nervous system (ANS) plays a major role in regulation and integration of internal organ functioning.[1] Whenever autonomic nervous system function is altered, there occurs a cardiovascular adverse effects.[2] The parameters that reflect this interactions are Heart Rate (HR) response at rest, during peak exercise and during recovery (Heart rate recovery - HRR), and Heart rate variability (HRV).[3] In recent times, researchers are using heart rate variability and heart rate recovery as the non-invasive physiological markers to evaluate autonomic nervous system activity. The heart rate profile during exercise and recovery is a powerful predictor of sudden death.[4]

The resting HR is predominantly due to vagal tone[5]. The sympathetic activation during exercise is responsible for the increase in HR more than 100 beats per minute.[6] The initial increase in HR during exercise is due to parasympathetic withdrawal and sympathetic activation. The decline in heart rate immediately after exercise is due to the reactivation of parasympathetic nervous system.[14]

Heart rate recovery is defined as the ability to slow the heart rate after an exercise stress test and it has been suggested as a measure of chronotropic competence.[7] The lowest normal value for HRR is ≤ eighteen beats per minute[8]. The highest value for HRR is 22 beats per minute.[9] The HRR and chronotropic competence gives information prognostically. The HRR has been suggested as a predictor of CVD mortality in healthy adults.[10]

The prevalence of cardiovascular disease is more in individual with higher BMI.[11] Obesity is emerging as a global epidemic in children and adults called "New world syndrome." Obesity is said to be due to the imbalance between calorie intake and energy expenditure. Since, autonomic nervous system (ANS) is involved in both energy metabolism and in the cardiovascular regulation, it is believed that alterations in ANS may be involved in the pathophysiology of obesity at various levels from promotion of obesity to its role in cardiovascular diseases.[12] So, obesity is an independent risk factor for coronary artery disease, arrhythmias, and sudden death.[13]

The purpose of our study is to compare the cardiac autonomic functions between the individuals with normal BMI, Overweight and Obese BMI in South Indian population by measuring the resting heart rate, resting blood pressure, and peak exercise heart rate, blood pressure response to peak exercise, metabolic equivalent and exercise duration.

Methods

This study was conducted as a cross sectional study in the Department of Cardiology and Master Health Checkup in Chennai medical College and hospital, Trichy, a tertiary care teaching hospital during the period of December 2015 to December 2016. Study population includes healthy participants aged between 18-70 years of age from both genders with BMI more than 20 kg/m^2. Participants who gave history of alcohol consumption, smoking, hypertension, diabetes mellitus, pregnant and lactating mothers and cases with psychiatric disorders were excluded from the study. The sample size was calculated using mean maximum HR for various BMI categories as reference and the calculated sample size was 41 in each group. Considering the non-response rate of 10%, the total sample size was 135.

All participants were thoroughly explained about the study and its need in their native language and informed written consent were obtained from them. The study population was divided into three groups according to BMI.

The participants were clearly instructed to avoid heavy meals, caffeinated drinks, for at least 3 hours prior to the test.[14] After obtaining the demographic details about the subject, a brief instruction about the test procedure was given.

Before Exercise:

The participants were asked to take rest for 5 minutes in the supine position with eyes closed. At the end of the 5 minutes, resting heart rate and blood pressure was recorded. Resting heart rate were recorded by counting the pulse in the radial artery for one full minute. The normal resting heart rate ranges between 60-100 beats per minute. The resting heart rate ≥ 100 beats/minute was considered as abnormal. The maximum predicted HR was calculated as 220 minus age.[7] Resting blood pressure was recorded 3 times by auscultation method by using mercury sphygmomanometer (Diamond). The averages of the 3 readings were used.

During Exercise:

Treadmill exercise test was performed according to the Bruce protocol in WISPER MILL 594 XL. Individuals were encouraged to do the exercise until they experience limiting symptoms, even if 85% of maximum predicted heart rate was achieved. The heart rate and Blood pressure were recorded during peak exercise. The exercise duration and metabolic equivalent level were recorded. The exercise was terminated when the subject experienced chest pain, syncope, dyspnea, and fatigue, changes in ECG, failure to increase systolic blood pressure more than 10 mm Hg or fall of blood pressure below the resting level.[15]

After Exercise:

After cessation of exercise, the subject was asked to lie down in a couch. The heart rate and blood pressure were recorded within one minute and at three minutes of post exercise. Then, the resting blood pressure and heart rate, peak blood pressure and heart rate, heart rate recovery and blood pressure at 1 minute & 3rd minute, exercise capacity, and duration of exercise were calculated. The subjects were monitored for 6-8 minutes until heart rate, blood pressure, subject's symptom returns to pre-exercise level because the ECG changes that do not occur during the exercise might occur during the recovery period.[16]

Data was entered and analyzed using Statistical Package for Social Sciences (SPSS) version 18. Descriptive statistics and ANOVA tests were used, appropriately to calculate the statistical significance. P value of< 0.05 was considered as statistically significant.

Results

Age, gender and height were similar among the cases in normal, overweight and obese categories of BMI whereas weight and BMI were found to be significantly different between the three groups, as expected. (Table 1)

Table 1: Clinical profile and baseline characteristics of study participants

Parameters	Normal N=63	Overweight N=69	Obese N=46	P value
Age (in years)	44.52 ± 12.25	46.62 ± 8.87	48.04 ± 8.91	0.194
Gender (F/M)	26/37	12/57	13/33	-
Height (in cms)	161.76 ± 8.97	164.17 ± 9.28	163.76 ± 9.49	0.293
Weight (in Kgs)	59.52 ± 9.13	72.48 ± 10.41	82.43 ± 10.16	0.001*
BMI	22.57 ± 1.73	27.19 ± 1.39	31.34 ± 1.63	0.001*
At resting state				
Heart rate	82.51 ±18.68	82.29 ± 17.11	82.89 ± 15.56	0.983
SBP	122.06 ±16.48	124.35 ± 11.94	125.87± 13.43	0.36
DBP	77.14 ±0.23	80.55 ± 8.39	83.04 ± 7.56	0.003*

*Significant; F/M- female/male, BMI- Body Mass Index, SBP- systolic blood pressure, DBP- diastolic blood pressure

Heart rate at peak exercise, after one and three minutes were found to be similar among normal, overweight and obese individuals. Systolic blood pressure was similar during peak exercise and after one minute of exercise whereas after three minutes of exercise it showed significant difference. Diastolic blood pressure was found to be significantly different among normal, overweight and obese individuals during peak exercise and after one and three minutes (Table 2).

Table 2: Comparison of HR, SBP and DBP during and after the event of exercise with BMI categories

Event of exercise	Normal	Overweight	Obese	P value
Heart rate				
Peak exercise	155.76 ± 22.13	155.00 ± 19.4	150.3 ± 20.24	0.35
1 min after exercise	124.59 ± 22.24	126.68 ± 21.74	122.78 ± 23.55	0.65
3 mins after exercise	116.75 ± 19.2	105. 19 ± 19.5	103.28 ± 19.01	0.46
Systolic Blood Pressure				

Peak exercise	158.46 ± 18.26	161.88 ± 17.17	158.26 ± 19.59	0.46
1 min after exercise	156.67 ± 18.92	162.87 ± 15.66	162.17 ± 15.90	0.08
3 mins after exercise	137.78 ± 18.53	144.93 ± 14.72	147.39 ± 15.98	0.006**
Diastolic Blood Pressure				
Peak exercise	93.97 ± 10.09	96.01 ± 7.86	98.74 ± 9.44	0.03*
1 min after exercise	92.86 ± 10.23	95.94 ± 8.46	97.61 ± 8.48	0.02*
3 mins after exercise	85.97 ± 9.95	87.39 ± 8.69	89.35 ± 9.98	0.19

*Significant

Heart rate recovery after one and three minutes of exercise and duration of exercise, MET and chronotropic competence were found to be reduced among overweight and obese individuals but the difference was found to be statically insignificant (Table 3).

Table 3: Comparison of HRR and Indicators of exercise performance With BMI categories

Event of exercise	Normal	Overweight	Obese	P value
Heart Rate Recovery				
HRR after 1 min	31.10 ± 10.84	29.72 ± 14.35	29.11 ± 14.22	0.715
HRR after 3 mins	52.63 ± 14.85	51.58 ± 14.38	47.67 ± 14.72	0.198
Parameters	**Normal**	**Overweight**	**Obese**	**P value**
Duration of exercise (in mins)	8.17 ± 2.2	8.29 ± 2.06	7.51 ± 2.48	0.16
MET	10.46 ± 8.67	9.41 ± 2.01	8.61 ± 2.40	0.21
Chronotropic competence	149.15 ± 10.41	147.37 ± 7.54	146.16 ± 7.57	0.19

Discussion

In most of the studies the participants spent 1-2 minutes in a cool down walk after achieving peak workload. Even this minimal amount of work done during cool down walk would affect the sensitivity of HRR. So, participants were not allowed to undergo cool down walk in this study.

Resting Heart Rate and Blood Pressure

In our study, the diastolic BP is significantly higher in obese when compared to normal and overweight subjects. This finding is observed in many studies which have concluded that diastolic BP is positively correlated with BMI.[17,18] Das et al have shown that both systolic and diastolic BP correlated positively with BMI which supports our study.[18] Our findings are supported by Freedman et al[19], who studied the correlation between systolic BP and BMI and they have found a positive correlation between SBP and BMI. Our findings are further supported by Chorin et al who has concluded in their study that the mean diastolic BP increases by 3-4 mm Hg with increasing BMI.[20] Peripheral resistance is one of the most important parameter contributing to diastolic BP The peripheral resistance is normally higher in obese individuals and this probably substantiates for the statistically significant increase in diastolic blood pressure in resting state.[21]

We have found that the baseline heart rate (resting heart rate) and systolic BP was almost the same across the participants of various BMI groups. The resting HR reflects the balance between sympathetic and para sympathetic influence of the SA node. A higher HR indicates reduced parasympathetic influence or sympathetic over activity.[22] In the present study, the resting HR was recorded by examining the pulse rate and was found to be normal irrespective of age, gender and BMI.

Ogluwarde et al has stated that the increased resting HR is a predictor of cardiovascular disease mortality in subjects with and without diagnosed cardiovascular disease.[22] Since our study participants showed no difference in resting heart rate among the groups, it is evident that the study participants were well comparable and there were no other confounding factors that could interfere with the study results grossly.

Heart rate changes during and after exercise

The current study showed no significant difference in the heart rate during peak exercise, at 1 minute after exercise and at 3 minutes after exercise in overweight and obese groups. Our study is supported by LA Gondon et al who found that there were no significant differences in resting and peak HR between obese, overweight and normal subjects[14]. Peak heart rate refers to the maximal Heart rate achieved at termination of a graded maximal exercise test (Tanaka et al). The maximal Heart rate achieved at a given age is commonly assessed by the formula "220 – age." Nes et al found that the maximum heart rates achieved in physically active subjects are slightly lower than that of their inactive counterparts.[23]

Though the values are not statically significant, still the peak heart rate in obese individual is comparatively less than that of normal subjects. The heart rate recorded after 1 minute and 3 minutes of exercise showed that the heart rate is decreased in obese and overweight individuals. Our study results are similar to the study conducted by Rajalakshmi et al who found that obese and overweight had higher heart rate response to exercise than the normal subjects.[24]

Systolic Blood Pressure and Diastolic Blood Pressure

In our study, the systolic BP recorded at 3 minutes after exercise is significantly higher in obese group when compared to normal and overweight groups. Systolic BP recorded at 1 minute after exercise is increased in overweight and obese individuals. Our study results are similar to the study conducted by J.P.W. Burger et al who found that the systolic blood pressure was increased in obese subjects after exercise[25]. This study is further supported by Rajalakshmi et al who found that the systolic blood pressure recorded after 1 and 5 minutes of exercise was increased in overweight and obese groups[24]. The possible explanation may be an increase in sympathetic activity in overweight and obese groups. It is evident that the increase in Systolic BP in overweight and obese groups might be due to autonomic dysfunction since the excepted one following exercise is a fall in systolic BP.[21]

In our study, the systolic blood pressure recorded during peak exercise was almost same in subjects belonging to normal, overweight and obese groups. Rajalakshmi et al who found that the systolic blood pressure recorded during dynamic exercise was increased in overweight and obese groups which was not similar to our study[24]. This was not supported by study conducted by J.P.W. Burger et al who found that the systolic blood pressure recorded during peak exercise was increased in obese subjects when compared to lean subjects[25]. A major factor that increases during exercise to supply blood to all body regions is the cardiac output. The rise in systolic BP is due to sympathetic system mediated cardio-acceleration[15].

In our study, the diastolic blood pressure recorded during peak exercise and 1 minute after exercise was increased significantly in overweight and obese groups when compared to normal groups. Diastolic BP also seems to be increased 3 minutes after exercise in obese group though it was not statistically significant. Our study results are similar to study conducted by J.P.W. Burger et al who found that the diastolic blood pressure was increased during maximal exercise and after exercise in obese subjects when compared to lean subjects[25]. Our findings are not supported by Rajalakshmi et al reported that the diastolic blood pressure recorded after 1 and 5 minutes of exercise was decreased in overweight and obese groups.[24] Diastolic blood pressure depends upon peripheral resistance. During severe exercise, a fall in diastolic BP occurs due to vasodilation in the exercising muscle. The possible explanation may be alterations in autonomic nervous system in overweight and obese groups.

Heart Rate Recovery

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The present study reveals that the HRR in overweight and obese individuals are impaired after 1 minute and 3 minutes of exercise when compared to normal individuals though statistically not significant. Our study findings are supported by Tereza et al who found that the heart rate recovery measured immediately after exercise was impaired in obese individuals[26]. Our study was further supported by Sedova et al, postulated that, in response to a stress load, diet- induced obesity led to an abnormal HRR that was positively associated with an increase in body weight in spontaneously hypertensive rats[27]. Our findings are related to the study conducted by Garg et al who found that the obese subjects presented with slower heart rate recovery regardless of their fitness level[28]. Brinkworth et al. suggested that HRR is negatively associated with changes in body weight. HRR is an adjustable risk factor that can be improved with weight reduction in healthy, overweight and obese men without established CVD.[29] One possibility of less decrease in HRR in overweight and obese individual after exercise is that obesity is associated with chronic inflammation of adipose tissue that leads to accumulation of inflammatory adipokines (TNF, interleukins 6) that promote sympathetic hyperactivity.[26] Another possible explanation for impaired HRR in obese individuals may be vagal nerve dysfunction.

The heart rate recovery after exercise is due to rapid central vagal reactivation. Patients with an abnormal HRR had 8% mortality at 5.2 years and patients with normal HRR had a mortality of 2% at 5.2 years.[30] Both increased resting HR and delayed HRR have been proved as a powerful predictor of all-cause mortality and cardiovascular mortality in many previous studies.

Chronotropic Competence

Our data suggest that overweight and obese individuals were not able to achieve 85% of their age predicted HR during exercise (chronotropic competence) when compared to normal subjects. Tereza et al found that the maximum HR achieved during exercise was reduced in overweight and obese groups when compared to normal subjects which supports our study.[26] Garg et al suggested that the chronotropic index were lower in obese individuals which further supports our study[28]. The possible explanation may be a reflection of autonomic tone modulation. Lauer et al studied the HR response to exercise stress testing in healthy subjects and suggested that the chronotropic incompetence predicts cardiovascular mortality.[31]

Exercise Capacity- MET

In our study, the duration of exercise and exercise capacity is lowered in case of overweight and obese individuals when compared to normal individuals and hence their MET values are also lower. Tereza et al found that the MET values were reduced in overweight and obese groups when compared to normal subjects which supports our study.[26] Our findings are related to the study conducted by Garg et al who found that the obese subjects presented with slower heart rate recovery which is the most important factor that influences exercise capacity.[28] Framingham risk score proposed that there is a decrease in mortality risk by 17% for every 1 MET increase[32]. In a study conducted by Sedova et al who found that, more than 10 METs achieved during exercise stress test was associated with lower prevalence of ischemia.[28]

Conclusion

To conclude, systolic blood pressure among obese participants is significantly high even after 3 minutes of exercise whereas diastolic blood pressure showed significant difference between the all BMI categories during and after the exercise. HRR after 1 and 3 minutes of exercise, duration of exercise, MET value and Chronotropic incompetence were found to show no significant difference among normal, overweight and obese participants. Thus we conclude obesity and overweight affects the cardiovascular system in many ways and it predicts all- cause mortality and cardiovascular mortality. To reduce the burden of cardiovascular disease in the population, we must overcome this by promoting weight reduction.

References

1. Zygmunt, A. and Stanczyk, J., 2010. Methods of evaluation of autonomic nervous system function. Archives of medical science: AMS, 6(1), p.11.
2. Dimopoulos, S., Manetos, C., Panagopoulou, N., Karatzanos, L. and Nanas, S., 2015. The prognostic role of heart rate recovery after

exercise in health and disease. Austin J Cardiovasc Dis Atherosclerosis, 2(2), p.1014.

3. Freeman, J.V., Dewey, F.E., Hadley, D.M., Myers, J. and Froelicher, V.F., 2006. Autonomic nervous system interaction with the cardiovascular system during exercise. Progress in cardiovascular diseases, 48(5), pp.342-362.
4. Cunha, F.A., Midgley, A.W., Gonçalves, T., Soares, P.P. and Farinatti, P., 2015. Parasympathetic reactivation after maximal CPET depends on exercise modality and resting vagal activity in healthy men. SpringerPlus, 4(1), pp.1-9.
5. Jouven, X., Empana, J.P., Schwartz, P.J., Desnos, M., Courbon, D. and Ducimetière, P., 2005. Heart-rate profile during exercise as a predictor of sudden death. New England journal of medicine, 352(19), pp.1951-1958.
6. Ghaffari, S., Kazemi, B. and Aliakbarzadeh, P., 2011. Abnormal heart rate recovery after exercise predicts coronary artery disease severity. Cardiology Journal, 18(1), pp.47-54.
7. Cole, C.R., Blackstone, E.H., Pashkow, F.J., Snader, C.E. and Lauer, M.S., 1999. Heart-rate recovery immediately after exercise as a predictor of mortality. New England journal of medicine, 341(18), pp.1351-1357.
8. Watanabe, J., Thamilarasan, M., Blackstone, E.H., Thomas, J.D. and Lauer, M.S., 2001. Heart rate recovery immediately after treadmill exercise and left ventricular systolic dysfunction as predictors of mortality: the case of stress echocardiography. Circulation, 104(16), pp.1911-1916.
9. Yawn, B.P., Ammar, K.A., Thomas, R. and Wollan, P.C., 2003. Test-retest reproducibility of heart rate recovery after treadmill exercise. The Annals of Family Medicine, 1(4), pp.236-241.
10. Nishime, E.O., Cole, C.R., Blackstone, E.H., Pashkow, F.J. and Lauer, M.S., 2000. Heart rate recovery and treadmill exercise score as predictors of mortality in patients referred for exercise ECG. Jama, 284(11), pp.1392-1398.
11. Mathew, B., Francis, L., Kayalar, A. and Cone, J., 2008. Obesity: effects on cardiovascular disease and its diagnosis. The Journal of the American Board of Family Medicine, 21(6), pp.562-568.
12. Grewal, S. and Gupta, V., 2011. Effect of obesity on autonomic nervous system. Int J Cur Bio Med Sci., 1(2), pp.15-18.
13. Rissanen, P., Franssila-Kallunki, A. and Rissanen, A., 2001. Cardiac parasympathetic activity is increased by weight loss in healthy obese women. Obesity research, 9(10), pp.637-643.
14. Gondoni, L.A., Titon, A.M., Nibbio, F., Augello, G., Caetani, G. and Liuzzi, A., 2009. Heart rate behavior during an exercise stress test in obese patients. Nutrition, Metabolism and Cardiovascular Diseases, 19(3), pp.170-176.
15. Vivekananthan, D.P., Blackstone, E.H., Pothier, C.E. and Lauer, M.S., 2004. Heart rate recovery after exercise is a predictor of mortality, independent of the angiographic severity of coronary disease. ACC Current Journal Review, 1(13), pp.30-31.
16. Syme, A.N., Blanchard, B.E., Guidry, M.A., Taylor, A.W., VanHeest, J.L., Hasson, S., Thompson, P.D. and Pescatello, L.S., 2006. Peak systolic blood pressure on a graded maximal exercise test and the blood pressure response to an acute bout of submaximal exercise. The American journal of cardiology, 98(7), pp.938-943.
17. Wu, Y., Zhang, D., Pang, Z., Jiang, W., Wang, S., Li, S., von Bornemann Hjelmborg, J. and Tan, Q., 2015. Multivariate modeling of body mass index, pulse pressure, systolic and diastolic blood pressure in Chinese twins. Twin Research and Human Genetics, 18(1), pp.73-78..
18. Das, R.K. and Nessa, A., 2013. Blood pressure in different levels of BMI. Mymensingh medical journal: MMJ, 22(4), pp.699-705.
19. Freedman, D.S., Goodman, A., Contreras, O.A., DasMahapatra, P., Srinivasan, S.R. and Berenson, G.S., 2012. Secular trends in BMI

and blood pressure among children and adolescents: the Bogalusa Heart Study. Pediatrics, 130(1), pp.e159-e166.

20. Chorin, E., Hassidim, A., Hartal, M., Havakuk, O., Flint, N., Ziv-Baran, T. and Arbel, Y., 2015. Trends in adolescents obesity and the association between BMI and blood pressure: a cross-sectional study in 714,922 healthy teenagers. American journal of hypertension, 28(9), pp.1157-1163.
21. Fletcher, G.F., Balady, G.J., Amsterdam, E.A., Chaitman, B., Eckel, R., Fleg, J., Froelicher, V.F., Leon, A.S., Piña, I.L., Rodney, R. and Simons-Morton, D.A., 2001. Exercise standards for testing and training: a statement for healthcare professionals from the American Heart Association. Circulation, 104(14), pp.1694-1740.
22. Ogunlade O, Ayoka AO, Akintomide A, Akomolafe RO, Akinsomisoye OS, Oyebola jg DO. Akunne, j.i., 2013. Effect of life style modification measures in adult hypertensive patients attending 44 Nigerian army reference hospital kaduna. Faculty of Family Medicine.
23. Nes, B.M., Janszky, I., Wisløff, U., Støylen, A. and Karlsen, T., 2013. Age‐predicted maximal heart rate in healthy subjects: The HUNT Fitness Study. Scandinavian journal of medicine & science in sports, 23(6), pp.697-704.
24. Rajalakshmi, R., Nataraj, S.M., Vageesh, V. and Dhar, M.U.R.A.L.I., 2011. Blood pressure responses to steady treadmill exercise in overweight and obese young adults. Indian J Physiol Pharmacol, 55(4), pp.309-314.
25. Burger, J.P.W., Serne, E.H., Nolte, F. and Smulders, Y.M., 2009. Blood pressure response to moderate physical activity is increased in obesity. Age (years), 24(4.6), pp.24-0.
26. Lins, T.C.B., Valente, L.M., Sobral Filho, D.C. and e Silva, O.B., 2015. Relation between heart rate recovery after exercise testing and body mass index. Revista Portuguesa de Cardiologia (English Edition), 34(1), pp.27-33.
27. Šedová, L., Bérubé, J., Gaudet, D., Dumont, M., Tremblay, J., Hamet, P. and Pausová, Z., 2004. Diet‐induced obesity delays cardiovascular recovery from stress in spontaneously hypertensive rats. Obesity Research, 12(12), pp.1951-1958.
28. Garg, R., Malhotra, V., Goet, N., Dhar, U. and Tripathi, Y., 2013. A study of autonomic function tests in obese people. International Journal Of Medical Research & Health Sciences, 2(4), pp.750-755.
29. Brinkworth, G.D., Noakes, M., Buckley, J.D. and Clifton, P.M., 2006. Weight loss improves heart rate recovery in overweight and obese men with features of the metabolic syndrome. American Heart Journal, 152(4), pp.693-e1.
30. Shetler, K., Marcus, R., Froelicher, V.F., Vora, S., Kalisetti, D., Prakash, M., Do, D. and Myers, J., 2001. Heart rate recovery: validation and methodologic issues. Journal of the American College of Cardiology, 38(7), pp.1980-1987;
31. Lauer, M.S., Francis, G.S., Okin, P.M., Pashkow, F.J., Snader, C.E. and Marwick, T.H., 1999. Impaired chronotropic response to exercise stress testing as a predictor of mortality. Jama, 281(6), pp.524-529.
32. Gulati, M., Pandey, D.K., Arnsdorf, M.F., Lauderdale, D.S., Thisted, R.A., Wicklund, R.H., Al-Hani, A.J. and Black, H.R., 2003. Exercise capacity and the risk of death in women: the St James Women Take Heart Project. Circulation, 108(13), pp.1554-1559.