Assessment of aerobic capacity in overweight young females: A cross-sectional study

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Abstract

Context: Overweight/obese people are prone to develop cardiovascular, respiratory and other chronic diseases at young age because of abnormal weight. Aerobic capacity (VO\(_2\) max) is an accepted index of cardio respiratory fitness. Decrease in VO\(_2\) max can be an early marker for altered cardiovascular physiology. Objectives: The present study was carried out with the objective of evaluating aerobic capacity in overweight young females and comparing it with that of normal weight females. Materials and Methods: Twenty-three female subjects aged 18–20 years were enrolled in each group. Group 1 comprised overweight subjects and group 2 comprised normal weight subjects. Analysis to assess the difference in VO\(_2\) max between the groups was done by unpaired t-test. Results: Mean age of group 1 and 2 was 18.91 ± 0.67 years and 18.83 ± 0.78 years, respectively. Mean BMI in group 1 and 2 was 26.18 ± 1.06 kg/m\(^2\) and 20.65 ± 1.5 kg/m\(^2\) respectively. VO\(_2\) max in groups 1 and 2 was 34.52 ± 3.26 ml/min/kg and 37.51 ± 2.88 ml/min/kg respectively. The difference in VO\(_2\) max found in overweight girls was statistically significant with P value of 0.002. Conclusion: Overweight girls had significantly reduced, cardio-respiratory fitness when compared to normal weight young females.

Key words: Body mass index, cardio-respiratory fitness, obesity, VO\(_2\) max

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Introduction

Obesity is associated with chronic lifestyle related disorders particularly cardiovascular and respiratory diseases.\(^{[1-2]}\) The course of early life overweight/obesity toward development of cardiovascular disease involves gradual decrease in cardio-respiratory efficiency. Physical fitness, which may be used as an indicator of cardiopulmonary efficiency has been found to be more effective than assessment of physical activity in the prediction of health outcome in an individual. Physical fitness in children has been found to be associated with better health outcomes in terms of blood pressure, muscle strength, blood lipids, serum insulin or blood vasculature characteristics.\(^{[3-5]}\) Cardio-respiratory efficiency can be assessed by evaluating maximal oxygen consumption (VO\(_2\) max) also called as aerobic capacity and indicates the physical fitness of a person.\(^{[6-9]}\) Evaluating VO\(_2\) max in overweight people can help in early detection of physiological alterations which can help in designing appropriate intervention strategies. Thus, there is a need to assess the change in cardio-respiratory efficiency at an early stage in life. The present study was carried out to evaluate aerobic capacity in overweight young females and comparing it with that of normal weight females.

Materials and Methods

The present cross-sectional study was carried out after taking due permission from the Institutional Ethics Committee. Sample size was calculated for detecting a large effect size (Cohen’s d = 0.9) with \(\alpha\) as 0.05 and power of study as 80% for two tailed hypothesis testing.

Twenty-three apparently healthy female subjects aged 18–20 years who gave informed consent for the study...
were enrolled in each group. Group 1 comprised overweight subjects (25 ≤ body mass index [BMI] < 30) and group 2 comprised normal weight subjects (18.5 ≤ BMI < 25) as per BMI reference range of World Health Organization. Subjects with history of cardiopulmonary disease, hepatic or renal impairment, medication, chronic illness, any major surgery, undergoing any physical conditioning program or involved in sports activity were not included in the study groups. Weight was recorded to the nearest 1 kg with clothing on a standard scale and height was measured to nearest one cm without footwear. BMI was calculated by Quetelet’s index (kg/m²). All the recordings were done in the clinical laboratory of the Department of Physiology. To account for diurnal variation, all the readings were taken in the morning after light breakfast. The study duration was 2 months.

Estimation of VO₂ max by queens college step test

It was performed using stepping bench with 16.25 inches height. Stepping was done for total duration of 3 min at the rate of 22 steps ups/min. After completion of exercise, carotid pulse rate was measured from 5th to 20th second of recovery period. It was converted into pulse rate per minute. Following equation as described along with the procedure in McArdle, Katch and Katch’s Exercise Physiology was used to estimate VO₂ max expressed in milliliters per kilogram body weight per minute.

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\text{VO}_2 \text{ max (ml/kg/min)} = 65.81 - (0.1847 \times \text{pulse rate in beats/min}).
\]

Statistical analysis was performed using Student’s unpaired t-test using online GraphPad (GraphPad Software Inc. California, USA) software.

RESULTS

All the subjects were able to complete the Queens College Step Test protocol for full 3 min without break. Table 1 shows the characteristics of each group. Mean age and BMI in overweight group was 18.91 ± 0.67 years and 26.18 ± 1.06 kg/m² respectively. Mean age and BMI in normal weight group was 18.83 ± 0.78 years and 20.65 ± 1.5 kg/m² respectively.

The VO₂ max in overweight females was 34.52 ± 3.26 ml/kg/min whereas it was 37.51 ± 2.88 ml/kg/min in normal weight females. Thus, the overweight females had significantly lower aerobic capacity (VO₂ max) when compared with normal weight females with a P = 0.002 as shown in Table 2.

DISCUSSION

In the present study, VO₂ max relative to body weight was found to be significantly less in overweight young females when compared to normal weight females. This indicates that the ability to carry out exhausting work is considerably less in overweight young females. Reduction in cardiopulmonary fitness even in young overweight females who are below the cut off values of BMI of 30 kg/m² to be labeled as obese is a significant alert. However, it can be explained by the influence of excess fatty tissue on the physiology of cardiac and respiratory systems, which has been explored by various research works done in overweight and obese individuals.

Obesity has been found to be associated with a spectrum of various cardiovascular abnormalities, which range from a state of hyperdynamic circulation to evidence of subclinical changes in cardiac structure. Wong et al. have reported that even if we adjust for age, gender, mean arterial pressure and left ventricular mass, being overweight is independently associated with subclinical changes in left ventricular structure in subjects without an overt heart disease. Cardiac function has been found to correlate with BMI as well as the duration of obesity. This signifies the importance of early detection and intervention at an early stage to prevent the cardiac disease in overweight/obese individuals. Obesity adversely affects the respiratory system causing a deviation in respiratory mechanics, decreasing the endurance and strength of respiratory muscles, decreased gas exchange and limitations in the lung function and the exercise capacity. Lung function impairment is supposed to be caused by the extra amount of adipose tissue in chest wall and the abdominal cavity, which may compress the thoracic cage, diaphragm, and lungs. This may limit diaphragm displacement and compliance of the lung and chest wall. This results in a decrease in lung volumes.
The impact on respiratory function worsens with an increase in the BMI of individual.[22]

Study by Davies et al.[23] has reported that on maximal exercise, obese females showed a marked decrease in the exercise performance when compared with control group. Furthermore, it was observed that absolute VO$_2$ max was similar in obese and control subjects but VO$_2$ max per kilogram body weight was reduced significantly in the obese group. Goran et al.[24] also found VO$_2$ max expressed relative to body weight was reduced significantly in obese individuals and that VO$_2$ max expressed relative to body weight improved significantly by around 15% after weight reduction in the obese group.

Limitations of this study include the cross-sectional design which cannot comment upon the cause-effect relationship between overweight status and aerobic capacity status. Larger and longitudinal studies need to be done to further the understanding on the subject. Interventional studies can help in evaluating whether there can be improvement of cardiopulmonary efficiency in obesity with weight loss.

To summarize the lower aerobic capacity in overweight females may be an early indicator of cardio-respiratory dysfunction. We can hypothesize that obesity may lead to reduced ability to maximally consume oxygen and therefore has detrimental effect on VO$_2$ max. Thus, it is necessary to take steps for primary prevention for the control of the overweight/obesity syndrome.

**REFERENCES**

1. Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, et al. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. JAMA 2003;289:76-9.

2. Field AE, Coakley EH, Must A, Spadano JL, Laird N, Dietz WH, et al. Impact of overweight on the risk of developing common chronic diseases during a 10-year period. Arch Intern Med 2001;161:1581-6.

3. Ruiz JR, Ortega FB, Meusel D, Harro M, Oja P, Sjöström M. Cardiorespiratory fitness is associated with features of metabolic risk factors in children. Should cardiopulmonary fitness be assessed in a European health monitoring system? The European Youth Heart Study. J Public Health 2006;14:94-102.

4. Watts K, Jones TW, Davis EA, Green D. Exercise training in obese children and adolescents: Current concepts. Sports Med 2005;35:375-92.

5. Froberg K, Andersen LB. Mini review: Physical activity and fitness and its relations to cardiovascular disease risk factors in children. Int J Obes (Lond) 2005;29 Suppl 2:S34-9.

6. Anderson KM, Shephard RJ, Denolín H, Varnauskas E, Masironi R. Fundamentals of Exercise Testing. Geneva: WHO; 1971.

7. Astrand PO. Textbook of Work Physiology, Physiological Bases of Exercise. 3rd ed. New York: McGraw Hill; 1986.

8. Shephard RJ. World standards of cardiorespiratory performance. Arch Environ Health 1966;13:664-72.

9. Banerjee PK, Chatterjee S, Chatterjee P, Maitra SR. Maximal oxygen uptake in boys. Indian J Med Res 1982;75:380-6.

10. WHO. The international classification of adult overweight, obesity and obesity according to BMI. BMI Classification, Global Data Base on Body Mass Index. Geneva: World Health Organization; 2006.

11. McArdle WD, Katch IF, Katch LV. Exercise Physiology: Energy, Nutrition and Human Performance. 5th ed. Philadelphia: Lippincott Williams and Wilkins; 2001.

12. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: A 26-year follow-up of participants in the Framingham Heart Study. Circulation 1983;67:968-77.

13. Kenchaiah S, Evans JC, Levy D, Wilson PW, Benjamin EJ, Larson MG, et al. Obesity and the risk of heart failure. N Engl J Med 2002;347:305-13.

14. Alpert MA. Obesity cardiomyopathy: Pathophysiology and evolution of the clinical syndrome. Am J Med Sci 2001;322:225-36.

15. Wong CY, O’Moore-Sullivan T, Leano R, Byrne N, Beller E, Marwick TH. Alterations of left ventricular myocardial characteristics associated with obesity. Circulation 2004;110:3081-7.

16. Scaglione R, Dichiara MA, Indovina A, Lipari R, Ganguzzza A, Panninello G, et al. Left ventricular diastolic and systolic function in normotensive obese subjects: Influence of degree and duration of obesity. Eur Heart J 1992;13:738-42.

17. Faintuch J, Souza SA, Valezi AC, Sant’Anna AF, Gama-Rodrigues JJ. Pulmonary function and aerobic capacity in asymptomatic bariatric candidates with very severe morbid obesity. Rev Hosp Clin Fac Med Sao Paulo 2004;59:181-6.

18. Koenig SM. Pulmonary complications of obesity. Am J Med Sci 2001;321:249-79.

19. Ladosky W, Botelho MA, Albuquerque JP Jr. Chest mechanics in morbidly obese non-hypoventilated patients. Respir Med 2001;95:281-6.

20. Lotti P, Gigliotti F, Tesi F, Stendardi L, Grazzini M, Duranti R, et al. Respiratory muscles and dyspnea in obese nonsmoking subjects. Lung 2005;183:311-23.

21. Rasslan Z, Junior RS, Stirbulov R, Fabbri RM, Lima CA. Evaluation of pulmonary function in class I and II obesity. J Bras Pneumol 2004;30:508-14.

22. Jones RL, Nzekwu MM. The effects of body mass index on lung volumes. Chest 2006;130:827-33.

23. Davies CT, Godfrey S, Light M, Sargeant AJ, Zeidifard E. Cardiopulmonary responses to exercise in obese girls and young women. J Appl Physiol 1975;38:373-6.

24. Goran M, Fields DA, Hunter GR, Fabbri RM, Lima CA. Evaluation of pulmonary function in class I and II obesity. J Bras Pneumol 2004;30:508-14.