Childhood Obesity And Cardiovascular Autonomic Nervous System
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Abstract: ANS (Autonomic Nervous System) is involved in energy metabolism and regulation of cardiovascular system. ANS of obese individuals is chronically altered. Various studies demonstrate important modifications in the autonomic control of obese adults and adolescents, there is scarce information on obese children, and the findings remain inconclusive. So work should be done to investigate the cardiovascular autonomic nervous functions in obese school children in comparison with age, sex and ethnically matched control group. The reduction in cardiovascular autonomic nervous system observed in obese children may be an early sign for the prediction of the risk for cardiovascular and metabolic disease.

Key words: Obesity, ANS (Autonomic Nervous System), CVS (Cardiovascular System)

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Introduction: Obesity is emerging as a global epidemic in both children and adults. This has been called “New world syndrome” and is a reflection of massive social, economic and cultural problems currently facing developing and developed countries. Obesity is regarded as a complex disease because it arises from multifaceted interactions of genetic and environmental factors. As the prevalence of this disorder grows worldwide, obesity is increasingly considered a major public health problem. The consequences of obesity in childhood and adolescence include arterial hypertension, atherosclerosis, dyslipidemia, diabetes, obstructive sleep apnoea, alterations in the musculoskeletal system, depression and a reduction in quality of life. Obesity is characterised by an excessive deposition and storage of fat in the body. Body mass index is the marker for body fat content.

Definition and classification of obesity: Obesity is defined as an excessive accumulation of fat that causes a generalized increase in body mass. Several definitions and cut off points have been used to define overweight and the degree of obesity. The most commonly used indicator of general adiposity is body mass index (BMI), calculated as weight in kilograms divided by the square of the height in meters. World Health Organization (WHO) (1995) and National Institutes of Health (NIH) (1998) have recommended the classification of BMI using cut off points of 25 kg/m² and 30 kg/m², i.e. overweight BMI 25-30 kg/m², and obesity BMI of 30 kg/m² or higher. The risks of morbidity and mortality seem to increase with increasing BMI.

Classification of obesity

<table>
<thead>
<tr>
<th>BMI (Kg/m²)</th>
<th>Class</th>
<th>Health risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI &lt; 25</td>
<td>Normal weight</td>
<td>Very low</td>
</tr>
<tr>
<td>25 &lt; BMI &lt; 30</td>
<td>Overweight</td>
<td>Low</td>
</tr>
<tr>
<td>30 &lt; BMI &lt; 35</td>
<td>Moderate obesity</td>
<td>Moderate</td>
</tr>
<tr>
<td>35 &lt; BMI &lt; 40</td>
<td>Severe obesity</td>
<td>High</td>
</tr>
<tr>
<td>BMI &gt; 40</td>
<td>Morbid obesity</td>
<td>Very high</td>
</tr>
</tbody>
</table>

Classification of adult underweight, overweight and obesity according to BMI and risk of obesity-related co-morbidities (adapted from the International Classification)

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI (Kg/m²)</th>
<th>Risk of Obesity related morbidities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>Less than 18.5</td>
<td>Low risk</td>
</tr>
<tr>
<td>Normal range</td>
<td>18.50 to 24.99</td>
<td>Average risk</td>
</tr>
<tr>
<td>Overweight</td>
<td>Greater than or equal to 25.00</td>
<td>Increased risk</td>
</tr>
<tr>
<td>Obese</td>
<td>Greater than or equal to 30.00</td>
<td>Medium to high risk</td>
</tr>
<tr>
<td>Morbidly obese</td>
<td>Greater than or equal to 40.00</td>
<td>Very high risk</td>
</tr>
</tbody>
</table>

Causes of childhood obesity
- Lack of regular exercise.
- Sedentary habits.
- Over consumption of high calorie food.
- Genetic, prenatal and life factors.

Effect of obesity on cardiovascular autonomic nervous system: ANS is involved in energy metabolism and regulation of cardiovascular system. Several studies in literature suggest that ANS of obese individuals is chronically altered. Peterson et al. report an association...
between the increase in body fat and hypogastric activity of sympathetic and parasympathetic components of ANS. It has been observed that individuals with low resting muscle sympathetic nerve activity may be at risk for body weight gain resulting from lower metabolic rate. The hypothalamus is a regulatory centre of satiety and of the ANS. Therefore, abnormalities in the hypothalamus may cause obesity and autonomic dysfunction. A reduction in parasympathetic activity among obese children has also been reported. According to Nagai and Moritani a causal relationship between alteration in ANS activity and obesity cannot be confirmed, however authors suggest that a reduction in autonomic activity may be an etiological factor in the onset and development of obesity.

Study done by Simran Grewal, Vidhushi suggested impaired autonomic nervous system function in obese. The impaired CPT in obese which could possibly because of hypofunctional sympathetic nervous system. Some workers that showed increase in blood pressure during exposure to cold pressor test on loss of around 30% of excess weight following a period of hypocaloric diet. In another study, there was reduced sympathetic responsiveness associated with thermoregulation demonstrated by abnormal heart rate variability on cold exposure. In another study obese children possessed a decreased sympathetic and parasympathetic activity and parasympathetic activity increased with weight loss in obese women. It is documented that low sympathetic activity and a low activity of adrenal medulla leads to development of central adiposity. Some workers who observed decreased parasympathetic response in terms of RV (Heart Rate Variability analysis) of 5 minute R-R interval before and after head up tilt manoeuvre. In another study it has been seen that Cardiac parasympathetic dysfunction present in obese subjects could be associated with higher carbohydrate intake and lower fat and protein intake which results in parasympathetic abnormality. It is also demonstrated that parasympathetic activity increased with weight loss in obese.

Vanderlei LCM et al study suggests that obese children have autonomic dysfunctions characterized by a reduction of both sympathetic and parasympathetic activity. A reduction in parasympathetic activity among obese children has been reported by authors. The mechanisms by which vagal dysfunction is related to obesity are under debate, and it is uncertain whether this dysfunction is a consequence of obesity or facilitates their development. A reduction in vagal activity is associated with an increased risk for all-cause morbidity and mortality and for the development of several risk factors. Therefore, the reduction observed in obese children may be an early sign for the prediction of the risk for cardiovascular and metabolic disease. A reduction in sympathetic activity in obese children is also described in the literature. The sympathetic ANS is associated with the mobilization of body energy and participates in the control of glucose and fat metabolism.

Conclusion: The continuing increase in the number of obese children is alarming due to the potential risk of premature health problems. Moreover, obese children have a high likelihood of becoming obese adults and obese adults who were once obese children have a lower treatment response than those who became obese in adulthood. These concerns about the effects of obesity reinforce the need for the prevention and treatment of the condition in childhood. So autonomic function test can be made an important tool for obesity in children. More studies should be done to get more authentic data to show how autonomic functions are in obese children.

References


31. Nagai N, Matsumoto T, Kita H, Moritani T. Autonomic nervous system activity and the state...