



Impact of obesity on autonomic modulation, heart rate and blood pressure in obese young people



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ABSTRACT

Introduction: Obesity can be understood as a comorbidity of increasing systemic impact, including a deficit in the autonomic nervous system.

Objective: To analyze cardiac autonomic behavior and hemodynamic parameters in obese young people.

Methods: 92 individuals (20.58 ± 1.48 years) were evaluated, divided into two groups: obese and eutrophic. Heart rate (HR) was captured for 30 min in the supine position whilst breathing spontaneously. Blood pressure (BP) values were obtained prior to performance of the protocol. For the autonomic analysis, 1000 beats were used for the calculation of heart rate variability indices in the time (Mean RR, SDNN and RMSSD) and frequency (LF, HF and LF/HF) domains, in addition to the Poincaré plot (SD₁, SD₂, SD₁/SD₂ and qualitative visual analysis). **Results:** The obese group presented higher baseline BP and HR values compared to the eutrophic. Regarding autonomic modulation a significant decrease was observed in the RMSSD; SD₁, HFms and HFnu indices in the obese group, indicating a decrease in vagal activity and reduced SDNN and SD₂ rates, with statistical significance for the former, suggesting a reduction in overall variability. The high value of the LFnu index and decrease in Mean RR in the obese group pointed to relative sympathetic predominance in these individuals. The visual analysis of the Poincaré plot showed less dispersion of the points in the obese group.

Conclusion: The obese group presented higher BP and HR values at rest and autonomic impairment, characterized by a reduction in parasympathetic activity and relative predominance of sympathetic activity.

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1. Introduction

Obesity, defined as the excessive accumulation of body fat resulting from the positive energy balance (WHO. World Health Organization, 2003), refers to the risk of severe chronic diseases such as type II diabetes, hypertension, stroke, certain types of cancer and disorders of the ventricles, morphological changes and psychosocial damage (Melo et al., 2010; Sokmen et al., 2013; Tumuklu et al., 2007; Wong et al., 2004). It is also considered a potential predictor for cardiovascular disease and, when established early in life, leads to short and long-term damage, resulting from the abnormal lipid profile presented by these individuals (Santos et al., 2008).

Another important manifestation of obesity is impairment in the autonomic nervous system (ANS), present in all age groups, each of which has its own profile (Farah et al., 2013; Vanderlei et al., 2010). Studies show that the presence of obesity in children under 12 years old causes autonomic imbalance, characterized by decreased parasympathetic modulation; the sympathetic modulation presents inconclusive results (Dangardt et al., 2011; Souza et al., 2012). Autonomic behavior in obese adults is characterized by a decrease in parasympathetic activity, with autonomic imbalance (Wu et al., 2008). Although studies show that there are important changes in autonomic control in adults and obese children, studies related to ANS behavior in obese young people are scarce in the literature (Krishna et al., 2013).

These alterations, together with other systemic effects of obesity, can promote early damage to the integrity of the body. Therefore, understanding the scenario of autonomic dysfunction present in these individuals is of fundamental clinical importance both from a preventive and therapeutic point of view, and may provide support for professionals working with this population, ensuring greater outcomes from

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their efforts. Thus, this study aimed to analyze ANS behavior and hemodynamic parameters in obese young people.

2. Method

For this study, the data were analyzed from 92 young volunteers of both genders, with a mean age of 20.58 ± 1.48 years. The volunteers were non-randomly distributed into two groups – the obese group GO ($n = 44$) and the eutrophic group – GE ($n = 48$). The classification criteria adopted for formation of the groups was the value of the body mass index (BMI) (World Health Organization, 2006).

Volunteers who presented any of the following: tobacco smokers, alcohol drinkers, the use of illicit drugs, medications or those who presented a framework of infections or known diseases of the metabolic cardiorespiratory systems which could affect cardiac autonomic control, were not included from the study.

The volunteers were informed about the procedures and objectives of this study and, after agreeing to participate, signed a free and informed consent form. All procedures were approved by the Institutional Ethics Committee (Protocol No. 11/2011).

All study volunteers were instructed to abstain from alcoholic beverages and/or stimulants such as coffee and tea for 12 h prior to the experimental protocol so as to avoid any direct influence on cardiac autonomic behavior at the time of the protocol.

The protocol was preceded by the collection of data from volunteers, including information on name, age, telephone numbers and the presence of comorbidities. Next, the volunteers underwent a physical examination to determine: body weight, by means of digital electronic scale (Welmy, Brazil); height, obtained in the upright position by means of a stadiometer (Sanny, Brazil); blood pressure (BP) – measured indirectly using a aneroid sphygmomanometer (WelchAllyn, Germany) and stethoscope (Littmann, USA); and heart rate (HR) obtained using a heart rate monitor Polar S810i (Polar Electro, Kempele, Finland) (Vanderlei et al., 2008). The height and weight measurements were used to determine BMI, given by the formula $BMI = \text{weight}/\text{height}^2$ (kg/m^2) (World Health Organization, 2006).

The subsequent stages of data collection, physical and hemodynamic parameters, were aimed at obtaining the RR intervals for analysis of heart rate variability (HRV). Data collection was performed in a room with a temperature between 21°C and 23°C and humidity of 40–60%. The movement of people was controlled to reduce the anxiety of the participants.

To capture the RR intervals a capture strap was positioned on the chest of the volunteer and an HR receptor, Polar S810i, on the wrist. After placement of the equipment, the participants were instructed to remain in the supine position on a mat, where they remained, breathing spontaneously for 30 min. At the end of the collection period the volunteer was released.

The HRV analysis was performed from the HR beat-to-beat recorded during the protocol, and only series with more than 95% sinus beats were used for the analysis (Godoy et al., 2005). After collection of the data, the HRV analysis was performed from the final 25 min of the recording, with the first 5 min discarded to exclude the initial influences of the protocol on HR behavior.

The selected portion was subjected to digital filtering performed using Polar Precision Performance SW software (version 4.01.029), supplemented by manual filtering to eliminate premature ectopic beats and artifacts. Finally, for the final data analysis, the first 1000 consecutive RR intervals derived from the series of RR intervals were used, performed using the software Kubios HRV (Kuopio University, Finland). The analysis was performed using linear methods, analyzed in the time domain, frequency domain and by the Poincaré plot.

In the time domain the following indices were used for HRV analysis: Mean RR, RMSSD and SDNN. The RMSSD index is defined as the root mean square of successive differences between adjacent normal RR intervals in a given time interval, in milliseconds and the SDNN

represents the standard deviation of normal to normal RR intervals, in milliseconds (Vanderlei et al., 2009), being interpreters of parasympathetic activity and global activity, respectively.

For HRV analysis in the frequency domain, the following were calculated: the total power (Ptot) in ms^2 translated by the band from 0.003 to 0.4 Hz and the spectral components: low frequency LF: 0.04 to 0.15 Hz and high frequency HF: 0.15 to 0.40 Hz in normalized units, which refers to the relative value of each spectral component in relation to the total power, minus the very low frequency VLF components multiplied by 100, and in milliseconds squared, and the ratio between them (LF/HF). The spectral analysis was calculated using the Fourier Transform algorithm (Billman, 2011; Vanderlei et al., 2009).

Finally, the Poincaré plot was analyzed quantitatively, expressed by the indices: SD_1 (standard deviation of the instantaneous beat-to-beat variability, representing parasympathetic modulation), SD_2 (standard deviation of the long-term continuous RR intervals, representing global variability) and the SD_1/SD_2 ratio and qualitative analysis by analyzing the figures formed by its attractor, or (Billman, 2011; de Carvalho et al., 2014; Vanderlei et al., 2009):

- 1) A figure in which an increase in dispersion of RR intervals is observed with an increase in intervals, characteristic of a normal plot.
- 2) A figure with little global beat-to-beat dispersion and without an increase in the dispersion of RR intervals in the long-term.

The population profile data were described using descriptive statistics and the results presented as mean, standard deviation and absolute number values. For the group comparisons, initially the normality of the data was determined and when the normal distribution was accepted the Student's t-test for unpaired data was applied. In situations where normal distribution was not accepted, the Mann–Whitney test was used. Differences in these tests were considered statistically significant when the value of $p < 0.05$.

The calculation of the study power (GraphPad Software StatMate 2:00 version for Windows, GraphPad Software, San Diego California USA), with the sample number and significance level of 5% (two-tailed test) guaranteed a test power greater than 80% to detect differences between the variables.

3. Results

Table 1 presents the characteristics of the sample. Significant differences were observed for BMI, this resulting only from the weight

Table 1
Anthropometric and hemodynamic variables of the obese and eutrophic groups submitted to the protocol.

	Obese (Men = 24/women = 20)	Eutrophic (Men = 24/women = 24)	p value
Age (years)	20.45 ± 1.57 (20.5) [19.97–20.93]	20.70 ± 1.39 (21) [20.30–21.11]	0.4155
Weight (kg)	102.30 ± 20.82 (98.5) ^a [95.96–108.64]	62.89 ± 10.47 (62.4) [59.85–65.94]	<0.0001
Height (m)	1.70 ± 0.10 (1.72) [1.67–1.74]	1.68 ± 0.10 (1.67) [1.65–1.72]	0.3774
BMI (kg/m^2)	34.67 ± 3.87 (33.53) ^a [33.50–35.85]	21.91 ± 1.86 (22.21) [21.37–22.45]	<0.0001
SBP (mm Hg)	126.59 ± 14.93 (120) ^b [122.05–131.13]	107.91 ± 13.60 (110) [103.96–111.87]	<0.0001
DPB (mm Hg)	83.31 ± 11.26 (80) ^b [79.89–86.74]	68.33 ± 10.45 (70) [65.29–71.37]	<0.0001
HR (bpm)	80.22 ± 10.09 (80) ^b [77.15–83.29]	73.06 ± 10.35 (72) [70.05–76.07]	0.0012

Mean $\pm$ standard deviation (median) [confidence interval]; ^a value with statistical difference compared to the eutrophic group (Mann Whitney test, $p < 0.05$); ^b value with statistical difference compared to the eutrophic group (t test for unpaired samples; $p < 0.05$). Abbreviations: kg = kilograms; m = meter; BMI = body mass index; kg/m^2 = kilogram per meter squared ratio; SBP = systolic blood pressure; DPB = diastolic blood pressure; mm Hg = millimeters of mercury; HR = heart rate; bpm = beats per minute.

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