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Effects of obesity on breathing pattern, ventilatory neural drive and mechanics

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ABSTRACT

The purpose of this study was to assess whether obesity induces changes in breathing pattern and ventilatory neural drive and mechanics. Measurements performed in 34 male obese subjects (BMI, $39 \pm 6\ kg/m^2$) and 18 controls (BMI, $23 \pm 3\ kg/m^2$) included anthropometric parameters, spirometry, breathing patterns, mouth occlusion pressure, maximal inspiratory pressure and work of breathing. The results show that spirometric flow (FEV_1% pred, FVC% pred) and maximal inspiratory pressure (P_{Imax}) were significantly lowers ($p < 0.001$) in obese subjects compared to controls. The (fR/VT) ratio was higher in obese subjects than in controls ($p < 0.001$). The increase in (fR/VT) was associated with an increase in the ratio of mean inspiratory pressure to maximal inspiratory pressure (P_I/P_{Imax}) and the duty cycle (T_I/T_{TOT}) ($p < 0.001$). The energy cost of breathing (W_{rest}/W_{crit}), which reflects the oxygen consumed by the respiratory muscle was greater in obese subject than in controls ($p < 0.001$) inducing an increase in the effective inspiratory impedance on the respiratory muscles. It is concluded that obese subjects show impairment in breathing pattern and respiratory mechanics as assessed by rapid shallow breathing leading to ventilatory failure.
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1. Introduction

Excessive adipose tissue around the chest wall can alter normal pulmonary function by reducing both expiratory reserve volume (ERV) and functional residual capacity (FRC) due to alterations in chest wall mechanics (Collins et al., 1995; Parameswaran et al., 2006). Furthermore, the mass loading of obesity decreases chest wall compliance and increase the work of breathing. Among causes of ventilatory failure, the breathing pattern has been shown to occupy a major role (Rochester, 1993) to rapid shallow breathing, or the ratio of breathing frequency to tidal volume (fR/VT) seems to be one of the predictive criteria of ventilatory failure outcome (Hayot et al., 1998). Mechanical impediments to breathing predispose to an increase in ventilatory drive (De Troyer et al., 1982; Burki, 1983) and respiratory muscle weakness which is a prominent feature of most diseases that predispose to ventilatory failure. In subjects with obesity, we hypothesized that alterations in the thoraco-pulmonary to mechanical properties modify the ventilatory response to a given neural output, breathing pattern (DeLorey and Babb, 1999), mouth occlusion pressure (Burki, 1984; Burki and Baker, 1984), maximal inspiratory strength and work of breathing (Rochester, 1993).

Studies on the respiratory muscles of obese individuals are rare and have produced conflicting results. Previous studies have demonstrated that, among obese individuals, the respiratory system is subjected to mechanical overload and that, when faced with this, some individuals increase the activity of their respiratory muscles (Wannamethee et al., 2005), although few studies have related body weight to breathing pattern, ventilatory neural drive and mechanics. Several studies have evaluated the relation of waist circumference (WC) and waist-to-hip ratio (WHR) to pulmonary function testing variables (Santana et al., 2001) and respiratory muscle function (Wannamethee et al., 2005). This study aimed to determine the predictability of BMI for pulmonary function, breathing pattern, ventilatory neural drive and mechanics in obese subjects.

2. Materials and methods

2.1. Protocol

The entire experiment was performed in the afternoon in all subjects. The subjects first underwent spirometric measurements at rest. They were then seated and, after a period of familiarization with the experimental equipment (mouthpiece and nose clips), ventilatory and pressure parameters were recorded for 5 min. At least 10 occlusions were performed for each subject, at the rate of

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2–3 per minute. After 5 min of rest, P_{Imax} were measured. Measurement was performed in the sitting position.

2.2. Subjects

This study was carried out in 34 male obese subjects (BMI, $39 \pm 6\,kg/m^2$) to 18 control males' subjects (BMI, $23 \pm 3\,kg/m^2$). Subjects provided verbal and written informed consent in accordance with the guidelines established by the institutional review board. The experimental procedures complied with the ethical standards of the 1975 Helsinki Declaration. Obese subjects (51 ± 7 years) were asked to rest in a chair for approximately 10 min after arriving to the laboratory. Obese subjects had no history of obstructive sleep apnea, lung disease history of asthma, cardiovascular disease, or musculoskeletal abnormalities or any other serious illness and they did not currently smoke and were clinically stable at the time of evaluation. Subjects not meeting these guidelines were excluded. The control group consisted of sedentary male subjects (48 ± 12 years) with no history of cardiovascular or pulmonary disease or smoking.

2.3. Body composition

The anthropometric measurements included height, weight. Subjects were measured in light clothing without shoes. Height and weight were both measured standing. Height was measured with a Harpenden stadiometer to the last complete 0.1 cm and weight with a Soehnle digital electronic scale to the last complete 0.1 kg. BMI (in kg/m^2) was calculated for each subject.

2.4. Pulmonary function testing

Spirometric test were performed using a CPX/D System (Medical Graphics Corp., St Paul, MN, U.S.A.), with flow measurement using a calibrated pneumotachograph (Fleisch No. 3; Fleish, Lausanne, Switzerland). The subject completed at least three acceptable maximal forced expiratory maneuvers; technical procedures, acceptability and reproducibility criteria were those recommended by the American Thoracic society (ATS, 1995)

Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and peak expiratory flow (PEF) were recorded at body temperature and ambient pressure, and saturated with water vapor (BTPS). Predictive values were taken from Roca et al. (1998)

2.5. Occlusion pressure

The subjects were asked to breathe quietly, with the nose occluded, through a mouthpiece connected to the pneumotachograph (Fleish, Lausanne, Switzerland) with a two-way low-resistance breathing valve ($0.9\,cmH_2O\,L^{-1}\,s$, dead space of 50 ml, model 9340 occlusion valve, Hans Rudolph Inc., Kansas City, MO, U.S.A). During the exhalation phase of breathing, a balloon was rapidly inflated in the inspiratory limb of the breathing circuit to occlude the subsequent inspiratory flow. It was closed during exhalation and automatically opened about 150 ms after the onset of the subsequent inspiration. Occlusion pressure ($P_{0.1}$) was measured with a differential pressure transducer (Druck, LPM 9000 series, $\pm 50\,cmH_2O$, Leicester, England). The balloon was inflated with helium from a small gas cylinder, and the valve was controlled manually with a small switch. The subject was asked to continue to breathe normally despite the occlusions. After this manoeuvre was repeated 10–15 times over a period of 3 min, testing was completed. The subject wore headphones and listened to music to dampen any noise from the switching device controlling the balloon, and could see neither the occlusion valve nor the operator and therefore was unable to anticipate neither airway occlusion nor change in respiratory pattern. The analysis portion of the computer program displayed flow, volume, and pressure waveforms values. The Labview interface (Labview, National Instruments Corporation, Austin, TX, U.S.A.) which provided a visual feedback was used to identify the onset of inspiration (where pressure crossed $0\,cmH_2O$). Mouth occlusion pressure was measured at the mouth 100 ms after the onset of inspiration. Inspiratory time (T_I) and total time (T_{TOT}) were measured for the breath immediately preceding the occlusion manoeuvre.

Measurement of $P_{0.1}$ was introduced by Whitelaw and Derenne (1993) because the parameter is measured at zero flow and is thus independent of respiratory system compliance and resistance, it is an estimate of the neuromuscular drive to breathe. High $P_{0.1}$ values reflect increased neuromuscular activation of the respiratory system and indicate a strong likelihood of inspiratory muscle fatigue (ATS/ERS, 2002).

In obese subjects, when supine, gravity similarly would be expected to increase the mass effect of the abdomen on the abdominal surface of the diaphragm. Although mass loading and reduced compliance of the respiratory system are the presumptive causes of the restrictive lung disease that is found in obese subjects when seated, neither TLC nor FRC show the expected further fall when recumbent (Watson and Pride, 2005). Occlusion pressure $P_{0.1}$, is dependent on the contractile state and function of the respiratory muscles and consequently on the lung volume at which it is measured. Sitting position not influence occlusion pressure if $P_{0.1}$ was assessed at the level of functional residual capacity (FRC) (Whitelaw and Derenne, 1993).

The valve was connected by the expiratory circuit to a breath-by-breath automated exercise metabolic system (CPX, Medical Graphics Corp, MN, U.S.A.). Expired gases were analysed for oxygen with a zirconia solid electrolyte analyser and for carbon dioxide with an infrared analyser. The CPX continuously measured oxygen uptake, carbon dioxide output, and respiratory exchange ratio. Before each test, the gas analysers were calibrated with two gas mixtures of 16% O_2 and 4% CO_2. The data were averaged during the last 30 s of each load over an integral number of breaths.

Subjects were seated in comfort, breathing quietly during 2–3 min. After a stable respiratory ratio was attained, the following parameters were determined from an average of 30 s during 5 min: minute ventilation ($\dot{V}E$), tidal volume (VT), breathing frequency (fR), inspiratory time (T_I), expiratory time (T_E), and total time of respiratory cycle (T_{TOT}).

2.6. Maximal inspiratory pressure

At rest, maximal inspiratory pressure (P_{Imax}) was measured at the functional residual capacity (FRC) on seated subjects at rest, with a differential pressure transducer (Druck, LPM 9000 series, $\pm 350\,cmH_2O$, Leicester, England) using the technique of Black and Hyatt (1969). Subjects were asked to perform a maximal inspiratory effort against an occluded airway and to maintain it for at least 1 s. Maneuvers were made until three technically satisfactory and reproducible measurements were obtained (variation <5%). The highest score was kept for analysis.

2.7. Derived parameters

The breathing pattern was determined from an average of 10 respiratory cycles: tidal volume, inspiratory time (T_I), and total time of the respiratory cycle (T_{TOT}) were measured. We then calculated respiratory frequency (fR); minute ventilation ($\dot{V}E$); the duty cycle (T_I/T_{TOT}), which represents the time fraction during which the inspiratory muscles are in motion (index of the respiratory rhythm); and mean inspiratory flow (VT/T_I), which corresponds to an inten-

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