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Mini-Symposium: Obesity and the Respiratory System

Obesity and its impact on the respiratory system

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EDUCATIONAL AIMS

The reader will come to appreciate that:

- Evidence for detrimental effects on respiratory function during childhood are evident in the literature but do not appear to be of the same magnitude as that encountered in adults.
- Increased adiposity may influence lung volumes even in mildly obese subjects.
- Lung compliance is reduced in obese subjects
- Expiratory flow limitation may be seen with methacholine challenge in non-asthmatic obese subjects.
- Obese children achieve peak exercise earlier at a lower respiratory quotient and at a lower workload.

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SUMMARY

Obesity has complex and incompletely understood effects upon the respiratory system in childhood, which differs in some aspects to those seen in adults. There is increasing evidence that excess adiposity will impact negatively upon static and dynamic respiratory function as measured through lung volumes, lung compartment mechanics, measures of airway function and exercise capability to varying degrees. Further information is needed to better understand the effects in children, and the importance of onset and duration of obesity on subsequent outcomes. Consensus about how best to express adiposity is also an essential part of this process and fat distribution is another important factor. From a clinical standpoint this creates challenges in distinguishing a deconditioned obese young person from a non-atopic asthmatic because of symptom overlap and lung function testing results, including responses seen during airway challenges. There is evidence to support the role of weight loss in achieving normalisation of lung function parameters, but as always with obesity there are enormous challenges in realising this goal for many subjects.

INTRODUCTION

Childhood obesity has reached epidemic proportions in many parts of the world [1], with current World Health Organisation estimates that approximately two billion people worldwide are either obese or overweight. This is a growing problem for both developed and developing countries where data is available [2–5]. There is also data to suggest that this problem is not limited to the school age population, but is starting to affect pre-school children in some parts of the world [2]. Childhood obesity has been shown to track through to adulthood [6], and influences not only later adult cardiovascular health [7], but multiple other organ systems, including the respiratory system [8]. Obesity has been shown to increase the risk of respiratory symptoms in adults and in school aged children, in the absence of known respiratory illness [9–11]. Furthermore, obesity is an important risk factor for asthma [12], which itself affects approximately 15% of children worldwide [13].

This article will discuss the changes that occur in respiratory function tests in the setting of obesity. Specifically, emphasis will be placed upon changes detected by both routine tests, such as

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spirometry and plethysmography, available within most paediatric lung function laboratories, and more specialized tests, typically limited to the research setting or specialist clinical laboratories.

As with many aspects of paediatric medicine, understanding of the pathophysiology is largely based on available adult literature, where considerable work has occurred into the effects of obesity on different aspects of lung function. By contrast the available paediatric literature is less, and in part may reflect the challenges of obtaining reproducible measurements in younger subjects for some of the tests discussed. The available paediatric literature is summarized in Table 1. Where possible, comparison of adult to paediatric data will be made, and differences highlighted. Finally, future directions will be proposed to try and stimulate the research required to better understand the effects of obesity on paediatric respiratory health and its complex interactions with co-existing respiratory disease.

The simplest method to measure obesity is body mass index (BMI) which is defined as mass (kg)/height (metres)^2. In adults a fixed threshold for obesity is used, defined as a BMI > 30 kg/m^2. Whilst this may be appropriate for adolescents [6], it is not used in younger children due to the fact that BMI changes through childhood and is age and gender specific. In younger children centile charts are used and overweight is considered to include children with a BMI$>$ 85% for gender and age, whilst obesity is considered to include a BMI $>$95%.

EFFECTS ON LUNG VOLUMES

Spirometry provides a measure of inspired and expired lung volume. Measurements of the absolute lung volumes, residual volume (RV), functional residual capacity (FRC) and total lung capacity (TLC) require additional techniques such as plethysmography, inert gas washout or gas dilution methods [14].

Across the adult obesity literature, the most common abnormality reported for lung volumes is a reduction in the functional residual capacity [FRC]. FRC reflects resting end expiratory lung volume and is the equilibrium lung volume determined by the balance of the elastic recoil of the lung and the normal tendency of the chest wall to spring outwards [15]. In obesity, accumulation of adipose tissue around the rib cage, abdomen and within the visceral cavity produces a mass loading effect on both the thorax and within the upper abdomen by limiting the outward movement of the chest wall and downward movement of the diaphragm, respectively. The resultant effects of mass loading were investigated in studies performed 50 years ago by Sharp *et al.* [16] by placing shot bags on the chest and abdomen of healthy subjects and examining the change in static pressure-volume curves and resting lung volume (FRC). Both thoracic and abdominal mass loading were found to contribute to their subsequently observed changes in actual obese subjects [16]. Teasing out the relative contributions of these two factors is challenging.

Some research groups, using dual-energy X-ray absorptiometry (DEXA) to quantify extent and distribution of fat distribution, have found it difficult to separate out the contributions of thoracic wall and abdominal fat [17]. Others, however, using differing methods, have described a detectable greater effect of thoracic wall fat on TLC (plus FEV1 and FVC) and abdominal wall fat on FRC, in mildly obese adult subjects [18].

In adults the inverse relationship between FRC and BMI appears to be exponential, and detectable even in overweight adult individuals [19]. This is accompanied by a corresponding decrease in expiratory reserve volume (ERV, Figure 1). In extremes of obesity resting end expiratory lung volume approaches residual volume (RV). In the original study this happened earlier for some obese individuals. In contrast, relatively smaller changes in total lung capacity (TLC) and residual volume (RV) are seen, such that TLC remained within the lower part of the normal range for the vast majority of adults ($>$90%) [19]. As ERV falls, an accompanying increase in inspiratory capacity (IC) is observed. The exact reason for this reduction in TLC is unclear but several reasons have been postulated, including reduced downward movement of the diaphragm with increased abdominal mass [20], and deposition of fat in the subpleural space (i.e. subpleural fat pads, described in other conditions [21] but not obesity to date). In the paediatric literature, decreased FRC values have been shown directly [22] and indirectly (decrease in ERV and increased IC) [23] but not in all studies [24–27]. In the study by Li et al, a significant negative correlation between FRC and degree of obesity was described, based on DEXA-based assessment of obesity [22].

EFFECTS ON LUNG MECHANICS

The methodology used to assess lung mechanics is more specialized and seldom used in the routine paediatric or adult lung function laboratory. A detailed explanation of the techniques used is beyond the scope of this review, but a brief background is provided prior to discussing available literature. Tests of respiratory system mechanics may be passive or dynamic in nature. Passive respiratory mechanics are measured during spontaneous breathing using occlusion techniques. Occlusion of the airways at lung volumes above the resting lung volume relaxes the respiratory muscles by stimulating the slowly adapting stretch receptors and the vagally-mediated Hering-Breuer inflation reflex. Passive tests performed in this way assess the mechanical properties of the entire respiratory system. Reduced compliance (or increased stiffness) of the respiratory system is reported in obese subjects [28,29]. If passive tests are accompanied by simultaneous measurement of transpulmonary pressure (using an oesophageal balloon), then respiratory system compliance can be partitioned into lung and chest wall components. Lung compliance appears to be reduced in obesity [29–32], and in one study the same exponential relationship with BMI was seen, as described previously for FRC and RV (RV data not shown) [31]. Several potential mechanisms for reduced lung compliance have been postulated [20] including atelectasis due to closure of dependent airways, increased alveolar surface tension with reduction in FRC, and increased pulmonary blood flow.

There is, however, less consensus about whether chest wall compliance is altered, with some studies reporting normal findings and another reporting a reduction [28]. This lack of agreement may reflect the methodological challenges of obtaining accurate assessments of chest wall compliance, due to the need for complete relaxation and inactivity of the respiratory muscles. This may not be achieved during spontaneous breathing measurements in a plethysmograph, but is feasible under anaesthesia. Unfortunately, in anaesthetized subjects altered body position (i.e. supine not standing) may be a confounding factor when interpreting results [33]. The possible mechanisms for this reduction in chest wall compliance remain poorly understood. In the original mass loading experiments of Sharp *et al* the external thoracic mass loads in healthy subjects shifted the pressure-volume curve to the right, but did not change its shape [16], consistent with unchanged chest wall compliance but an increased inspiratory threshold effect [20]. In other words, once this increased threshold for inspiration is overcome the chest wall behaves normally.

Measurements of dynamic lung mechanics are based on analysis of dynamic waveforms obtained either during normal (tidal) breathing or via imposed oscillatory waveforms. The Forced Oscillation Technique (FOT) is an example of the latter approach and reports parameters such as respiratory system resistance (Rrs) and reactance (Xrs) which reflect airway caliber and lung stiffness, respectively [34]. Adult studies have reported increased Rrs and Xrs in obese subjects [35,36]. However, when interpreting these changes