

Dyspnea in overweight children: Is it asthma?

Richard M. Schwartzstein, MD,^{a,c} and Diane R. Gold, MD, MPH^{b,c}

Key words: Asthma, dyspnea, obesity, overweight, children, deconditioning

In a Dutch birth cohort study published in this issue of the *Journal*,¹ children had an increased risk of bronchial hyperresponsiveness or dyspnea at age 8 years if they had a high body mass index (BMI) at 6 to 7 years of age with or without increased BMI at an earlier age. Taking advantage of the longitudinal nature of their study of 3,756 children, the authors also report the encouraging finding that children who had a high BMI at a young age but whose BMI returned to normal did not have increased risk either of bronchial hyperresponsiveness or dyspnea. As always, extrapolation from an observational study to early-life interventions should be done cautiously. It is not known whether children whose BMI decreases without any known deliberate intervention are the subset of children who might benefit from BMI screening or from weight-reduction programs. Programs for screening and early intervention in obesity abound, but further prospective evaluation of their success in reducing weight and obesity-associated respiratory symptoms is needed.²

But what does overweight-associated dyspnea signify in the children from this birth cohort, and is dyspnea a symptom of asthma, as the investigators suggest? Mouse model data suggest that obesity can lead to bronchial hyperresponsiveness.³ Airway narrowing can occur through obesity-associated lung and tidal volume reduction, low-grade systemic inflammation, or obesity-related changes in adipose-derived hormones, including leptin and adiponectin. Nevertheless, studies of adults in which data suggest that asthma might be overdiagnosed in obese adults with dyspnea are accumulating. Misdiagnosis of asthma can result in inappropriate prescription of short-acting bronchodilators or corticosteroids in persons who are short of breath for obesity-related dyspnea that does not represent reversible airway obstruction. In a study of 16,171 participants (≥ 17 years of age) from the Third National Health and Nutrition Examination Survey, the highest BMI quintile (ie, the most obese participants) was associated with the greatest risk of self-reported asthma, bronchodilator use, and dyspnea with exertion (odds ratio, 2.66; 95% CI, 2.35-3.00).⁴ The highest BMI quintile, however, had the lowest risk for significant airflow

obstruction defined as percent predicted FEV₁ of less than 80%.⁴ A Canadian study demonstrated no objective evidence for asthma in a third of 540 adults with doctor-diagnosed asthma, regardless of whether they were obese.⁵ In a small study of 18 obese African American women with dyspnea and a doctor's diagnosis of asthma, only 1 demonstrated airway reversibility, and only 8 of 18 demonstrated methacholine responsiveness.⁶

Despite its potential lack of specificity in predicting reversible airway obstruction, dyspnea has been considered a possible asthma symptom not only by Scholtens et al¹ but others as well. In fact, the National Heart, Lung, and Blood Institute 2007 asthma guidelines⁷ recommend consideration of a diagnosis of asthma and performance of spirometry if there is a history of recurrent difficulty in breathing or recurrent chest tightness, even without a history of wheeze. However, the use of a history of difficulty in breathing or "dyspnea" in the epidemiologic diagnosis of asthma can be problematic, particularly in the epidemiologic study of children, when one is relying on parental interpretation of the perceptions of the child. A symptom, particularly dyspnea, in its purest form is a sensation reported by an individual; the sensation might be uncomfortable, and the term "symptom" connotes that the individual has interpreted the sensation as abnormal.⁸ Only the individual experiencing the sensation can say that it is definitely present. Seven-year-old children can, of course, report symptoms of chest tightness or shortness of breath to their parents, but evaluation of the symptom of dyspnea in young nonverbal children is more questionable. Physicians rightly rely on parents to report whether infants or toddlers are in general distress or are "not themselves." However, for younger nonverbal children, parental report of physical signs (eg, increased respiratory rate, wheeze, and use of accessory muscles of ventilation) is likely to be more reliable than report of a specific symptom, such as dyspnea.

Definition of asthma through the reporting of the prescription of inhaled corticosteroids for respiratory or lung problems can also be problematic. Because the prevalence of asthma is high in children and young adults and the side effects of inhaled bronchodilators and inhaled steroids in the short-term are relatively few, a therapeutic trial of these medications is commonly used in young persons with respiratory complaints. Nevertheless, formal testing of these individuals frequently demonstrates the absence of asthma.^{5,9}

Dyspnea, or breathing discomfort, is a complex symptom comprising multiple qualitatively distinct sensations,⁸ which might provide insight into the underlying physiologic abnormalities producing the sensation.¹⁰⁻¹² The dyspnea of asthma might be the consequence of stimulation of pulmonary receptors (caused by inflammation or bronchoconstriction) manifesting as a sensation of "chest tightness,"¹³⁻¹⁵ increased work or effort of breathing caused by increased airway resistance,^{13,15} or a sensation of air hunger or an inability to get a deep breath, which might reflect hyperinflation.¹⁶

From ^athe Division of Pulmonary, Critical Care and Sleep Medicine, Department of Medicine, Beth Israel Deaconess Medical Center; ^bChanning Laboratory, Department of Medicine, Brigham and Women's Hospital; and ^cHarvard Medical School, Boston, Mass. Disclosure of potential conflict of interest: R. M. Schwartzstein has provided expert testimony on the topic of pulmonary embolism. D. R. Gold has received research support from the National Institutes of Health and the Environmental Protection Agency and is a member of the American Thoracic Society.

Received for publication April 24, 2009; accepted for publication April 28, 2009.

Reprint requests: Richard M. Schwartzstein, MD, Beth Israel Deaconess Medical Center, Division of Pulmonary Medicine, Kirstein Building, Basement Rm 23, 330 Brookline Ave, Boston, MA 02215. E-mail: rschwartz@bidmc.harvard.edu.

J Allergy Clin Immunol 2009;123:1319-20.

0091-6749/\$36.00

© 2009 American Academy of Allergy, Asthma & Immunology

doi:10.1016/j.jaci.2009.04.034

In contrast to the qualities of dyspnea associated with asthma, cardiovascular deconditioning produces dyspnea characterized as “breathing heavy,” “gasping,” and “breathing more.”¹² Obese individuals might experience dyspnea caused by poor cardiovascular fitness (often resulting from a sedentary lifestyle), reduced compliance of the chest wall, increased airways resistance from narrowing of airways caused by the weight of the chest wall (particularly during exhalation), and hypoxemia caused by ventilation-perfusion mismatch resulting from atelectasis at the lung bases. Even patients with documented asthma might misconstrue dyspnea caused by poor fitness as exercise-induced asthma. Using formal cardiopulmonary exercise testing, Garfinkel et al¹⁷ demonstrated that individuals with asthma who believed they were limited by exercise-induced asthma were, in fact, experiencing poor fitness. Studies in children and young adults have shown that patients with asthma often are less physically fit than their peers.^{18,19}

Given these data, one might ask whether asthma is leading to a sedentary existence and causing a high BMI rather than whether obesity is causing asthma. The failure of Scholtens et al¹ to find a significant relationship between BMI and wheeze, BMI and inhaled corticosteroids, and BMI and rest dyspnea might support the hypothesis that the dyspnea being reported was primarily caused by factors other than asthma. The observation that children whose BMIs normalized no longer had an increased risk of dyspnea at age 8 years is consistent with the hypothesis that obesity, cardiovascular deconditioning, or both was the cause of their respiratory discomfort.

The cross-sectional relationship of high BMI at age 6 to 7 years with bronchial hyperresponsiveness at age 8 years is supportive of an association of obesity with asthma, but the directionality of the relationship remains uncertain, given the absence of strong evidence of a relationship of early-life increase of BMI with bronchial hyperresponsiveness at age 8 years. In the final analysis there are still many questions to be answered about the relationship between obesity and asthma. Future studies that focus on the prevalence of dyspnea in large populations would be well served by asking more explicit questions about the quality of the dyspnea experienced.

REFERENCES

- Scholtens S, Wijga AH, Seidell JC, Brunekreef B, de Jongste JC, Gehring U, et al. Overweight and changes in weight status during childhood in relation to asthma symptoms at 8 years of age. *J Allergy Clin Immunol* 2009;123:1312-8.
- Kipping RR, Jago R, Lawlor DA. Obesity in children. Part 1: epidemiology, measurement, risk factors, and screening. *BMJ* 2008;337:a1824.
- Shore S. Obesity and asthma: possible mechanisms. *J Allergy Clin Immunol* 2008;121:1087-95.
- Sin DD, Jones RL, Man SF. Obesity is a risk factor for dyspnea but not for airflow obstruction. *Arch Intern Med* 2002;162:1477-81.
- Aaron SD, Vandemheen KL, Boulet LP, McIvor RA, Fitzgerald JM, Hernandez P, et al. Overdiagnosis of asthma in obese and nonobese adults. *CMAJ* 2008;179:1121-31.
- Waggoner D, Stokes J, Romero F, Casale TB. Dyspnea and obesity in African American women. *Ann Allergy Asthma Immunol* 2008;101:644-5.
- National Heart, Lung, and Blood Institute. Guidelines for the diagnosis and management of asthma (EPR-3). Available at: www.nhlbi.nih.gov/guidelines/asthma.
- American Thoracic Society. Dyspnea: mechanisms, assessment and management. ATS Consensus Statement. *Am J Respir Crit Care Med* 1999;159:321-40.
- Chevalier B Sr. The role of the methacholine inhalation challenge: avoiding a misdiagnosis of asthma. *J Respir Dis* 2001;22:153-60.
- Simon PM, Schwartzstein RM, Weiss JW, Fencel V, Teghtsoonian M, Weinberger SE. Distinguishable types of dyspnea in patients with shortness of breath. *Am Rev Respir Dis* 1990;142:1009-14.
- Elliott MW, Adams L, Cockcroft A, MacRae KD, Murphy K, Guz A. The language of breathlessness. Use of verbal descriptors by patients with cardiopulmonary disease. *Am Rev Respir Dis* 1991;144:826-32.
- Mahler DA, Harver A, Lentine T, Scott JA, Beck K, Schwartzstein RM. Descriptors of breathlessness in cardiorespiratory diseases. *Am J Respir Crit Care Med* 1996;154:1357-63.
- Moy ML, Woodrow Weiss J, Sparrow D, Israel E, Schwartzstein RM. Quality of dyspnea in bronchoconstriction differs from external resistive loads. *Am J Respir Crit Care Med* 2000;162:451-5.
- Binks AP, Moosavi SH, Banzett RB, Schwartzstein RM. “Tightness” sensation of asthma does not arise from the work of breathing. *Am J Respir Crit Care Med* 2002;165:78-82.
- Killian KJ, Watson R, Otis J, St Amand TA, O’Byrne PM. Symptom perception during acute bronchoconstriction. *Am J Respir Crit Care Med* 2000;162:490-6.
- Lougheed MD, Lam M, Forkert L, Webb KA, O’Donnell DE. Breathlessness during acute bronchoconstriction in asthma. Pathophysiologic mechanisms. *Am Rev Respir Dis* 1993;148:1452-9.
- Garfinkel SK, Kesten S, Chapman KR, Rebuck AS. Physiologic and nonphysiologic determinants of aerobic fitness in mild to moderate asthma. *Am Rev Respir Dis* 1992;145:741-5.
- Ludwick SK, Jones JW, Jones TK, Fukuhara JT, Strunk RC. Normalization of cardiopulmonary endurance in severely asthmatic children after bicycle ergometry therapy. *J Pediatr* 1986;109:446-51.
- Orenstein DM, Reed ME, Grogan FT Jr, Crawford LV. Exercise conditioning in children with asthma. *J Pediatr* 1985;106:556-60.