

for 76% of the variance in the original data. These were reorganized to be compatible with present guidelines and reports resulting in the following domains with weights and maximum points allotted: days of symptoms and albuterol use (15%, 0–3 points), nights of symptoms and albuterol use (15%, 0–3 points), controller treatment (25%, 0–5 points), lung function (15%, 0–3 points), and exacerbations (includes oral corticosteroid burst and hospitalizations; 30%, 0–6 points). Final scores ranged from 0 to 20. At the ACE enrollment, the mean CASI score was 6.2 (SD = 3.0) and decreased by 23% after 3 weeks of guidelines-based intervention before randomization, mainly due to lower symptoms. After randomization and the final ACE visit 1 year later, the CASI score remained stable but redistributed to be higher in medication use and lower in symptoms. CASI was also more stable between visits compared with ACT. When externally validated by using data from ICATA, the CASI demonstrated a 0.67-point improvement in those on omalizumab and a 32% greater magnitude of effect compared with symptom days alone.

CONCLUSIONS. CASI provides an instrument to measure asthma severity as composite measure of control (risk and impairment) and the treatment required to achieve it.

REVIEWER COMMENTS. This is the first composite asthma index to account for future risk and impairment along with the level of treatment, resulting in a comprehensive measure of severity that follows the *Expert Panel Report—3* guidelines. Given the stability in the score over time, it is unique in that it can discriminate those with severe asthma even when well controlled under guidelines-directed therapy. CASI also shows greater effect sizes compared with symptom days alone because it measures the multidimensional aspect of asthma. This makes it useful in studies of new medications and changes in environmental exposure. It will also likely be practical in a clinical setting to help highlight risk and impairment when making management decisions. Additional validation in other populations is needed, and determining what constitutes an elevated CASI score or a clinically significant difference in scores remains an issue.

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Monitoring Pulmonary Function During Exercise in Children With Asthma

van Leeuwen JC, Driessen JM, de Jongh FH, et al. *Arch Dis Child*. 2011;96(7):664–668

PURPOSE OF THE STUDY. Exercise-induced bronchospasm (EIB) is defined as acute, reversible bronchoconstriction induced by exercise. Two variations have been described: one with

symptoms occurring during exercise and another with symptoms occurring after exercise. How often does each of these patterns occur in children with EIB?

STUDY POPULATION. The study included 30 Children with asthma (mean age 12.3 years) and a history of exercise induced symptoms.

METHODS. Participants underwent an exercise test of 12 minutes at 80% predicted maximum heart rate on a treadmill inside an ice rink where the air was cold and dry. Pulmonary function was measured before and each minute during exercise and at 1, 3 and 5 minutes after exercise. If EIB occurred (fall in FEV₁ >15% from baseline), exercise was terminated and albuterol administered.

RESULTS. EIB was revealed in 19 of the 30 subjects. In 12, EIB occurred during exercise between 6 and 10 minutes (mean, 7.75 minutes) with a mean fall in FEV₁, 1 minute after cessation of exercise, of 34% from baseline (range, 17%–54%). In 7, EIB did not occur until immediately after exercise with a mean fall in FEV₁ of 21% (range, 17%–34%).

CONCLUSIONS. In the majority of children with EIB in this study (ie, 12 of 19), bronchoconstriction started during, and not after, a submaximal exercise test.

REVIEWER COMMENTS. There are clearly 2 patterns of EIB: 1 where patients develop symptoms during exercise and another where the symptoms do not begin until shortly after exercise. These 2 patterns may correlate with the 2 proposed mechanisms for EIB: (1) exercise induced drying of the respiratory mucosa leading to degranulation of mast cells and (2) exercise induced airway cooling followed by vasodilatation in the airways on rewarming. Also, exercise induces the release of several bronchodilating mediators, such as Prostaglandin E₂ and nitric oxide, and deep inspirations themselves may protect against bronchoconstriction. Thus there is a balance between bronchoconstrictor and bronchodilator influences. If the bronchoconstrictor influences predominate during exercise, this would cause symptoms during exercise. If the bronchodilator influences are able to compensate for the bronchoconstrictor influences during exercise, the symptoms would not occur until after exercise. Also of interest is the fact that in 11 of 33 children with asthma, who complained of exercise-induced asthma symptoms, the symptoms could not be reproduced by this exercise challenge. Some of these children may not have asthma, or may not have EIB, but it may also be that the conditions of the exercise challenge were not the same as those that induce symptoms during a “real life” challenge such as a soccer game. Such patients may still warrant a trial of preexercise albuterol or inhaled corticosteroids.

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