

Pulmonary Perspective

“Reactive Airways Disease”

A Lazy Term of Uncertain Meaning That Should Be Abandoned

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The terms “reactive airways” and “reactive airways disease” have crept into the clinical lexicon in recent years. They are being used as synonyms for asthma. The terms are widely used in case presentations involving outpatients and inpatients, and even patients in intensive care units. They are in particular commonly used in the pediatric setting. The problem is that “reactive airways” and “reactive airways disease” are highly nonspecific terms that have no clinical meaning. As such, we view these terms as unhelpful and potentially harmful, and we recommend that they not be used.

Patients are usually labeled with “reactive airways” if they have a history of cough, sputum production, wheeze, or dyspnea. Sometimes, however, the only prompt for a diagnosis of “reactive airways disease” is the possession by the patient of an inhaler of some sort. Most often, physicians who use the terms do not have pulmonary function test results for the patient. Certainly, it is very rare that patients have had measurement of airway reactivity to methacholine, histamine, or hypertonic saline. Therefore, armed only with symptoms referable to the airway, or with a history of inhaler use, the doctor will present on rounds or write in the chart, in letters, or in discharge summaries that the patient has “reactive airways disease.” Unfortunately, this diagnosis often goes unchallenged. In fact, increasingly the term is being commonly used among specialists in pulmonary medicine.

The term “reactive airways disease” needs to be distinguished from reactive airways dysfunction syndrome (RADS) and from airway hyperreactivity—two terms that have value and meaning in pulmonary medicine. RADS is a specific term coined by Brooks and coworkers (1) in 1985 to describe an asthma-like illness developing after a single exposure to high levels of an irritating vapor, fume, or smoke. Patients with RADS have methacholine airway hyperreactivity, but other pulmonary function tests may or may not be abnormal. Symptoms and airway hyperreactivity can persist for years after the incriminating exposure. RADS differs from occupational asthma in that it typically occurs after a single exposure without a preceding period of sensitization. It should be noted that not all experts agree that RADS is a real clinical syndrome (2), arguing that the entity is based on case reports that lack control groups and that usually lack preexposure pulmonary function assessment. However, the weight of current scientific evidence supports RADS as a distinct clinical entity, and the

disorder is currently recognized as distinct by the American Thoracic Society and the American College of Chest Physicians (3).

Airway hyperreactivity is also a specific term that means that the airways are hyperreactive to a variety of stimuli including methacholine, histamine, hypertonic saline, distilled water, exercise, or eucapnic hyperventilation (4). Hyperreactivity in this context means a bronchoconstrictor response at “doses” that normally have no bronchoconstrictor effect. Airway hyperreactivity actually encompasses both airway sensitivity (the dose of agonist at which the FEV₁ begins to fall) and airway hyperresponsiveness (the slope of the dose–response curve thereafter). Airway hyperreactivity is a characteristic of asthma and to a lesser extent of chronic obstructive pulmonary disease (COPD) (5), but has also been described in patients with allergic rhinitis (6), but no asthma, in cystic fibrosis (7), and even in irritable bowel disease (8). Thus, although airway hyperreactivity is a highly specific term with definite meaning, it is not a disease diagnosis; rather it represents a physiological abnormality of the airway. It is, however, an important component of the diagnostic criteria for asthma.

The use of the term “reactive airways disease” in part reflects the difficulty with establishing a diagnosis of asthma in some situations. In the pediatric setting, especially in very young children, the diagnosis of asthma may be problematic because the history is difficult to obtain, because good quality pulmonary function tests cannot be obtained, or because asthma is a diagnosis that carries a negative connotation for the patients. Thus, the term “reactive airways disease” may be used as a nonspecific term in clinical contexts ranging from asthma, to wheezy bronchitis, to viral bronchiolitis, or even to pneumonia. In adult medicine, we suspect that the term is popular because of instances in which physicians obtain a history of wheeze, sputum production, or inhaler use, but a formal diagnosis of asthma is not in the patient record. A formal diagnosis of asthma requires documentation of reversible airway obstruction or airway hyperreactivity in the setting of a typical history of asthma. Frequently, the physiological information is missing or elements of a typical asthma history are missing. In the absence of these findings, physicians will provide a label of “reactive airways disease” to convey that the patient has some sort of airway problem.

The problem with the term reactive airways or reactive airways disease is not just that they represent an annoyance to purists of terminology. The problem is that using the terms may provide physicians with a false sense of diagnosis security. Ascribing a label of reactive airways to a patient may be harmful in this context, because it may prevent work-up of the cause of the symptom complex that led to the diagnosis of reactive airways disease in the first place. These patients may actually have asthma, chronic bronchitis, emphysema, or even pneumonia. Treatment usually prescribed for these specific

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diseases may or may not be prescribed if the diagnosis is “reactive airways disease.” Overtreatment may also be a side effect of this diagnosis. We suspect that many patients with a diagnosis of “reactive airways disease” receive treatment with inhaled β -agonists or with inhaled corticosteroids. However, if the patient does not have asthma there is no evidence that these treatments benefit the patient.

Finally, the terms “reactive airways” and “reactive airways disease” are now making their way from the clinical lexicon to the clinical literature. Two recent publications have used the term “reactive airway disease” (9, 10). In one instance reactive airway disease was used as a summary term to describe patients with asthma and/or COPD; in the other it was used synonymously with airway hyperreactivity (10). We find this trend troubling because many patients considered to have “reactive airways disease” do not have asthma, and the vast majority of patients with reactive airways have never had their airway reactivity measured. We believe it essential to preserve the integrity of asthma and airway hyperactivity as diagnostic terms in the clinical literature. In fact, in the context of clinical research, we believe the use of the terms “reactive airways” and “reactive airways disease” will complicate research on asthma, especially for clinical epidemiologists who are investigating the current worldwide epidemic of asthma.

In summary, at best the diagnostic label “reactive airways disease” is an annoyance to those of us who want to maintain diagnostic clarity in our discipline. At worst, the term represents a form of diagnostic laziness that may cause harm to patients.

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