LETTERS TO THE EDITOR

Bronchial Inflammation in Smokers: Clarifying Terms

To the editor: In a recent editorial on early diagnosis of bronchial inflammation in smokers, Pacheco Galván[1] makes some statements that we feel are debatable. He says that a diagnosis of tobacco-related chronic bronchitis (CB) may lack specificity and place too much emphasis on the part played by tobacco, as only some smokers develop rapid deterioration of lung function. We believe that a diagnosis of tobacco-related CB is appropriate in smokers with symptoms of CB not caused by another disease. If CB in smokers who develop chronic obstructive pulmonary disease (COPD) is caused by inhaling tobacco smoke, and quitting smoking is the only measure that achieves a slower rate of decline in forced expiratory volume in 1 second (FEV_1), we believe that tobacco smoke is the main causative agent even though it does not act in the same way in different settings.[2] We do not find it surprising that the decline in FEV_1 varies in different patients with this disease, just as liver function varies in patients with alcohol-related hepatitis. Pacheco Galván goes on to say that there is a high level of synergy between the inflammation caused by asthma and that caused by tobacco smoke, consistent with the Dutch hypothesis that asthma and COPD have a common origin. Inhaling tobacco smoke, like inhaling silica dust, can cause CB but it has not been shown to causes asthma. It therefore seems incautious to maintain that both processes have the same origin because there is no origin or beginning before the disorder starts. We consider that although the clinical and histopathological signs of asthma and tobacco-related COPD may be similar, their etiology, response to treatment, and evolution are distinct.

Pacheco Galván concludes by saying that he thinks it would be advantageous to conduct sputum induction and bronchial hyperreactivity tests in all smokers with chronic respiratory symptoms and normal spirometric results, in order to identify subgroups of patients who would benefit from antiinflammatory treatment. He proposes the term "chronic airflow limitation syndrome" to describe this condition. We do not believe that sputum induction should be carried out on all smokers with chronic respiratory symptoms, because its usefulness and significance are not well established. Furthermore bronchial hyperreactivity lacks specificity and may be the result of airway inflammation and not its cause. The respiratory symptoms of many smokers will disappear when they give up smoking. In patients who do not give up, a test of inhaled corticosteroid use can serve to clarify the cause of airway inflammation. We agree with Snider[3] in preferring "chronic airflow obstruction" to "chronic airflow limitation" but doubt these terms serve to clarify anything, as they only name a functional disorder common to several chronic pulmonary diseases.

In the same issue of ARCHIVOS DE BRONCONEUMOLOGÍA, Díaz Lobato and Mayorales Alises[4] discuss a question of great interest: the term COPD itself. Inhaling tobacco smoke causes CB and pulmonary emphysema in a large number of smokers. The English term COPD (*enfermedad pulmonar obstructiva crónica*—or EPOC—in Spanish) was established to describe CB and pulmonary emphysema when they have caused chronic airflow obstruction.

We think that the term COPD may have been chosen, among other reasons, so that doctors would recommend bronchodilators on a regular basis to patients with this disorder, even though their symptoms and lung function do not improve and in spite of the fact that the Lung Health Study did not demonstrate that these drugs have any effect on the course of airflow obstruction.[2] We believe that COPD caused by inhaling tobacco smoke has more similarities in its etiology and response to treatment to occupational lung diseases caused by long-term inhaling of organic or inorganic materials than it has to asthma. Although recently there has been great pressure on doctors to treat COPD and asthma in the same way, we are very skeptical about the benefits of using asthma medicines in COPD. The histopathological anomalies of CB and pulmonary emphysema caused by inhaling tobacco smoke are as varied as those presented by patients with pulmonary silicosis. These variations are possibly due to such factors as the length of time and intensity of exposure to the causative agent or to patient characteristics. It seems appropriate to keep in mind the excellent maxim from classical medicine: "There are no diseases, only people who are ill."

"Chronic tobacco-related pneumonitis"—along the lines of the name given to liver inflammation produced by the intake of alcohol—would seem to us to be a more appropriate term for this occupational disease not contracted at work that is caused by inhaling tobacco smoke. (We use "occupational" in the sense of activity or pastime according to the dictionary of the Spanish Royal Academy.)

In our opinion the term COPD would be appropriate for chronic airflow obstruction of unknown cause.

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3. Snider GL. Nosology for our day. Its application to chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2003; 167:678-83.
4. Díaz Lobato S, Mayorales Alises S. Un solo nombre para una sola enfermedad. Arch Bronconeumol 2003;39:376-7.