Chronic Obstructive Pulmonary Disease: Is Social Injustice the Elephant in the Room?

When patients with exacerbations of chronic obstructive pulmonary disease (COPD) are admitted to my hospital, their day-to-day care is most likely to be assigned to the most junior member of the house staff. As an educator, I would like to believe that this is done deliberately to ensure that all trainees become experts in the management of an increasingly common cause of hospitalization. As a realist, I suspect it is because the disorder rates low on the priority list to most physicians including our trainees. The discharge summaries reflect this; after listing the formulaic interventions borrowed from asthma care, the discharge note rarely lists the most basic of disease parameters, the patient's FEV_1. By contrast, smoking status and smoking history are almost always noted. Others have made similar observations. When Damarla and colleagues surveyed the charts of patients hospitalized in their institution for the care of COPD, they could find spirometry recorded for fewer than one-third of the patients (as compared with echocardiograms noted for almost 80% of heart failure patients) (1). When they did spirometry in the remainder, 30% did not have obstructive spirometry.

Two decades ago, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) noted that COPD was expected to become the third most common cause of death on the planet and introduced a simple and coherent diagnostic definition of the disease (2). A patient could be considered to have COPD if he or she had a post-bronchodilator FEV_1/FVC ratio less than 0.70 that could not be improved and that appeared to be the consequence of chronic exposure. The impact on clinical care has been mixed. The Burden of Obstructive Lung Disease (BOLD) initiative reports that COPD remains underdiagnosed (3). This under recognition is not limited to poorly resourced countries. In my relatively affluent province with universal, government-funded health care, a recent survey found that fewer than one-third of patients meeting spirometric criteria for COPD had previously been diagnosed by their physicians (4). Clearly most clinicians have chosen not to screen the at-risk population with spirometry but have chosen to rely on symptoms in smokers or former smokers to guide them to the correct diagnosis. We can debate the importance of the resulting underdiagnosis, but more concerning may be the other consequence of ignoring simple diagnostic tools, which is the risk of overdiagnosis. It seems that primary care clinicians in many Western nations have come to regard long-term exposure to tobacco smoke and a patient-reported respiratory symptom as sufficient grounds to begin COPD treatment. When we do spirometry in individuals treated for COPD in primary practices of the United Kingdom, the Netherlands, Australia, New Zealand, and Canada, we find that between 31% and 60% do not have obstructive spirometry (5–9). I cannot think of any other common cause of death treated with such diagnostic indifference.

With the first GOLD publication, the authors were careful to point out that tobacco smoking was not the only cause of the disorder. This important perspective has been reexamined by Burney and colleagues (pp. 1353–1365), and their findings are detailed in this issue of the *Journal* (10). Using data from the BOLD initiative, the investigators explored the risk factors associated with a finding of COPD. Unsurprisingly, the strongest association for both men and women was a history of tobacco smoking. The remaining associations, expressed as population attributable risk, would puzzle most of our junior trainees. In order of importance, poor education levels, employment in a dusty workplace for more than a decade, low body mass index, and a history of tuberculosis were all associated with an increased likelihood of COPD. Notably absent from the list was exposure to biomass fuels such as the smoke of cooking fires. This list has implications for clinicians and researchers.

As clinicians, we might ask ourselves if this common cause of mortality is often treated with diagnostic indifference because it is most likely to appear in the poorly educated blue-collar worker who smokes. As we pause to assimilate this uncomfortable thought, we might ask if these findings have other important implications for care. If tobacco smoking is by itself only one of several factors associated with the development of COPD, how comprehensive is our care when we approach the patient with COPD? Can we be satisfied that we have done our jobs if we address smoking cessation without addressing ongoing workplace exposures? The list of possible contributing factors to COPD should also prompt us to consider the neglected phenotyping of COPD. Does chronic airflow limitation related to past tuberculosis behave the same way as chronic airflow limitation caused by tobacco smoking? Is the same treatment pathway warranted? Perhaps the tuberculosis-associated disease precludes the use of inhaled corticosteroids and should prompt us to search more diligently for bronchiectasis as a comorbidity requiring our attention (11). Our current guidelines offer relatively little phenotypic guidance.

For research scientists, this study provides further impetus to parse out the many different pathways that seem to result in an obstructive injury. It is safe to assume that poor educational attainment is a surrogate for socioeconomic disadvantage but by what mechanism or mechanisms does this occur? Nutritional factors come to mind and would fit nicely with low body mass index and, in other studies, low birth weight as factors associated with COPD (12). Thus far, studies of fish oils and vitamin D have tantalized but have not yielded convincing and important pathways and interventions (13–15).

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The findings by Burney and colleagues should be reviewed carefully and should prompt reconsideration of how we approach this increasingly common disorder. As with any study, some limitations should be noted. More than 40% of the potentially eligible subjects did not participate in this large scale, multinational examination of risk factors. We do not know if the nonrespondents differed significantly from the respondents, but one must wonder if, for example, those exposed to the smoke of biomass fuels were represented similarly in the respondent and nonrespondent groups. A disparity might account for the failure to find indoor air quality as an important factor in the development of COPD. In addition, the study methodology was limited to patient-reported exposures. The actual levels of exposure, prenatal issues, childhood respiratory history, and many other plausible factors were not and could not be considered by this project (16, 17).

The work of Burney and colleagues should prompt all to ask if our approach to COPD is sufficiently comprehensive and whether we have sought out all potentially modifiable risk factors that might reduce the risk of further lung function decline. And for our trainees just learning to tackle the problem of COPD, we must hope that such findings convince them that tobacco smoking is not the sole cause of COPD and that a smoking history by itself is insufficient grounds to make a COPD diagnosis. I am impressed by the political and scientific awareness and activism of many current trainees. One can only hope that identifying COPD as a disorder so profoundly influenced by social disadvantage will help to move this lethal problem higher on their list of priorities. ■

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