

Conflicts of interest

The authors have no conflicts of interest to declare.

References

1. Kallstrom TJ, American Association for Respiratory Care (AARC). AARC Clinical Practice Guideline. Oxygen therapy for adults in the Acute Care Facility-2002 revision & update. *Respir Care*. 2002;47:717–20.
2. Spoletini G, Alotaibi M, Blasi F, Hill NS. Heated humidified high-flow nasal oxygen in adults: mechanisms of action and clinical implications. *Chest*. 2015;March, <http://dx.doi.org/10.1378/chest.14-2871> [Epub ahead of print].
3. Dysart K, Miller TL, Wolfs MR, Shaffer TH. Research in high flow therapy: mechanisms of action. *Respir Med*. 2009;103:1400–5.
4. Gotera C, Díaz Lobato S, Pinto T, Winck JC. Clinical evidence on high flow oxygen therapy and active humidification in adults. *Rev Port Pneumol*. 2013;19:217–27.
5. Sotello D, Rivas M, Mulkey Z, Nugent K. High-flow nasal cannula oxygen in adult patients. A narrative review. *Am J Med Sci*. 2015;349:179–85.
6. Joseph L, Goldberg S, Shitrit M, Picard E. High-flow nasal cannula therapy for obstructive sleep apnea in children. *J Clin Sleep Med*. 2015 Jun 11, pii:jc-00442-14. [Epub ahead of print].
7. McGinley B, Halbower A, Schwartz AR, Smith PL, Patil SP, Schneider H. Effect of a high-flow open nasal cannula system on obstructive sleep apnea in children. *Pediatrics*. 2009;124:179–88.
8. Corley A, Caruana LR, Barnett AG, Tronstad O, Fraser JF. Oxygen delivery through high-flow nasal cannulae increase end-expiratory lung volume and reduce respiratory rate in postcardiac surgical patients. *Br J Anaesth*. 2011;107:998–1004.
9. Roca O, Pérez-Terán P, Masclans JR, Pérez L, Galve E, Evangelista A, et al. Patients with New York Heart Association class III heart failure may benefit with high flow nasal cannula supportive therapy: high flow nasal cannula in heart failure. *J Crit Care*. 2013;28:741–6.

S. Díaz-Lobato*, J.M. Alonso, J.M. Carratalá, S. Mayoralas
Hospital Ramón y Cajal, Neumología, Carretera de Colmenar Viejo, Km 9, 100, 28034 Madrid, Spain

* Corresponding author.
E-mail address: sdiazlobato@gmail.com (S. Díaz-Lobato).

21 July 2015 22 October 2015

<http://dx.doi.org/10.1016/j.rppnen.2015.10.008>
2173-5115/

© 2015 Sociedade Portuguesa de Pneumologia. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

COPD: From the stethoscope to the spirometer



Letter to the editor:

In the world, about 3 billion of people, predominantly women, young girls and small children, are exposed to household air pollution, secondary to indoor cooking with biomass and coal.¹ Because the combustion of solid fuels produces a mixture of air pollutants, from respirable particles to gases, this exposure is associated with chronic obstructive pulmonary disease (COPD), and this association is well documented.² During their lifetime, some women can be exposed to biomass smoke for 30–40 years, or 60,000 h of exposure.³ This can be the biggest risk factor for COPD in the world, even predominates in developing countries.⁴ In rural areas, 90% of households continue to use this highly polluting biomass fuel. In 2008, in the Portuguese region of Lisbon, the BOLD study showed a GOLD stage 1 or higher COPD prevalence of 9.2% in never-smokers.⁵

Despite the fact that, within never-smokers, COPD has been neglected and excluded in the majority of large studies,⁶ the association observed between the exposure to biomass smoke and COPD is as great as the risk of active smoking. The prevalence of COPD among never-smokers varies widely across the countries, but at least one fourth of patients with COPD are never-smokers. Many are women, in whom this disease is understudied.⁷ But cooking with biomass and coal, rather than a recent phenomenon, is an old problem, still persisting in rural areas of many developed countries, as in Portugal, Spain, Canada, Australia or

US. Thereby, we wonder why COPD is a disease only recently concern, and usually associated with tobacco epidemic.

In fact, the recognition of emphysema and chronic bronchitis, and the evolution of their knowledge, cover nearly four centuries, since the Bonets's description of "voluminous lungs", in 1679.⁸ In 1821, Laënnec, the inventor of the stethoscope, describes the emphysema as a disease that was "very little known" and "completely overlooked".⁸ However, since the discovery and use of the stethoscope, he wrote, he believed to be a much more common problem. With Laënnec begins the modern era of COPD. In that time smoking was rare, but urban atmospheric pollution, in particular coal smoke, and domestic pollution from open fireplaces, in houses with poor ventilation, was a well-known phenomena, as was the conditions of work in cotton factories. In 1868, Manchester's first Medical Officer of Health said that the normal condition of the working man of middle age in Manchester was bronchitic.⁹ Occupation, domestic pollution and atmospheric pollution were, in the 19th century, well known risk factors for chronic bronchitis and emphysema.

Until recently, the diagnosis of emphysema and chronic bronchitis was based on symptoms of dyspnea, cough and expectoration, and on physical examination of an enlarged chest. The diagnosis of emphysema, according to Ronald Christie, in 1944, should be considered certain in the presence of dyspnea on exertion and of insidious onset, not due to bronchospasm or left ventricular failure, in a patient with physical signs of emphysema. But by the time clinical signs are present, COPD is in a moderate or advanced stage,¹⁰ and we miss not only the early diagnosis but most diagnosis. Furthermore, before the CIBA Guest Symposium,

in 1959, and the American Thoracic Society Symposium, in 1962, there was much confusion regarding the diagnosis of chronic bronchitis and emphysema, and the concept of airflow obstruction was only introduced at that time.¹¹ Gradually, doctors and scientists concern with the pathology and the clinic moved toward the pathophysiology, giving rise to the actual concept of COPD.

Cigarette mass production began circa 1880, and their consumption has increased until now, but the association with COPD was only established once the latent effects of cigarettes became apparent, in the second half of the 20th century. With the progressive increase of smoking, initially solely in the developed countries, an easier access to the quantification of exposure, and a well-documented association to COPD, tobacco smoking became the most important risk factor for COPD. But COPD, even under the old label of emphysema or chronic bronchitis, was probably very common before tobacco smoking was widespread, although little recognized.

In 1846 Hutchinson invented the spirometer, so important to the diagnosis of COPD, but only recently the industry and technology made possible the emergence of handheld spirometers, accurate and inexpensive, and their spread in the primary care medicine, although there is a lack of accessibility in our country, as suggested by the BOLD study in Lisbon.⁵ FEV₁/FVC, more than defining obstruction, is a sensitive test for early stages of airflow limitation, and, when <70%, heralds the onset of rapid decline in FEV₁. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) program was initiated in 1998, and the first report issued in 2001.¹² Rather than a new disease, the generalization of these two tools, the spirometer and the GOLD consensus report, enabled showing COPD as a common disease and a leading cause of morbidity and mortality worldwide, giving to COPD importance and visibility.

However, the term COPD is more than a new label for an old disease: is a new concept, as proposed by the GOLD working definition; is chronic and common; is preventable and treatable; is a complex disease based on inflammation, his pathological hallmark; is a heterogeneous disorder, and also heterogeneous in its presentation. And exacerbations and comorbidities contribute to the overall severity. Any individual patient may have emphysema, or peripheral airways disease, or chronic bronchitis, or all of these conditions,¹³ but the dominant clinical feature is persistent and usually progressive airflow limitation, that is the hallmark of COPD.

Conflicts of interest

The author has no conflicts of interest to declare.

References

1. Nour A, Balmes J, Mehta S, Cheema U, Sood A. Chronic obstructive pulmonary disease secondary to household air pollution. *Semin Respir Crit Care Med*. 2015;36:408–21.
2. Zeng G, Sun B, Zhong N. Non-smoking-related chronic obstructive pulmonary disease: a neglected entity. *Respirology*. 2012;17:908–12.
3. Salvi S, Barnes P. Is exposure to biomass smoke the biggest risk factor for COPD globally? *CHEST*. 2010;138:3–6.
4. Liu Y, Lee K, Perez-Padilla R, Hudson N, Outdoor Mannino D. Indoor air pollution and COPD-related diseases in high and low-income countries. *Int J Tuberc Lung Dis*. 2008;12:115–27.
5. Bárbara C, Rodrigues F, Dias H, Cardoso J, Almeida J, Matos M, et al. Prevalência da Doença Pulmonar Obstrutiva Crónica em Lisboa, Portugal: estudo Burden of Obstructive Lung Disease. *Rev Port Pneumol*. 2013;19:96–105.
6. Lee S, Kim S, Kong K, Ryu Y, Lee J, Chang J. Risk factors for chronic obstructive pulmonary disease among never-smokers in Korea. *Int J COPD*. 2015;10:497–506.
7. Varkey A. Chronic obstructive pulmonary disease in women: exploring gender differences. *Curr Opin Pulm Med*. 2004;10:98–103.
8. Petty T. The history of COPD. *Intern J COPD*. 2006;1(1):3–14.
9. Warren C. The Nature. Causes of chronic obstructive pulmonary disease: a historical perspective. *Can Respir J*. 2009;16:13–20.
10. Petty T. COPD in perspective. *CHEST*. 2002;121:116s–20s. Available from: www.journal.publications.chestnet.org/data/Journals/CHEST/21978/116S.pdf
11. Fletcher C, Pride N. Definitions of emphysema, chronic bronchitis, asthma, and airflow obstruction: 25 years on from the Ciba symposium. *Thorax*. 1998;39:81–5.
12. The global initiative for chronic obstructive lung disease (GOLD); updated 2015. Available from: www.goldcopd.org/upload/users/files/GOLD_Report_2015_Feb18.pdf
13. Dantzer D, Pingleton S, Pierce J, Kronenberg R, Plummer A, Kerby G, et al., on behalf of the Task Group appointed by ATS Scientific Assembly on Clinical Problems. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD) and asthma. Reprinted from *Am Rev Respir Dis* 1987;136(1):1–21. Available from: www.thoracic.org/statements/resources/archives/standards-for-diagnosis-and-care-of-COPD-and-Asthma-patients.pdf.

A. Araújo

Respiratory Department, H. S^a Oliveira, Guimarães, Portugal

E-mail address: duartearaujojr@sapo.pt

<http://dx.doi.org/10.1016/j.rppnen.2016.02.008>
2173-5115/

© 2016 Sociedade Portuguesa de Pneumologia. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).