

5. Friedman PJ. Adult pulmonary ligament pneumatocele: a loculated pneumothorax. *Radiology*. 1985;155:575–6.
6. Ravin CE, Smith GW, Lester PD, McLoud TC, Putman CE. Post-traumatic pneumatocele in the inferior pulmonary ligament. *Radiology*. 1976;121:39–41.
7. Meza MP, Benson M, Slovis TL. Imaging of mediastinal masses in children. *Radiol Clin N Am*. 1993;31:583–604.

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<http://dx.doi.org/10.1016/j.rppnen.2016.10.003>
2173-5115/

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High-flow nasal oxygen is not an oxygen therapy device



Traditionally the oxygen therapy systems have been classified as low and high flow. Low-flow systems do not provide all the inspiratory flow demanded by the patient; do not ensure stable levels of FiO₂ and it is not possible to control the temperature and moisture of inspired gas. The low flow oxygen system most widely used is the nasal prongs. By contrast, high flow systems are able to provide the entire atmosphere breathed by the patient and do ensure a stable FiO₂ and it is possible to control the temperature and moisture of the inspired gas. The typical high flow oxygen system is the venturi mask.¹ In recent years, a new way of supplying patients with oxygen has appeared which in our opinion has been incorrectly called high-flow nasal cannula oxygen therapy (HFNC).² This therapeutic approach has a lot of physiological effects which makes it a really active treatment for patients with both acute and chronic respiratory failure; it is more than a simple oxygen delivery system. Several studies have shown that HFNC generates a low level of positive airway pressure, improves oxygenation, increases the end-expiratory lung volume, reduces airway resistance, increases functional residual capacity and alveolar recruitment and flushes nasopharyngeal dead space, thus helping to decrease the work of breathing. There is a better control of FiO₂ and the gas humidification ensures better patient tolerance and comfort. Due to a better mucociliary clearance, pulmonary defense mechanisms are restored.³ Many of these effects are similar to those produced by non-invasive ventilation. Several studies have shown its utility in patients with acute hypoxemic respiratory failure, in the post-extubation period, in palliative care, in patients with acute heart failure, in chronic airway diseases and its indications are still rising. It is used in critical care areas, in the emergency department, in wards and it is being used at home in COPD patients.^{4,5}

As we can see, oxygen plays a secondary role in this treatment. The name of high-flow oxygen therapy is confusing. Although the efficacy of improvement in respiratory gas exchange and effectiveness in outcomes has been demonstrated, we have not fully understood the main pathophysiological principles of this therapy. The role and importance of each of the mechanisms related to the

high-flow therapy, the end expiratory positive pressure, the flushing of nasopharyngeal dead space or the humidification and warming of the inspired air, has not been clarified yet. Up to now, we have not been able to identify the functional significance of each of the three mechanisms, maybe this is a question that will never be answered because in different clinical situations the relevance of these potential mechanisms changes. What is clear is that high-flow is not an oxygen therapy device. Most international groups working in this field use the term ‘‘Nasal High Flow’’ avoiding the term ‘‘oxygen’’ in its definition.

We think such an active treatment should have a name that reflects the effects of the treatment better, in order to avoid confusion. And that deserves some careful reflection. High-flow can be applied through nasal cannula or tracheostomy, so the term ‘‘nasal’’ should not be part of the name. Perhaps the name ‘‘Active High Flow’’ would be more accurate so as to clearly distinguish it from the conventional or ‘‘passive’’ high flow like the venturi system. The Venturi system is usually administered by mask and high flow by cannula, which could be another distinguishing feature. Taking into account the different but also relevant effects of the therapy, we might ask why not include the term ‘‘humidification’’ or ‘‘heating’’ to the words ‘‘high flow’’. The positive expiratory pressure effect could probably be more interesting to incorporate into the name. Although the pressure achieved is highly dependent on the individual patient and the interface used, is low in absolute value and, above all, not determined by the prescribed parameters of the equipment, we do have quite a lot about its relevance. Certainly, this therapy has demonstrated that can be useful in treating sleep apnea syndrome.^{6,7} Its effect on alveolar recruitment has been clearly shown by Corley et al.⁸ Roca et al.⁹ have also indirectly demonstrated the positive intrathoracic pressure reached with the HFNC, showing a reduction greater than 20% in the estimated inspiratory collapse of the inferior vena cava from baseline. Perhaps the provocative name of ‘‘High-flow positive pressure’’ would better define a treatment that gives the patient a high flow of heated and wet gas, with a level of positive pressure, and a controlled FiO₂. Anyway, the name of high-flow oxygen therapy would be restricted to the classical high flow system such as the venturi system. We think that a consensus on a more precise name is required.

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.

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21 July 2015 22 October 2015

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

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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,