



Letters to the Editor

Scientific Studies are Required to Validate the Indications for Long-Term Oxygen Therapy at High Altitudes[☆]

Se requieren estudios científicos para validar las indicaciones de oxigenoterapia crónica en la altitud

To the Editor:

The letter from Dr. Salvador Díaz-Lobato and Dr. Sagrario Mayoralas Alises published in *Archivos de Bronconeumología*¹ discuss about the criteria for long-term oxygen therapy (LTOT) and whether these criteria should change depending on the altitude at which the patient lives—an important issue in respiratory medicine, especially because a rather large portion of the population resides at moderate to high altitudes. Let us remember that, in Latin America, at least, many people live at moderate altitude. For example, 1 out of 4 Mexicans lives in metropolitan areas in the Valley of Mexico (2240 m), and half of the population (approximately 55 million people) live at more than 1500 m above sea level; in Peru, 1 out of 3 inhabitants lives above 2500 m, while in the metropolitan area of La Paz, Bolivia, a little more than 2 million people live at an altitude of 3600 m.

There is definitely a need for well-designed double-blind randomized clinical trials studies—designed like the Nocturnal Oxygen Therapy Trial Group (NOTT)² or the Medical Research Council (MRC)³ but conducted at different altitudes—to document which groups could benefit, in terms of survival, from being included in a LTOT program. Lacking such studies, we are marooned in uncertainty and speculation and, as a result, conserving ever-scarce financial resources may be the basis for deciding who should receive oxygen. For example, the deterioration seen with a certain PaO₂ level (<55 mm Hg, for instance) might be similar at any residence altitude, and in that situation—the one underlying most recommendations—prescribing oxygen to improve survival would be the right course of action no matter how many individuals meet the criteria. However, study groups differ in their adaptation to altitude, possibly for genetic reasons. Tibetan populations and the Aymara people of Peru, for example, differ in their response to hypoxemia at altitudes of around 4000 m, the latter showing a higher incidence of polycythaemia and pulmonary artery hypertension than the Tibetans, which most likely indicates a less adaptive response.⁴ The Tibetans not only have a lower PaO₂ than the Aymara people but also have increased vasodilator response, which explains their lower incidence of pulmonary artery hypertension.⁵ Altitude is fertile ground for the expression of natural selection,

which has followed at least 2 pathways with different degrees of “efficiency.”

The Díaz-Lobato and Mayoralas letter quotes Dr. Thomas Petty, who said, “If we were to apply the LTOT criteria in Denver, Colorado (1609 m above sea level), we would have to put the entire population on O₂,” to which there would be financial and logistical rather than physiological or clinical objections. The PLATINO study measured oxygen saturation in a population sample of individuals over 40 years of age in Montevideo (35 m above sea level), Caracas (950 m), and Mexico City (2240 m).⁶ The main determinants of SpO₂ were age, body mass index, FEV₁, and—primarily—altitude. The incidence of hypoxemia, defined as an SpO₂ of ≤88%, was 6% in Mexico City, which is an enormous proportion, even recognizing the errors in evaluation due to inconsistency between oximetry and gasometry or to variations in its timing.

We believe that oxygen therapy criteria should be adjusted so that limited financial resources are directed to those who would benefit most from the oxygen. This would be based on not only a relatively arbitrary PaO₂ or SpO₂ level but also demonstration of a consequence of chronic hypoxemia, such as pulmonary artery hypertension or polycythaemia—that is, responses similar to those described for the Aymara people, on average, to higher altitudes, or biomarkers of chronic hypoxia. These are provisional criteria that require scientific validation.

In Mexico, only 8% of individuals with SpO₂ ≤88%—an almost universal criterion for LTOT—were receiving supplementary oxygen. In other words, hypoxemia is underdiagnosed in 92%; however, it was also documented that 50% of the individuals on LTOT should not have been receiving it—by the most relevant criterion, at least.⁶

In summary, we urge studies similar to NOTT or MRC to be conducted at moderate altitudes aiming to demonstrate (1) the benefit of supplementary oxygen and the circumstances under which it is reached and (2) the priority groups to whom limited financial resources for oxygen therapy should be directed.

Under the present circumstances, and in the absence of reliable information, we would consider the priority patient group to be those who have (1) a chronic PaO₂ of ≤55 mm Hg as well as evidence of chronic hypoxemia on non-invasive tests, such as polycythaemia or pulmonary artery hypertension on echocardiogram, or (2) a concomitant disease, such as coronary or cerebral vascular disease that would be exacerbated under conditions of hypoxemia. In the absence of these medical conditions, we run the risk of simply treating the PaO₂ and, above all, dramatically expanding the indications for oxygen.

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Post-Traumatic Pseudoaneurysm of the Innominate Artery: A Rare Presentation of Tracheal Stenosis*

Seudoaneurisma postraumático de la arteria innominada. Una presentación infrecuente de estenosis traqueal

Dear Editor,

We present the case of a 26-year-old male: a farm worker and non-smoker, with unremarkable medical and family history. He was referred by his family doctor to the pneumology clinic with symptoms of dyspnea progressing over the last 3 years. During that time, he had been treated with beta-agonist inhalers and inhaled corticosteroids for suspicion of bronchial asthma, in spite of which the dyspnea continued to progress, to the point that, in recent months, it has limited his daily activities. The patient had no cough, no expectoration, and no other respiratory symptoms. The only revelation from his medical history was that, 4 years earlier, he had been in a high-speed auto accident in which he suffered closed chest trauma, with no medical consequence of any kind at that time. Physical examination revealed a satisfactory general condition, vital signs within normal range, cardiopulmonary auscultation within normal limits, and oxygen saturation of 98% (fraction of inspired oxygen 21%), the rest of the examination being unremarkable. Simple chest X-ray showed a widening of the upper mediastinum, with compression and displacement of the trachea toward the left. A CT scan of the chest was ordered, which revealed the existence of a 3.5 cm × 3 cm × 2.5 cm saccular aneurysm of the innominate artery, surrounded by a thick wall (up to 15 mm). This wall showed small calcifications in its sinus. The aneurysm was causing severe compression of the trachea and displacing it toward the left side (Fig. 1). Taking the patient's history into consideration, these findings were consistent with a chronic post-traumatic pseudoaneurysm. Flow-volume curves confirmed a fixed extrathoracic obstruction. Regarding laboratory tests, both the haemogram and chemistries were within normal limits; arterial blood gases were also normal.

Pseudoaneurysm of the innominate artery is a rare complication of closed chest trauma. It has various clinical presentations—from superior vena cava syndrome to a chance finding of mediastinal widening on chest X-ray. Tracheal stenosis secondary to innominate artery aneurysm is an uncommon condition,^{1,2} and progressive dyspnea as the clinical presentation of a pseudoaneurysm is even more uncommon.³ The great vessels within the chest are rarely damaged as a result of closed trauma; when they are damaged, however, the innominate artery is the second-most

affected mediastinal vessel. Approximately 100 cases of innominate artery lesions secondary to closed chest trauma have been reported in the literature.⁴ Direct trauma to the great vessels results primarily from high-speed motor vehicle collisions. The injury is due to the effect of deceleration, during which the antero-posterior force applied in the chest reduces the space between the spine and the sternum, thus displacing the heart posteriorly and toward the left. This displacement increases the curvature of the aortic arch and the pressure in the thoracic outlet vessels. Clinical symptoms of closed trauma to the brachiocephalic arterial trunk—also known as the innominate artery—include diminished peripheral pulse, superior vena syndrome, dysphagia, bruits, and pulsatile suprasternal mass. On the other hand, the trauma may be asymptomatic and detected only as a finding on X-ray. Our patient, presenting 4 years later with the consequences of a closed chest trauma, is an extremely unusual case. There have been reported cases of acute respiratory distress from tracheal compression secondary to aortic aneurysm and other conditions, such as vascular rings. There have also been some cases related to innominate artery dilatation⁵ and even cases in which massive tracheal necrosis occurred.⁶ However, there has been no reported case of a slowly progressive dyspnea on exertion resulting from pseudoaneurysm of the innominate artery. This is a situation where CT scan of the chest is a sensitive detection tool that may yield an initial working diagnosis; however, the diagnosis must be confirmed by angiography. In addition, intraluminal clots may be found within the aneurysm, and the local effects of the hematoma or the aneurysm on adjacent structures may be apparent, enabling a true lumen to be distinguished from a false lumen. In conclusion, although tracheal stenosis is quite uncommon, it is a condition that should come to mind in patients with a history of

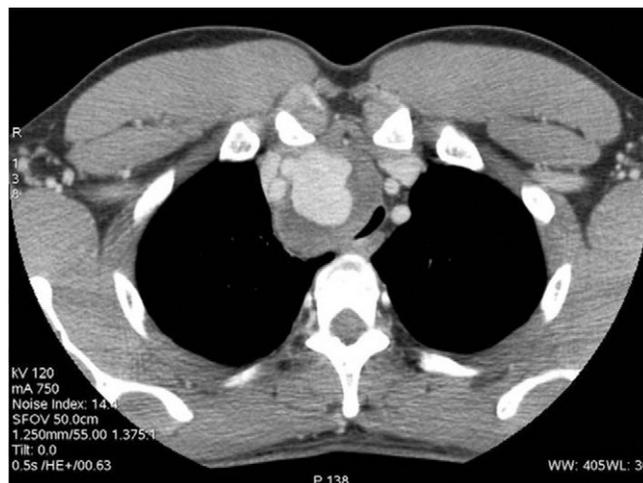


Fig. 1. Saccular aneurysm of the innominate artery.

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