ruled out. A diagnosis of spontaneous pneumomediastinum secondary to labor was made, and we decided on conservative management. Subsequent progress was favorable, and the patient was asymptomatic with complete resolution of the lesions within 2 weeks of follow-up.

Hamman's syndrome, described in 1939, is defined by the association of extensive subcutaneous emphysema and pneumomediastinum in the immediate postpartum period. It is a rare complication associated with labor, and occurs at an incidence of approximately 1 in 100,000 births,[1,5] although it has also been described in association with other processes not limited to the postpartum period, such as screaming, cough, hyperemesis, respiratory infections, or physical exertion.[6,7] It is more frequent in young primiparous women and is associated with the delivery of large babies.[8] It has been posited that it can also occur during the perinatal period in patients with hyperemesis gravidarum. However, most cases occur in the second stage of labor in primigravidas who have a prolonged and difficult labor, due to efforts made during the Valsalva maneuvers. Symptoms usually appear in the third or fourth stages of labor.[5] Table 1 shows the cases of Hamman's syndrome published in the past 10 years.

The pathophysiology of Hamman's syndrome is based on the rupture of alveoli due to a marginal increase in intraalveolar pressure with sustained Valsalva maneuvers (forced expiration against a closed glottis) associated with coughing, vomiting, screaming, and pushing during delivery. Intrathoracic pressure can rise to levels of 50 cmH_2O.[9] This increase in pressure in the presence of reduced vascular caliber establishes a pressure gradient in the vascular sheath along which air can dissect the mediastinum, with subsequent migration of air to the subcutaneous planes.

The most common clinical presentation of Hamman's syndrome consists of retrosternal chest pain, shortness of breath, facial or neck pain, odynophagia, and dysphagia. Since chest pain during childbirth can have different etiologies, urgent conditions such as pulmonary embolism, amniotic fluid embolism, myocardial infarction, pneumothorax, and aortic dissection, must be first be ruled out.[3] In the case of hyperemesis, esophageal rupture should be ruled out, since it can be precipitated by the same factors.

Chest X-ray is the initial diagnostic technique. CT is considered the gold standard to rule out mediastinal air because it can detect small amounts of air that are not visible on chest X-ray, which occurs in up to 30% of cases.[1,10]

Recurrence of Hamman's syndrome is rare and patients usually respond favorably to conservative treatment, consisting essentially of on-demand analgesia, oxygen therapy when necessary, and rest. Patients can be safely discharged to home with analgesic treatment. In very exceptional cases, when pneumothorax is also present, placement of a chest tube may be necessary.[11,12]

In conclusion, spontaneous pneumomediastinum, despite being a rare disease, should be considered in the differential diagnosis of chest pain in the immediate postpartum period. Chest X-ray is a useful tool but it is not always diagnostic, while chest CT is the diagnostic test of reference. Progress is favorable, recurrence is uncommon, and management is often conservative.

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https://doi.org/10.1016/j.arbr.2019.05.014
1579-2129/

Progress after the withdrawal of home oxygen therapy. The profile of patients requiring reintroduction☆

Evolución tras la retirada de la oxigenoterapia domiciliaria. Perfil del paciente en el que hay que reintroducirla

To the Editor,

Home oxygen therapy (HOT) improves survival in patients with chronic obstructive pulmonary disease (COPD) and severe respiratory failure.[1] The prevalence of HOT has increased in recent decades, leading to an increase in costs.[2,3] This intervention should be indicated in non-smoking patients in a stable phase who meet the established criteria for HOT and who are receiving optimal drug therapy. Arterial blood gas should be determined at rest and in a sitting position.[4] If HOT is prescribed in the acute phase, as often occurs after a hospital admission,[5] it is important that it is subsequently evaluated when the patient is stable, in order to avoid inefficient use,[6] since it has been demonstrated that in many cases, HOT may be withdrawn.[7–9] Unnecessary HOT can also be a burden for patients, and has been associated with feelings of discomfort, restrictions in daily activities, and social isolation, contributing to poor compliance.[10] In our hospital, we have a dedicated oxygen therapy clinic where all provisional prescriptions are reviewed within approximately 3 months. The aim of this study was to analyze the progress of patients who were withdrawn from HOT, and to study the characteristics of those in whom it was restarted, a topic not addressed previously in the literature.

☆ Please cite this article as: Lourido-Cebreiro T, et al. Evolución tras la retirada de la oxigenoterapia domiciliaria. Perfil del paciente en el que hay que reintroducirla. Arch Bronconeumol. 2019;55:663–665.

Table 1
Comparison of the characteristics of patients in whom oxygen therapy was correctly or incorrectly restarted and factors associated with restarting oxygen in COPD patients (multivariate analysis).

Comparison of patients in whom oxygen was correctly or incorrectly restarted			
	Restarted correctly	Restarted incorrectly	p Value
Age	75.4 ± 8.8	75.7 ± 10	0.86
FEV_1	46.7 ± 15.9	55.9 ± 19.6	0.007
pO_2	65.9 ± 5.2	68.3 ± 6.5	0.032

Factors associated with restarting oxygen in COPD patients (multivariate analysis)			
	OR	95% CI	p Value
Age	1	(0.95–1.05)	0.99
FEV_1	0.97	(0.95–1.00)	0.072
pO_2	0.97	(0.90–1.04)	0.46
Subsequent admission	5.46	(2.26–13.13)	0.00

95% CI: 95% confidence interval; COPD: chronic obstructive pulmonary disease; FEV_1: forced expiratory volume in 1 s; OR: odds ratio; pO_2: partial pressure of oxygen.

Between October 2015 and August 2017, 960 provisional prescriptions for oxygen therapy were made, of which 483 were withdrawn. These patients were followed for 1 year and the impact of HOT withdrawal was analyzed, taking into account the need for restart, hospital readmissions for cardiorespiratory reasons, and mortality. The characteristics of patients who required restart of HOT were also studied. After a descriptive analysis of patients, COPD patients in whom HOT was restarted were compared to those in whom it was not (Chi-squared test for qualitative variables, Student's t-test for age and FEV_1, and the Mann–Whitney U test for pO_2 determined in the clinic at the time of withdrawal). We also evaluated if HOT was restarted correctly or incorrectly, and conducted a logistic regression multivariate analysis to study the factors that were associated with restarting HOT in these patients. A p value < 0.05 was considered statistically significant. The SPSS® package version 20.0 was used for the statistical analysis.

Mean age of the 483 patients was 77.6 ± 10 years and 59% were men. Ninety percent of the prescriptions were made at the time of hospital discharge. The most common diseases were: COPD (40.5% of cases), pneumonia (20.5%), heart disease (14%), and asthma (10%). Mean pO_2 at the time of withdrawal of HOT was 69.4 ± 7 mmHg. In the first year after withdrawal, HOT was restarted on a provisional basis in 99 patients (21%), albeit incorrectly in 44 (43.5%), either due to failure to determine arterial blood gases at the time of the prescription, or because the patients did not meet the criteria for oxygen therapy.[4] Thirty-three percent of patients in whom HOT was restarted incorrectly had previously documented hypercapnia. Of the 55 patients in whom HOT was restarted correctly, 36 (65.5%) were diagnosed with COPD (mean FEV_1 46.4% ± 16%), and 75% were readmitted for exacerbations. Mean time to restart of HOT was 7 ± 4 months. COPD patients in whom HOT was correctly restarted had lower FEV_1: 46.7 ± 15.9 ($p < 0.007$), pO_2: 65.9 ± 5.2 ($p < 0.032$) and a greater number of hospitalizations ($p < 0.0001$) than the others. In the multivariate analysis, the restart of HOT was associated with a greater number of hospitalizations subsequent to withdrawal (Table 1), because HOT is often used to facilitate discharge home and reduce length of hospital stay. Of patients who were withdrawn from HOT during the follow-up period, 156 (33%) were admitted for cardiorespiratory causes, within a mean time of 6.2 ± 3.4 months. Seventy-nine patients (17%) died, the most frequent causes being respiratory (19 patients; 24%) and cardiac (14 patients; 18%). Mean time to death was 6.7 ± 3.7 months. Ten patients died in the first month: 1 in a traffic accident, 1 due to acute pulmonary edema, 2 of gastrointestinal bleeding, 1 due to stroke, and 1 due to cancer of the larynx, and 4 deaths occurred for unknown causes (death outside the hospital).

Our results seem to confirm that withdrawal of HOT is safe in patients who do not meet criteria for this treatment. However, COPD patients with severe exacerbations will require close monitoring, and respiratory day hospitals can be useful for this purpose.[11]

Oxygen therapy clinics, by correctly applying the criteria for indicating this intervention, have managed to significantly reduce the prevalence of HOT.[5,7,8] Most prescriptions are made in the acute phase, and often do not follow the established recommendations.[8,9,12] It is important to note that 33% of patients in whom HOT was incorrectly restarted had hypercapnia at the time of withdrawal. Administering oxygen to these patients can worsen hypercapnia and cause respiratory acidosis,[13] as arterial pO2 levels play an essential role in the control of ventilation.[14]

COPD exacerbations are more frequent and severe as the disease progresses, and the risk factors for a new admission are $FEV_1 < 50\%$, previous hospitalizations, and use of HOT,[15] characteristics that are in line with those of our study.

In summary, the withdrawal of HOT in patients who do not meet the indication appears safe, since it only has to be restarted in a small percentage of patients, nearly half of which do not meet the criteria for restarting HOT. COPD patients with exacerbations and severe airflow obstruction must be closely monitored. However, more studies are needed to analyze this highly prevalent problem.

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https://doi.org/10.1016/j.arbr.2019.05.015
1579-2129/

Extracorporeal CO_2 removal in combination with continuous renal replacement therapy☆

Sistema combinado de depuración de CO

To the Editor,

Extracorporeal carbon dioxide removal ($ECCO_2R$) systems are devices that provide partial respiratory support. They work with a blood flow of 250–1500 ml/min, less than that required for extracorporeal membrane oxygenation (ECMO), and use a smaller membrane surface (0.33–0.67 m^2). This system was first described in the 1980s by Gattinoni et al.,[1] while in 1990, Terragni et al.[2] published the first combined $ECCO_2R$ system. Using a neonatal membrane lung with a total membrane surface of 0.33 m^2 coupled with a continuous hemofiltration system in 32 patients with acute respiratory distress syndrome, they succeeded in reducing tidal volume (Vt) to less than 6 ml/kg ideal weight, achieving normalization of hypercapnia and a reduction of cytokines in bronchoalveolar lavage at 72 h, reflecting a reduction in mechanical ventilator-induced lung injury.

In patients with acute respiratory distress syndrome, these systems remove CO_2, allowing Vt to be reduced, so that protective or ultraprotective mechanical ventilation (MV) ($Vt \leq 6$ ml/kg or Vt 3–4 ml/kg, respectively) can be efficiently applied. These findings have been demonstrated in a recent international multicenter prospective study.[3] A greater reduction in Vt and plateau pressure would prevent alveolar overdistension, reduce mechanical ventilator-induced lung injury, and may reduce mortality in patients with acute respiratory distress syndrome.[4,5] These systems have several potential indications in hypercapnic patients.[4,5] In COPD, they could help avoid the use of MV, act as an alternative if non-invasive MV fails, or facilitate extubation.[6] In the bridge to lung transplant, they can improve physical conditions, obviating the complications derived from MV.[7,8]

Several $ECCO_2R$ systems are available, most of which are of the veno-venous type.[9] The use of this system combined with continuous renal replacement techniques (CRRT) has been shown to decrease vasopressor requirements,[10] in addition to sparing vascular access.

We report a case in which we used a combined $ECCO_2R$-CRRT system, describe the effects, and discuss the most important technical aspects.

Our patient was a 61-year-old woman admitted for an asthma exacerbation with progressive hypercapnia, who was intubated and connected to MV. On admission to the ICU, she had a pressure plateau of 35 cmH_2O and a peak pressure of 52 cmH_2O. Arterial blood gases with inspired oxygen fraction of 0.4 showed pH 7.3, $PaCO_2$ 120 mmHg, PaO_2 96 mmHg, bicarbonate 28.1 mmol/l, base deficit −7 mmol/l, and oxygen saturation 98%. She developed acute renal failure with urea 107 mg/dl and creatinine 1.36 mg/dl.

Antibiotic therapy, both empirical and targeted at pulmonary aspergillosis, was started, and she received corticosteroids, salbutamol, ipratropium, ketamine, and magnesium. MV was optimized by starting ECMO with ultraprotective MV, which was withdrawn on day 11. After 1 week, the patient's status deteriorated, with pH 7.32; $PaCO_2$, 83 mmHg; PaO_2, 181 mmHg; and bicarbonate, 37 mmol/l. A 13.5 Fr femoral Shaldon catheter was inserted for a combined $ECCO_2R$-CRRT system, with an 0.9 m^2 AN69 hemofilter, and CO_2 membrane lung with surface area of 0.32 m^2, blood flow of 350 ml/min, air 10 l/min, and anticoagulation with sodium heparin for an activated partial thromboplastin time (aPTT) of 2.1. After starting therapy, respiratory acidosis was corrected, with development of respiratory alkalosis after effective reduction of $PaCO_2$ to 30 mmHg in the first 3 h, allowing us to start protective MV with a Vt of 5 ml/kg and PEEP 8 cmH_2O. In the following hours, blood flow was reduced to 300 ml/min due to the development of alkalosis, and the fraction of inspired oxygen was reduced. Despite aPTT remaining within a good range, the hemofilter clotted at 24 h, so the system had to be removed. The patient died in the following 24 h due to severe global respiratory failure caused by pulmonary aspergillosis and septic shock, after ruling out the reintroduction of extracorporeal respiratory support systems, although no complications derived from the use of the system were observed.

In the case described, CO_2 removal was effective in the first hour, with maximum effect at 3 h, but effectiveness was later lost due to hemofilter clotting. It is important to emphasize that $ECCO_2R$ systems contribute only marginally to the improvement of oxygenation by several mechanisms.[11] The diffusing capacity of CO_2 is 20 times higher than that of oxygen, and these systems are theoretically able to eliminate 200–250 ml/min of CO_2 in an adult with a flow of 500 ml/min.[11,12] Hypercapnia should

☆ Please cite this article as: López-Sánchez M, Rubio-López MI. Sistema combinado de depuración de CO_2 y reemplazo renal continuo. Arch Bronconeumol. 2019;55:665–666.