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SCIENTIFIC LETTER

Stress relaxation, another cause of ''Pseudo auto-PEEP''?

Estrés de relajación, otra causa de P̈seudo auto-PEEP?̈

In critically ill patients receiving mechanical ventilation, positive end-expiratory pressure greater than that applied externally (auto-PEEP) reflects dynamic hyperinflation of the lungs. The term ''Pseudo auto-PEEP'' (Bilen & Cohen, 1993[1] was coined to describe a progressive increase in expiratory plateau pressure in prolonged occlusion, attributed to a retrograde flow of extraluminal gas into the airways. However, the underlying mechanisms have not yet been closely studied. Herein, we describe another mechanism for this phenomenon, explained by stress relaxation due to inequalities in the expiratory time constant (τE), manifested in inspiration and expiration.

In mechanical ventilation, a final expiratory volume greater than the relaxation volume generates an end-expiratory pressure known as ''auto-PEEP''.[2] Airway dynamic hyperinflation could be the pathophysiological explanation for this phenomenon, as could expiratory dynamic collapse, insufficient expiratory time, or increased expiratory time constant (τE), which have important consequences on cardiovascular function and respiratory mechanics.[3,4]

Auto-PEEP is usually detected by end-expiratory occlusion (static auto-PEEP) or end-expiratory flow amputation (dynamic auto-PEEP).[5] Another term associated with this is occult positive end-expiratory pressure,[6] resulting from severe airway obstruction, as in patients with asthma, which can be observed using the end-inspiratory occlusion maneuver. Interestingly, in a recent article, Abella & Gordo[7] highlights the importance of recognizing the occult PEEP in mechanically ventilated patients with airflow obstruction due to the impact on the hemodynamic, respiratory mechanics, effort, and asynchronies. Bilen et al.[1] described another related phenomenon, termed pseudo auto-PEEP caused by a backward gas flow from the extrapulmonary to the alveolar space, illustrated by the progressive increase in end-expiratory pressure during prolonged expiratory occlusion.

In this study, we aimed to describe the pathophysiological mechanism of a phenomenon that reproduces findings similar to those described by Bilen et al.[1] (Fig. 1A). We hypothesize that stress relaxation caused by regional inequalities in τE generates anterograde contra-pulmonary flow from fast emptying areas to slow emptying areas (''pendelluft''). This phenomenon manifests in both inspiratory and expiratory occlusions, as suggested by the logarithmic fit of the pressure released during the relaxation stress (Fig. 1B).

A 42-year-old female patient with a medical history of pulmonary interstitial disease was admitted to the ICU after surgical intervention involving a right unilateral pulmonary transplant. The patient had multiple postoperative complications, including hemothorax, bacterial and fungal pneumonia, and bronchial prosthesis due to stenosis of the transplanted lung, which required support with venous-arterial extracorporeal membrane oxygenation during the perioperative period. Moreover, prolonged mechanical ventilation and ICU stay were required. To evaluate respiratory mechanics, advanced monitoring by esophageal pressures and electrical impedance tomography (EIT) were performed during controlled ventilation with neuromuscular blockade. Respiratory mechanical parameters were obtained via multiple linear regression, and the results showed high elastance and resistance (Ers: 43.18 cmH_2O/L, EL 40.09 cmH_2O/L, and total resistance: 15.83 $cmH_2O/L/s$). A prolonged end-expiratory occlusion maneuver showed a progressive increase in pressure until a plateau (6 s) was reached. This finding corresponds to stress relaxation during inspiratory occlusion (Fig. 1A). The values of transpulmonary pressure correspond to a logarithmic function (natural logarithm, ln) of the release pressure in stress relaxation, and the values of the end-expiratory occlusion pressure revealed equivalent but inverse log functions, i.e., 0.525*ln(x) and −0.514*ln(x), respectively (Fig. 1B). A specific form of auto-PEEP is the so-called pseudo-PEEP, which was described by Bilen in 1993[1] and is attributed to the retrograde flow of extraluminal gas into the airway. To the best of our knowledge, no additional studies have been conducted on this concept. We provide a representative case, which shows similar findings that can be explained by relaxation stress due to inequalities in time constants and thus complements the original description.

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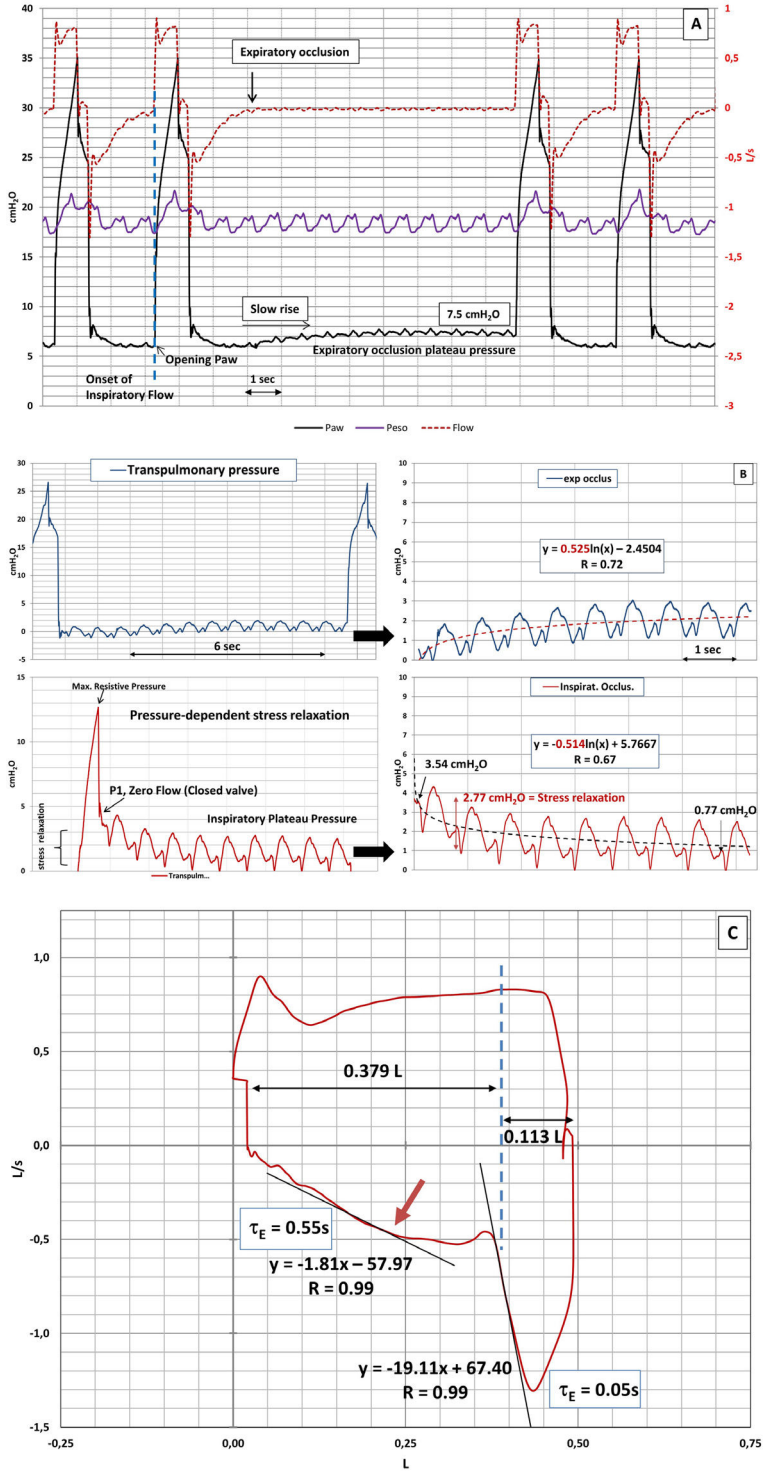

Figure 1 Respiratory signal monitoring during volume-controlled ventilation.
Panel A. Recordings of airway pressure (Paw), esophageal pressure (Peso), and flow during expiratory occlusion. Note the following remarkable features: the near-zero end-expiratory flow state in regular cycles and the slow rate of the rise of the Paw to reach a stable plateau (≅ 6 sec).
Panel B. Traces of transpulmonary pressure during expiratory occlusion (upper panel) and inspiratory occlusion maneuver (lower panel). Note similarity fitting by logarithmic regression of inspiratory stress relaxation and plateau pressure during prolonged expiratory occlusion, with an apparent mirror image.
Panel C. Flow-volume loop. Remarkable emptying compartments were noticeable in the expiratory time constants (τE) and exhaled volume, evidence of initial rapid emptying and a subsequent delay, possibly due to the pendelluft phenomenon (arrow). Note the absence of an end-expiratory flow limitation.

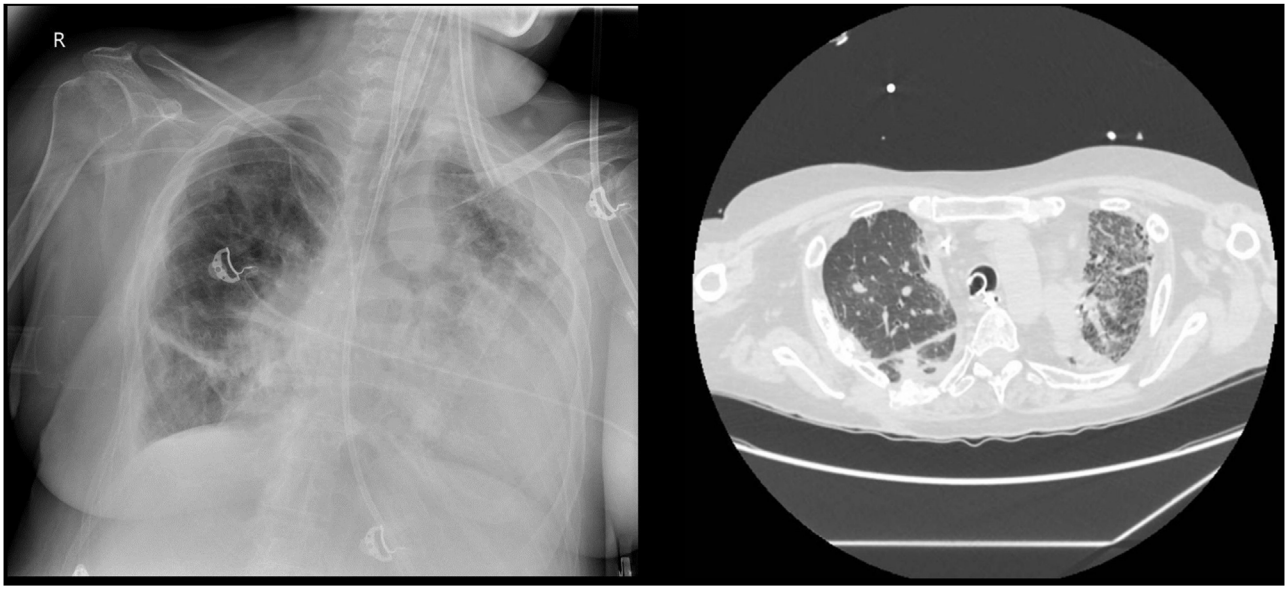

Figure 2 Radiological images. On the left are the conventional radiological findings and on the right are the computed tomography findings. Note the apparent disparity between lungs.

The equilibrium state of the inspiratory and expiratory plateau pressure depends on the relaxation stress due to thoracic-pulmonary viscoelastic resistance, surface tension, and differences in regional time constants.[8] The stress relaxation has a nonlinear relationship with the pressure-dependent viscoelastic properties of the respiratory system, with a focus on the differences in energy distribution.[9]

Our case represents a phenomenon equivalent to pseudo auto-PEEP, which is rationally explained by stress relaxation due to extreme inequalities in time constants. The flow-volume loop suggested an obstructive mechanism due to its biphasic morphology[2,6] (Fig. 1C). However, this can be explained by the presence of two different lung-emptying compartments. These findings were confirmed by monitoring with EIT (video in Electronic Supplementary Material, ESM). Notice in Fig. 1A shows how the end-expiratory flow reached a level near 0. As shown in the radiological images (Fig. 2), there was a predominantly unilateral pathology with notable involvement of the left native lung. Finally, Fig. 1B shows how stress relaxation has an equivalent logarithmic fitting of both the inspiratory and expiratory phases, manifested in the progressive increase in end-expiratory pressure during the prolonged hold, which simulates the pseudo auto-PEEP phenomenon. In addition, this finding can be used as a parameter to evaluate the severity of lung injury.[9]

The clinical and physiological consequences of pseudo-auto-PEEP remain to be determined. Not recognizing these may affect the calculation of respiratory mechanics. Although our purpose was not to evaluate inspiratory effort, this phenomenon could increase muscle workload.

Author contributions

JM SS designed the study, conducted the study, collected and analysed the patient data, interpreted all data, and wrote the manuscript. MVM interpreted the data, revised and wrote the manuscript. CBR interpreted the data, revised the manuscript. JRNI revised the manuscript ARC revised the manuscript. JFMC revised the manuscript.

Ethics approval and consent to participate

The study procedures and data collected for this report were approved by the Ethics Committee of the University Hospital, Reina Sofia, Cordoba, Spain (Refer.: Musc-Txp23). The patient could not execute the permission to publish patient information, so the surrogates (family legally responsible, sister) were informed about the study with enough detail that the patient was admitted, understood the entire procedure, and signed a written informed consent form before inclusion in the study. All methods were performed according to the guidelines and regulations of the Declaration of Helsinki.

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Conflicts of interest

None.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.medin.2024.06.009.

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Manuel Valdivia Marchal[a], María Carmen Bermúdez Ruiz[a], José Ricardo Naranjo Izurieta[a], Ashlen Rodríguez Carmona[b], Juan Francisco Martínez Carmona[c], José Manuel Serrano Simón[a,*]

[a] *Intensive Care Unit, Hospital Universitario Reina Sofía, Córdoba. Spain*
[b] *Unidad Terapia Intensiva, Hospital El Carmen, Mendoza. Argentina*
[c] *Intensive Care Unit, Hospital Regional Universitario de Málaga, Málaga. Spain*

Corresponding author.
E-mail addresses: mvaldiviamarchal@gmail.com (M. Valdivia Marchal), carmen95berm@gmail.com (M.C. Bermúdez Ruiz), jose.naranjo.10@gmail.com (J.R. Naranjo Izurieta), Ashlen.rodriguez00@gmail.com (A. Rodríguez Carmona), jf.mtnez88@gmail.com (J.F. Martínez Carmona), jm.serranosimon@gmail.com (J.M. Serrano Simón).

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In-hospital cardiac arrest simulation program in a cardiopulmonary critical care unit: A pilot experience

Programa de simulación de paro cardíaco intrahospitalario en una unidad de cuidados críticos cardiopulmonares: una experiencia piloto

In-hospital cardiac arrest (IHCA) has an incidence of 1–6/1000 hospital admissions. Approximately one in four IHCA patients survive to discharge, but the neurological outcomes after the return of spontaneous circulation (ROSC) are often poor.[1] Outcomes are influenced by patient characteristics, the timing and location of the cardiac arrest, and the performance of the cardiac arrest team.[2] Improving the performance of the cardiac arrest team can significantly increase patient survival rates. Among educational methods, simulation is considered the most effective strategy for enhancing team communication, collaboration, teamwork, and leadership-fellowship relations.[3]

In Fondazione Toscana Gabriele Monasterio (FTGM), a public tertiary-level center specializing in cardiology, pulmonology, and heart surgery with locations in Pisa and Massa, Italy, we have initiated a pilot IHCA simulation program. This center includes a cath-lab hub for acute coronary syndrome, an adult and pediatric cardiac surgery center, and serves as a referral center for heart failure and primary pulmonary hypertension patients (123 beds; more than 5,000 hospital admissions per year). The aim of the program is to evaluate the intervention times of the intra-hospital emergency team and the Chest Compression Fraction (CCF) during simulated scenarios.

During six simulation sessions, each consisting of four clinical scenarios (see Supplementary Table) conducted in ward and outpatient settings, we recorded the following times (median with interquartile range):

- 8 [6–10] seconds from early recognition/evaluation to the activation of the emergency response;
- 30 [17–37] seconds from activation of the emergency response to the arrival of the defibrillator;
- 67 [46–78] seconds from activation of the emergency response to the arrival of the advanced medical response team.

In FTGM, nurses are often the first responders in cases of IHCA. Therefore, their competence during the initial