The Effect of Time Between Sample Extraction and Arterial Blood Gas Analysis in Clinical Practice☆

Impacto del tiempo entre la extracción y el análisis de la gasometría arterial en la práctica clínica

To the Editor,

Arterial blood gas (ABG) analysis is an essential determination for all healthcare professionals specializing in the respiratory system. Errors in measuring and interpreting ABG analysis may cause direct injury to the patient, so accurate calculations are essential.

We are still far from achieving standardization in both the collection and subsequent analysis of samples. Although all clinical practice guidelines in the literature agree that samples should ideally be analyzed as soon as possible, we are still surprised to find ambiguities and discrepancies in the maximum time permitted before analysis, without the sample deteriorating.[1–6]

It is generally accepted that ABG values change over time: oxygen is consumed and carbon dioxide increases. It is assumed that erythrocyte metabolism continues outside the body, causing pCO_2 levels to increase in the sample.[7] Because of this assumption, most clinical practice guidelines recommend storing the blood samples on ice, in the hope that this will slow down metabolism and avoid wide variations in ABG values.[5,8]

Our goal was to analyze the impact of time on ABG values between the extraction of samples and analysis, and the factors that influence such variations. We therefore designed a prospective study of the analyses requested by our department over a period of 1 month. The recommendations and the SEPAR protocol[1,6] were followed throughout the entire process. Two analyses were conducted on each sample using a BD-Preset syringe; 1 baseline sample, performed within the normal timeframe, and the other 30 min after the first. During this interval, the samples were preserved on ice, according to protocol.

Data were collected from several variables: the patient (age, sex, height, weight, smoking habit, associated diseases, and lung function), the environment (barometric pressure and temperature), and ABG determinations (FiO_2, pO_2, pCO_2, pH, O_2Hb, COHb).

The sample size was calculated based on the difference between the final pO_2 and the initial pO_2; for a power of 80% and an alpha error <0.05, the minimum *N* was 43.[9]

An intra-subject *t*-test for dependent variables was conducted, and correlations among the different variables were also compared using the Pearson's test.

A total of 69 patients was finally included. In the baseline measurement, mean ABG values were: pO_2 63 mmHg (SD 15), pCO_2 45 mmHg (SD 10), and pH 7.42 (SD 0.037). Mean differences between the final measurement and the initial analysis were observed in pO_2, +2.26 mmHg (66.02 final vs 63.78 initial, *P*<.001), and pCO_2, −0.30 mmHg (45.48 final vs 45.78 initial; *P*=.017). There was a 0.007 difference in pH compared to the baseline value (7.416 final vs 7.424 initial; *P*<.001).

No correlation was observed between differences in pO_2 and time to analysis. In contrast, a significant correlation was found between barometric pressure and differences in pCO_2 and pO_2 (*P*<.05) (Fig. 1).

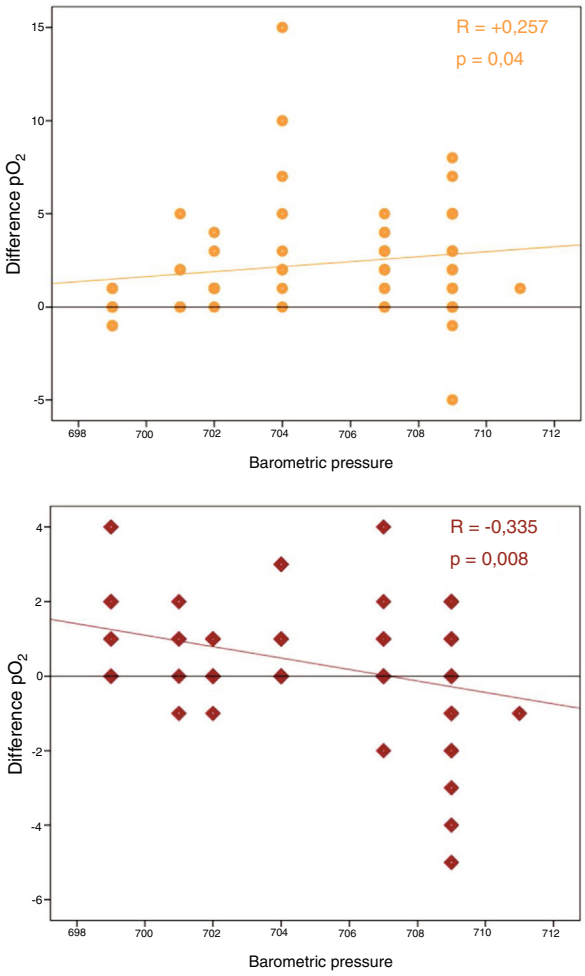

Fig. 1. Positive correlation between differences in pO_2 and barometric pressure and negative correlation between differences in pCO_2 and barometric pressure.

No other associations were found between differences in gases and the other variables studied, including comorbidities and various respiratory pathologies.

In contrast to the recommendations of some clinical practice guidelines and widespread medical beliefs, the changes observed in ABG (pO_2, pCO_2 and pH) were opposite to expected. An increase was observed in pO_2 and a decrease in pCO_2 in the second analysis compared to baseline.

This fact, while remarkable, has already been previously described in other studies: Liss and Payne[8] conducted an analysis similar to ours, and found similar results. Knowles et al.,[10] Schmidt and Muller-Plathe,[11] and Pretto and Rochford[12] went further, and reported that the variation in gases differed depending on the material of the syringe. Indeed, they showed that no such variations occurred with glass syringes.

Different theories have been proposed to explain these modifications in the gases of the samples, but all have a common denominator: the diffusion of gases through the porous plastic material of the syringes. Perhaps the most widely accepted hypothesis is that gas diffusion as a function of oxygen content in the sample is due to a purely physical mechanism, as demonstrated in the study of Mahoney et al.[9] Fletcher and Barber[13] also supported physical diffusion, to the detriment of the theories that take into

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account blood cell metabolism: these authors studied changes in the concentration of pO_2 of oxygenated water, avoiding the use of blood per se, and obtained similar results.

It is important to note that in studies with a higher initial pO_2 than ambient pO_2, the diffusion gradient of the gases is reversed and pO_2 diminishes with time,[12,14] a finding that only goes to reinforce the hypothesis of simple gas diffusion.

Our results also clearly support this physical diffusion theory, since pO_2 variability increases depending on atmospheric pressure in a statistically significant manner, a factor that had never been previously analyzed.

In summary, it seems clear that over time, pO_2 in ABG tends to increase, and that this variation could be directly associated with plastic syringes and the diffusion of gases through this porous material. Our study supports this theory and reveals a direct relationship of these variations with atmospheric pressure.

Despite the fact that ABG determinations vary significantly with time, they do so to an extent that is insignificant in clinical practice. It may be of interest, in the future, to expand the study to different time points.

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Ana Gómez-García,[a] Tomás Ruiz Albi,[b] José Ignacio Santos Plaza,[a] Andrea Crespo Sedano,[a] Ana Sánchez Fernández,[c] Graciela López Muñiz,[a] Tania Álvaro de Castro,[a,b,c] Pilar Revilla Gutiérrez,[a] Josefa Villar Muñoz,[a] Gloria Martínez González,[a] Félix del Campo Matías[b,*]

[a] *Hospital Universitario Río Hortega, Valladolid, Spain*
[b] *Universidad de Valladolid, Spain*
[c] *Hospital Universitario de Salamanca, Spain*

* Corresponding author.
E-mail address: fsas@telefonica.net (F. del Campo Matías).

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Pulmonary Emphysema in a Child With Alpha-1 Antitrypsin Deficiency: Evaluation of 2 Years of Intravenous Augmentation Therapy

Enfisema pulmonar en un niño con deficiencia de alfa 1 antitripsina: evaluación tras dos años con terapia de aumento

Dear Editor:

Alpha-1 antitrypsin deficiency (AATD) is an autosomal codominant condition, known to predispose to early onset pulmonary emphysema and chronic obstructive pulmonary disease. Generally, lung manifestations of the disease affect smokers at the third or fourth decade of life and so far, intravenous injections of purified alpha-1 antitrypsin are the single target therapy for AATD. Given that lung function in children is typically normal, augmentation therapy has never been systematically studied in pediatric population.

We report a case of a male neonate born at 40 weeks of gestation, weighing 2750 g, with an uneventful postnatal period. His first year of life was marked by downward crossing of weight percentile, recurrent suppurative otitis media and two hospitalizations due to respiratory infections. At 9 months of age, routine laboratory investigation revealed anemia (11.8 g/dL) and elevated liver enzymes (aspartate transaminase 596 U/L, alanine transaminase 722 U/L, alkaline phosphatase 239 U/L and gamma-glutamyl transpeptidase 75 U/L). Due to decreased alpha-1 antitrypsin (AAT) serum concentration (50 mg/dL) gene sequencing (*SERPINA1*) was performed and revealed a ZPlowell genotype. There was no ultrasound evidence of liver impairment. The child's follow-up was interrupted due to a problematic social situation. His family medical history was unremarkable. However, a significant passive smoke exposure due to paternal smoking was reported. He returned to our observation at age 10 years, complaining of recurrent productive coughing, wheezing as well as dyspnea on exertion. On physical examination the patient presented with pale skin, weak appearance and low weight for his age (10th percentile). The chest auscultation revealed diffuse expiratory wheezing and bilateral basal crackles. Pulmonary function tests indicated a non-reversible mild airflow obstruction (FEV1 71.3% pred, FEV1/FVC 74% pred). Laboratory studies showed aggravated anemia, peripheral eosinophilia, elevated total IgE and sensitization to common inhalant allergens (RAST positive). At this time serum level of AAT was 36.7 mg/dL. Chest computed tomography (CT) imaging showed lower lobe predominant panlobular emphysema and cystic bronchiectasis (Fig. 1).

He started inhaled therapy with medium-dose corticosteroid (budesonide 160 mcg) plus long-acting β-agonist (formoterol 4.5 mcg) and leukotriene receptor antagonist (montelukast 10 mg). Despite optimized anti-inflammatory and bronchodilator therapy