

Acid-base variations of whole blood and isolated plasma induced by acute changes in partial pressure of carbon dioxide in critically ill patients and healthy controls: an in-vitro study.

Dott.ssa SERENA BRUSATORI (1), Dott. THOMAS LANGER (1)(2), Dott.ssa ELEONORA CARLESSO (1), Dott. FRANCESCO ZADEK (1), Dott.ssa VALENTINA CASTAGNA (1), Dott.ssa ROSAMARIA LIMUTI (1), Dott. GIORGIO GIUDICI (1), Dott. TOMMASO MAURI (1)(2), Dott. ALBERTO ZANELLA (1)(2), Prof. GIACOMO GRASSELLI (1)(2), Prof. ANTONIO PESENTI (1)(2)

(1) University of Milan, Milan, Italia.

(2) Fondazione IRCCS Ca' Granda, Ospedale Maggiore Policlinico, Milan, Italia.

Argomento: Insufficienza respiratoria acuta e ventilazione meccanica

Introduction Acute changes in PCO_2 are buffered by non-carbonic weak acids (A_{TOT}), i.e., albumin, phosphates and hemoglobin.

Aim Describe acid-base variations induced by *in-vitro* PCO_2 changes in critically ill patients' blood and isolated plasma, compare them with healthy controls and quantify the contribution of different buffers.

Methods Blood samples were collected from patients admitted to the ICU and controls. Blood and isolated plasma were tonometered at 5 and 20% of CO_2 in air. Electrolytes, pH, blood gases, albumin, hemoglobin and phosphates were measured. The Strong Ion Difference (SID) was calculated [1] and non-carbonic buffer power was defined as $\beta = -\Delta[HCO_3^-]/\Delta pH$ [2]. T-tests and linear regression were used for analysis.

Results Seven patients and 10 controls were studied. Hemoglobin, hematocrit and albumin were lower in patients ($p < 0.001$), while SID and phosphates were similar. PCO_2 changed from 29 ± 4 to 108 ± 13 mmHg, causing different blood pH variations in patients and controls (0.43 ± 0.06 vs. 0.36 ± 0.02 , $p = 0.03$). Patients had lower blood and plasma β (20 ± 5 vs. 30 ± 4 , $p < 0.001$ and 2 ± 2 vs. 4 ± 1 , $p = 0.03$, respectively). Figure 1 shows changes in $[HCO_3^-]$ and SID induced in blood by PCO_2 variations. In both populations, $82 \pm 12\%$ of $[HCO_3^-]$ change was due to SID variations, while only $18 \pm 12\%$ to changes in A_{TOT} dissociation. A significant correlation between hematocrit and ΔSID ($r^2 = 0.51$, $p = 0.001$) was observed in the whole study population.

Conclusions The β of ICU patients was lower, likely due to reduced albumin and hemoglobin concentrations. Similar PCO_2 increases caused therefore greater pH variations in this population. Electrolyte shifts, likely deriving from red blood cells [3], were the major buffer system in our *in-vitro* model of acute respiratory acidosis.

References

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