

# Role of different buffers during acute respiratory disorders in septic patients: an in-vitro study.

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Argomento: Altro

**Introduction.** According to Stewart's theory, pH variations during acute respiratory disorder are limited by non-carbonic weak acids, whose buffer power depends on their concentration ( $A_{TOT}$ ) and acid dissociation constant (pKa). Recent studies showed that also Strong Ion Difference (SID) variations have a role in buffering acute  $P_{CO_2}$  changes.<sup>[2]</sup>

**Aim.** Describe acid-base variations induced by *in-vitro*  $P_{CO_2}$  changes in whole blood and isolated plasma of septic patients, compare them with healthy controls and quantify the contribution of different buffers.

**Methods.** Blood samples of 5 septic patients and 4 controls were tonometered at  $CO_2$  ranging from 2 to 20%. Total non-carbonic buffer power ( $\beta$ ) and its components due to SID variation ( $\beta_{SID}$ ) and  $A_{TOT}$  ( $\beta_{Atot}$ ) were calculated via linear regression.<sup>[1]</sup> The pKa and  $A_{tot}$  were computed via non-linear regression performed by SAS 9.4 software solving the equation.<sup>[3]</sup> **Results.**  $P_{CO_2}$  variations ranged from  $18 \pm 2$  to  $127 \pm 3$  mmHg (Fig.1). Haemoglobin and total protein were lower in patients ( $10.3 \pm 1.0$  vs.  $14.8 \pm 0.8$  g/dL,  $p < 0.001$  and  $4.6 \pm 0.3$  vs.  $7.0 \pm 0.5$  g/dL,  $p < 0.001$ , respectively). Septic patients had lower blood and plasma  $\beta$  ( $19 \pm 2$  vs.  $29 \pm 2$  mEq/L,  $p < 0.001$  and  $2 \pm 1$  vs.  $4 \pm 1$  mEq/L,  $p = 0.005$ ) and blood  $\beta_{SID}$  ( $15.3 \pm 1.8$  vs.  $23.5 \pm 0.7$  mEq/L,  $p < 0.001$ ). Blood pKa values were  $6.37 \pm 0.21$  vs.  $6.58 \pm 0.16$  ( $p = 0.139$ ), with  $A_{TOT}$   $15.3 \pm 3.7$  vs.  $18.5 \pm 1.5$  mmol/L ( $p = 0.159$ ) and  $\beta_{Atot}$   $3.8 \pm 1.7$  vs.  $5.3 \pm 0.7$  ( $p = 0.169$ ), in patients and controls respectively.

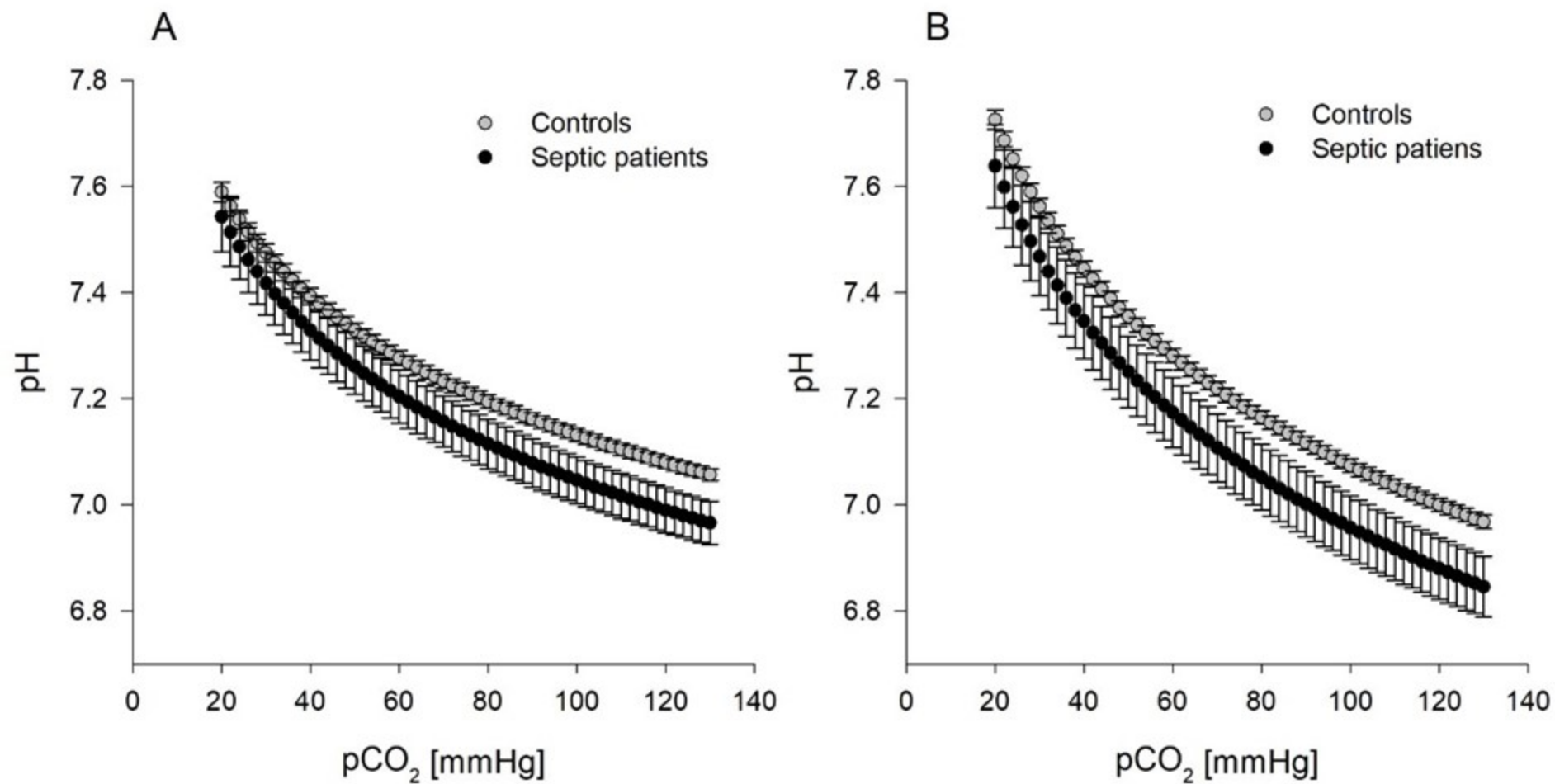
**Conclusions** Septic patients had lower  $\beta$  as compared to controls. This was due both to quantitative reductions in buffers and, possibly, alterations of their function, as suggested by lower  $pK_a$  values. In both groups, SID variation ( $\beta_{SID}$ ) was the main mechanism limiting pH variations.

## Bibliography

<sup>[1]</sup> Van Slyke DD J Biol Chem 52:525-570, 1922

<sup>[2]</sup> Langer T et al. J Crit Care 30(1):2-6, 2015

<sup>[3]</sup> Staempfli HR J Appl Physiol 95:620-630, 2003



**Figure 1.** Average pH-pCO<sub>2</sub> curves computed from the fittings of individual recordings in blood (A) and plasma (B). Data are presented as mean±SD.