Non-carbonic buffer power of whole blood is increased in experimental metabolic acidosis: an in-vitro study

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Introduction

Non-carbonic buffer power (β) of blood is used in the Van Slyke equation to calculate base excess (BE). According to this equation, β depends on hemoglobin and proteins, and is independent from pH.

Objectives

To assess *in-vitro* the effect of metabolic acidosis on β of whole blood.

Methods

- Whole blood of 9 healthy volunteers was used.
- CO₂ tonometery was performed with the original sample and after *in-vitro* acidification.
- Hydrochloric acid (HCl) and lactic acid (HLac) were used to induce moderate (7.5 mEq/L) or severe (15 mEq/L) reduction of strong ion difference (SID).
- $\beta = -d[HCO_3^-]/dpH$ at pH = 7.2 (Figure 1).



Figure 1: An example of CO₂ tonometry.



Figure 2: The increase in β ($\Delta\beta$) as compared to control blood of each volunteer.



- β of control samples was 28.0 ± 2.5 mmol/L.
- In moderate hyperchloremic and lactic acidosis β increased by 2.8 ± 0.9 and 2.3 ± 0.9 mmol/L (p<0.001, Figure 2).
- In severe hyperchloremic and lactic acidosis β increased by 5.6 ± 1.0 and 5.7 ± 1.6 mmol/L (p<0.0001, Figure 2).
- β did not differ significantly when the same degree of acidosis was achieved by a different acid.

Conclusions

The non-carbonic buffer power of whole blood is affected by its acid-base status. In particular, it is significantly increased during *in-vitro* metabolic acidosis. This may affect the calculation of BE.