Border flux balance approach to acid-base status assessment

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The use of quantitative relations of acid-base chemistry as they were described by Stewart often leads to erroneous conception of causality in the clinical practice. There is often confusion between a causal relationship and a relationship of an independent and dependent variable in the mathematical sense (e.g. SID and pH), which are necessarily not the same. Other problem is posed by quantitative data often being limited to plasma only.

Last year, we have presented our alternative approach to assessment of acid-base disorders. We have tried to unite the classical approach of the so called "Danish school" with the quantitative concept of Stewart and his followers. Our approach enables us to causally explain (with better precision than usual) disorders of acid-base and ionic balance.

We have created a computer model of acid-base chemistry to give quantification to our approach.

When modeling acid-base status of blood, we need to consider, besides other things, fluxes of bicarbonates and of strong acids (or bases). We searched for a state variable that would change a simple way during influx/outflux of hydrogen ions or bicarbonates. As such a state variable, we have chosen "standard Base Excess", defined as follows: StBE is the amount of hydrogen ions that we need to add to (remove from) 1 liter of blood at given hemoglobin concentration and given total concentration of plasmatic buffers (albumins mostly) to reach pH of 7.4, all that at fixed pCO_2 = 40 Torr, temperature = 37°C and O_2 saturation = 100%. Siggaard-Andersen's original definition of BE has implied the opportunity to compare acid-base status of blood samples with various hemoglobin concentrations (two blood samples with the same pH can have different BE, if their hemoglobin concentration differs). We have broadened the definition to encompass various concentrations of plasmatic buffers as well.

Identified model has the same behaviour as the Siggaard-Andersen's nomogram with one dimension added – that is concentration of plasmatic buffers. The model of whole blood is made by combination of a model of plasma and a model of red blood-cells. Since the experimental data to its direct identification are not available so far (obtaining them is our future goal; it seemingly requires construction of a robotized measuring apparatus), we had to do with what is available. We used the data that are available for the model of plasma. For the identification of the model of red blood-cells, we used experimental data of behaviour of red blood-cells at various levels of pCO_2 and pH (measured outside the cells, ie. in plasma) derived from original Siggard-Andersen nomogram. The model of blood can be included into a more complex model of acid-base chemistry, to depict equilibration with interstitial fluid, exchange of hydrogen, potassium and sodium ions on cellular membrane, respiratory and renal part of acid-base balance. The model was made for the purpose of medical education as an integral part of the project of Internet Atlas of Normal, Pathological and Clinical Physiology. It can explain pathogenesis of states as:

Hypochloremic dilution acidemia
Hyperchloremic acidosis
Hypoalbuminemic alcalemia