

6. Závěr

AI (Artificial Intelligence) – umělá inteligence je popisovaná jako věda a inženýrství sestavování inteligentních strojů. AI se zrodila po druhé světové válce. Termín AI je aplikovatelný k široké škále postupů v medicíně, jako jsou robotika, medicínská diagnostika, medicínská statistika a humánní biologie – včetně všech různých „omics“. Společenské a etické aspekty těchto aplikací vyžadují další reflexi, prokázání medicínské užitečnosti, ekonomické hodnoty a rozvoj interdisciplinárních strategií pro jejich širší aplikaci.

AI (a robotika) zcela mění prostředí medicíny a zdravotní péče. Vlna inovací transformuje proces klinického rozhodování, monitorování pacientů a péče, mění i přístup zdravotní péče o celou populaci (preventivní péče). Tyto změny také budou měnit role ošetřujícího personálu. Bude se měnit potřebné vzdělání a dovednosti. AI uvolní kliniky pro ostatní typy práce, což jim umožní více se věnovat smysluplné péči o pacienta (1, 3, 6).

Literatura:

- [1.] KARŠULÍNOVÁ, V. (2014) *Využití umělé inteligence v lékařství*. Brno: Masarykova univerzita, Filozofická fakulta. (nepublikováno)
- [2.] LEGG, S., HUTTER, M. (2007) *A Collection of Definitions of Intelligence*. [Cs], June 25. <http://arxiv.org/abs/0706.3639>.
- [3.] HAMET, P., TREMBLAY, J. (2017) *Artificial intelligence in medicine*. *Metabolism*. Apr;69S:S36-S40. DOI: 10.1016/j.metabolism.2017.01.011
- [4.] HAVEL, I., M. (1992) *Artificial Intelligence and Connectionism*. In: *Advanced Topics in AI* (Mařík, Štěpánková, Trapp, editoři), *Lecture Notes in Artificial Intelligence*, Springer-Verlag 1992, s. 25–41.
- [5.] NILSSON, N., J. (2010) *The Quest for Artificial Intelligence: A History of Ideas and Achievements*. Cambridge; New York: Cambridge University Press.
- [6.] PwC (2017) *What doctor? Why AI and robotics will define New Health*. (staženo 2017: www.pwc.com)

Kontakt:

Prof. Jan Hendl
FSV UK – katedra sociologie
U Kříže 8 a 10
158 00 Praha 5 – Jinonice
e-mail: jan.hendl@fsv.cuni.cz

ACID-BASE EQUILIBRIUM MODELING BASED ON THE BALANCE CONCEPT

Jiří Kofránek, Filip Ježek

Abstract

Two approaches are applied to the clinical evaluation of acid-base equilibria: one is the traditional “Danish school” approach of Siggaard-Andersen et al., the other is the “modern” approach by Stewart and Fencl. The two theories are interlinked by what is called the balance approach, described in detail in [1]. A simulation model can be applied in order to model various pathogeneses of acid-base disorders and to monitor their manifestations from both the traditional and the modern acid-base theory aspects.

Key words

Acid-base equilibrium, Balance approach, Danish school of acid-base equilibrium, Model, Stewart's theory

1. Introduction

Two approaches are applied to the evaluation of an acid-base equilibrium (ABE): the traditional approach of the Danish school of Astrup, Siggaard Andersen and collaborators, using BE and compensation diagrams, and Stewart's approach, sometimes referred to as “modern”, based on physico-chemical calculations of acid-base and electroneutral equilibria of ions and buffers in plasma. Both theories basically target the same topic, only from different aspects [2–4]. Nevertheless, advocates of one or the other theory sometimes trigger fierce arguments. Kamel and Halperin, in their most recent monograph [5], thereby conclude that the traditional theory augmented with ion balance correction calculations is quite well suited to the evaluation of ABEs, while Stewart's approach basically brings no new contribution to the concept. By contrast, many other authors consider Stewart's approach a breakthrough and resentfully reject any criticisms. Advocates of the second approach prevail in the Czech Republic. [6–8].

Stewart published two papers [9,10] that maybe would have passed unnoticed had Mr. Vladimír Fencl, a renowned professor with Czech roots teaching at Boston University, not devoted a great effort to the promotion and an additional expansion of Stewart's ideas [11–16]. This is why any criticism of the Stewart-Fencl theory is widely perceived as blasphemy, particularly in the Czech milieu.

We have put forward a new concept, based on balances, which, as we believe, interlinks the two theories and provides an explanation of pathophysiological processes involved in ionic and acid-base equilibrium disorders [1,17].

In order to verify this theory (and to persuade clinicians) we are developing a simulator, accessible from our website, that will enable our approach to be explained interactively.

2. In which aspect is the Stewart theory wrong?

The balance concept will enable changes in acid-base parameters and ions, which are wrongly explained by the Stewart theory and inadequately explained by the traditional theory, to be explained on a pathophysiological basis.

The mathematical causality being mistaken for pathophysiological causality is the major weakness of Stewart's approach (Figure 1). Mathematical causality enables the pH and bicarbonate plasma concentration to be calculated from following input variables: partial pressure of carbon dioxide ($p\text{CO}_2$), the Strong Ion concentration Difference (SID) and the total non-bicarbonate buffer concentration [A_{tot}]. The explanation of the pathophysiology of acid-base disorders by a mechanistic adoption of the dependences of pH and HCO_3^- on the input variables $p\text{CO}_2$, SID and A_{tot} is seemingly simple, but fails to explain the real causal chain of the disorders (Figure 2).

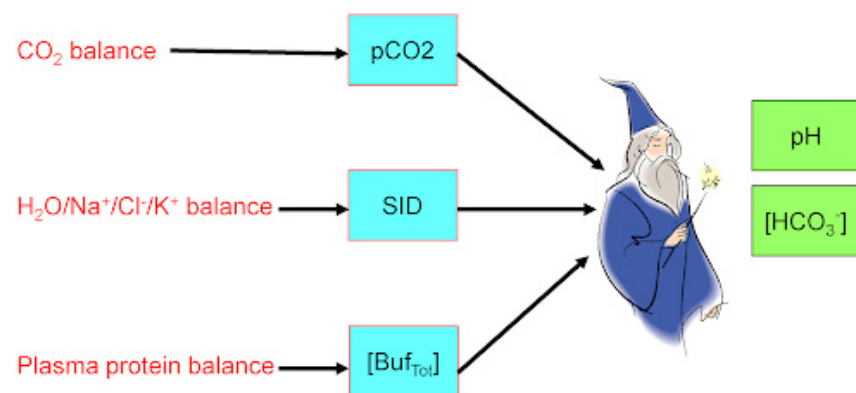


Figure 1– Stewart's approach is based on mathematical relations that can be used to calculate the pH and $[\text{HCO}_3^-]$ levels from the mutually independent $p\text{CO}_2$, SID and $[A_{\text{tot}}]$ data.

Indubitably, the changes in the SID, A_{tot} and/or $p\text{CO}_2$ levels will bring about changes in pH and in the bicarbonate concentration. This, however, does not imply that the body controls its pH and bicarbonates by controlling the balance of strong ions and by subsequently affecting the SID or controlling the albumin and phosphate levels and, in turn, by affecting the A_{tot} parameter. The internal environment stability control depends on the balance of various components involved in the internal environment (i.e., ECF composition). Ionic, volumetric and osmotic homeostasis of the internal environment depends on a controlled balance of various constituents (ions, water, ...). Acid-base homeostasis is no exception. Once again, the control of material (CO_2 , H^+ and HCO_3^-) flow balance is involved. The CO_2 , H^+ and HCO_3^- flows are interrelated due to the presence of the bicarbonate buffer system in the body fluids.

The CO_2 flow is controlled by respiration – CO_2 balance disorders bring about respiratory disorders of the acid-base equilibrium. However, remember that the CO_2 level in venous blood together with equilibrated IST depends not only on the respi.

ration-dependent arterial concentration but also on perfusion – the hypo-perfused tissues experience hypercapnic acidosis even in normal acid-base situations in arterial blood (hypercapnic hypo-perfusive tissue acidosis results in the bonding of H^+ ions to proteins in the cells with damaged performance of the enzymes). In normal situations, the metabolic H^+ formation is in equilibrium with the kidney-generated bicarbonate flow during urine acidification. $\text{H}^+/\text{HCO}_3^-$ flow balance disorders result in metabolic acid-base equilibrium disorders.

So, the body **does not control the body fluid pH or the HCO_3^- level through the SID change and the albumin level control. Instead, the body controls those parameters by controlling the flow balance of CO_2 (respiratory ABE control) and H^+ and HCO_3^- (the metabolic component of ABE control). Disorders of those balances bring about acid-base disorders. The Balance theory provides a pathophysiological explanation of the causes of the changes in the ionogram, SID and A_{tot} accompanying various acid-base disorders.**

3. In what respect is the explanation of the ABE disorder pathophysiology insufficient?

The Danish school focuses on the ABE disorder diagnosis primarily based on the **concentrations** of the buffer system components in blood (and, in turn, in the ECF) and the **concentrations** of ions in the ECF. This school inadequately describes

the diagnosis of mixed disorders (particularly where the two disorders act in opposite directions) and, in particular, their causes, and fails when evaluating hemodilution and hemoconcentration states[1].

4. What does the balance theory newly contribute?

The balance concept is based on the following six principles enabling the changes in the acid-base parameters and ions to be explained on a pathophysiological basis (see [1], Section 6):

1. All **trans-membrane transports** take place in a steady state in the overall balance by an **electroneutral route**.
2. **Electroneutrality does not change during biochemical reactions** in the human body. So, for instance, the negatively charged lactate or citrate metabolizes to water and CO_2 while taking up H^+ ions, hence, like lactic or citric acid.
3. The acid-base equilibrium depends on the **balance of the CO_2 , proton and bicarbonate flows interconnected via the bicarbonate buffer system**. This is the same as in the traditional theory and enables ABE disorders to be classed into respiratory disorders (CO_2 balance) and metabolic disorders ($\text{H}^+/\text{HCO}_3^-$ balance).
4. The **plasma SID and BB levels describe the same entities, calculated in different ways**, the SID and BB (BE) level changes are identical and describe the balance of the proton and bicarbonate flows. An addition of 1 mmol/l of protons will reduce the SID and BB by 1 mmol/l, whereas an addition of 1 mmol/l of bicarbonate will increase the SID and BB by 1 mmol/l (and vice versa).
5. The bicarbonate buffer system in the body fluids captures and releases protons. Therefore, **proton flows are equivalent to bicarbonate flows**.
6. The **buffer systems are “storerooms” for situations involving bicarbonate flow disbalances** with appropriate changes in the acid-base equilibrium.

The balance theory is NOT based on evaluation of the bicarbonate concentration in the ECF, as it might seem at the first glance. What interests us is the **balance** of the $\text{H}^+/\text{HCO}_3^-$ flows and **electroneutrally interlinked flows** of the remaining ions. The hydrogen and bicarbonate ion flows are interlinked via the bicarbonate system, which is ubiquitous in the body fluids. The diffusion flow rate depends on the concentration gradient. The proton concentration gradient is many orders of magnitude lower than the bicarbonate concentration gradient, and as such the proton diffusion flows are lower than the bicarbonate diffusion flows. Owing to this, the **acid-base metabolic balance can be described in terms of the bicarbonate flow balance**. A negative bicarbonate balance characterizes metabolic acidosis, whereas a positive balance characterizes metabolic alkalosis – in this, our theory agrees with the traditional Danish ABE school.

The **buffer systems are true buffers (dampeners) of variations in the acid-base balances of the $\text{H}^+/\text{HCO}_3^-$ flows during metabolic acid-base equilibrium disorders**. When the bicarbonate balance is negative/positive (metabolic acidosis/alkalosis), the buffer systems provide/accumulate bicarbonates, and the subsequent change in the equilibrium within the buffer systems result in pH changes (see Figures 3-7 taken from ref. [1]).

Not only the concentrations of the components but also the total amount plays a crucial role in the balance concept.

Both the traditional and the Stewart ABE theories primarily focus on the concentrations of the substances, which sometimes leads to incorrect conclusions. Not only the concentrations but also the total amount of the buffer bases (particularly bicarbonates in the ECF) is of importance with respect to the performance of the buffer systems as $\text{H}^+/\text{HCO}_3^-$ flow balance variation buffers during metabolic ABE disorders. As, e.g., Halperin and Kamel point to [5,18], ECF volume reduction will be accompanied by a reduction in the total amount of bicarbonates, which are able, through H^+ bonding to dampen the $\text{H}^+/\text{HCO}_3^-$ balance fluctuations during metabolic acidosis. With the ECF volume reduced, the bicarbonate concentration decrease – or the BE (or SID) decrease – may not match the severity of the metabolic acidosis.

Furthermore, Kamel and Halperin [5] point to the role of bicarbonate reserves in the ECF in the prevention of the hydrogen ion bonding to brain tissue proteins during metabolic acidoses. Bicarbonates in the ECF, particularly of the muscle tissue, bind the hydrogen ion load during acidoses, while the brain tissue, which contains relatively small amounts of HCO_3^- but is subject to large blood flows, only binds a minimal quantity of hydrogen ions. Whenever the volume is endangered (effective arterial blood volume is reduced), pCO_2 increases particularly in the muscle tissue, and the brain tissue is threatened by H^+ bonding to proteins. A difference > 6 mmHg in the pCO_2 levels between arterial blood and a brachial vein indicates endangered volume.

Balance theory provides a different explanation of metabolic disorders of the acid-base equilibrium.

5. Dilution acidosis and contraction alkalosis according to the balance principle

A number of authors, such as [19] (and also Kazda in the Czech Republic [20]), explain dilution acidosis occurring after adding saline in terms of SID being reduced as a result of excess chloride and bicarbonate vanishing due to electroneutrality. However, bicarbonate in the buffer reaction only vanishes through binding with H^+ , and this is released from the non-bicarbonate AH buffers – whereby the negative charge of A- increases – so that electroneutrality does not change in the buffer reactions and hence, this explanation is wrong.

The true cause of dilution acidosis / contraction alkalosis has nothing to do with chloride: it is related to the dilution/concentration of the buffers, the concentration

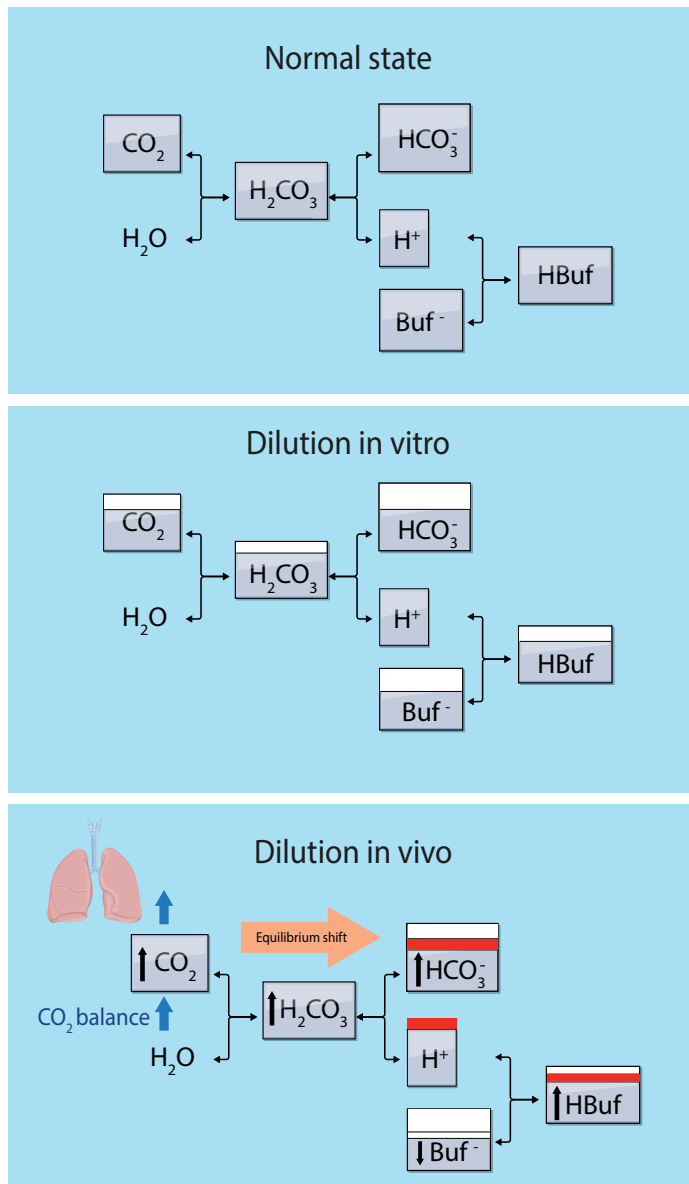


Figure 8 – Pathogenesis of dilution acidosis. The buffering capacity is reduced on buffer dilution, the pH, however, does not change. Since a constant weak acid level is maintained by respiration, the reaction equilibrium is shifted to the right side and acidification results. Taken from [1].

of the weak acid (CO_2 and H_2CO_3) not being changed as indicated in Figure 8 [1].

Buffers as “chemical engines” maintain a constant pH, which is dependent on the weak acid $[\text{HA}]$ to its conjugated base $[\text{A}^-]$ concentration ratio: $[\text{H}^+] = K_a [\text{HA}]/[\text{A}^-]$ (K_a is the dissociation constant). The concentrations of the weak acid and its conjugated base change identically on dilution or concentration – their concentration ratio remains constant and hence, so does the pH. In blood buffering systems, however, the CO_2 concentration and hence, the weak acid (H_2CO_3) concentration in arterial blood

(and also in venous blood and in the tissues if the flow rate does not change) are constant, whereby the equilibrium in the buffer systems shifts to the right (acid) side on dilution or to the left (alkaline) side on concentration.

If saline (NaCl) is added to the plasma, then the initiating cause of dilution does not rest in the fact that chloride increases relatively to Na^+ : the decisive phenomenon is the dilution of the buffers (including the conjugated bases, giving rise to SID decrease), while neither CO_2 nor H_2CO_3 is diluted. This gives rise to a shift of equilibria within the plasma buffer system – to a HCO_3^- concentration decrease and a matching non-bicarbonate base concentration increase (without any change in the SID, reduced by dilution, or BE), and pH decrease. The dilution effect manifests itself in the same way on the dilution of crystalloids (without changing the Na^+/Cl^- concentration ratio).

We implemented the Watson model [21] as a teaching aid as a part of our Atlas of

Physiology and Pathophysiology [22] (http://www.physiome.cz/atlas/acidobaze/02/ABR_v_plazme1_2.html), enabling the plasma dilution (or concentration) simulation experiment to be interactively replayed (see Figure 9).

In contrast to what occurs in the plasma model, the pH changes occurring on dilution or hemoconcentration are dampened by engaging intracellular buffers [23,24] and by the response of the kidneys.

The pathogenetic explanation of dilution acidosis is presented in Figure 10, while the pathogenetic explanation of contraction alkalosis disorders is shown in Figure 11.

6. Pathogenesis of hyperchloremic acidosis and hypochloremic alkalosis

The pathogenetic causes of hyperchloremic alkalosis are shown in Figure 12. According to Stewart, the bicarbonate concentration and pH adapt to the SID and pCO_2 . In the balance approach the bicarbonate level is determined by the bicarbonate loss/supply balance, and by the appropriate equilibrium in the buffer system. 1 mmol/l of bicarbonate removal does not imply bicarbonate level reduction by 1 mmol: the

reduction will be somewhat smaller because the non-bicarbonate base levels will also be lowered. 1 mmol/l of bicarbonate removal, however, will reduce the SID by 1 mmol.

In Stewart's concept, hypochloremic alkalosis is a result of SID lowering owing to the relative chloride concentration increase. Bicarbonate will adapt to that and will lower its concentration. In the balance concept, the basic cause is bicarbonate accumulation accompanied by an equimolar chloride loss (see Figure 13).

7. Pathogenesis of metabolic acidosis accompanied by an increase in strong unmeasured anions

According to Stewart, the pathogenetic cause of acidosis accompanied by an increase in the strong unmeasured anions rests in the lowering of the SID: the bicarbonate level will respond by decreasing, whereupon the pH will decrease as well. In balance theory this phenomenon basically results from the retention of the strong dissociated acids by the ECF. Hydrogen ions bind immediately to bicarbonate, thereby reducing the concentration of the latter. So, retention of strong acid anions is accompanied by an equimolar loss of bicarbonates that release hydrogen ions from the retained acids (Figure 14).

8. Pathogenesis of hypoalbuminemic alkalosis

Albumin is a negatively charged protein. Albumin synthesis and degradation are electroneutral processes. While synthesized, albumin is accompanied by hydrogen ions (and adequate consumption of bicarbonates). During catabolism, albumin is degraded by an electroneutral route in the metabolism along with the hydrogen ions (the consumption of hydrogen ions is accompanied by formation of bicarbonates). Normally, the albumin synthesis/degradation rates are in equilibrium and the balance of the bicarbonates and of the negatively charged albumin anions is not disturbed. The pathogenetic cause of hypoalbuminemic alkalosis is a negative albumin formation/degradation balance (e.g. on an increase in the catabolism of albumin as a rapid aminoacid source during major surgical procedures). This results in a pH shift to the alkaline side, while the SID value remains unchanged and the albumin level decreases. In this respect the explanation in balance theory differs from that in Stewart's concept, where the albumin level decrease while the SID value is constant is the initiating event (Figure 15).

9. Pathogenesis of hyperphosphatemic alkalosis

Hyperphosphatemic alkalosis is due to accumulation of phosphates together with hydrogen ions. Hydrogen ions are released by bicarbonates, and thus a growth of phosphates is accompanied by an equimolar consumption of bicarbonates, the SID remaining unchanged (see Figure 16).

10. Why create acid-base equilibrium models according to the balance approach?

We see the major balance concept contribution in the causal explanation of the interlinking between the acid-base disorders and ion and volume disorders, and in the understanding of internal environment disorders from the integrative physiology aspect. We agree that a clinician needs a phenomenological description of the actual acid-base equilibrium for the diagnosis and selection of appropriate therapy. Stewart's approach offers a fairly simple and practically applicable, although pathophysiologically incomplete explanation of acid-base disorders by using 3 indepen-

dent variables. The balance concept enables you to understand the pathogenetic causes of the phenomena described by Stewart's method.

It is particularly combined disorders that require the interrelations and disorders of the volume and reserves (not only concentrations) of the components to be considered, and this cannot be done without the balance concept.

Where new views upon the diagnosis are to be used and calculations applying mathematical models to patient data are to be made, you cannot do without the balance method.

A number of rather complex acid-base equilibrium models have been published [25–30] However, they are actually too complex to find application in the clinical practice and are unable to serve as a guide for selecting the best treatment method against acid-base equilibrium disorders. Such complex models are sometimes only created as scientific outcomes to be cited in other papers. Sometimes the authors claim to have created something useful for clinicians, but this is hardly more than just a statement.

We do not wish to set up models to augment the number of papers in scientific journals and to be cited in later papers, but to make a real contribution for clinical doctors:

- To use the models as simulators enabling the pathogenesis of the most diverse internal environment disorders to be incrementally monitored and to enable clinicians, by looking “under the bonnet”, to understand what is actually going on during the development of a pathogenesis. So, our efforts also include the development of a model fitted with a convenient user interface for presentation on a website.
- The creation of models as tools for diagnosis and for corrective therapy calculations. We feel that a wealth of hitherto unused information is not only hidden in the clinically measured data, but also in the dynamics of responses to the initiating events, such as reactions to infusions.

References

- [1.] Kofránek J. Bilanční pojetí acidobazické rovnováhy (Balance concept of acid-base equilibrium, in Czech). *Medsoft*. 2017;29: 34–56.
- [2.] Matousek S, Kofranek J, Rees SE. Independence of Variables in Stewart's Model of the Acid-Base Chemistry of the Blood Plasma. *IFAC Proceedings Volumes*. Elsevier; 2009;42: 246–250.
- [3.] Matousek S, Handy J, Rees SE. Acid–base chemistry of plasma: consolidation of the traditional and modern approaches from a mathematical and clinical perspective. *J Clin Monit Comput*. Springer Netherlands; 2010;25: 57–70.
- [4.] Kellum JA. Clinical review: reunification of acid-base physiology. *Crit Care*. *BioMed Central*; 2005;9: 500.
- [5.] Kamel KS, Halperin ML. *Fluid, Electrolyte and Acid-Base Physiology: A Problem-Based Approach*. Elsevier Health Sciences; 2016.
- [6.] Matoušovic K, Martinek V, Kvapil M. Acidobazická rovnováha tělesných tekutin a její kvantitativní fyzikálně-chemické hodnocení (Acid-base equilibrium of body fluids and its quantitative physico-chemical evaluation, in Czech). *Aktual Nefrol*. 2002;4: 150–156.
- [7.] Schück O, Matoušovic K. Vztah mezi pH a diferencí silných iontů (SID) ve vnitřním prostředí (Relation between pH and Strong Ion Difference (SID) in Body Fluids, in Czech). *Klin Biochem Metab*. *nts.prolekare.cz*; 2005; Available: http://nts.prolekare.cz/cis/odkazy/kbm0501_32.pdf
- [8.] Engliš M, Jabor A, Kubáč P, Červinka I. Příspěvek k hodnocení metabolické složky poruch acidobazického metabolismu podle Stewartovy a Fenclovy koncepce (Contribution to the evaluation of metabolic components of acid-base metabolism according to Stewart and Fenclo's concepts, in Czech). *Klin Biochem Metab*. 2006;14: 225–227.
- [9.] Stewart PA. *How to Understand Acid-base: A Quantitative Acid-base Primer for Biology and Medicine*. Edward Arnold; 1981.
- [10.] Stewart PA. Modern quantitative acid-base chemistry. *Can J Physiol Pharmacol*. 1983;61: 1444–1461.
- [11.] Rossing TH, Maffeo N, Fenclo V. Acid-base effects of altering plasma protein concentration in human blood in vitro. *J Appl Physiol*. 1986;61: 2260–2265.
- [12.] McAuliffe JJ, Lind LJ, Leith DE, Fenclo V. Hypoproteinemic alkalosis. *Am J Med*. 1986;81: 86–90.
- [13.] Fenclo V, Rossing TH. Acid-base disorders in critical care medicine. *Annu Rev Med*. 1989;40: 17–29.
- [14.] Fenclo V, Leith DE. Stewart's quantitative acid-base chemistry: applications in biology and medicine. *Respir Physiol*. 1993;91: 1–16.
- [15.] Figge J, Jabor A, Kazda A, Fenclo V. Anion gap and hypoalbuminemia. *Crit Care Med*. *journals.lww.com*; 1998;26: 1807–1810.
- [16.] Fenclo V, Jabor A, Kazda A, Figge J. Diagnosis of Metabolic Acid–Base Disturbances in Critically Ill Patients. *Am J Respir Crit Care Med*. 2000;162: 2246–2251.
- [17.] Kofránek J, Matoušek S, Andrlík M. Border flux balance approach towards modelling acid-base chemistry and blood gases transport. In: Zupanic VB, Karba S, Blažič S, editors. *Proceedings of the 6th EUROSIM Congress on Modeling and Simulation*, <http://www.physiome.cz/references/ljubljana2007.pdf>. Ljubljana: University of Ljubljana.; 2007. pp. CD Rom Proceedings: TU–1–P7–4: 1–9.
- [18.] Halperin ML, Kamel KS. Some observations on the clinical approach to metabolic acidosis. *J Am Soc Nephrol*. 2010;21: 894–897.
- [19.] Russo MA. Dilutional Acidosis A Nonentity? *Anesthesiology*. 1997;87: 1010–1011.
- [20.] Kazda A, Jabor A. Hodnocení vztahů mezi ionty Natria a chloridů při posuzování náleží acidobazické rovnováhy (Evaluation of relationships between sodium and chloride ions during assessing acid-base equilibrium findings, in Czech). *Klin Biochem Metab*. 2001;9: 199–201.
- [21.] Watson PD. Modeling the effects of proteins on pH in plasma. *J Appl Physiol*. *Am Physiological Soc*; 1999;86: 1421–1427.
- [22.] Kofránek J, Matoušek S, Rusz J, Stodulka P, Privitzer P, Mateják M, et al. The Atlas of Physiology and Pathophysiology: Web-based multimedia enabled interactive simulations. *Comput Methods Programs Biomed*. 2011;104: 143–153.
- [23.] Rosenbaum BJ, Makoff DL, Maxwell MH. Acid-base and electrolyte changes induced by acute isotonic saline infusion in the nephrectomized dog. *J Lab Clin Med*. 1969;74: 427–435.
- [24.] Garella S, Tzamaloukas AH, Chazan JA. Effect of isotonic volume expansion on extracellular bicarbonate stores in normal dogs. *Am J Physiol*. 1973;225: 628–636.
- [25.] Wooten EW. Calculation of physiological acid-base parameters in multicompartment systems with application to human blood. *J Appl Physiol*. 2003;95: 2333–2344.

- [26.] Wooten EW. Strong ion difference theory: More lessons from physical chemistry. *Kidney Int.* Elsevier; 1998;54: 1769–1770.
- [27.] Morgan TJ. Partitioning standard base excess: a new approach. *J Clin Monit Comput.* 2011;25: 349–352.
- [28.] Wolf MB, Deland EC. A mathematical model of blood-interstitial acid-base balance: application to dilution acidosis and acid-base status. *J Appl Physiol.* 2011;110: 988–1002.
- [29.] Wolf MB. Whole body acid-base and fluid-electrolyte balance: a mathematical model. *Am J Physiol Renal Physiol.* 2013;305: F1118–31.
- [30.] Wolf MB. Comprehensive diagnosis of whole-body acid-base and fluid-electrolyte disorders using a mathematical model and whole-body base excess. *J Clin Monit Comput.* 2015;29: 475–490.

Contact

Jiří Kofránek

Laboratory of Biocybernetics, Institute of Pathophysiology,
First Faculty of Medicine, Charles University,
Prague, Czech Republic,
Praha U Nemocnice 5 128 53, Praha 2
e-mail: kofranek@gmail.com

MODELOVÁNÍ EHEALTH PROCESŮ V POMOCÍ HIERARCHICKÝCH STAVOVÝCH AUTOMATŮ (STATECHARTS)

Jiří Kofránek, Jiří Berger, Jiří Polák, Adam Vojtěch

Abstrakt

Při návrhu informačních systémů pro eHealth je podstatné dobře popsat strukturu procesů navrhovaných systémů. Modely využívající hierarchické stavové automaty (statecharts) jsou efektivním nástrojem pro jejich dynamický popis a následnou simulaci, která je podkladem interdisciplinárního porozumění mezi architekty informačních systémů, lékaři a tvůrci legislativy. Dobře navržené procesy umožní vyhnout se chybám v navrhované architektuře i nezbytné legislativní podpoře, které se později těžko napravují.

1. Nerozorané brázdý mezi obory

„Po mnoho let jsme byli dr. Rosenblueth a já přesvědčeni, že neplodnějšími obory pro rozvoj věd jsou ty, které byly zanedbávány jako země nikoho mezi různými, pevně vymezenými vědními oblastmi“ – napsal Norbert Wiener v úvodu své knihy, který dala název novému vědnímu oboru – kybernetice [1]. Měl pravdu – právě v nerozoraných brázdách na styku oborů se dají, obrazně řečeno, nalézt zlaté valounky řešení složitých problémů. Jednou z oblastí, kde mezioborová spolupráce je klíčem k řešení spleťtých problémů, je rozvoj elektronického zdravotnictví (eHealth). Zdravotnické informační systémy mají ulehčit práci zdravotníkům. Prvním problémem je mezioborové porozumění mezi zdravotníky a informatiky. Informační systémy proto musí vznikat v neustálém vzájemném dialogu. Bez určitého překryvu znalostí se to ale neobejde. Informatičtí musí pochopit potřeby zdravotníků a nabídnout efektivní informatické řešení jejich potřeb, zdravotníci musí pochopit možnosti a způsob fungování navrhovaného informatického řešení.

Nejde ale jen o spolupráci zdravotníků a informatiků.

Pro občana platí, že činnosti, které nejsou legislativně upraveny, je dovoleno vykonávat libovolným způsobem. Ve veřejné správě je to naopak. Veřejná správa může vykonávat jen to, co jí zákon umožňuje. Ve veřejné správě je nutné veškerou činnost mít ošetřenou legislativně (veřejná správa může dělat jen ty činnosti, která má popsány v zákonech a nařízeních). Realizace národní strategie elektronického zdravotnictví proto musí být legislativně ošetřena – to ovšem vyžaduje také mezioborové porozumění i mezi informatiky, zdravotníky a legislativci, a konec konců i mezi politiky.

Legislativci musí dobře pochopit strukturu a funkci navrhovaného informatického řešení a na základě tohoto pochopení legislativně dostatečně jednoznačně popsat veškeré procesy v budovaném národním zdravotnickém informačním systému. Zde jsou zatím bohužel ještě velké problémy.

Z nedostatečné vzájemné komunikace a částečného nepochopení pak vznikají problémy, které nakonec někdy vedou i ke zbytečným sporům na půdě