Acid-base status evaluation

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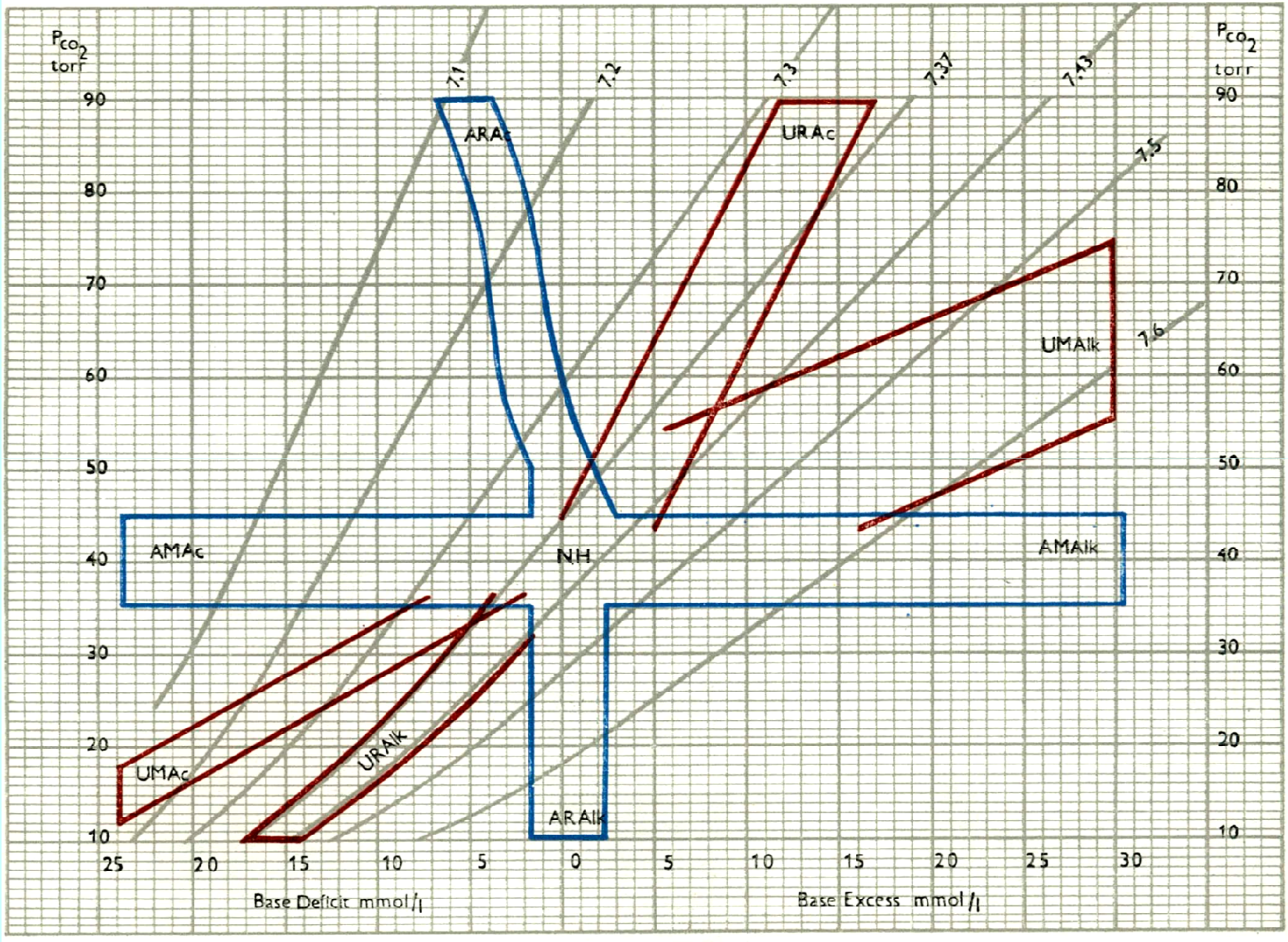
Graph of acid-base regulation



Graf acidobazické regulace (M. Engliš, Prakt. lék. 1972)

Buffer system in blood

buffers	acid	base	% relative	present in
hydrogen-carbonic	H_2CO_3	HCO^-_3	51	plasma
hemoglobin	oxyhemoglobin	deoxyhemoglobin	35	erytrocytes
phosphates	$H_2PO^-_4$	HPO^{2-}_4	7	erytrocytes (plasma)
proteins	-COOH	$-COO^-$	7	plasma (erytrocytes)

Henderson-Hasselbach's equation:

$$pH = pK + \log \frac{[base]}{[acid]}$$

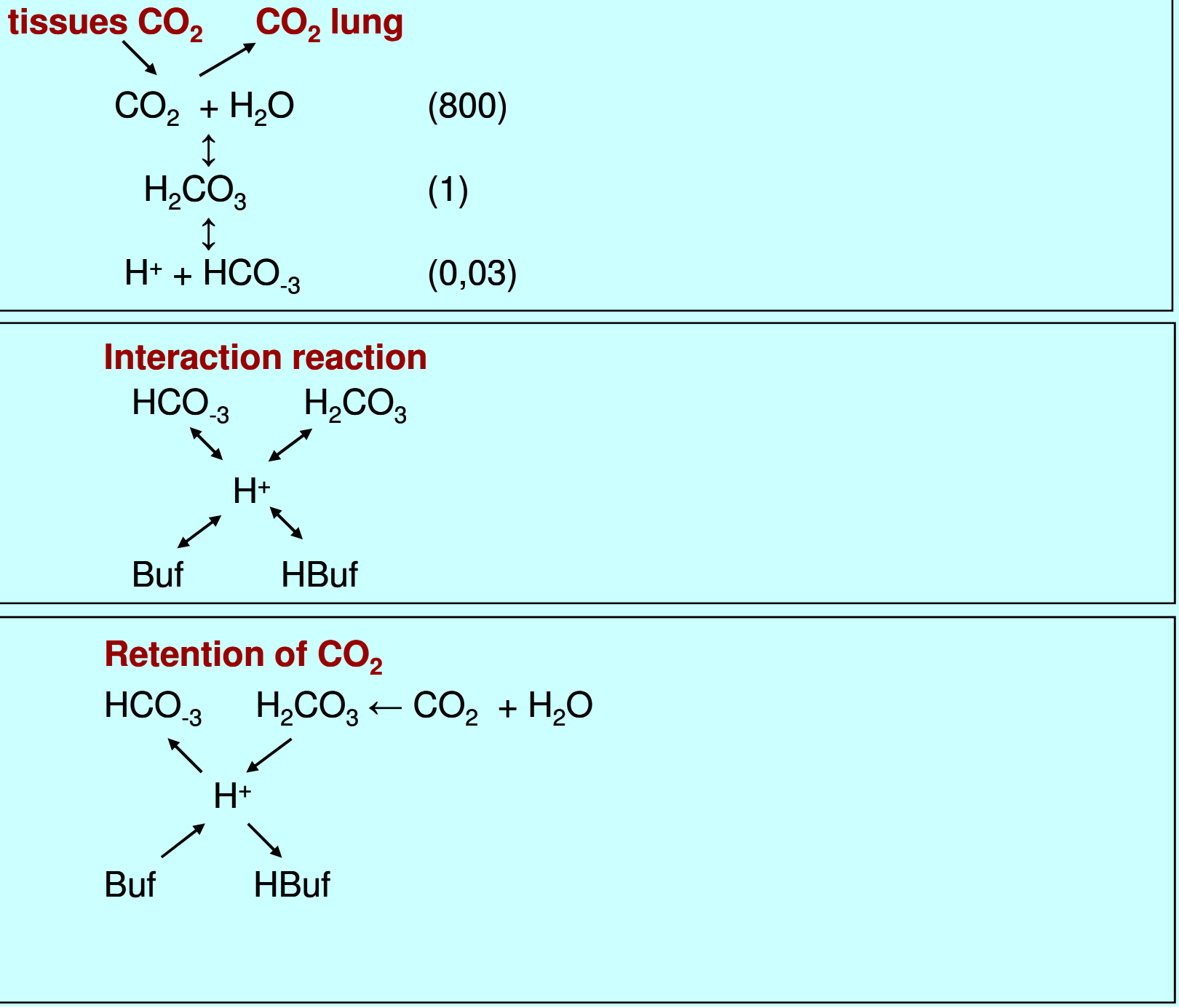
Henderson-Hasselbach's equation:

$$pH = pK + \log \frac{[HCO_{-3}]}{[H_2CO_3]}$$

Carbonic acid concentration can be expressed as a function of pCO_2

$$pH = pK + \log \frac{[HCO_{-3}]}{\alpha \, . \, pCO_2}$$

α = coeficient of solubility of CO_2, 0,0306 $mol^{-1}1^{-1}torr^{-1}$, tj. 0,2295 $mol^{-1}1^{-1}kPa^{-1}$
pK = approximately 6,1


tissues CO_2
CO_2 lung
$CO_2 + H_2O$ (800)
H_2CO_3 (1)
$H^+ + HCO_{-3}$ (0,03)
Interaction reaction
HCO_{-3}
H_2CO_3
H^+
Buf
HBuf
Retention of CO_2
HCO_{-3}
$H_2CO_3 \leftarrow CO_2 + H_2O$
H^+
Buf
HBuf

SYSTEMS USED FOR INTERPRETATION OF ACID-BASE DATA

How to identify and to quantitate the various cause of metabolic acid-base disturbances has been puzzle in interpreting acid-base date. Two systems are commonly used:

metabolic alkalosis / acidosis is detected by

1. **a high / low bicarbonate concentration ($[HCO_3^-]$),**
2. **a base excess / deficit (BE)**

These systems share two important limitations.

1. The first limitation $[HCO_3^-]$ and BE (as well as pH) are determined in plasma by several variables that can change independently of one another. Both alkalinizing and acidifying disturbances can exist simultaneously among these variables. Combined abnormalities may escape detection because they offset one another°s effects on $[HCO_3^-]$, BE, and pH.

2. The second limitation is in their ability to identify the various primary causes of metabolic alkaloses and acidoses. BE and $[HCO_3^-]$ do not give direct information about individual primary cause of metabolic acid-base derangements.

STEWART`S APPROACH

Acid-base state in a body fluid is determined by independent variables.

In blood plasma in vivo, the independent variables are:

1. PCO_2

2. the strong ion difference (SID), i.e. the difference between the sums of all the strong (fully dissociated, chemically nonreacting) cations (Na^+, K^+, Ca^{2+}, Mg^{2+}) and all the strong anions (Cl^- and other strong anions),

3. concentrations of nonvolatile weak acids.

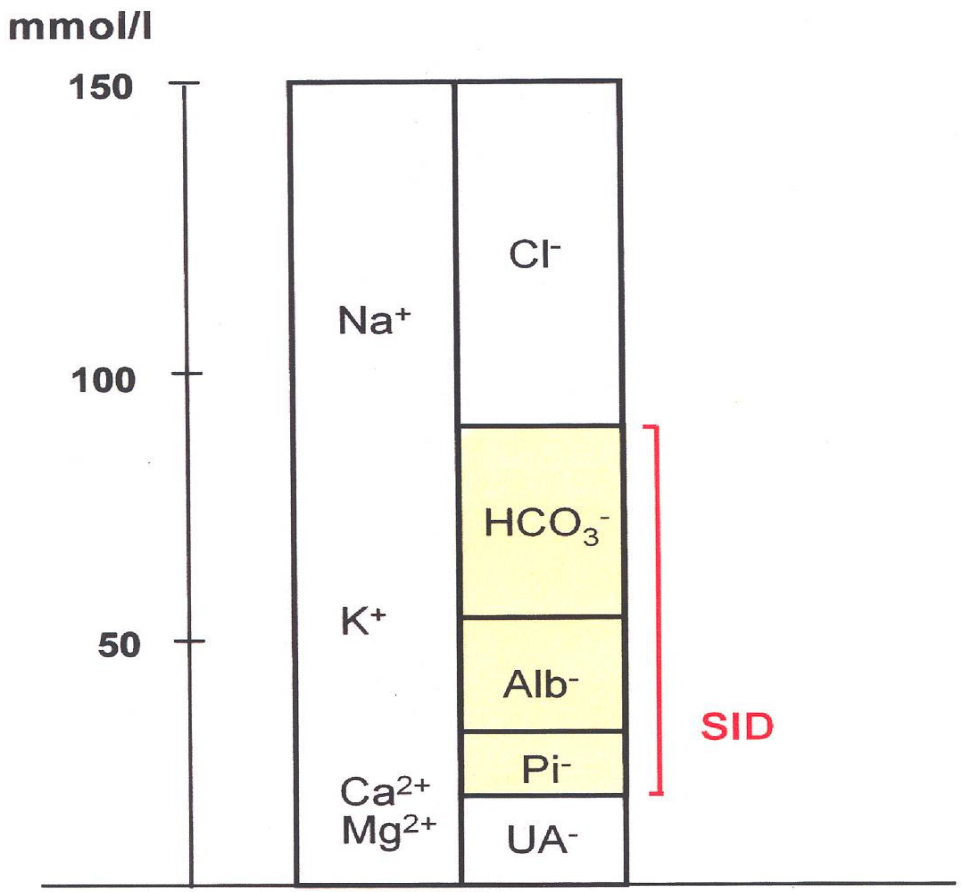
None of the other acid-base variables (e.g. pH, [HCO_3^-], BE, ect.) can change primarily. They are dependent variables that change, if one or more of the independent variables changes.

CLASSIFICATION OF PRIMARY ACID-BASE DISTURBANCES

Disturbance	Acidosis	Alkalosis
I. Respiratory	↑ PCO_2	↓ PCO_2
II. Non-respiratory (metabolic)		
1. Abnormal SID		
a. Water excess/deficit	↓ SID	↑ SID
	↓ $[Na^+]$	↑ $[Na^+]$
b. Imbalance of strong anions		
- Chloride excess/deficit	↓ SID	↑ SID
	↑ $[Cl^-]$	↓ $[Cl^-]$
- Unidentified anion excess	↓ SID	
	↑ $[UA^-]$	
2. Nonvolatile weak acids		
a. Serum albumin	↑ [Alb]	↓ [Alb]
b. Inorganic phosphate	↑ [Pi]	↓ [Pi]

METABOLIC ACID-BASE DISTURBANCES AND THEIR CLINICAL REASONS AFTER STEWART

Diagnosis	Clinical cause
dilutional acidosis	every cause of hyponatremia
concentrational alkalosis	every cause of hypernatremia
hyperchloremic acidosis	infusions rich in chloride diarrhea tubular dysfunction during acute or chronic renal failure compensation of chronic RAL
hypochloremic alkalosis	loss of gastric juice diuretics compensation of chronic RAC
acidosis from elevation of UA	uremia, ketoacidosis, lactic acidosis, intoxications (salicilates, methanol, ethanol)
hyperphosphatemic acidosis	every cause of hyperphosphatemia (renal failure)
hypoalbuminemic alkalosis	hepatic insufficiency, malnutrition, nephrotic sd, burns

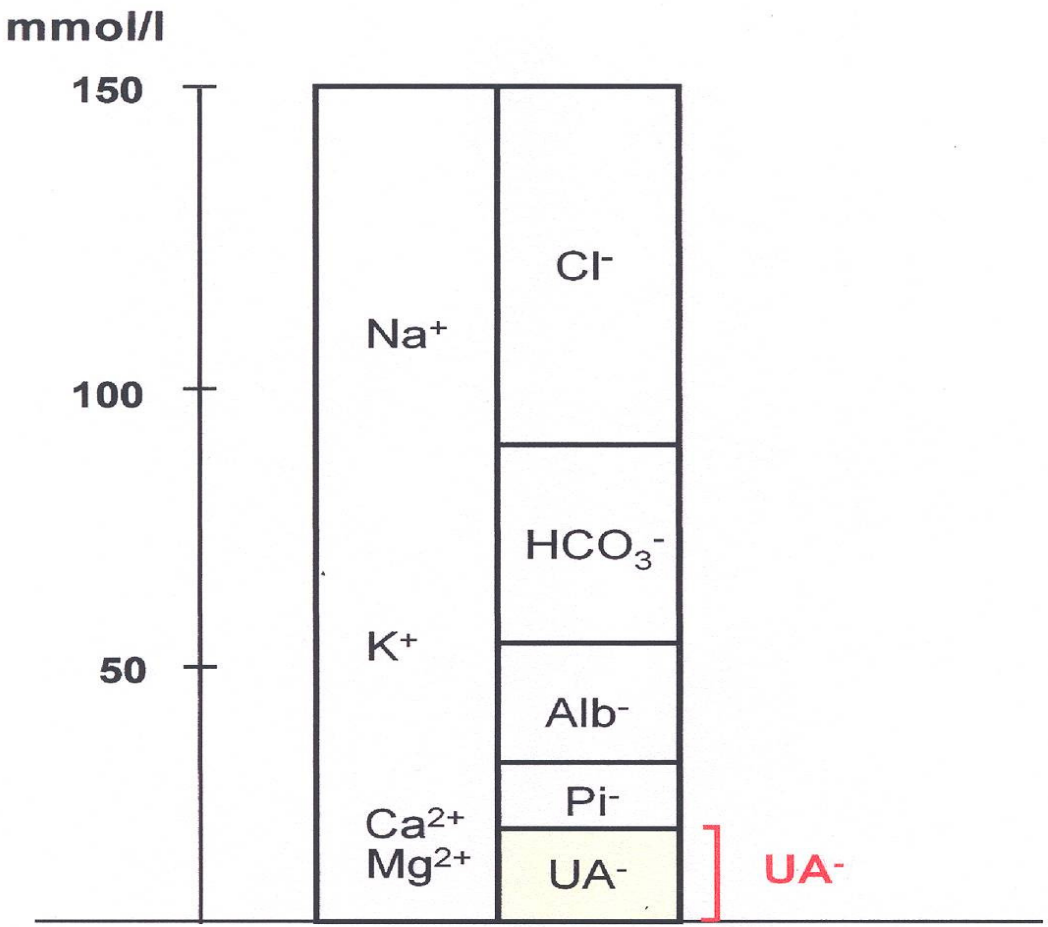


STRONG ION DIFFERENCE – SID

mmol/l

SID = $[Na^+] + [K^+] + [Ca^{2+}] + [Mg^{2+}] - ([Cl^-] + [UA^-])$

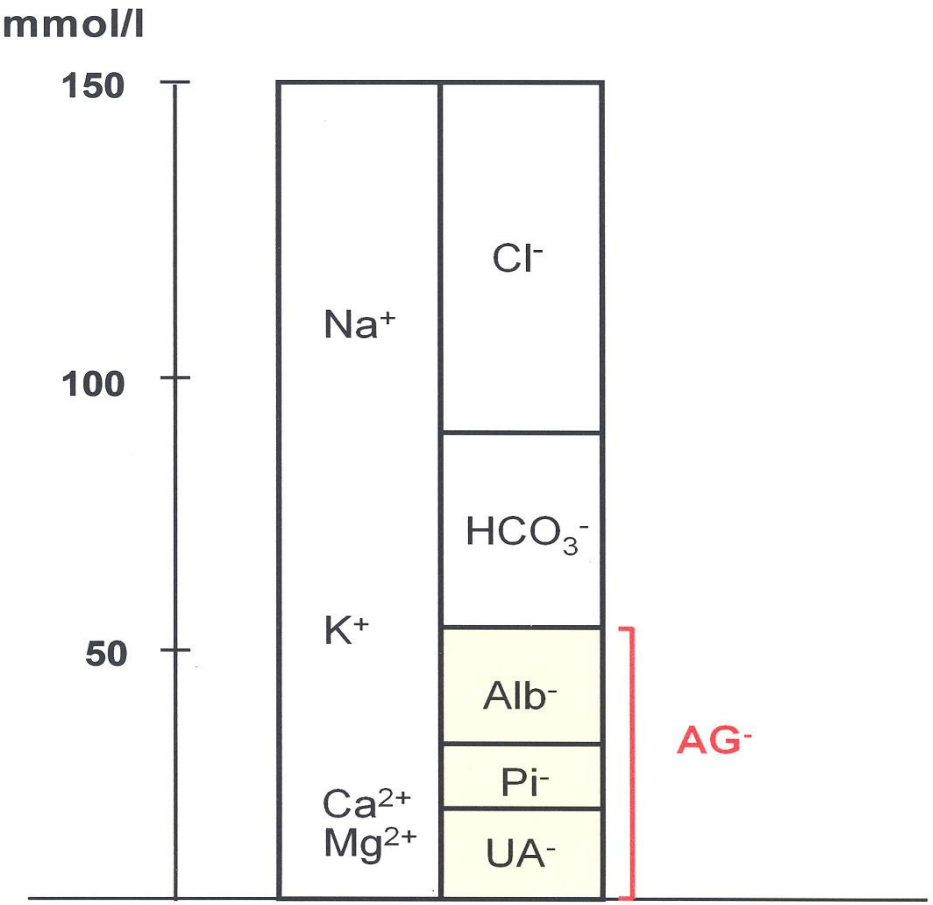
SID = $[HCO_3^-] + 0.28 \times [Alb]g/l + 1.8 \times [Pi]$



UNMEASURED ANIONS - UA^-

mmol/l

$[UA^-] = [Na^+] + [K^+] + [Ca^{2+}] + [Mg^{2+}] - ([Cl^-] + SID)$



ANION GAP - AG

mmol/l

AG = $[Na^+] + [K^+] - ([Cl^-] + [HCO3^-])$

AG = $[Na^+] - ([Cl^-] + [HCO_3^-])$

AGadjusted = AG + 0.25 x $([Alb]_{normal}-[Alb]_{measured})$

(where $[Alb]_{normal}-[Alb]_{measured}$ represent normal and actualy measured plasmatic albumin in g/l)

ANION GAP ADJUSTED - AG_{adj}

$$AG = [Na^+] + [K^+] - ([Cl^-] + [HCO_3^-])$$

$$AG_{adj} = AG + 0.25 \times ([Alb]_{normal} - [Alb]_{measured})$$

concentrations in plasma (ions mmol/l, albumin g/l)	Na^+	K^+	Cl^-	HCO_3^-	UA^-	Alb	AG	AG_{adj}
physiologic state	140	4.4	104	24	8	40	16.4	16.4
decreased albumin	140	4.4	104	29	8	20	11.4	16.4
decreased albumin + increased lactate	140	4.4	104	24	13	20	16.4	21.4

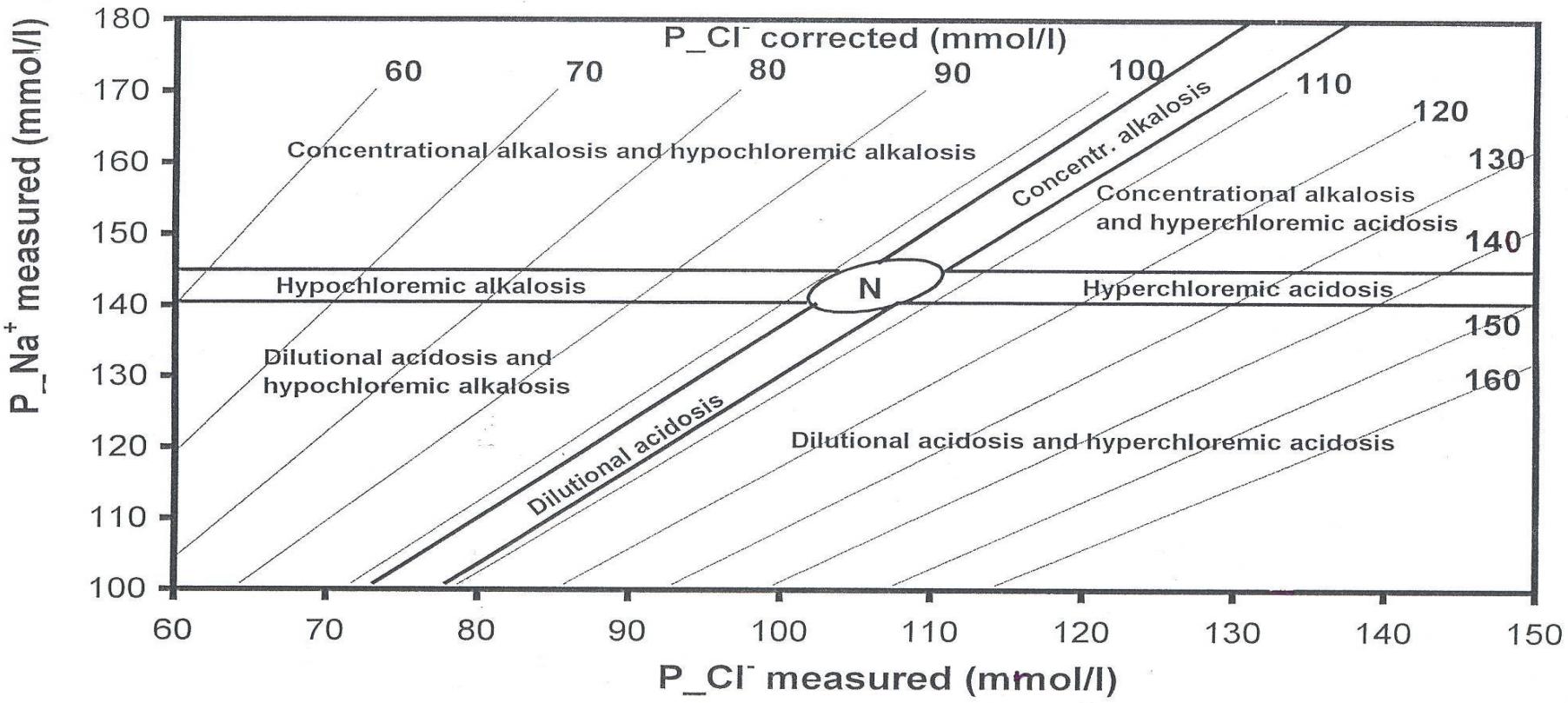
CORRECTION OF CHLORIDE ANION (Cl^-)

Calculation estimates, how in cases of hyponatraemia or hypernatraemia the level of chlorides in plasma (serum) would change with normalisation of Na^+ dysbalance.

Calculation estimates, whether during dysbalance of Na^+ the change of Cl^- is proportional to change of Na^+ or if it is changed more or less than Na^+.

$$[Cl^-]_{corr} = [Cl^-]_{meas} \times [Na^+]_{norm}/[Na^+]_{meas}$$

Classification of sodium and chloride disturbances



Examples

of hyperchlorid hyperchloremic acidosis and hypochloremic alkalosis, dilutional acidosis and concentrational alkalosis and their mutual combinations

Situation	Na^+	K^+	Ca^{2+}	Mg^{2+}	Cl^-	Cl^-_{korig}
physiologic	140	4,4	2,0	0,8	102	102
hyperchloremic acidosis	140	4,4	2,0	0,8	112	112
hypochloremic alkalosis	140	4,4	2,0	0,8	92	92
dilutional acidosis (water surplus)	119	3,7	1,7	0,7	86,7	102
dilutional acidosis + hyperchloremic acidosis	119	3,7	1,7	0,7	95	117,1
dilutional acidosis + hypochloremic alkalosis	119	3,7	1,7	0,7	78	91,7
concentrational alkalosis (water deficit)	161	5,1	2,3	0,9	117,3	102
concentrational alkalosis + hypochloremic alkalosis	161	5,1	2,3	0,9	111	96,5
concentrational alkalosis + hyperchloremic acidosis	161	5,1	2,3	0,9	125	108,7

Biochemical parameters necessary for evaluation of acid base finding

1. Acido-basic:
   pH, pCO_2, BE, HCO_3^- actual

2. For calculations of independent variables in serum (plasma):
   Na^+, K^+, Cl^-, P, Ca, Mg, albumin

3. For additional informations:
   lactate, oxygen parameters, urea, creatinin;
   in urine pH and ketonuria

parameter	reference range
anion gap	14-18
strong ion difference	37-41
Cl^- corrected	102-105
unmeasured anions	6-10
charge of albumin	11,2 (for pH 7,4 and P_alb 40 g/l)
charge of phosphates	1,8 (for pH 7,4 and P_phosphates 1,0 mmol/l)

Patient No 1, diagnosis:

Bronchopneumonia. Cirrhosis hepatis.
Chronic ethylism.

Measured parameters		Calculated parameters	
Na^+	125 mmol/l	HCO_3^-	24 mmol/l
K^+	5,2 mmol/l	$AG_{observed}$	8 mmol/l
Ca^{2+}	1,6 mmol/l	$AG_{adjusted}$	16 mmol/l
Mg^{2+}	0,6 mmol/l	BE	0 mmol/l
Cl^-	98 mmol/l	SID	29 mmol/l
Pi	0,9 mmol/l	Cl^-_{corr}	111 mmol/l
Albumin	13 g/l	UA^-_{corr}	8 mmol/l
pH	7,40		
pCO_2	5,2 kPa		

Evaluation: Acid-base values in reference intervals.

- SID value is decreased as result of water excess([Na^+] = 125 mmol/l) and of Cl^- surplus, recognized after Cl^- correction.
- This acidosis caused with two reasons is fully compenzated by hypoalbuminemic alkalosis.

Conclusion: Mixed disturbance. Acidosis caused by water excess and hyperchloremia
+ hypoalbuminemic alkalosis.

Patient No 2, diagnosis:

State after surgery for ulcus pylori penetrans ad vesicam felleam. During two weeks after surgery diarrhoea, dehydration, confusion, somnolence. This examination in 3rd week, to date few tenuous stools daily, diuresis about 1500 ml/day.

Measured parameters				Calculated parameters	
$[Na^+]$	159 mmol/l	[urea]	29 mmol/l	$[HCO^-_3]$	25,5 mmol/l
$[K^+]$	4,6 mmol/l	[kreat]	390 mmol/l	$Ag_{observed}$	26,1 mmol/l
[Ca]	2,1 mmol/l	[lakt]	2,2 mmol/l	$Ag_{adjusted}$	28,1 mmol/l
[Mg]	0,87 mmol/l	pH	7,41	BE_b	+1 mmol/l
$[Cl^-]$	112 mmol/l			BE_{ecf}	+ 1,3 mmol/l
[Pi]	2,5 mmol/l			SID	38,7 mmol/l
[Alb]	32 g/l			$[Cl^-]_{corr}$	98,6 mmol/l
pCO_2	5,13 kPa			$[UA^-]_{corr}$	14 mmol/l

Evaluation: Acid-base values in reference intervals.

- values of AG and UA are elevated – give evidence for metabolic acidosis
- hypernatremia and hypoalbuminemia – causes of metabolic alkalosis

Conclusion: Mixed disturbance. Metabolic acidosis accompanying renal insuficiency (with hyperfosfatemia) and metabolic alkalosis from two reasons.

Thank you for your attention