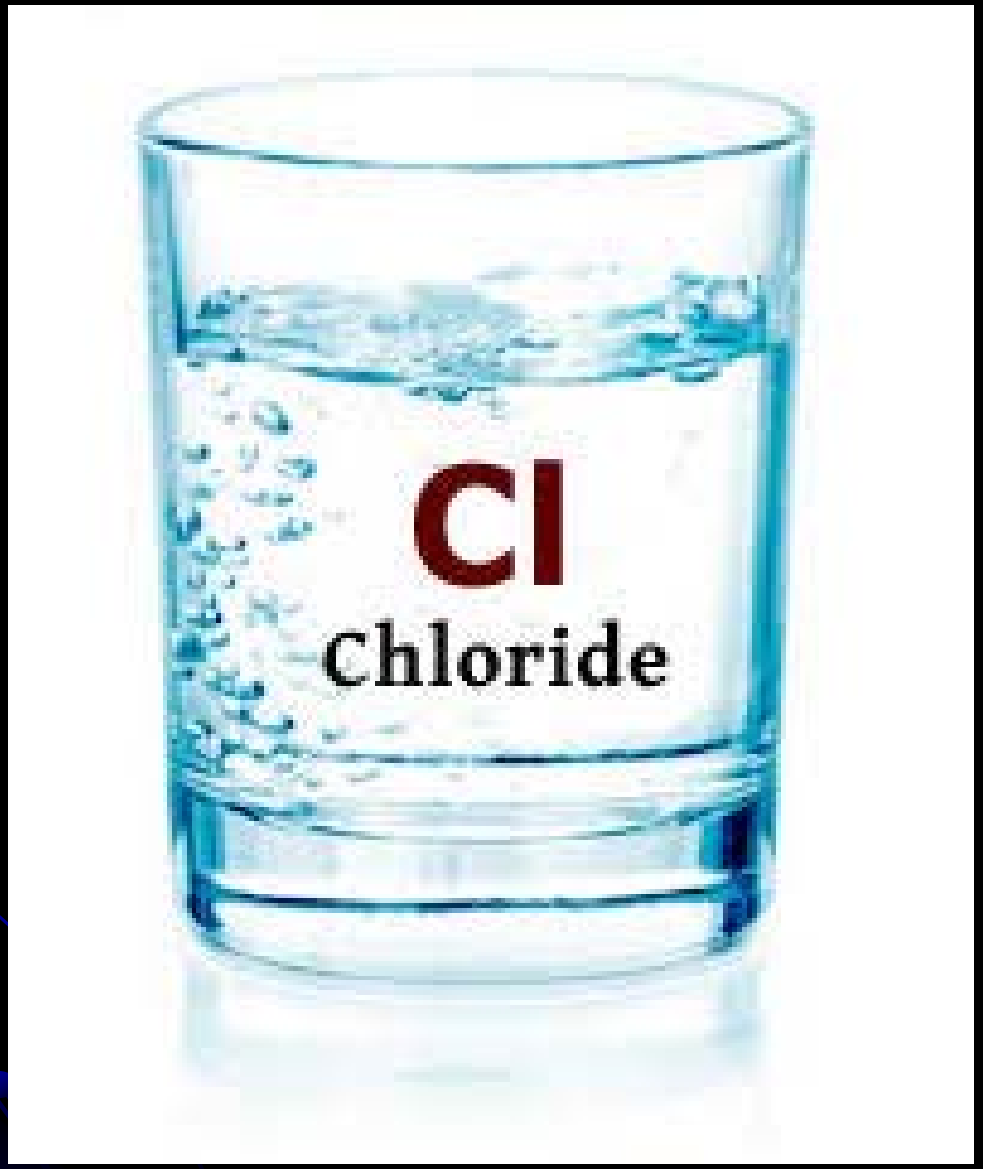
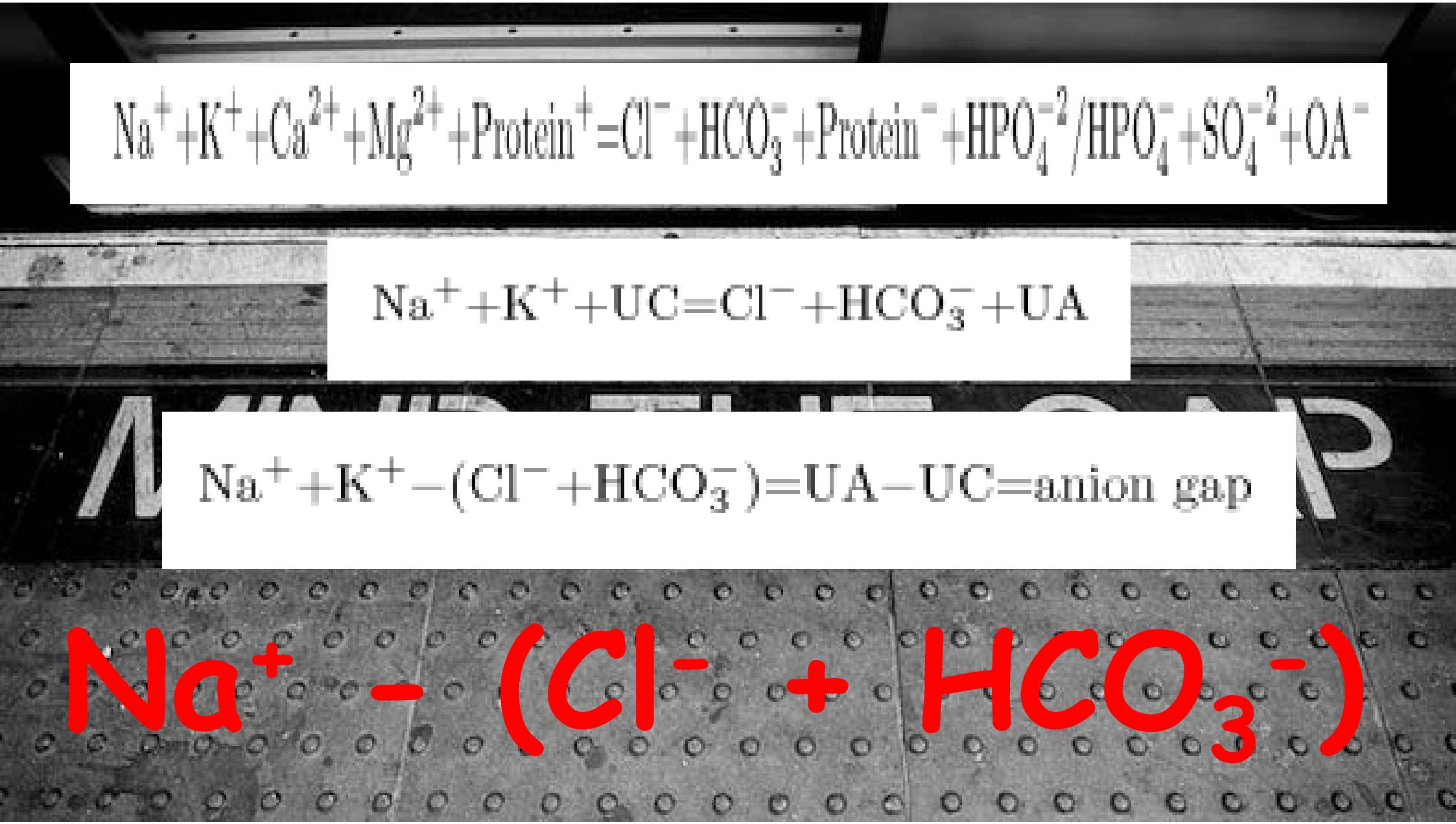
ΟΙ ΠΟΛΛΕΣ ΑΠΟΧΡΩΣΕΙΣ ΤΗΣ ΥΠΕΡΧΛΩΡΑΙΜΙΚΗΣ ΜΕΤΑΒΟΛΙΚΗΣ ΟΞΕΩΣΗΣ

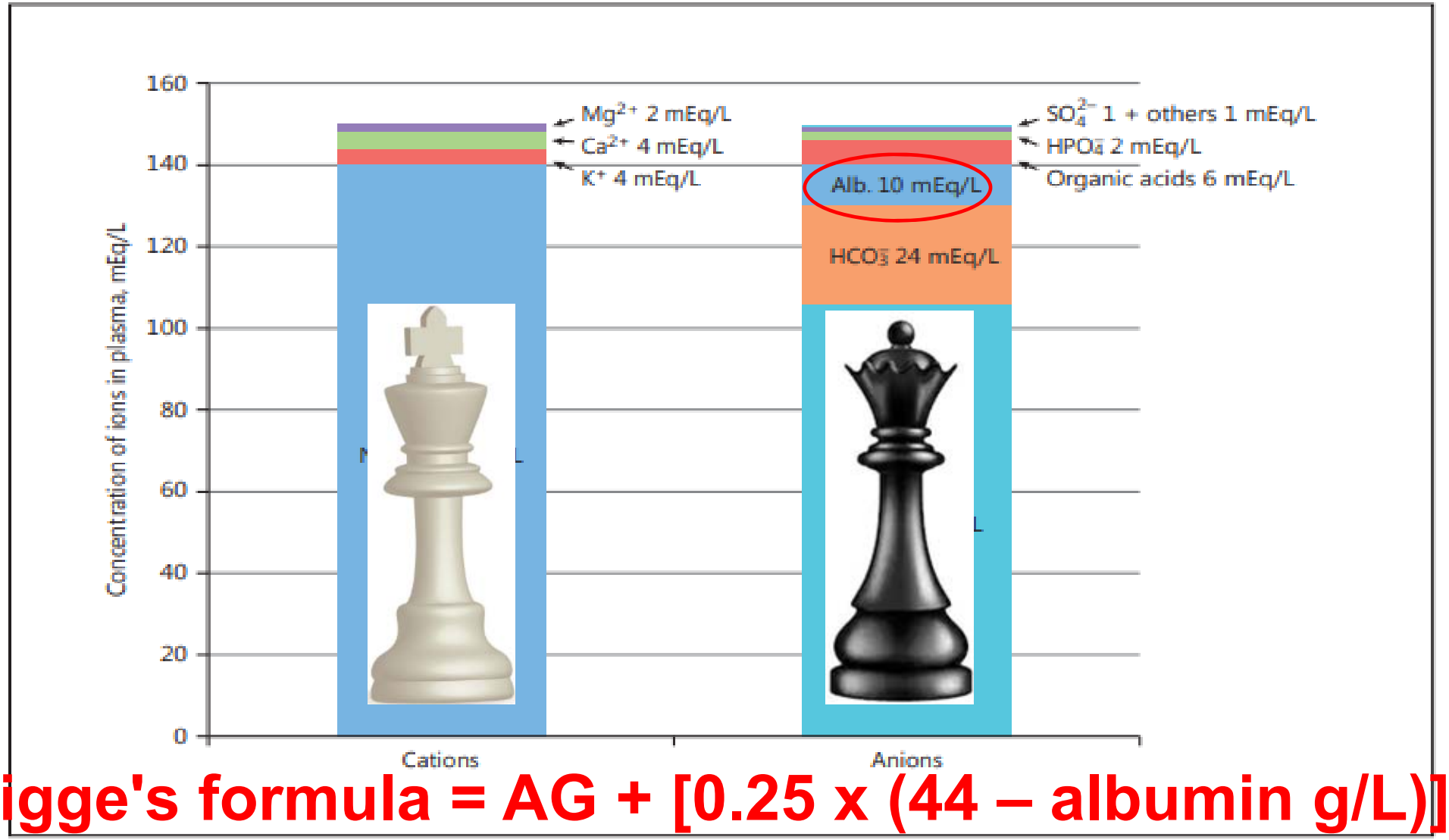
Χρήστος Νίχλος
Ειδικευόμενος Ιατρός
Ε' Παθολογική Κλινική, ΓΝΑ «ο Ευαγγελισμός»
Επιστημονικά και διοικητικά υπεύθυνος: Ιωάννης Κουτσουβέλης

Με μια ματιά…

- Αρχές
- Αίτια
- Διαγνωστική προσέγγιση
- Επιπτώσεις
- Αντιμετώπιση;


Cl
Chloride

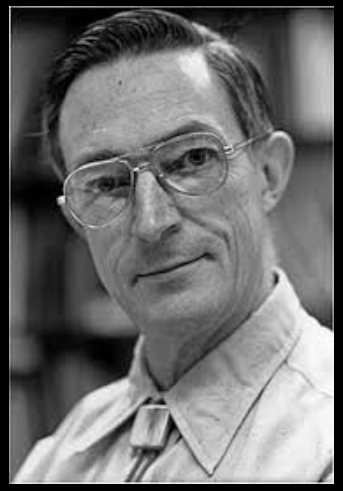

$Na^+ + K^+ + Ca^{2+} + Mg^{2+} + Protein^+ = Cl^- + HCO_3^- + Protein^- + HPO_4^{-2}/HPO_4^- + SO_4^{-2} + OA^-$
$Na^+ + K^+ + UC = Cl^- + HCO_3^- + UA$
$Na^+ + K^+ - (Cl^- + HCO_3^-) = UA - UC = \text{anion gap}$
$Na^+ - (Cl^- + HCO_3^-)$

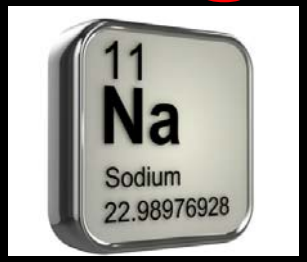


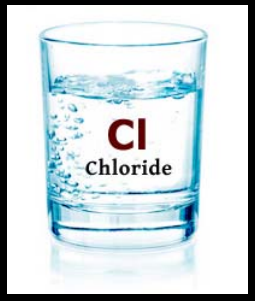
Figge's formula = AG + [0.25 x (44 – albumin g/L)]

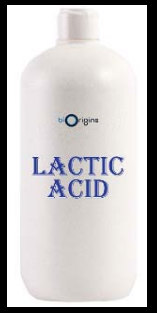
Fig. 1. Gamblegram: balance between anions and cations in plasma.

Strong Ion Difference (SID)

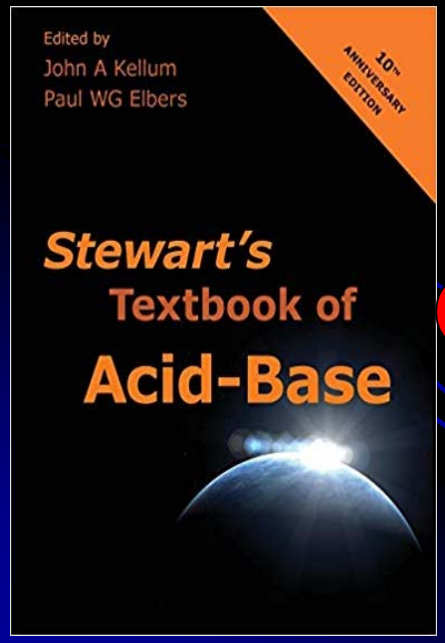




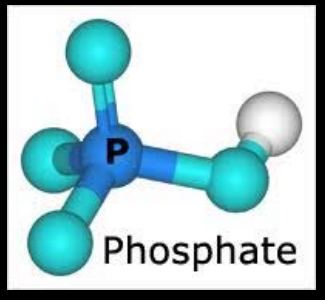




Partial pressure of carbon dioxide

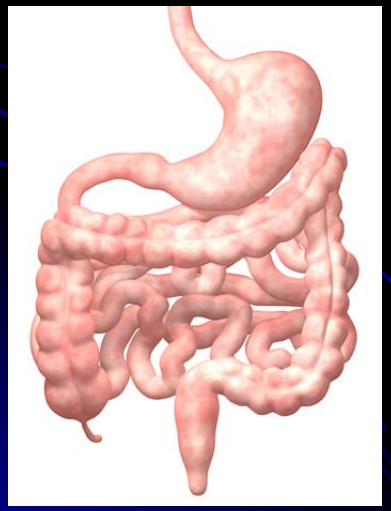


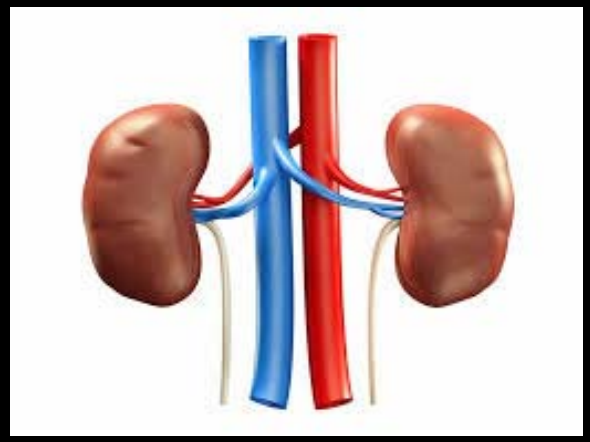
Concentrations of non-volatile weak acids (Atot)



- Non-Anion Gap-Metabolic Acidosis
- Normal Anion Gap Metabolic Acidosis
- Dilutional Metabolic Acidosis
- Hyperchloremic Metabolic Acidosis

$\underline{AG} = Na^+ - (Cl^- + HCO_3^-)$





Αίτια

CAGE
Chloride
Acetazolamide/Addison's
GI Loss
Extras-RTA, ingestion of oral acidifying salts, recovery phase of DKA

ABCD
Addison's
Bicarb loss (GI or renal, incl RTA)
Chloride
Drugs (e.g. acetazolamide, acids)

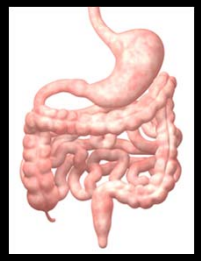
HARDUP
Hyperchloraemia
Acetazolamide, Addison's disease
Renal tubular acidosis
Diarrhoea, ileostomies, fistulae
Ureteroenterostomies
Pancreatoenterostomies

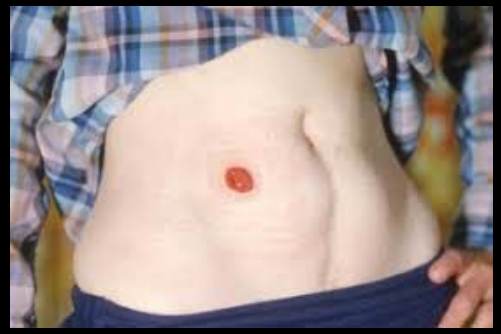
USEDCRAP
Ureteroenterostomies
Small bowel fistula
Excess Chloride
Diarrhoea
Carbonic anhydrase inhibitors
Renal tubular acidosis
Addisson's disease
Pancreatoenterostomies

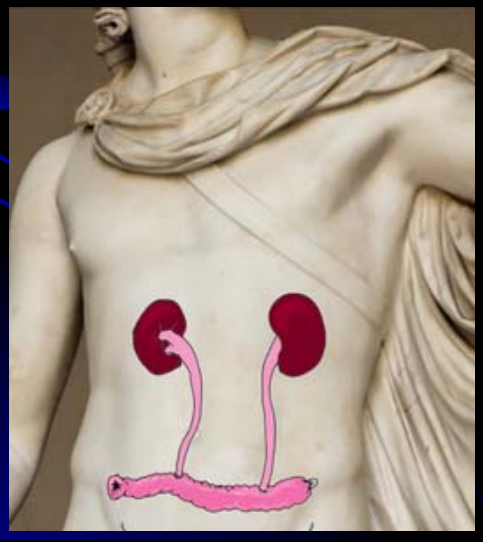
	Serum bicarbonate, mEq/L	Plasma K^+	Ca^{2+} excretion	Urine AG mEq/L	Urine osmolol gap,mosm/kg in metabolic acidosis	Urinary NH_4^+, mEq/day	Minimal urine pH	Ability to acidify urine in response to acidemia	Urine-blood pCO_2, mm Hg	Comment
Health	Normal	Normal	Normal	+20 to +90	10–100	30–40	4.5–6	Yes	>30	
Severe diarrhea	<24	Low	Normal	−20 to −50	>200	High	>5.5	Yes		
Toluene/hippurate	<24	Low	Normal	Positive	>200	High		Yes		
Defective CA II activity/proximal RTA (type 2)	12–20	Low/ normal	Normal	Negative −20 to −50	>150	Normal	<5.5	Yes		Urine pH >6.5 during early phase with bicarbonaturia
Fanconi syndrome/ proximal RTA (type 2)	12–18	Low	↑	Negative −20 to −50	>150	Normal	<5.5	Yes		Hypophosphatemia/ phosphaturia, hypouricemia/ hyperuricosuria renal glucosuria (glucosuria with a normal serum glucose concentration), aminoaciduria
Hypokalemic distal RTA (type 1)	10–20	Low	↑	Positive	<150 (usually <50–100)	Low	>5.5 (often >6.5)	No	<30	
Back diffusion	8–15	Low	?	Positive	?	Low		No		
Hyperkalemic distal RTA (voltage-dependent RTA)	8–15	High	Normal or ↑	Positive	<150 (usually <50–100)	Low	>5.5 (often >6.5)	No		
RTA type 3	Low	Low	↑			Low	>5.5	No		
RTA type 4	16–22	High	Normal	Positive	<150 (usually <50–100)	Low	<5.5	Yes		Often increased creatinine

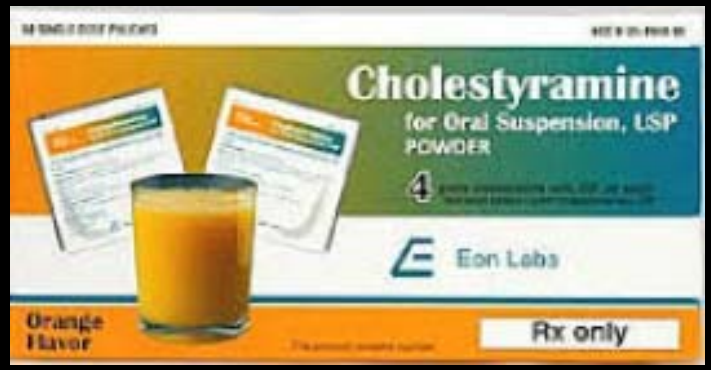
CA, carbonic anhydrase; RTA, renal tubular acidosis; urine-blood pCO_2, urine-blood pCO_2 after bicarbonate loading.

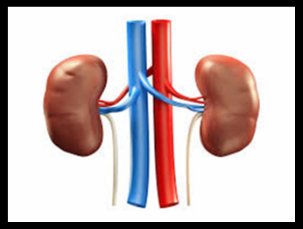
Αίτια



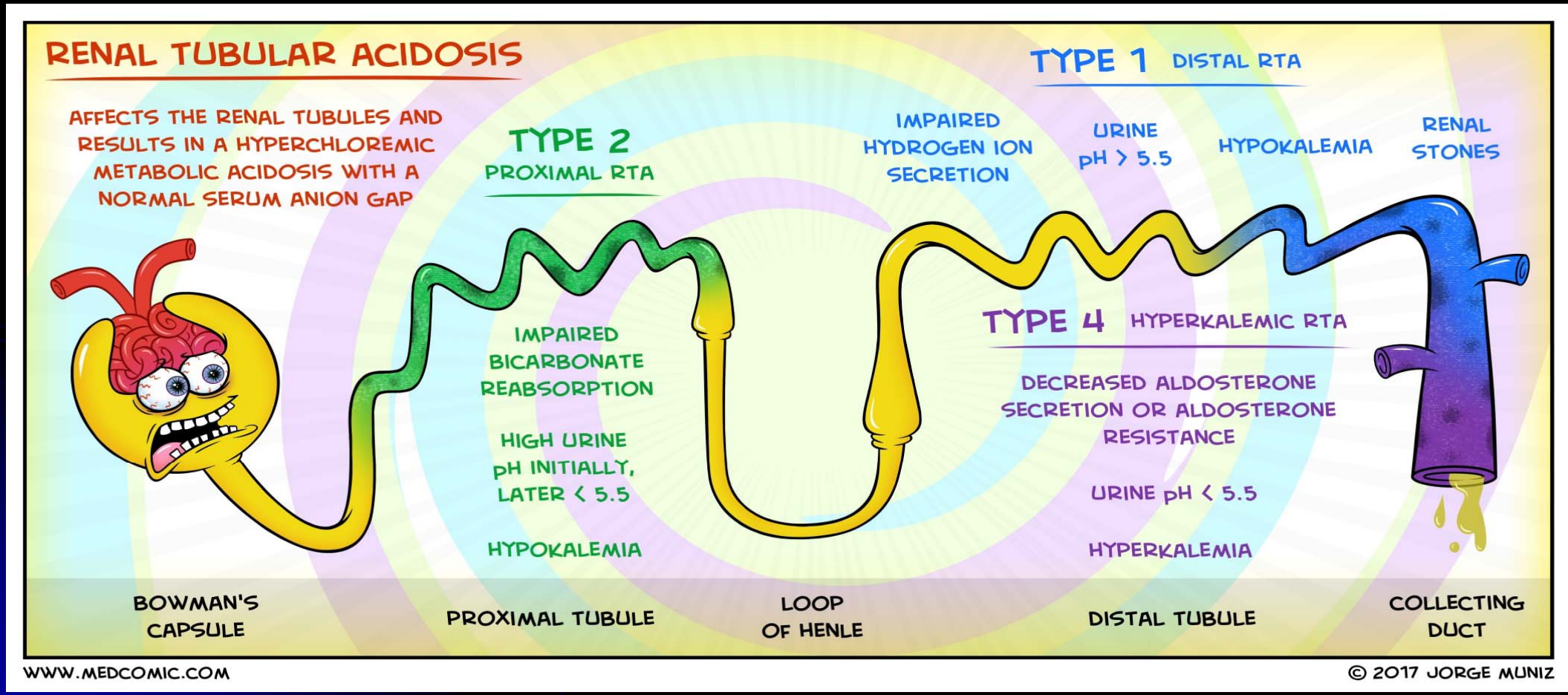





Cholestyramine
for Oral Suspension, USP
POWDER
4
Eon Labs
Orange
Flavor
Rx only



Αίτια


RENAL TUBULAR ACIDOSIS
AFFECTS THE RENAL TUBULES AND RESULTS IN A HYPERCHLOREMIC METABOLIC ACIDOSIS WITH A NORMAL SERUM ANION GAP
TYPE 2
PROXIMAL RTA
IMPAIRED BICARBONATE REABSORPTION
HIGH URINE pH INITIALLY, LATER < 5.5
HYPOKALEMIA
TYPE 1
DISTAL RTA
IMPAIRED HYDROGEN ION SECRETION
URINE pH > 5.5
HYPOKALEMIA
RENAL STONES
TYPE 4
HYPERKALEMIC RTA
DECREASED ALDOSTERONE SECRETION OR ALDOSTERONE RESISTANCE
URINE pH < 5.5
HYPERKALEMIA
BOWMAN'S CAPSULE
PROXIMAL TUBULE
LOOP OF HENLE
DISTAL TUBULE
COLLECTING DUCT
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	Τύπου I (άπω)	Τύπου II (εγγύς)	Τύπου IV
Αίτιο	CA-II μετάλλαξη Cl^-/HCO_3^- ανταλλαγέας μετάλλαξη H^+-ATPάση μετάλλαξη (& αυτοάνοση) Ελλιπής κλίση $[H^+]$ (αμφοτερικίνη)	CA-II μετάλλαξη Na^+-HCO_3^- συμμεταφορέας μετάλλαξη CIC-5 διαύλων Cl^- μετάλλαξη	Υποαλδοστερονισμός Αντίσταση στην ALD Υψηλές δόσεις αμιλορίδης ή τριμεθοπρίμης
Βασική βλάβη	Μειωμένη δυνατότητα οξινοποίησης των ούρων στο άπω σωληνάριο	Μειωμένη επαναρρόφηση HCO_3^- στο εγγύς σωληνάριο	Ανεπάρκεια ALD ή αντίσταση στη δράση της
pH ούρων	>5,3	>5,3 αν υπερβαίνει την ουδό επαναρρόφησης αν όχι <5,3	Συνήθως <5,3
$[HCO_3^-]$	Μπορεί να είναι <10 mEq/L	Συνήθως από 14 έως 20 mEq/L	Συνήθως >15 mEq/L
Κλασματική απέκκριση HCO_3^- με $[HCO_3^-]$ κφ	Ενήλικες: <3% Παιδιά: 5-10%	>15-20 %	<3%
Διάγνωση	Απόκριση στη χορήγηση $NaHCO_3$ ή NH_4Cl	Απόκριση στη χορήγηση $NaHCO_3$	Μειωμένη ALD πλάσματος
$[K^+]$ ορού	Συνήθως μειωμένο ή φυσιολογικό	Φυσιολογικό ή μειωμένο	Αυξημένο
Δόσεις HCO_3^- για την διόρθωση της $[HCO_3^-]$	Ενήλικες: 1-2 mEq/KgΣΒ/24ωρο Παιδιά: 4-14 mEq/KgΒΣ/24ωρο	10-15 mEq/KgΣΒ/24ωρο	1-2mEq/KgΣΒ/24ωρο (ίσως δεν χρειάζονται αν διορθωθεί η υπερκαλιαιμία)
Άλλες επιπλοκές	Νεφρολιθίαση	Ραχίτιδα ή οστεομαλακία	Καμία

Causes of proximal RTA		Causes of distal RTA		Causes of RTA type 4
isolated defect	generalized defect	with hypokalemia	with hyperkalemia	
Autosomal dominant Proximal RTA from unknown gene mutation	*Primary (genetic) inborn errors of metabolism* (Cystinosis, Wilson disease, galactosemia, hereditary fructose intolerance, methylmalonic acidemia, glycogen storage diseases)	*Calcium-induced tubular damage* Idiopathic hypercalciuria Primary hyperparathyroidism Hypervitaminosis D Medullary sponge kidney		
Autosomal recessive Sodium bicarbonate symporter (NBC1) protein mutation in the SLC4A4 gene	*Dysproteinemic states* (Myeloma, monoclonal gammopathy)	*Autoimmune diseases* Sjögren syndrome Rheumatoid arthritis SLE Polyarteritis nodosa Thyroiditis Primary biliary cirrhosis Chronic active hepatitis Cryoglobulinemia	Decreased effective intravascular volume of any cause Sickle cell disease Urinary tract obstruction SLE Renal transplant rejection Amyloidosis	*Aldosterone deficiency* Addison disease 21-Hydroxylase deficiency *Hyporeninemia* Diabetic nephropathy Tubulointerstitial disease HIV IgM monoclonal gammopathy
Inherited CA II deficiency caused by mutations in the CA2 gene – associated with mental retardation, cerebral calcifications and osteopetrosis (Sly syndrome)	Honeybee stings	*Idiopathic causes* Marfan syndrome Wilson disease Ehlers-Danlos syndrome		*Aldosterone resistance* Obstructive uropathy Sickle cell nephropathy Amyloidosis Diabetic nephropathy Lupus nephritis Pseudohypoaldosteronism
	Secondary hyperparathyroidism with chronic hypocalcemia Vitamin D deficiency			
	Tubulointerstitial diseases (Sjögren syndrome, medullary cystic disease, renal transplantation)			
	Nephrotic syndrome			
	Amyloidosis			
	Paroxysmal nocturnal hemoglobinuria			
	Toxins (lead, mercury, copper, cadmium, glue sniffing)			

Drugs as causes of proximal RTA		Drugs as causes of distal RTA	Drugs as causes of RTA type 4
isolated defect	generalized defect	with hypokalemia	
CA inhibitors	Ifosfamide, aminoglycosides, expired tetracycline, streptozocin, azacitidine (antimetabolites), mercaptopurine, valproic acid, ranitidine, lead, cadmium, mercury, antiretroviral drugs, propylene glycol-containing drugs	Amphotericin B, lithium carbonate, methicillin (meticillin), foscarnet, ifosfamide, toluene	*Reduced NH_4^+ production* (hypoaldosteronism) K^+-sparing diuretics (spironolactone, eplerenone, amiloride, triamterene), cotrimoxazole, ACEI, angiotensin II receptor type 1 antagonists, renin inhibitors, NSAIDs, ciclosporin, tacrolimus, heparin

ACEI, angiotensin-converting enzyme inhibitors; CA, carbonic anhydrase; SLE, systemic lupus erythematosus.

Αίτια

Διαγνωστική προσέγγιση

Urinary Ammonium/Urine AG

([Na +] + [K +] – [Cl –])

+20 to +90 mmol/L: ΦΤ, **RTA Typ1 + 4**

–30 to –50 mEq/L: **GI, RTA Typ2**

Fractional HCO3 excretion

0.5–1.0 mEq/kg/h

$$\text{Fractional } HCO_3 \text{ excretion} = \frac{(\text{urine } HCO_3^-) \times (\text{plasma creatinine})}{(\text{plasma } HCO_3^-) \times (\text{urine creatinine})} \times 100.$$

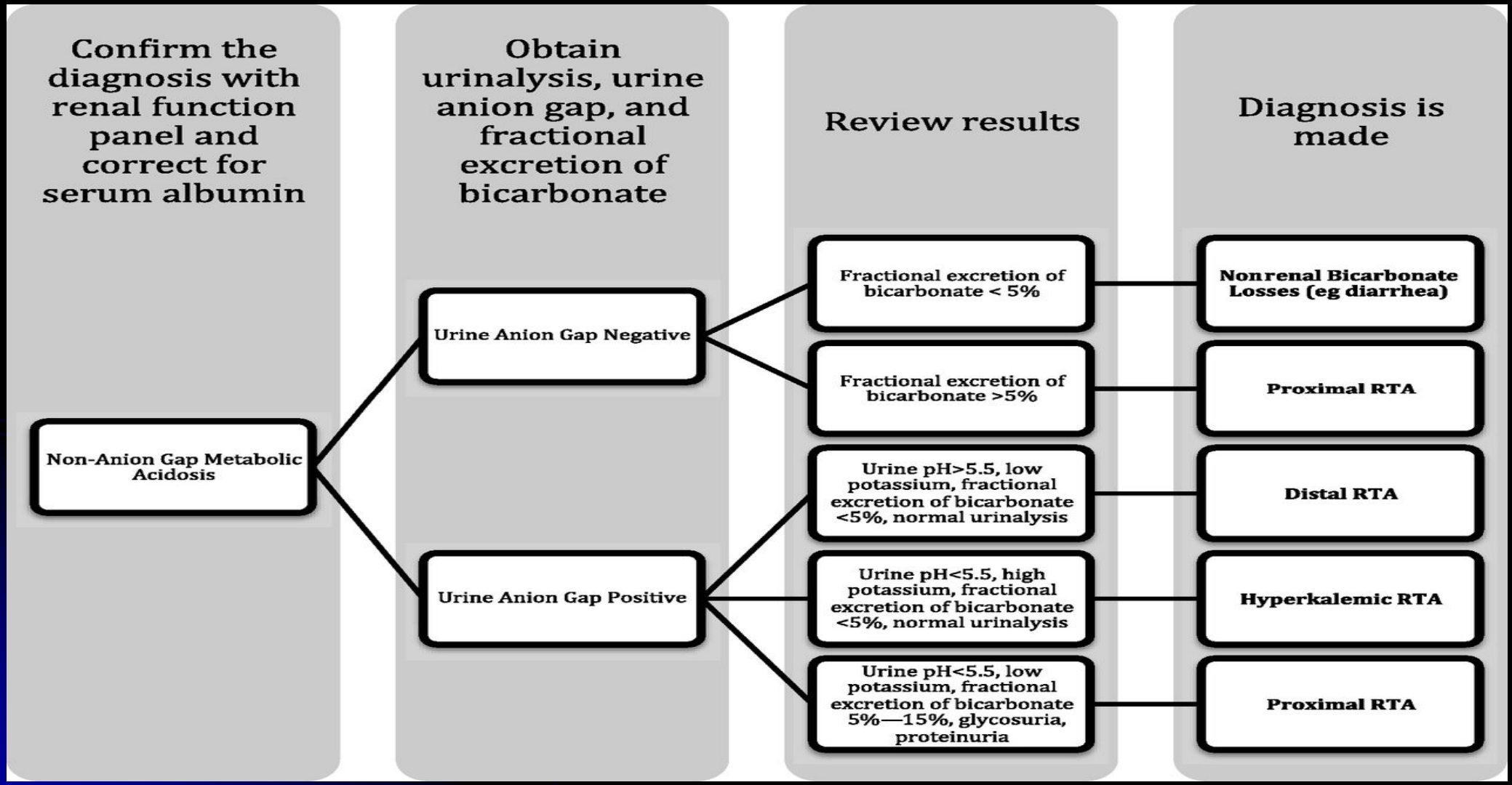
Urine pH >7.5 or fractional excretion of HCO3 >15%: **RTA Typ2**
Urine pH unchanged: **RTA Typ 1**
Fractional excretion of HCO3 <5%**:** **NO RTA Typ2**

Urinary Osmolal Gap

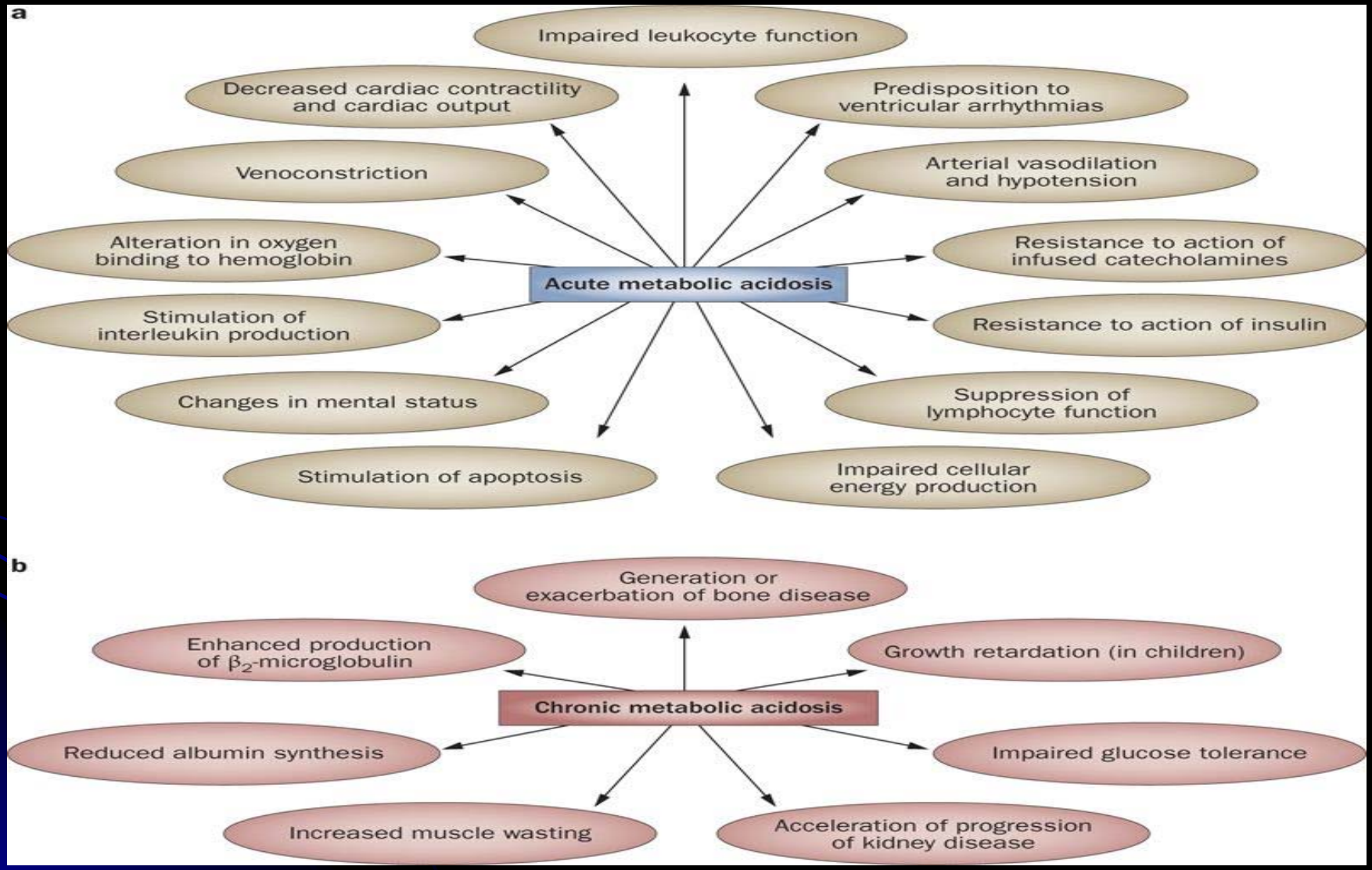
Urine-Blood pCO2 during NaHCO3 Loading

Furosemide and Fludrocortisone

Διαγνωστική προσέγγιση



Επιπτώσεις


a
Impaired leukocyte function
Decreased cardiac contractility and cardiac output
Predisposition to ventricular arrhythmias
Venoconstriction
Arterial vasodilation and hypotension
Alteration in oxygen binding to hemoglobin
Resistance to action of infused catecholamines
Acute metabolic acidosis
Stimulation of interleukin production
Resistance to action of insulin
Changes in mental status
Suppression of lymphocyte function
Stimulation of apoptosis
Impaired cellular energy production
b
Generation or exacerbation of bone disease
Enhanced production of β_2-microglobulin
Growth retardation (in children)
Chronic metabolic acidosis
Reduced albumin synthesis
Impaired glucose tolerance
Increased muscle wasting
Acceleration of progression of kidney disease

Γαστρεντερικό

Anesth Analg. 2001 Oct;93(4):811-6.

The effects of balanced versus saline-based hetastarch and crystalloid solutions on acid-base and electrolyte status and gastric mucosal perfusion in elderly surgical patients.

Wilkes NJ[1], Woolf R, Mutch M, Mallett SV, Peachey T, Stephens R, Mythen MG.

Anesth Analg. 1999 May;88(5):999-1003.

The effect of intravenous lactated Ringer's solution versus 0.9% sodium chloride solution on serum osmolality in human volunteers.

Williams EL[1], Hildebrand KL, McCormick SA, Bedel MJ.

Anesth Analg. 2000 Jan;90(1):74-9.

Metabolic acidosis and respiratory acidosis impair gastro-pyloric motility in anesthetized pigs.

Tournadre JP[1], Allaouchiche B, Malbert CH, Chassard D.

Διαβητική Κετοξέωση

Comparison of Plasma-Lyte A and Sodium Chloride 0.9% for Fluid Resuscitation of Patients With Diabetic Ketoacidosis.

Oliver WD[1], Willis GC[1,2], Hines MC[1], Hayes BD[3].

⊕ Author information

Abstract

Purpose: The aim of this study was to compare Plasma-Lyte A (PL) and sodium chloride 0.9% (NS) in regard to time to resolution of diabetic ketoacidosis (DKA) when one fluid was used predominantly over the other for resuscitation. **Methods:** We performed a retrospective analysis of the records of patients treated for DKA at a large, academic medical center between July 1, 2013, and July 1, 2015. Patients were placed into the PL or NS group based on the predominant fluid they received during fluid resuscitation. Serum biochemistry variables were categorized as follows: initial, 2 to 4 hours, 4 to 6 hours, 6 to 12 hours, and 12 to 24 hours. The primary outcome was mean time to resolution of DKA. **Results:** Eighty-four patients were included in the study. The primary outcome of mean time to resolution of DKA was similar between the PL (19.74 hours) and NS (18.05 hours) groups (P = .5080). Patients treated with PL had a significantly greater rise in pH within the 4- to 6-hour and 6- to 12-hour periods. The chloride level was significantly higher and the anion gap was significantly lower for the NS group in the 6- to 12-hour period. **Conclusion:** These data suggest that the use of PL for fluid resuscitation in DKA may confer certain advantages over NS.

J Crit Care. 2012 Apr;27(2):138-45. doi: 10.1016/j.jcrc.2012.01.007.

Plasma-Lyte 148 vs 0.9% saline for fluid resuscitation in diabetic ketoacidosis.

Chua HR[1], Venkatesh B, Stachowski E, Schneider AG, Perkins K, Ladanyi S, Kruger P, Bellomo R.

⊕ Author information

Abstract

PURPOSE: The purpose of the study was to determine the effects of Plasma-Lyte 148 (PL) vs 0.9% saline (NS) fluid resuscitation in diabetic ketoacidosis (DKA).

METHODS: A multicenter retrospective analysis of adults admitted for DKA to the intensive care unit, who received almost exclusively PL or NS infusion up until 12 hours, was performed.

RESULTS: Nine patients with PL and 14 patients with NS were studied. Median serum bicarbonate correction was higher in the PL vs NS groups at 4 to 6 hours (8.4 vs 1.7 mEq/L) and 6 to 12 hours (12.8 vs 6.2 mEq/L) from baseline ($P < .05$). Median standard base excess improved by 10.5 vs 4.2 mEq/L at 4 to 6 hours and by 16.0 vs 9.1 mEq/L at 6 to 12 hours in the PL and NS groups, respectively ($P < .05$). Chloride levels increased significantly in the NS vs PL groups over 24 hours. Potassium levels were lower at 6 to 12 hours in the PL group. Mean arterial blood pressure was higher at 2 to 4 hours in the PL group, whereas cumulative urine output was lower at 4 to 6 hours in the NS group. There were no differences in glycemic control or duration of intensive care unit stay.

CONCLUSION: Patients with DKA resuscitated with PL instead of NS had faster initial resolution of metabolic acidosis and less hyperchloremia, with a transiently improved blood pressure profile and urine output.

Φλεγμονώδης αντίδραση

Fluid resuscitation with lactated Ringer's solution vs normal saline in acute pancreatitis: A triple-blind, randomized, controlled trial.

de-Madaria E, et al. United European Gastroenterol J. 2018.
Show full citation

Abstract

Background: Little is known regarding the optimal type of fluid resuscitation in acute pancreatitis (AP).

Objective: The objective of this article was to compare the effect of lactated Ringer's solution (LR) vs normal saline (NS) in the inflammatory response in AP.

Methods: We conducted a triple-blind, randomized, controlled trial. Patients ≥ 18 admitted with AP were eligible. Patients were randomized to receive LR or NS. Primary outcome variables were number of systemic inflammatory response syndrome (SIRS) criteria at 24 hours, 48 hours and 72 hours and blood C-reactiv protein (CRP) levels at 48 hours and 72 hours. In vitro complementary experimer were performed to further explore the interaction between pH, lactate and inflammation.

Results: Nineteen patients receiving LR and 21 receiving NS were analyzed. The median (p25-p75) number of SIRS criteria at 48 hours were 1 (1-2) for NS vs 1 (0-1) for LR, p = 0.060. CRP levels (mg/l) were as follows: at 48 hours NS 166 (78-281) vs LR 28 (3-124), p = 0.037; at 72 hours NS 217 (59-323) vs LR 25 (3-169), p = 0.043. In vitro, LR inhibited the induction of inflammatory phenotype c macrophages and NF-κB activation. This effect was not observed when using Ringer's solution without lactate, suggesting a direct anti-inflammatory effect of lactate.

Conclusions: Lactated Ringer's is associated with an anti-inflammatory effect in patients with acute pancreatitis.

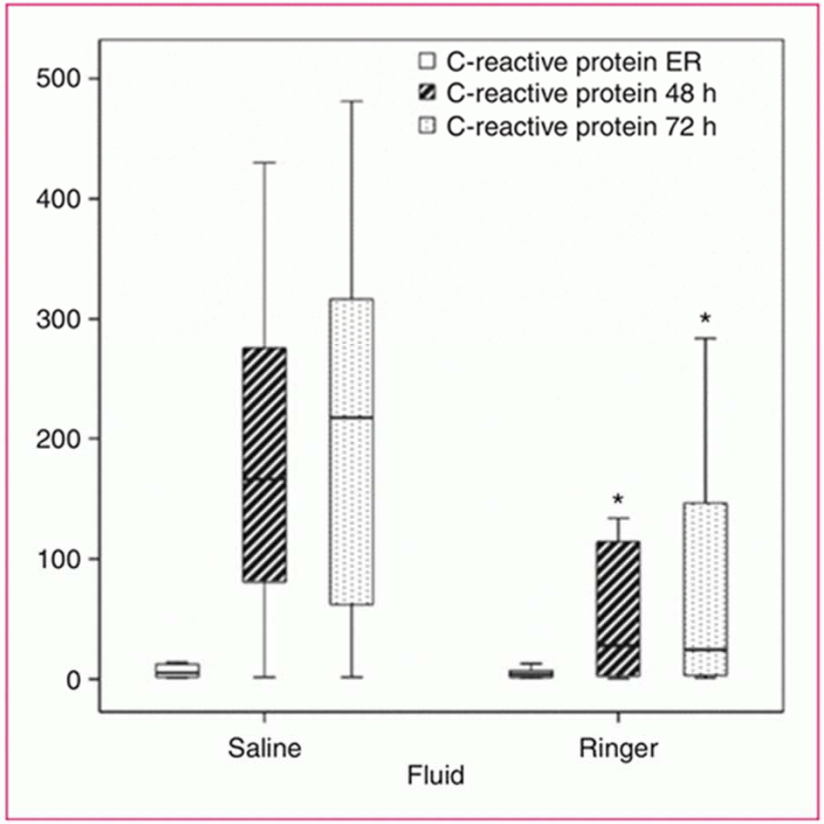


Figure 2. C-reactive protein (CRP, mg/l) at randomization (emergency room, ER), 48 hours and 72 hours. *$p < 0.05$ between patients receiving normal saline (saline) or lactated Ringer's solution (Ringer).

de-Madaria E et al. 2018 PMID 29435315

Αιμοδυναμική αστάθεια

Br J Anaesth. 2018 Feb;120(2):274-283. doi: 10.1016/j.bja.2017.11.088. Epub 2017 Dec 2.

Normal saline versus a balanced crystalloid for goal-directed perioperative fluid therapy in major abdominal surgery: a double-blind randomised controlled study.

Pfortmueller CA[1], Funk GC[2], Reiterer C[3], Schrott A[4], Zotti O[3], Kabon B[3], Fleischmann E[3], Lindner G[5].

Anesth Analg. 2015 Jan;120(1):123-9. doi: 10.1213/ANE.0000000000000419.

An acetate-buffered balanced crystalloid versus 0.9% saline in patients with end-stage renal disease undergoing cadaveric renal transplantation: a prospective randomized controlled trial.

Potura E[1], Lindner G, Biesenbach P, Funk GC, Reiterer C, Kabon B, Schwarz C, Druml W, Fleischmann E.

Chest. 2004 Jan;125(1):243-8.

Effects of hyperchloremic acidosis on arterial pressure and circulating inflammatory molecules in experimental sepsis.

Kellum JA[1], Song M, Venkataraman R.

⊕ Author information

Σήψη

Ann Intensive Care. 2018; 8: 43.
Published online 2018 Mar 27. doi: 10.1186/s13613-018-0388-4

Hyperchloraemia in sepsis

Christos Filis,[1] Ioannis Vasileiadis,[2] anc

of the human organism. Hyperchloraemia, whether a result of the sepsis process or a consequence of its treatment with supraphysiologic chloride fluids, appears to have a negative impact on the clinical outcom of septic patients. The detrimental effect of hyperchloraemic acidosis on the inflammatory response, on

Crit Care Med. 2015 Sep;43(9):1938-44. doi: 10.1097/CCM.0000000000001161.

Association of Hyperchloremia With Hospital Mortality in Critically Ill Septic Patients.

Neyra JA[1], Canepa-Escaro F, Li X, Manllo J, Adams-Huet B, Yee J, Yessayan L; Acute Kidney Injury in Critical Illness Study Group.

CONCLUSIONS: In critically ill septic patients manifesting hyperchloremia (Cl ≥ 110 mEq/L) on ICU admission, higher Cl levels and within-subject worsening hyperchloremia at 72 hours of ICU stay were associated with all-cause hospital mortality. These associations were independent of base deficit, cumulative fluid balance, acute kidney injury, and other critical illness parameters.

J Crit Care. 2011 Apr;26(2):175-9. doi: 10.1016/j.jcrc.2010.04.013. Epub 2010 Jul 8.

Is hyperchloremia associated with mortality in critically ill patients? A prospective cohort study.

Boniatti MM[1], Cardoso PR, Castilho RK,

CONCLUSIONS: Hypoalbuminemia and hyperchloremia were associated with mortality. This result involving chloride is something new and should be tested in future studies.

Αιμόσταση-Αιμοποίηση

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Saline versus Plasma-Lyte A in initial resuscitation of trauma patients: a randomized trial.

Young JB[1], Utter GH, Schermer CR, Galante JM, Phan HH, Yang Y, Anderson BA, Scherer LA.

J Trauma. 2009 Jul;67(1):202-8; discussion 208-9. doi: 10.1097/TA.0b013e3181a602a7.

Coagulopathy by hypothermia and acidosis: mechanisms of thrombin generation and fibrinogen availability.

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Lactated Ringer's is superior to normal saline in the resuscitation of uncontrolled hemorrhagic shock.

Todd SR[1], Malinoski D, Muller PJ, Schreiber MA.

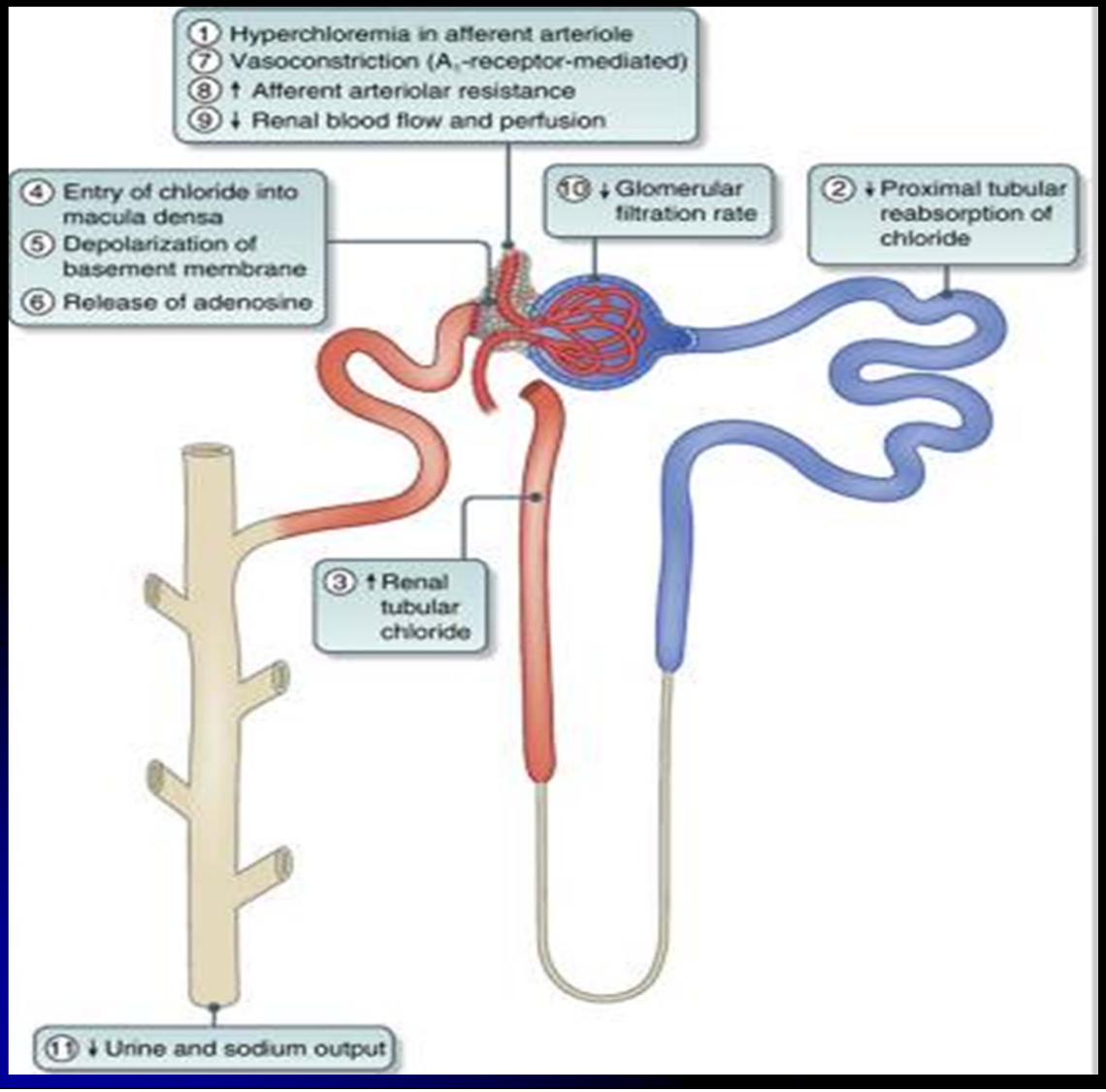
ELSEVIER

Journal of Critical Care
Volume 26, Issue 2, April 2011, Pages 175-179

Risks/Mortality/Outcomes

Is hyperchloremia associated with mortality in critically ill patients? A prospective cohort study

Νεφρική δυσλειτουργία



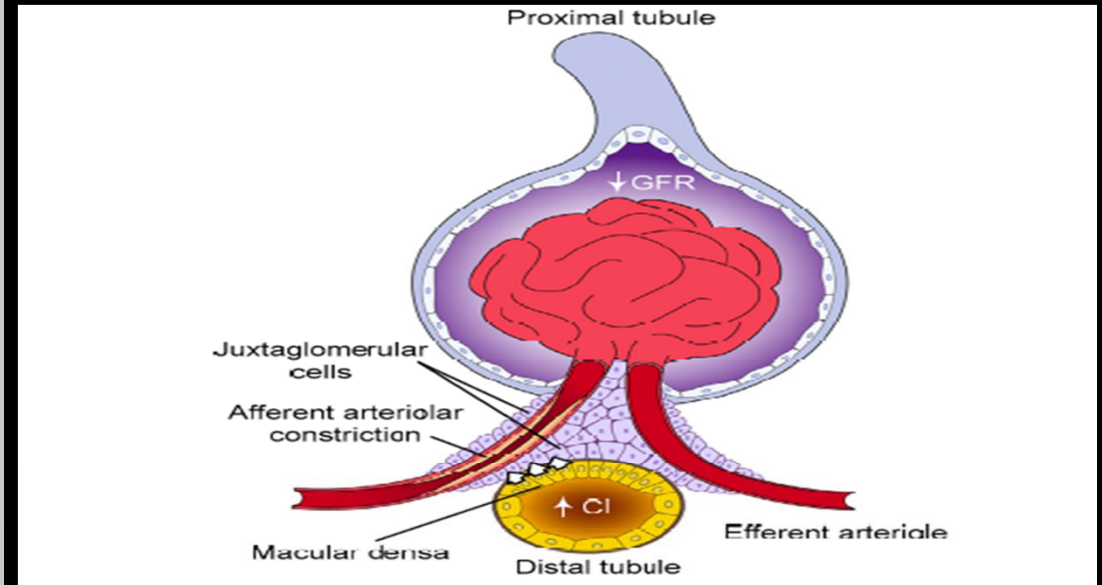


Fig. 2 A disproportionate increase in Cl and hyperchloremic acidosis caused by 0.9% saline infusion
The Cl filters into renal tubules. Through macular densa cells, the Cl signal is transmitted to afferent arterioles causing vasoconstriction, resulting in glomerular filtration rate (GFR) reduction and reduction in urine output. Regional ischemia due to saline-associated kidney volume expansion could further stimulate renin secretion from juxtaglomerular cells, leading to activation of the intra-renal renin angiotensin system (RAS). RAS activation would further enhance the proximal tubular Na absorption and afferent arteriolar vasoconstriction via tubuloglomerular feedback (TGF) activation

ORIGINAL ARTICLE

Balanced Crystalloids versus Saline in Noncritically Ill Adults

Wesley H. Self, M.D., M.P.H., Matthew W. Semler, M.D., Jonathan P. Wanderer, M.D., Li Wang, M.S., Daniel W. Byrne, M.S., Sean P. Collins, M.D., Corey M. Slovis, M.D., Christopher J. Lindsell, Ph.D., Jesse M. Ehrenfeld, M.D., M.P.H., Edward D. Siew, M.D., Andrew D. Shaw, M.B., Gordon R. Bernard, M.D., et al., for the SALT-ED Investigators*

SALT-ED

ORIGINAL ARTICLE

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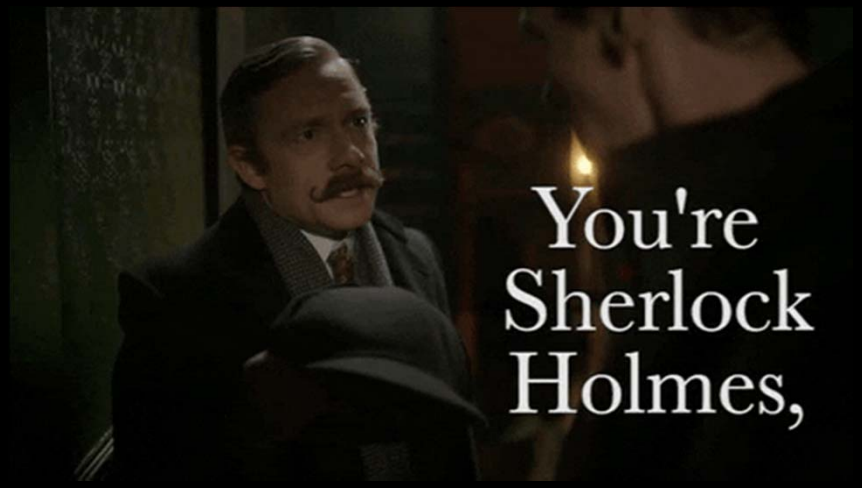
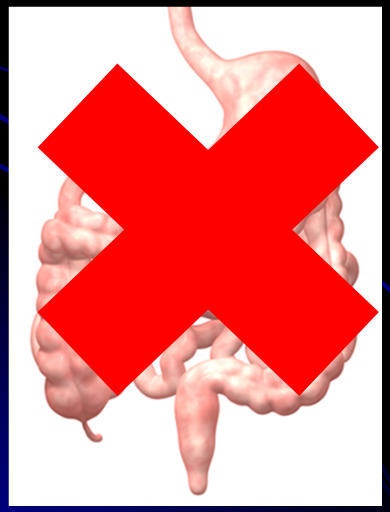
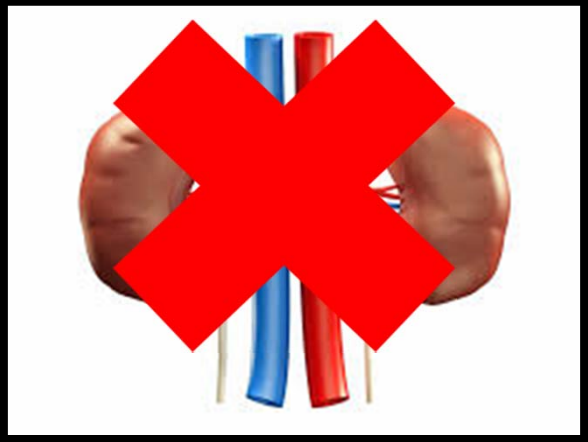
SMART

Αντιμετώπιση;

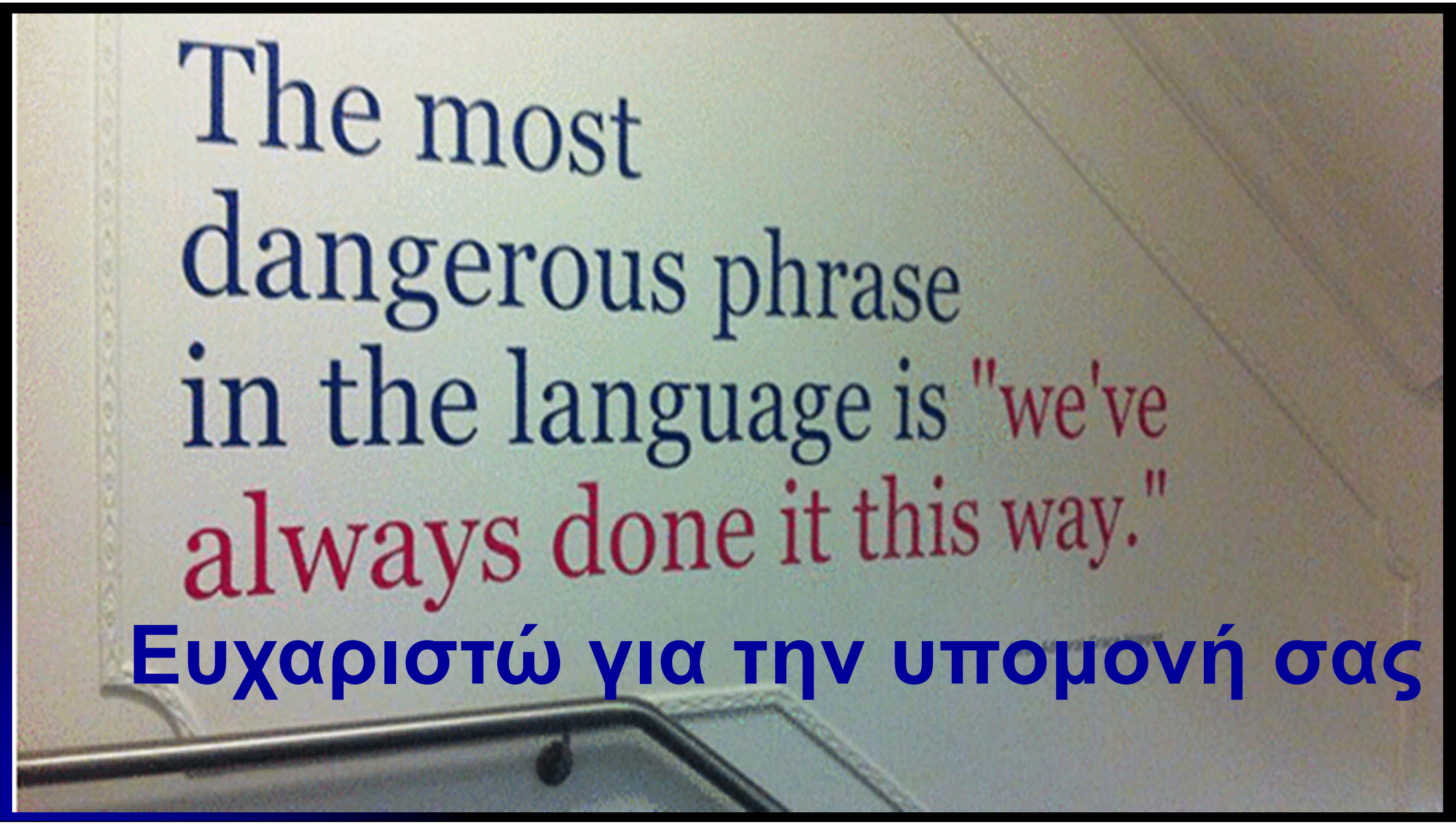






Take home messages…

- Η οξέωση με φυσιολογικό ΧΑ είναι πολύ συχνά υποδιαγνωσμένη
- Είναι γαστρεντερικής, νεφρικής ή ιατρογενούς αιτιολογίας
- Η ΔΔ της μπορεί να βοηθήσει στην εντόπιση του υποκείμενου αιτίου
- Επηρεάζει την φυσιολογία του οργανισμού ποικιλοτρόπως
- «Ισορροπημένα» διαλύματα vs NS?


The most
dangerous phrase
in the language is "we've
always done it this way."
Ευχαριστώ για την υπομονή σας