Hyponatremia okai és kezelési szempontjai

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Emberi Erőforrások Minisztériuma – Egészségügyért Felelős Államtitkárság

EGÉSZSÉGÜGYI SZAKMAI KOLLÉGIUM

Egészségügyi szakmai irányelv – A hyponatraemia diagnosztikájáról és kezeléséről

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Clinical practice guideline on diagnosis and treatment of hyponatraemia

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Egészségügyi szakmai irányelv – A hyponatraemia diagnosztikájáról és kezeléséről

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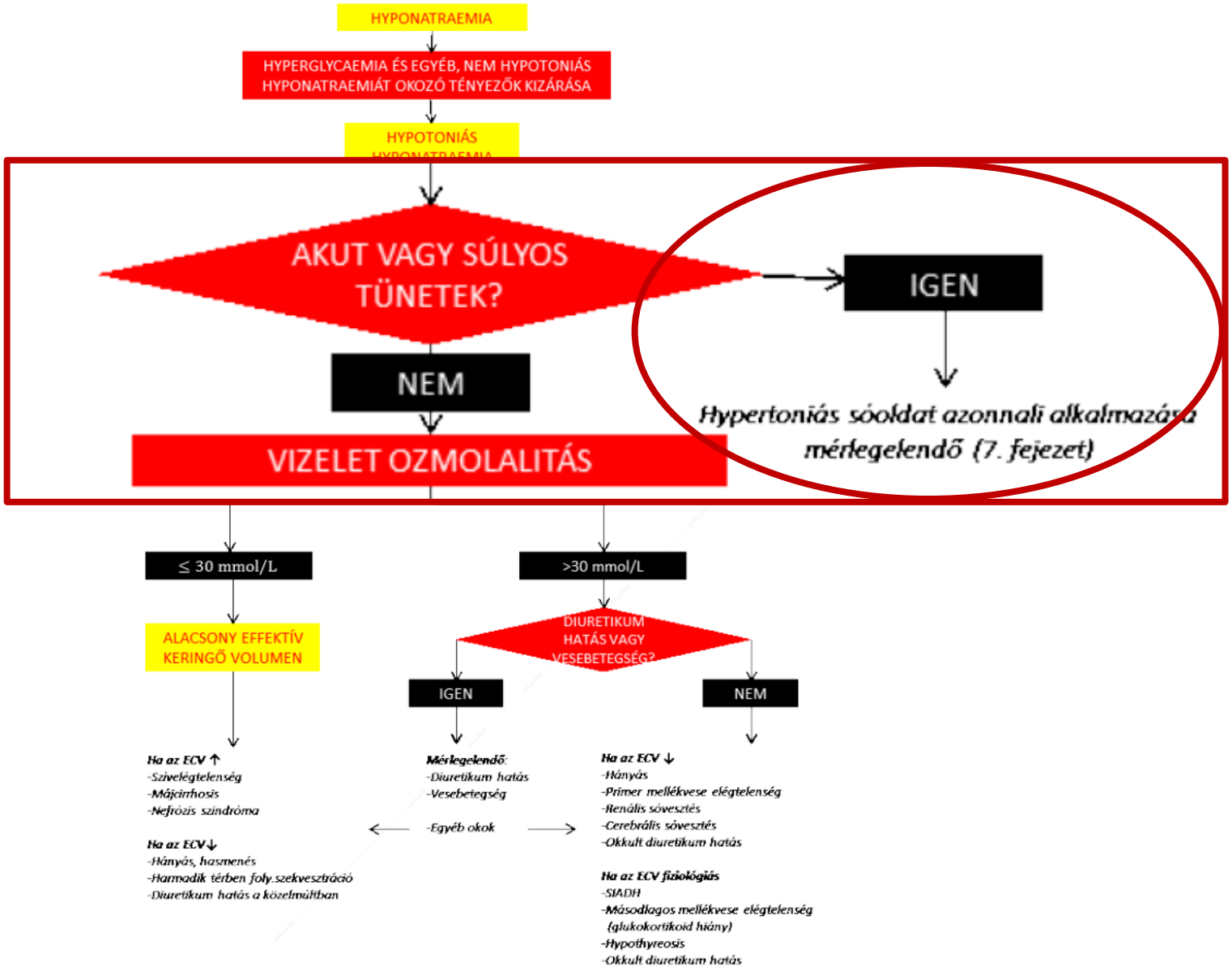
Súlyos	hányás keringési-légzési elégtelenség mély szomnolencia görcsök kóma (Glasgow Coma Scale (≤ 8)

Egészségügyi szakmai irányelv – A hyponatraemia diagnosztikájáról és kezeléséről



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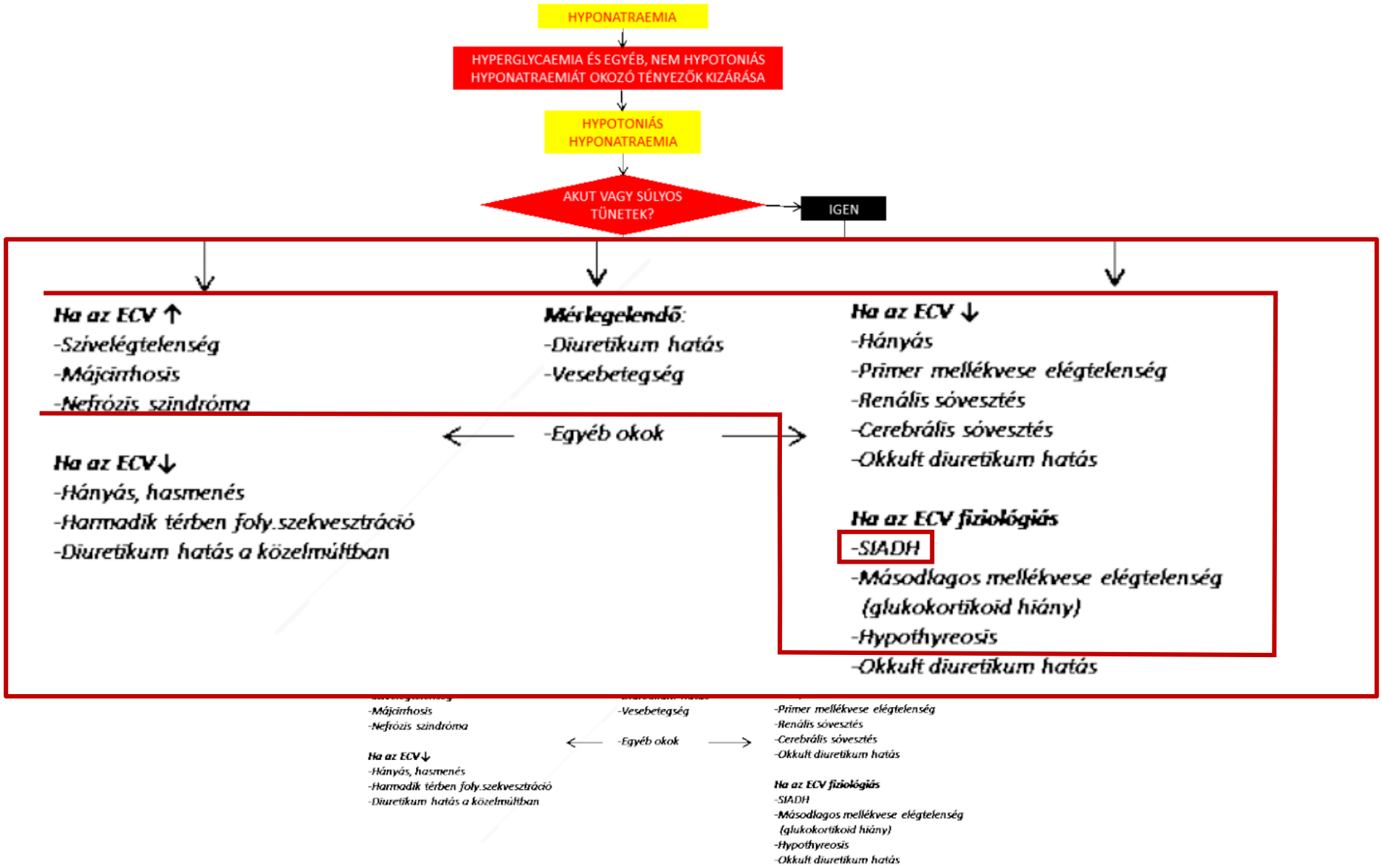

Causes of the syndrome of inappropriate antidiuresis.

Malignant diseases

- Carcinoma
  - Lung
  - Oropharynx
  - Gastrointestinal tract
    - Stomach
    - Duodenum
    - Pancreas
  - Genitourinary tract
    - Ureter
    - Bladder
    - Prostate
    - Endometrium
  - Endocrine thymoma
- Lymphomas
- Sarcomas
  - Ewing's sarcoma
- Olfactory neuroblastoma

Pulmonary disorders

- Infections
  - Bacterial pneumonia
  - Viral pneumonia
  - Pulmonary abscess
  - Tuberculosis
  - Aspergillosis
- Asthma
- Cystic fibrosis
- Respiratory failure associated with positive-pressure breeding

Disorders of the nervous system

- Infection
  - Encephalitis
  - Meningitis
  - Brain abscess
  - Rocky Mountain spotted fever
  - AIDS
  - Malaria
- Vascular and masses
  - Subdural hematoma
  - Subarachnoid haemorrhage
  - Stroke
  - Brain tumours
  - Head trauma
- Other
  - Hydrocephalus
  - Cavernous sinus thrombosis
  - Multiple sclerosis
  - Guillain–Barré syndrome
  - Shy–Drager syndrome
  - Delirium tremens
  - Acute intermittent porphyria

Drugs

- Vasopressin release or action stimulants
  - Antidepressants
    - SSRIs
    - Tricyclic
    - MAOI
    - Venlafaxine
  - Anticonvulsants
    - Carbamazepine
    - Oxcarbazepine
    - Sodium valproate
    - Lamotrigine
  - Antipsychotics
    - Phenothiazides
    - Butyrophenones
  - Anticancer drugs
    - Vinca alkaloids
    - Platinum compounds
    - Ifosfamide
    - Melphalan
    - Cyclophosphamide
    - Methotrexate
    - Pentostatin
  - Antidiabetic drugs
    - Chlorpropamide
    - Tolbutamine
  - Miscellaneous
    - Opiates
    - MDMA (XTC)
    - Levamisole
    - Interferon
    - NSAIDs
    - Clofibrate
    - Nicotine
    - Amiodarone
    - Proton pump inhibitors
    - MABs
- Vasopressin analogues
  - Desmopressin
  - Oxytocin
  - Terlipressin
  - Vasopressin

Other causes

- Hereditary
  - Gain-of-function mutation of the vasopressin V2 receptor
- Idiopathic
- Transient
  - Exercise-associated hyponatraemia
  - General anaesthesia
  - Nausea
  - Pain
  - Stress

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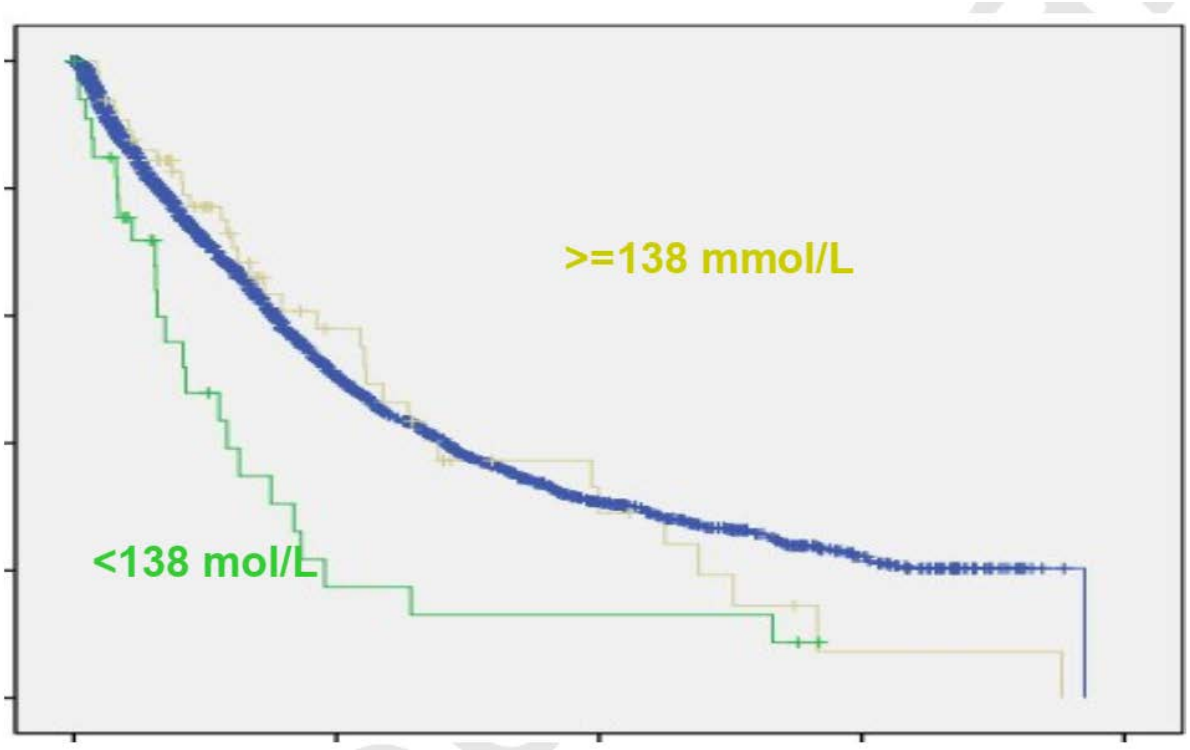
Hyponatraemia–SIADH in lung cancer Diagnostic and treatment algorithms



http://dx.doi.org/10.1016/j.critrevonc.2015.04.005

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  - Ureter
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  - Prostate
  - Endometrium
- Endocrine thymoma

Lymphomas
Sarcomas
- Ewing's sarcoma

Olfactory neuroblastoma

Pulmonary disorders

Infections
- Bacterial pneumonia
- Viral pneumonia
- Pulmonary abscess
- Tuberculosis
- Aspergillosis

Asthma
Cystic fibrosis
Respiratory failure associated with positive-pressure breeding

Pulmonary disorders

Infections
Bacterial pneumonia
Viral pneumonia
Pulmonary abscess
Tuberculosis
Aspergillosis
Asthma
Cystic fibrosis
Respiratory failure associated with positive-pressure breeding

Pozitív nyomású lélegeztetéslégzési elégtelenségben

n stimulants
- NSAIDs
- Clofibrate
- Nicotine
- Amiodarone
- Proton pump inhibitors
- MABs

Vasopressin analogues
- Desmopressin
- Oxytocin
- Terlipressin
- Vasopressin

Other causes

Hereditary
- Gain-of-function mutation of the vasopressin V2 receptor

Idiopathic
Transient
- Exercise-associated hyponatraemia
- General anaesthesia
- Nausea
- Pain
- Stress

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Pneumonia and the Syndrome of Inappropriate Antidiuretic Hormone Secretion: Don't Pour Water on the Fire

Water moves to maintain osmotic equilibrium between all body compartments. When plasma osmolality falls, as measured by a drop in the serum sodium (hyponatremia), water moves from plasma into the intracellular compartment. This results in cell swelling, which has potentially catastrophic consequences for the central nervous system where swelling is limited by the rigid calvarium. In order to maintain a normal total body water and plasma osmolality, water intake must be exactly balanced by water excretion. Water loss is regulated predominantly by the action of the posterior pituitary hormone, vasopressin, that increases water reabsorption in the renal collecting tubule. Under normal circumstances, excessive water intake produces hypoosmolality and leads to decreased synthesis and release of vasopressin. Subsequently, a water diuresis restores total body water and plasma osmolality to normal.

including acute bronchospasm, intrathoracic infections, neoplastic processes, acute and chronic respiratory failure, and mechanical ventilation have all been associated with hyponatremia (4). While the pathophysiology of the hyponatremia is not entirely clear, in virtually all studies where it has been measured, plasma vasopressin is elevated. This nonosmotic release of vasopressin then impairs renal water excretion and in the presence of excessive water intake leads to hyponatremia.

The mechanism(s) whereby pulmonary disorders lead to increased plasma vasopressin concentration is not entirely known. In the absence of an osmotic stimulus, increased plasma vasopressin levels are generally caused by either nonosmotic stimulation of central vasopressin release or ectopic vasopressin production (4). Alterations in vasopressin metabolism and clearance in the presence of pulmonary disease have not been

nonosmotic release of vasopressin associated with pulmonary disease.

Pulmonary infections are a well-documented cause of hypoosmolality and hyponatremia (4). Although hyponatremia has frequently been reported in association with legionella pneumonia, it is not unique to this microbe and occurs with a variety of other bacterial, viral, and fungal pathogens (4, 6). While several reports have documented that vasopressin levels are elevated in hyponatremic patients with pneumonia, the mechanism of this effect has not been explained. The study by Dreyfuss and associates (6) reported in this issue is clearly the most comprehensive study of the effect of pneumonia on vasopressin release and renal water excretion yet performed. In this study of nine normonatremic euvolemic patients with normal renal function, pneumonia was found to elevate plasma vasopressin levels and impair renal water excretion. The abnormal water excre-

AM REV RESPIR DIS 1988; 138:512-513

Causes of the syndrome of inappropriate antidiuresis.

Malignant diseases	Pulmonary disorders	Disorders of the nervous system	Drugs	Other causes
Carcinoma	Infections	Infection	Vasopressin release or action stimulants	Hereditary
Lung	Bacterial pneumonia	Encephalitis	Antidepressants	Gain-of-function mutation of the vasopressin V2 receptor
Oropharynx	Viral pneumonia	Meningitis	SSRIs	Idiopathic
Gastrointestinal tract	Pulmonary abscess	Brain abscess	Tricyclic	Transient
Stomach	Tuberculosis	Rocky Mountain spotted fever	MAOI	Exercise-associated hyponatraemia
Duodenum	Aspergillosis	AIDS	Venlafaxine	General anaesthesia
Pancreas	Asthma	Malaria	Anticonvulsants	Nausea
Genitourinary tract	Cystic fibrosis	Vascular and masses	Carbamazepine	Pain
Ureter	Respiratory failure associated with positive-pressure breeding	Subdural hematoma	Oxcarbazepine	Stress
Bladder		Subarachnoid haemorrhage	Sodium valproate	
Prostate		Stroke	Lamotrigine	
Endometrium		Brain tumours	Antipsychotics	
Endocrine thymoma		Head trauma	Phenothiazides	
Lymphomas		Other	Butyrophenones	
Sarcomas		Hydrocephalus	Anticancer drugs	
Ewing's sarcoma		Cavernous sinus thrombosis	Vinca alkaloids	
Olfactory neuroblastoma		Multiple sclerosis	Platinum compounds	
		Guillain–Barré syndrome	Ifosfamide	
		Shy–Drager syndrome	Melphalan	
		Delirium tremens	Cyclophosphamide	
		Acute intermittent porphyria	Methotrexate	
			Pentostatin	
			Antidiabetic drugs	
			Chlorpropamide	
			Tolbutamine	
			Miscellaneous	
			Opiates	
			MDMA (XTC)	
			Levamisole	
			Interferon	
			NSAIDs	
			Clofibrate	
			Nicotine	
			Amiodarone	
			Proton pump inhibitors	
			MABs	
			Vasopressin analogues	
			Desmopressin	
			Oxytocin	
			Terlipressin	
			Vasopressin	

Anticancer drugs
- Vinca alkaloids
- Platinum compounds
- Ifosfamide
- Melphalan
- Cyclophosphamide
- Methotrexate
- Pentostatin

Miscellaneous
- Opiates
- MDMA (XTC)
- Levamisole
- Interferon
- NSAIDs
- Clofibrate
- Nicotine
- Amiodarone
- Proton pump inhibitors
- MABs

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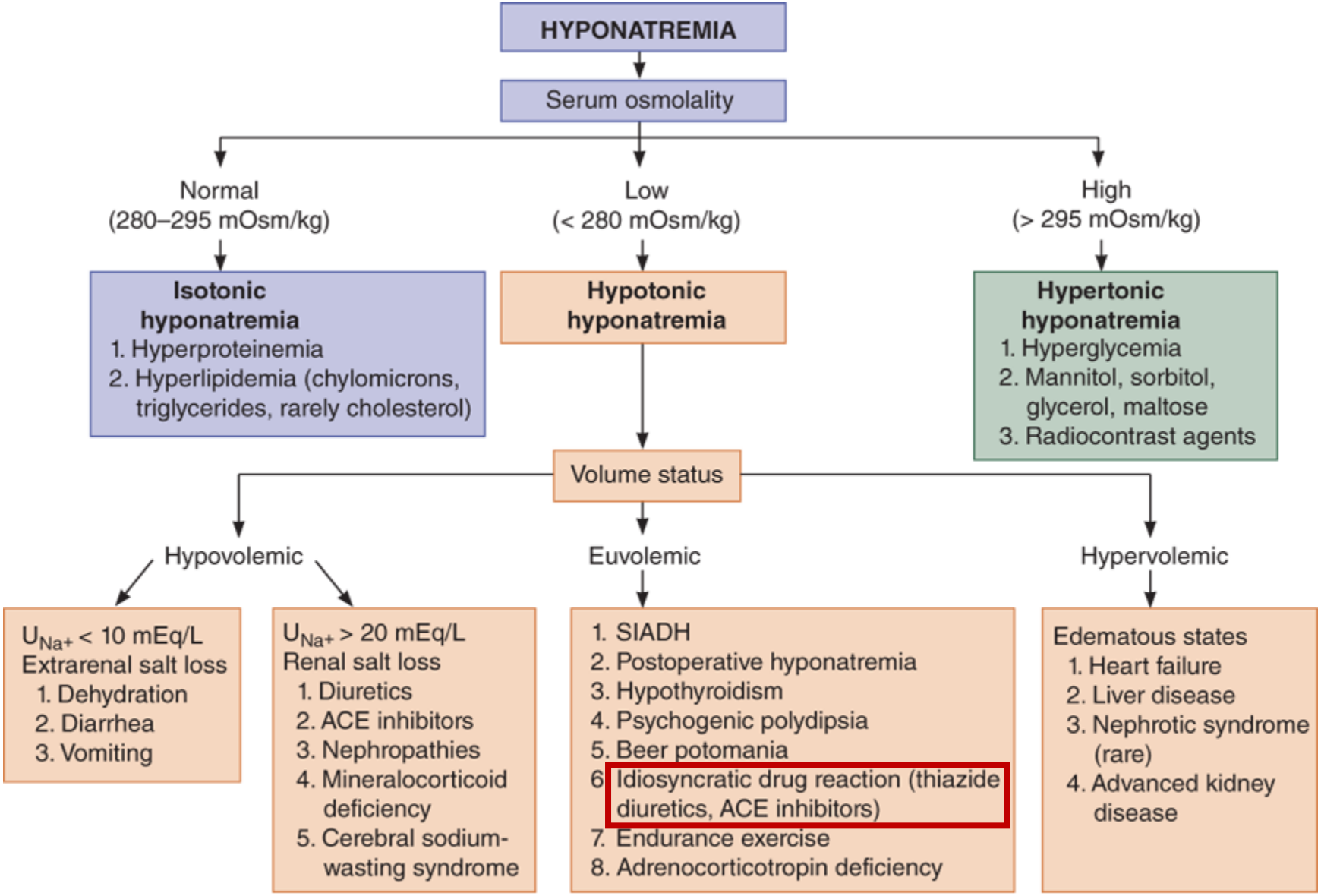

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Shepshelovich D et al. Br J Clin Pharmacol. (2017)

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Vázlat

- A hyponatraemia okai
- A hyponatraemia a napi gyakorlatban

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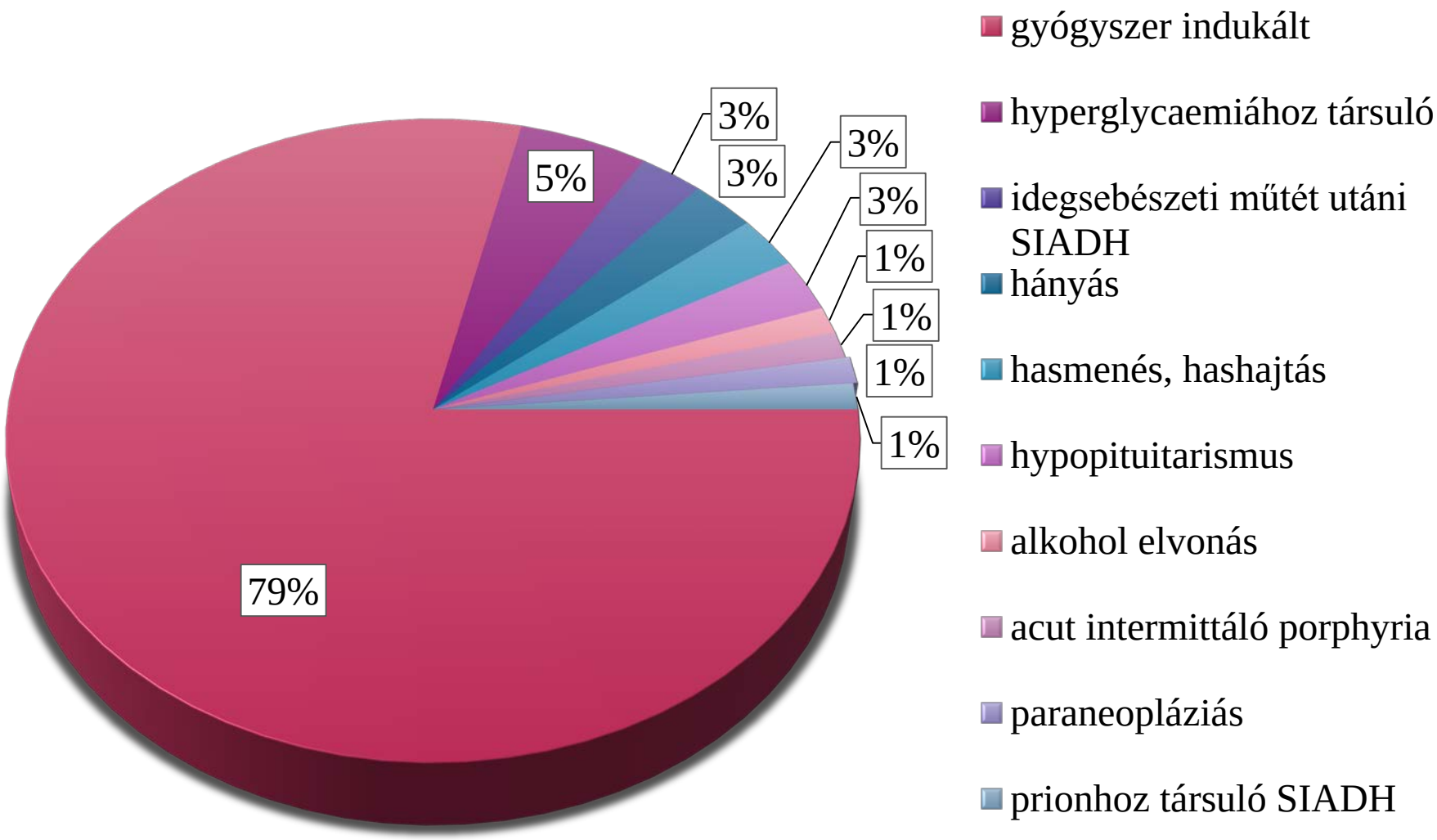
Epikrízis

A 66 éves COPD-s, hypothyreosis miatt hormon substitutioban részesülõ férfi két napja az Infectológiai Osztályra került felvételre lázas állapot, hyponatraemia, gyengeség, száraz köhögés, hányinger, hányás miatt. Láza a parenterális levofloxacin mellett megszûnt, gyulladásos paraméterei csökkentek. A mellkas rtg-n pneumonia nem volt igazolható. Vizelet Legionella, Streptococcus antigén vizsgálata negatív lett. Ezt követõen a beteg a kezelését a II. Belklinikán kívánta tovább folytatni. Így került ma délután felvételre osztályunkra. Laborjaiban progrediáló hyponatraemiát, változatlanul magas CRP értéket, valamint normalizálódó fehérvérsejt számot észleltünk (az infectológián leucopeniás volt). Bentfekvése során a beteg agitáltsága, nyugtalansága fokozódott, hõemelkedést észleltünk. A megbeszélt has, kismedence, mellkas, valamint koponya CT vizsgálat elõtt pszichiátriai javaslatra im. haloperidolt adtunk, azonban a beteg agitáltsága miatt a CT vizsgálathoz intenzív osztályos segítséget kellett kérnünk és elõzetes megbeszélés alapján (Dr. Kiss Tamás-Dr. Gyimesi Tamás) a beteget további ellátás céljából a 3/C Intenzív Osztályra helyezzük. Osztályunkon vérnyomása stabil, oxigén szaturációja megfelelõ volt. A szállítása elõtt kért Astrup vizsgálat technikai okok miatt sikertelen volt.

a PDK-ban történt MR vizsgálathoz képest nem változott. A mindkét felsõ tüdõlebeny érintõ interstitialis eltérések atípusos gyulladásos folyamatra utalhatnak. Paraseptalis emphysema a felsõ lebenyekben. Kismedencei folyadék. Ligamentum arcuatum mediale

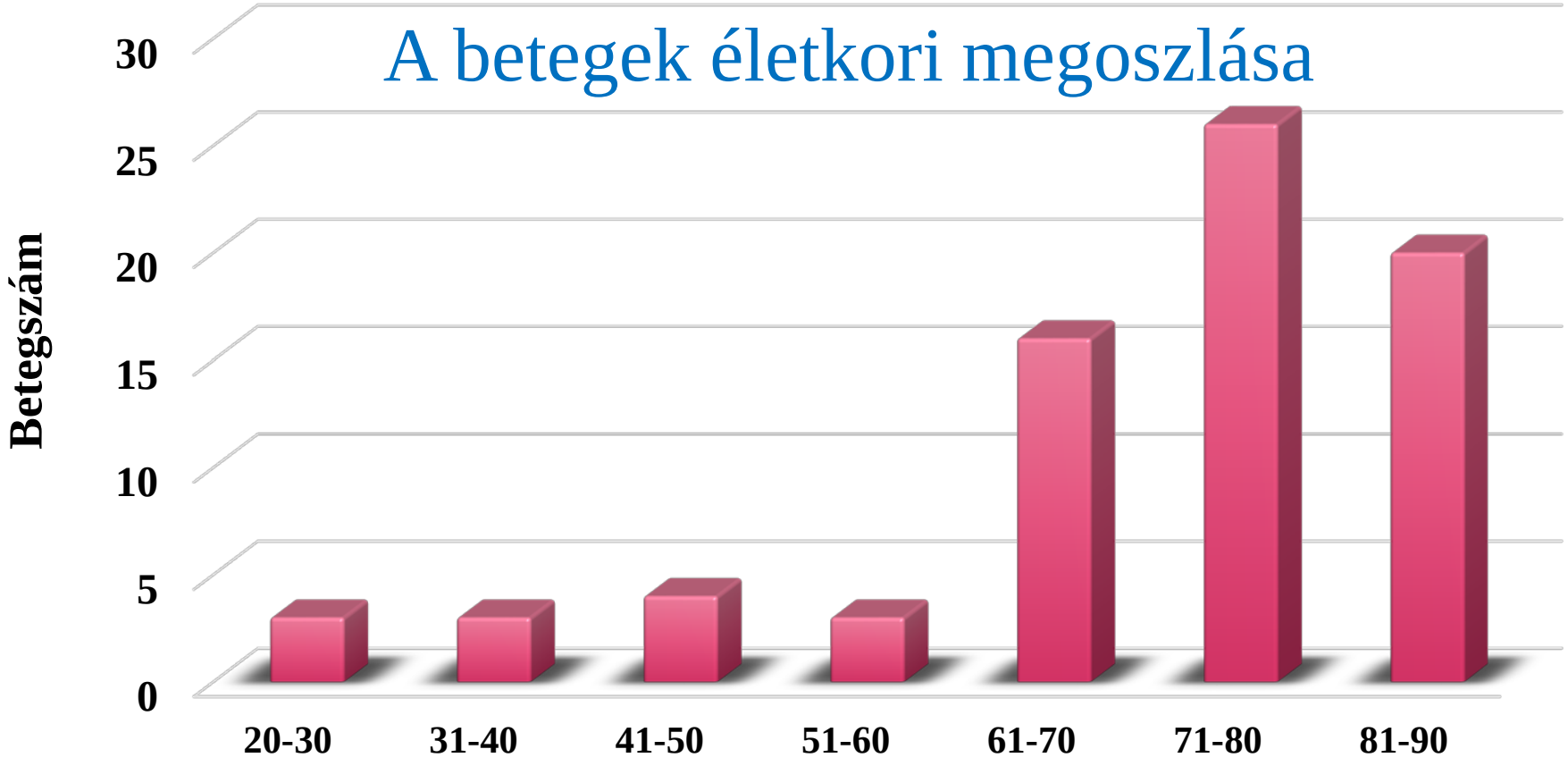
ACTH: >1250,00(A) pg/ml; CORT: 89,2(L) nmol/l

A hyponatraemia feltételezett okai



Magyar Belorvosi Archívum 2014; 67: 399-405

52 nő és 23 férfi



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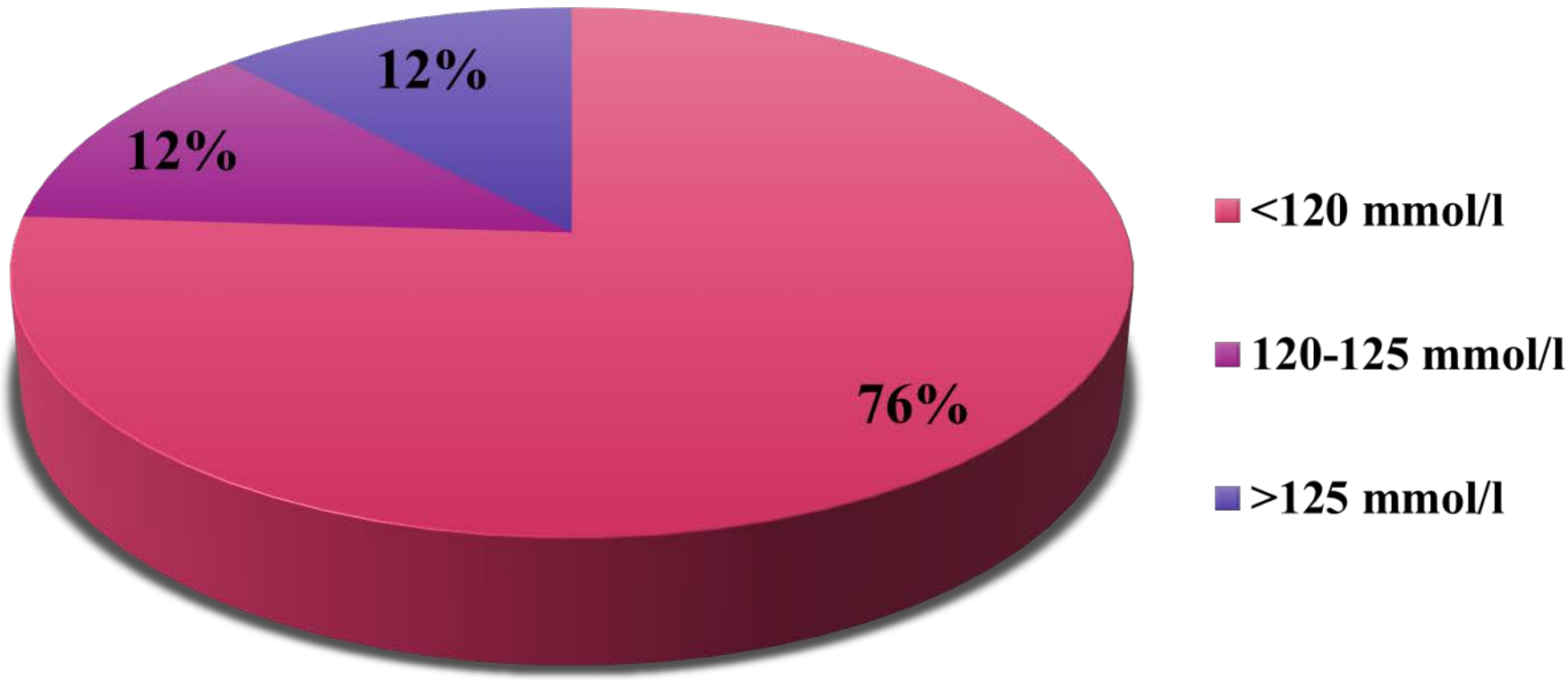
A hyponatraemia súlyossága



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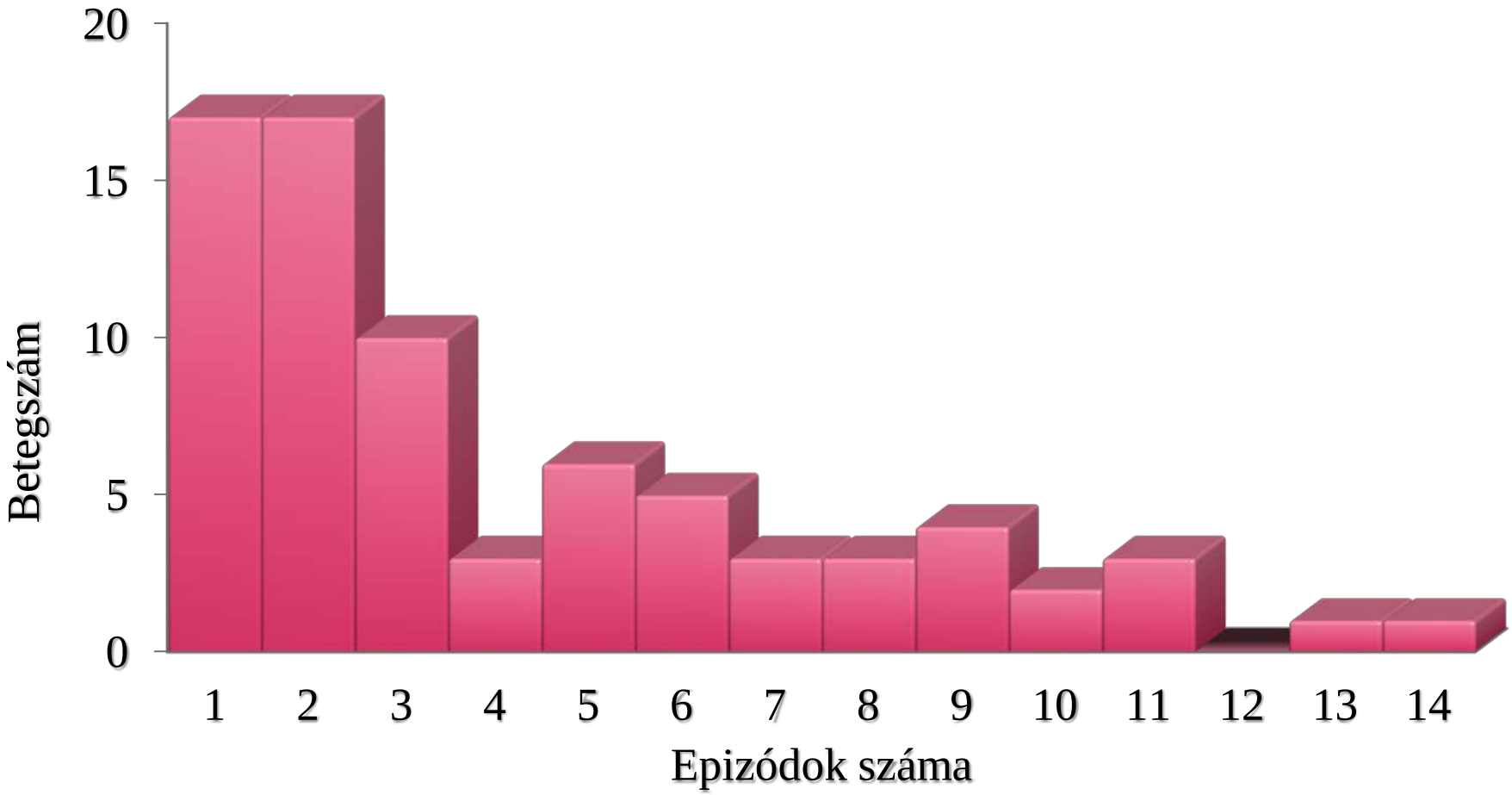
A hyponatraemiás epizódok ismétlődése



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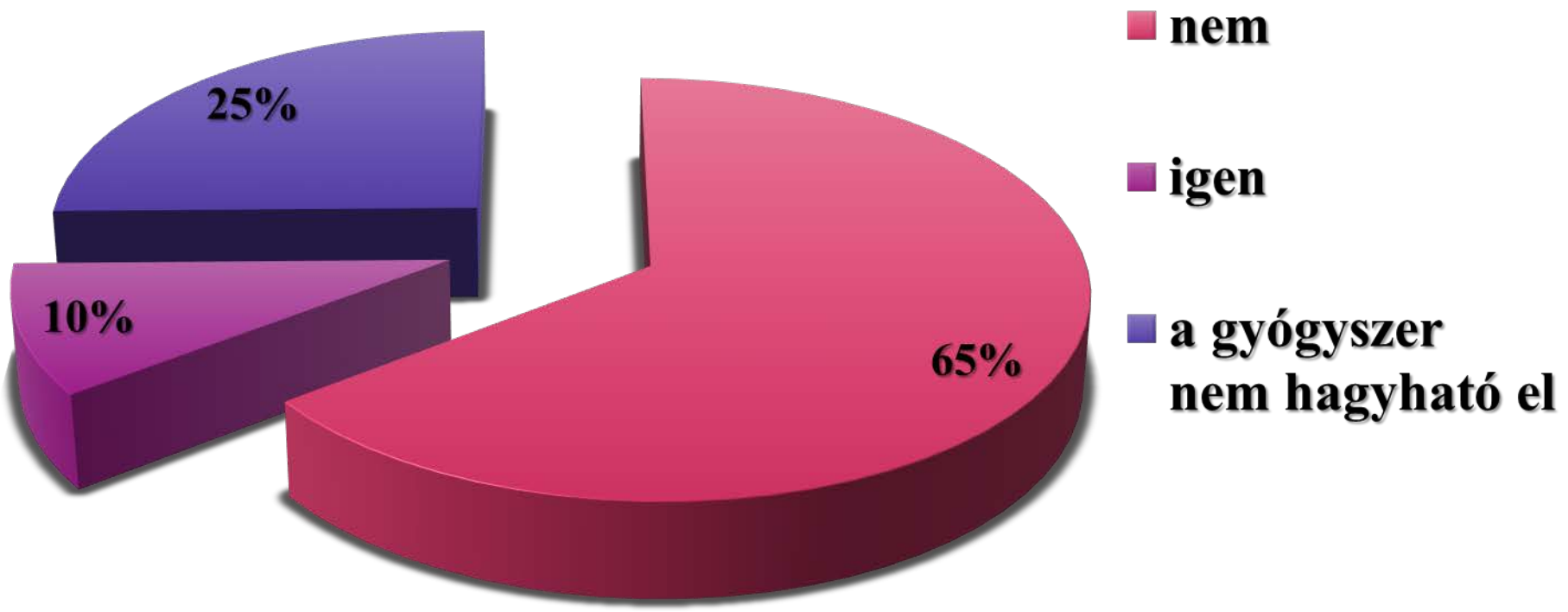

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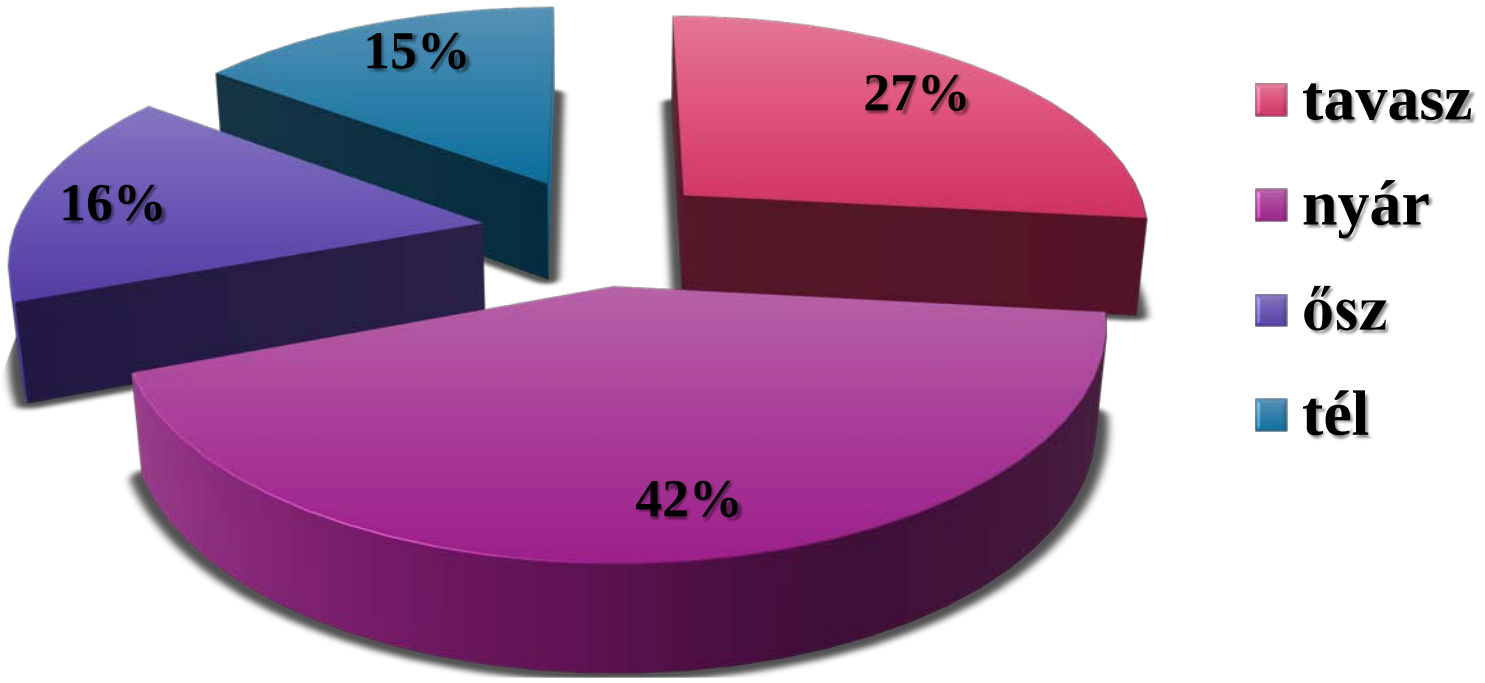

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A hyponatraemia szövődményei

- **Elesés: 13 eset (17,3%)**
- **Csonttörés: 8 eset (10,6%)**
- **Halálozás: 7 beteg (9,3%)**

Magyar Belorvosi Archívum 2014; 67: 399-405

* A thiazid ill. indapamid kezelés mellett kialakuló hyponatraemia patomechanizmusában a disztális csatorna higítóképességének csökkenése, az akvaporin 2 csatornák számának növekedése, szomjúságérzés és megnövekedett vízfogyasztás játszanak szerepet.

Vázlat

- A hyponatraemia okai
- A hyponatraemia a napi gyakorlatban
- A hyponatraemia ellátása

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Thiazide-Induced Syndrome of Inappropriate Secretion of Antidiuretic Hormone

Laboratory Flow Sheet

Day	Serum Sodium, mEq/L (Normal, 135-148)	Serum Potassium, mEq/L (Normal, 3.5-5.3)	Serum Osmolality, mOsm/kg H_2O (Normal, 275-300)	Urine Osmolality, mOsm/kg H_2O (Normal, 50-1,200)	BUN, mg/dL (Normal, 7-22)	Creatinine, mg/dL (Normal, 0.5-1.0)	Clinical Action
1	107	2.6	224	274	9	0.7	Withdrawal of diuretic potassium repletion, water restriction
4	135	5.5	. . .	. . .	. . .	. . .	Water restriction discontinued
5	124	5.2	256 (calculated)	312	. . .	. . .	Water restriction reinstituted
9	135	4.6	289	338	. . .	. . .	Water restriction discontinued
13	136	4.3	. . .	. . .	. . .	. . .	. . .

Egészségügyi szakmai irányelv – A hyponatraemia diagnosztikájáról és kezeléséről

$$Szérum\ Na\ változás = \frac{infúzió\ [Na+]\ - szérum\ [Na+]}{teljes\ test\ víztartalom + 1}$$

Hypokalemia esetén:

$$Szérum\ Na\ változás = \frac{(infúzió\ [Na+] + infúzió\ [K+]) - szérum\ [Na+]}{teljes\ test\ víztartalom + 1}$$

† [Na+], nátrium koncentráció mmol/L; [K+], kálium koncentráció mmol/L

§ Az 1. képlet számlálója a 2. képlet egyszerűsítése. A becsült teljes víztér (literben) a testtömegből arányosan kalkulált érték. Ez az arány nem-idős férfiaknál 0,6; nem-idős nőknél 0,5; idős férfiaknál 0,5; idős nőknél 0,45. Élettani körülmények között a test teljes vízmennyiségének 40%-a extracellulárisan, 60%-a intracellulárisan helyezkedik el.

Az Adrogué-Madias képlet a fent leírt formában az *1 liternyi* infúzió adásának hatására bekövetkező szérum nátrium ($[Na]_{se}$) szint változást adja meg. Ettől eltérő mennyiségű infúzió adása esetén a képlet:

[1] $\Delta\ [Na]_{se} = V_{inf} \times ([Na]_{inf} + [K]_{inf} - [Na]_{se}) / (\text{teljes test víz} + V_{inf})$

[2] Elektrolit mentes víz clearance: $V_{viz}\ [1 - ([Na]_{viz} + [K]_{viz}) / [Na]_{se}]$

[3]
$$\Delta\ [Na]_{se} = \frac{V_{inf} \times (1{,}11[Na]_{inf} + [K]_{inf} - [Na]_{se}) + V_{viz} \times [Na]_{se} - V_{viz} \times ([Na]_{viz} + [K]_{viz})}{\text{Teljes test víz} + V_{inf} - V_{viz}}$$

Populationwide Sodium Guidance 'Makes No Sense' in Most Countries

Shelley Wood | Sep 04, 2013

AMSTERDAM — A new analysis from the large international PURE trial should intensify the ongoing debate over the link between sodium and blood pressure. Presenting **PURE SODIUM** here at the European Society of Cardiology (ESC) 2013 Congress, **Dr Andrew Mente** (McMaster University, Hamilton, ON) and colleagues found that only certain subgroups will actually experience blood-pressure benefits from restricting their sodium consumption. As such, sweeping recommendations for daily sodium limits are likely pointless in many parts of the world.

"A targeted approach would be more appropriate for populations that consume a moderate amount of sodium in the 3-to-5-g/day range, because there would be very little benefit or a modest benefit of getting them down to low levels," Mente told **heart*wire*** . "For people who consume high amounts of sodium, it's important to get them down to moderate levels, so . . . a population-based approach would be more effective: you'd get better bang for the buck."

In one of the most striking findings in their study of almost 100 000 subjects, Mente and colleagues found that none of the populations surveyed had a "usual intake" of sodium that fell at or below the 2.3 g/day recommended in most guidelines.

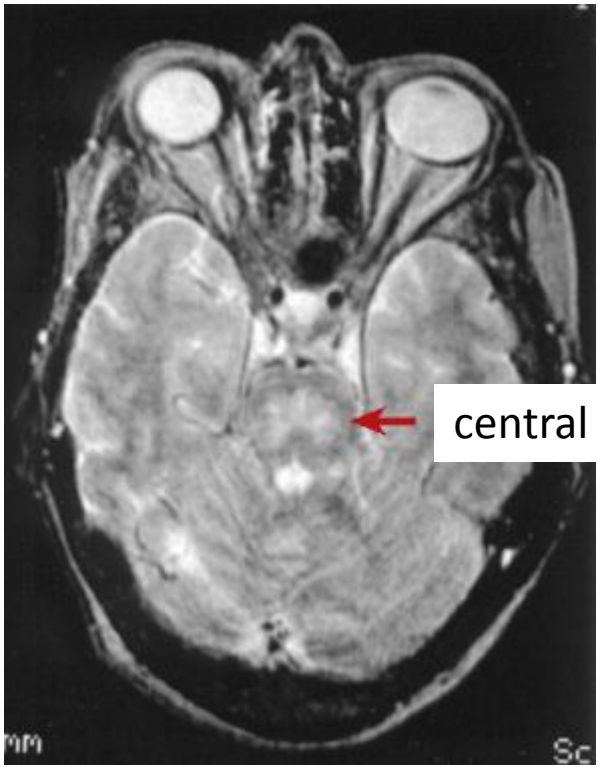
Teendők hyponatraemiában

- Klinikai kép értékelése
- Minden gyógyszert ellenőrizni (EESZT és/vagy behozatni)!
- Parenterális sópótlás általában (legalább) 125 mmol/l-ig
- Első 24 órában ≥2X Na meghatározás
- ≥1X Na meghatározás naponta, amíg infúzió szükséges
- Stabilizálódás ellenőrzése

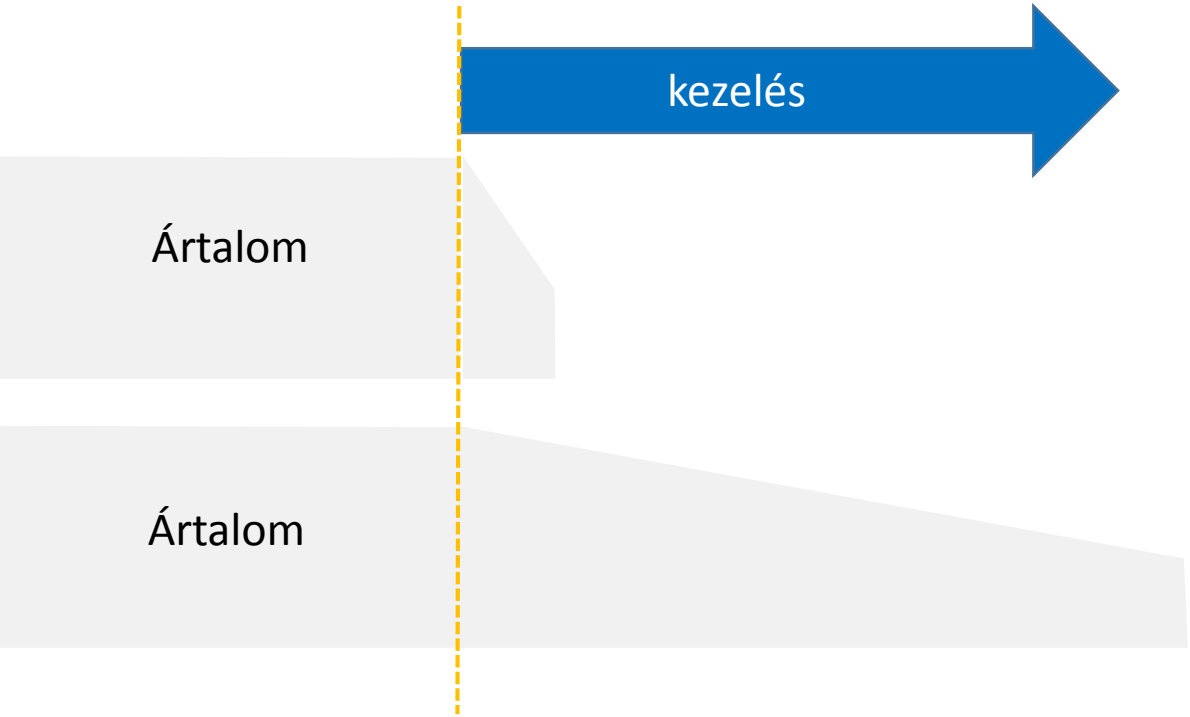
(500)-1000 ml Salsol

Na változás	
<3 mmol/l	+(500)-1000 ml Salsol +2 g NaCl
3-5 mmol/l	+(500)-1000 ml Salsol
5-10 mmol/l	+ 0 ml Salsol
>10 mmol/l	+(500)-1000 ml hipotóniás oldat

} + sze. furosemid



central pontine myelinolysis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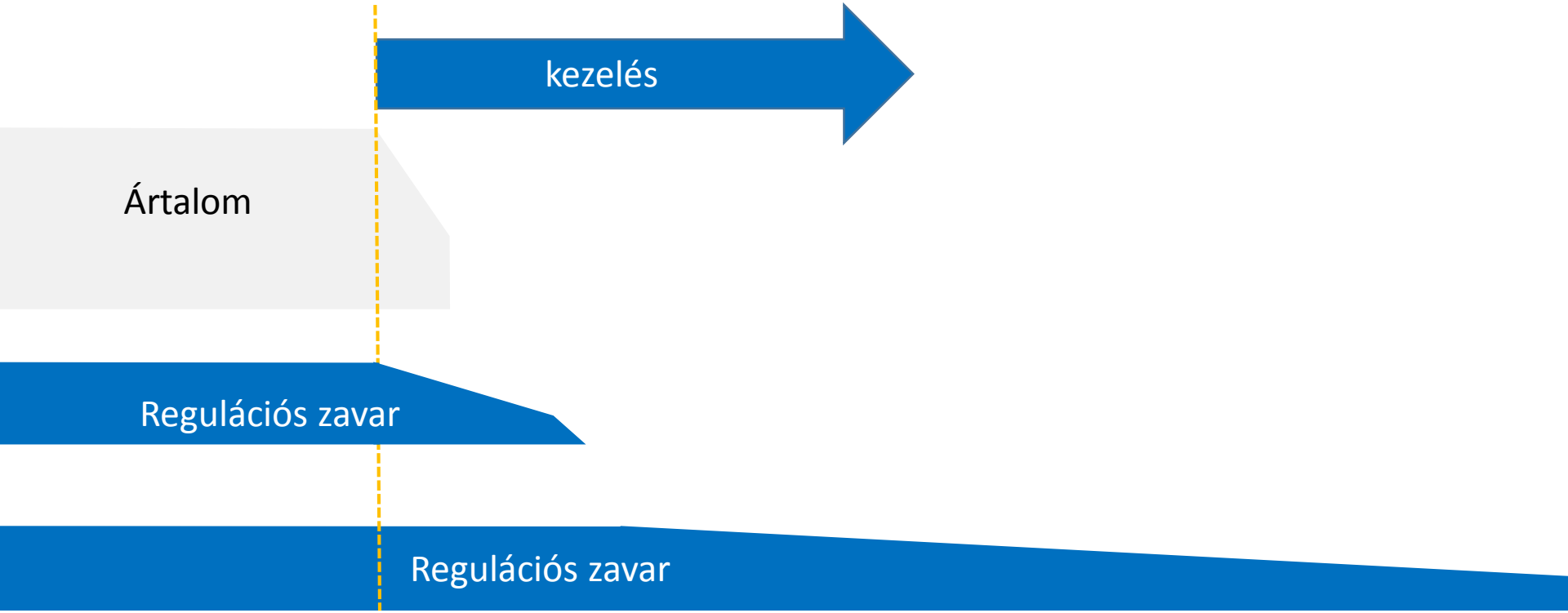
Ártalom
Ártalom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Ártalom
Regulációs zavar
Regulációs zavar

AITI sajátja | Metabólikus szindróma | Összesített Labor Eredmény | Összesített Mikrobiológiai Labor Eredmény | Onkológia | Nátrium, kálium | Ra

Analízis	Egys	Referencia tartomány	2009.05.25 12:58	2019.01.20 15:04	2019.01.21 08:13	2019.01.22 08:19	2019.01.23 08:25	2019.01.24 08:53	2019.01.25 08:09
AITI II. laborkérőlap									
Nátrium	mmol/l	136-145 -	142	121	117	113	109	119	127
Kálium	mmol/l	3,50-5,10 -	4,02	3,14	3,09	2,95	3,43	3,66	5,32
Kalcium	mmol/l	2,15-2,55 -					2,01		2,00
Glükóz	mmol/l	3,90-7,00 -		10,11					
Karbamid	mmol/l	2,14-8,21 -	4,92	2,22	2,76		3,00	3,02	2,92
Kreatinin	umol/l	44-80 -	59	32	30		21	30	34
Fehérvérsejt	Giga/l	4,000-10...	5,250	3,250					6,060
Vörösvértest	T/l	3,90-5,30 -	4,22	3,92					3,93
Hemoglobin	g/l	120-157 -	135	134					127
Hematokrit	%	34,1-44,9 -	39,9	35,0					36,3
Trombocita	Giga/l	140,0-44...	189,0	138,0					190,0
Ultraszenzitiv CRP	mg/l	<5,00 -	1,70	59,70			8,70		
Protrombin INR	.	0,90-1,15 -		1,20					
Összfehérje	g/l	66,0-87,0 -							57,5
Albumin	g/l	35,0-52,0 -							36,6
Összbilirubin	umol/l	5,0-20,0 -	6,6						
GOT	U/l	<44 -	15	22					
GPT	U/l	<50 -	13	12					
Gamma-GT	U/l	<70 -	18						
Alkalikus foszfatáz	U/l	100-300 -	182						
LDH	U/l	<450 -	348						
D-dimer	ug/l	<500 -		539					

Összesített Labor Eredmény: Virág Bertalanné - Googl

https://vemedapp1.kk.pte.hu:9444/pte/UpdateFrame.fl

Összesített Labor Eredmény: Virág Bertal

AITI sajátja | Metabólikus szindróma | Összesített Labor Eredmény | Összesített Mikrobiológiai Labor Eredmény | Onkológia | Nátrium, kálium

Analízis	Egys	Referencia tartomány	2019.01.20 15:04	2019.01.21 08:13	2019.01.22 08:19	2019.01.23 08:25	2019.01.24 08:53	2019.01.25 08:09
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Glükóz	mmol/l	3,90-7,00 -	10,11					
Karbamid	mmol/l	2,14-8,21 -	2,22	2,76		3,00	3,02	2,92
Kreatinin	umol/l	44-80 -	32	30		21	30	34
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Protrombin INR	.	0,90-1,15 -	1,20					
Összfehérje	g/l	66,0-87,0 -						57,5
Albumin	g/l	35,0-52,0 -						36,6
GOT	U/l	<44 -	22					
GPT	U/l	<50 -	12					
D-dimer	ug/l	<500 -	539					

Teendők idült hyponatraemiában

1. Folyadék-megszorítás (kivéve platina alapú kezelés)
2. Sózás (fludrokortizon?) + furosemid
3. Urea (Ure-Na by Nephcentric) ?
4. Tolvaptan (Samsca)??

Vázlat

- A hyponatraemia okai
- A hyponatraemia a napi gyakorlatban
- A hyponatraemia ellátása
- A hyponatraemia hátterében álló endokrin eltérések

bajnok.laszlo@pte.hu


OUT OF
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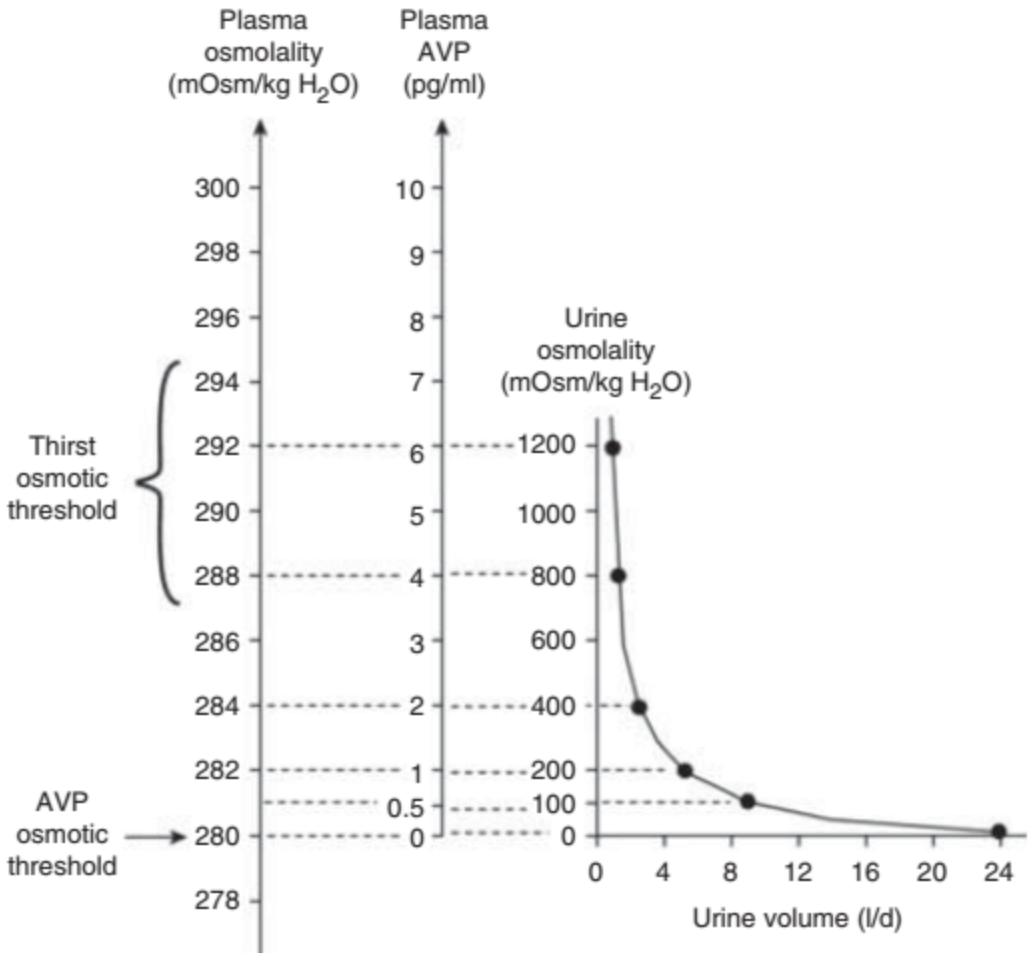
BOX


Plasma osmolality (mOsm/kg H_2O)
Plasma AVP (pg/ml)
Urine osmolality (mOsm/kg H_2O)
300
298
296
294
292
290
288
286
284
282
280
278
10
9
8
7
6
5
4
3
2
1
0.5
0
1200
1000
800
600
400
200
100
0
0
4
8
12
16
20
24
Urine volume (l/d)
Thirst osmotic threshold
AVP osmotic threshold

Hyponatremia: A Prospective Analysis of Its Epidemiology and the Pathogenetic Role of Vasopressin

ROBERT J. ANDERSON, M.D.; HSIAO-MIN CHUNG, M.D.; RUDIGER KLUGE, M.D.; and ROBERT W. SCHRIER, M.D.; Denver, Colorado

We prospectively evaluated the frequency, cause, and outcome of hyponatremia (plasma sodium concentration, < 130 meq/L), as well as the hormonal response to this condition, in hospitalized patients. Daily incidence and prevalence of hyponatremia averaged 0.97% and 2.48%, respectively. Two thirds of all hyponatremia was hospital acquired. Normovolemic states (so-called syndrome of inappropriate secretion of antidiuretic hormone) were the most commonly seen clinical setting of hyponatremia. The fatality rate for hyponatremic patients was 60-fold that for patients without documented hyponatremia. Nonosmotic secretion of vasopressin was present in 97% of hyponatremic patients in whom it was sought. In edematous and hypovolemic patients, plasma hormonal responses (increases in plasma renin activity and aldosterone and norepinephrine levels) were compatible with baroreceptor-mediated release of vasopressin. Hyponatremia is a common hospital-acquired electrolyte disturbance that is an indicator of poor prognosis. Nonosmotic secretion of arginine vasopressin is a major pathogenetic factor in this electrolyte disturbance.

Nonosmotic secretion of vasopressin was present in 97% of hyponatremic patients…

logic, and psychiatric inpatients per day throughout the study. Of the patient population approximately one third were from the local community, one third were referral patients, and one third indigent patients. A central laboratory computer provided a list of all hospitalized patients with a plasma sodium concentration less than 130 meq/L at 0800 h and 1600 h daily. Rec- valu- f the

ation less than 130 meq/L. This concentration was arbitrarily selected because it is clearly abnormal. Plasma sodium concentrations averaged 140.1 ± 0.43 in 27 patients awaiting elective surgery who were randomly selected from the medical-surgical wards during the study. Moreover, a plasma sodium concentration less than 130 meq/L would lower plasma osmolality by approximately 20 mosmoles/kg of water. This magnitude of decrement in osmolality is approximately the threshold at which clinical symptoms have occurred in patients in experimental studies of hyponatremia (5). After initial evaluation and follow-up observation, patients were divided into groups according to the following criteria.

Annals of Internal Medicine. 1985;102:164-168.

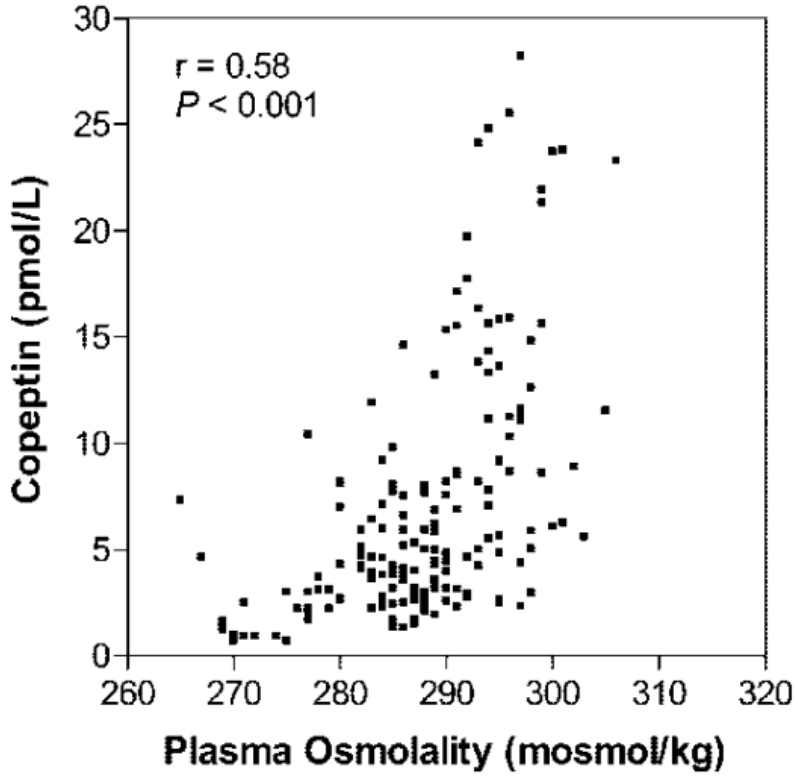
0021-972X/07/$15.00/0
Printed in U.S.A.

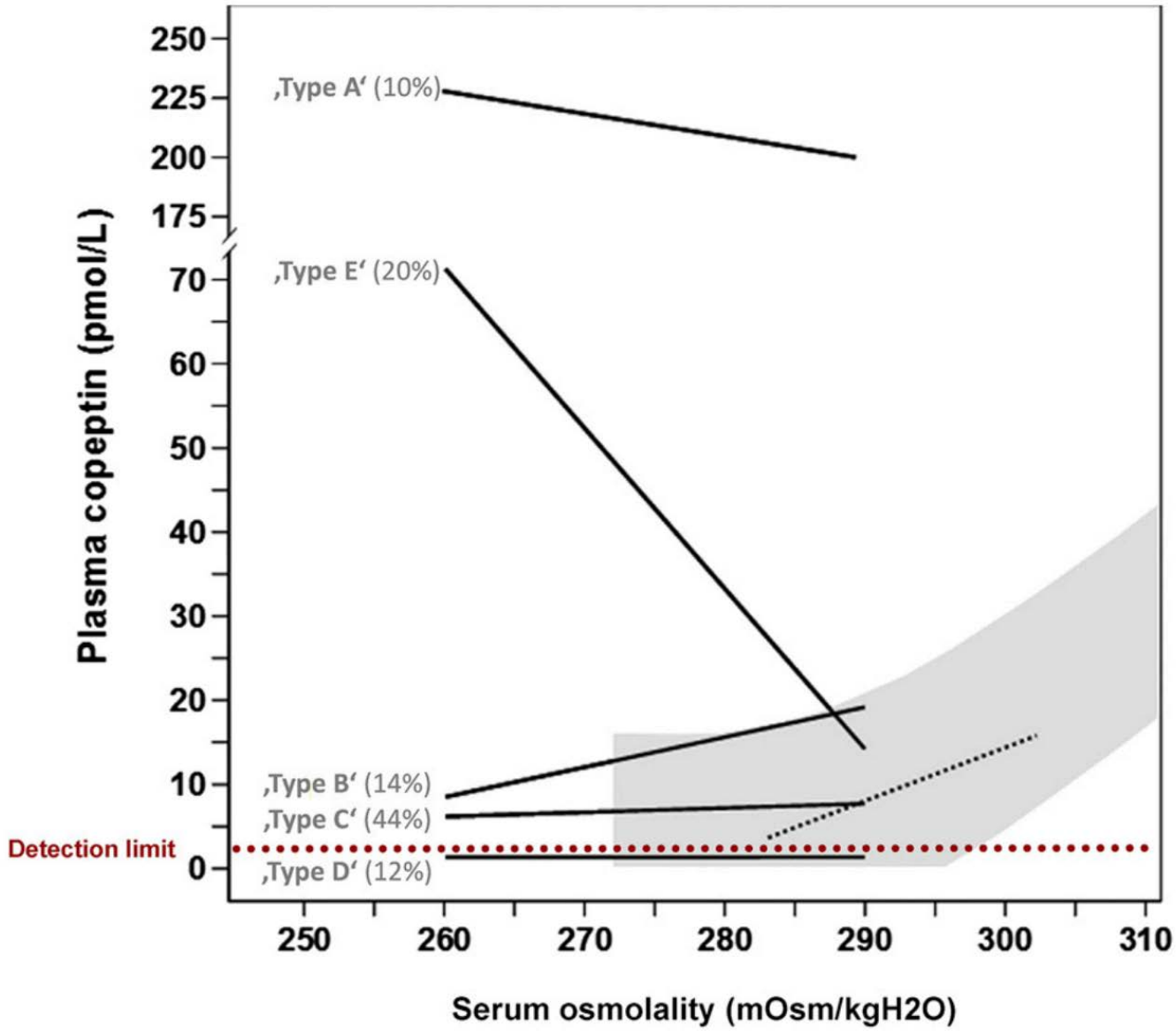
The Journal of Clinical Endocrinology & Metabolism 92(10):3973–3978

doi: 10.1210/jc.2007-0232

Changes in Plasma Copeptin, the C-Terminal Portion of Arginine Vasopressin during Water Deprivation and Excess in Healthy Subjects

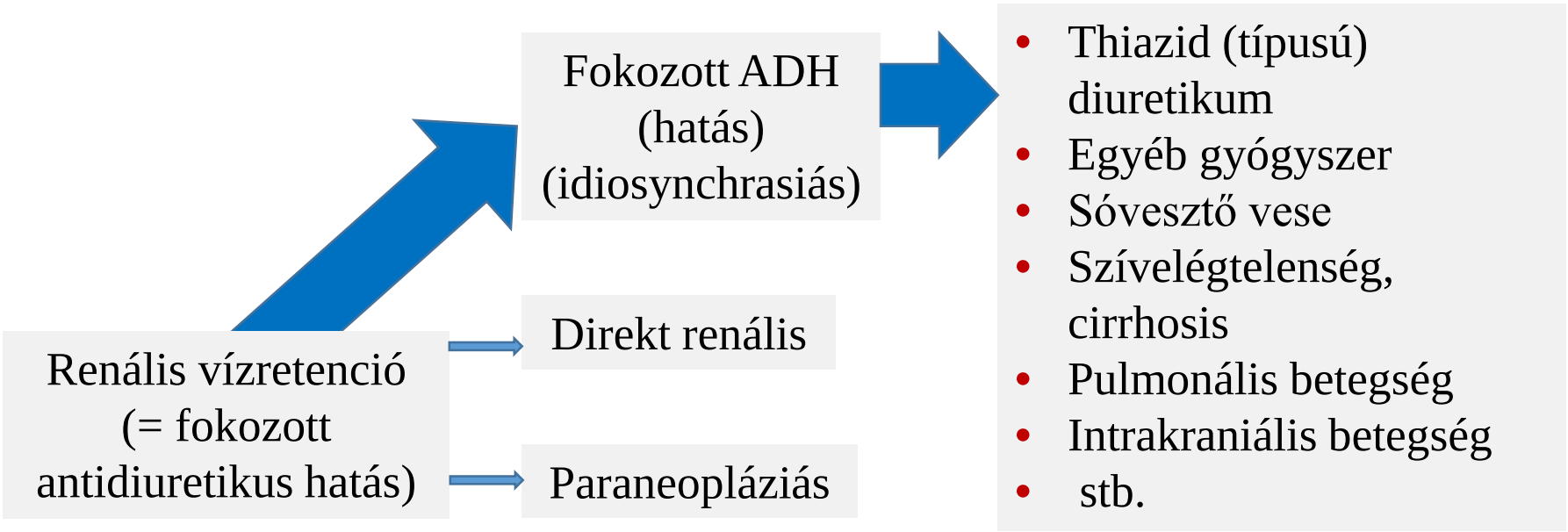
Gabor Szinnai, Nils G. Morgenthaler, Kaspar Berneis, Joachim Struck, Beat Müller, Ulrich Keller, and Mirjam Christ-Crain





J Am Soc Nephrol 28: 1340–1349, 2017.

A hyponatraemia okai (szerintem)

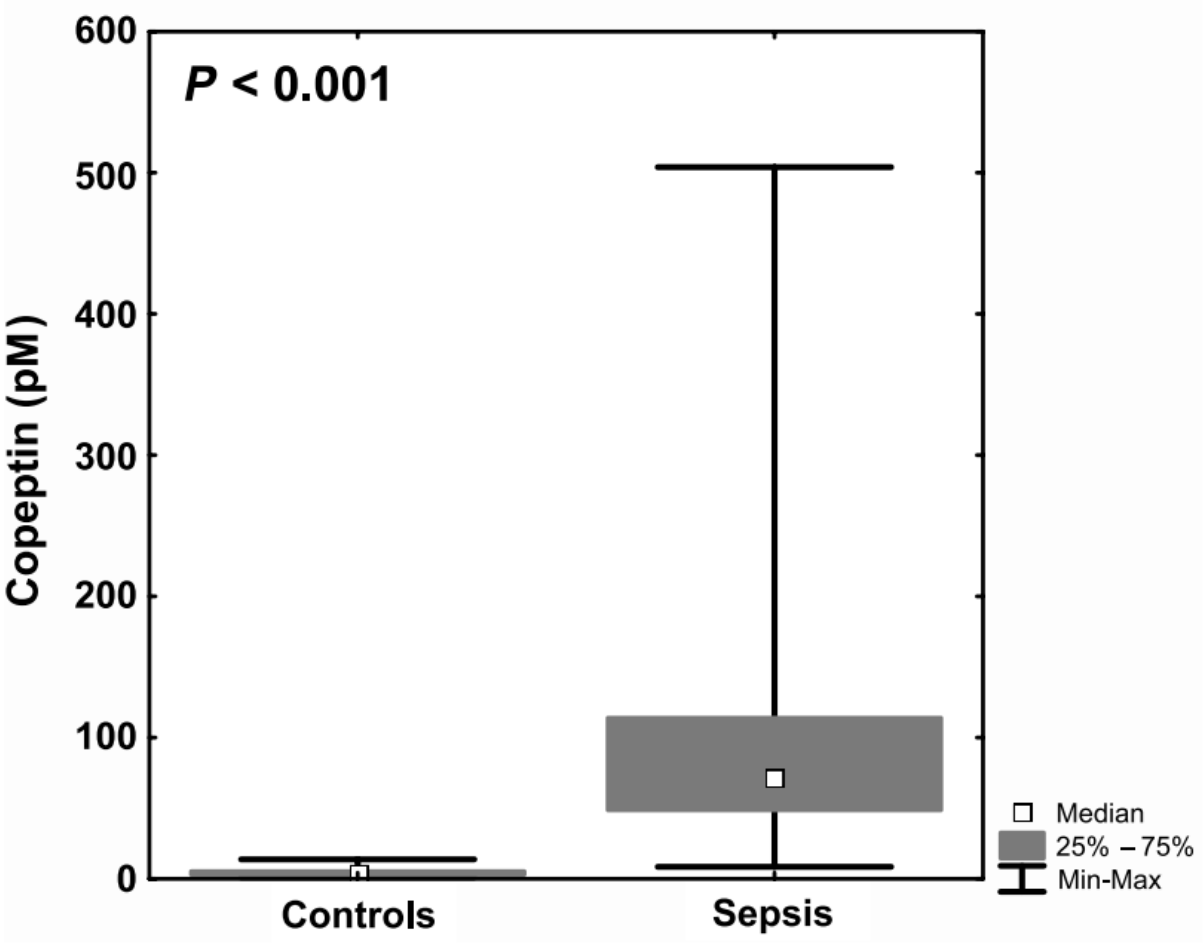


Tiszta sóvesztéses

SHOCK, Vol. 28, No. 2, pp. 219–226, 2007

COPEPTIN, A STABLE PEPTIDE OF THE ARGININE VASOPRESSIN PRECURSOR, IS ELEVATED IN HEMORRHAGIC AND SEPTIC SHOCK

Nils G. Morgenthaler,* Beat Müller,† Joachim Struck,* Andreas Bergmann,* Heinz Redl,‡ and Mirjam Christ-Crain†



CLINICAL RESEARCH www.jasn.org

J Am Soc Nephrol 19: 1015–1024, 2008.

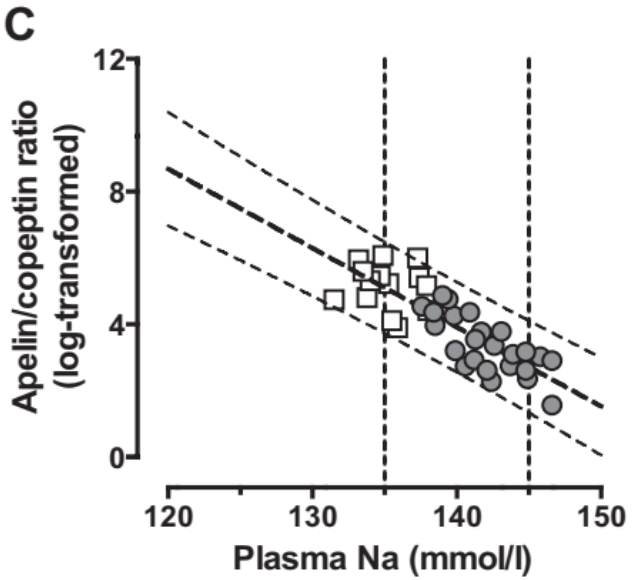
Reciprocal Regulation of Plasma Apelin and Vasopressin by Osmotic Stimuli

Michel Azizi,* Xavier Iturrioz,† Anne Blanchard,* Séverine Peyrard,* Nadia De Mota,† Nicolas Chartrel,‡ Hubert Vaudry,‡ Pierre Corvol,§ and Catherine Llorens-Cortes†

An Abnormal Apelin/Vasopressin Balance May Contribute to Water Retention in Patients With the Syndrome of Inappropriate Antidiuretic Hormone (SIADH) and Heart Failure

Anne Blanchard,* Olivier Steichen, Nadia De Mota, Emmanuel Curis, Cedric Gauci, Michael Frank, Grégoire Wuerzner, Peter Kamenicky, Amélie Passeron, Michel Azizi, and Catherine Llorens-Cortes

(*J Clin Endocrinol Metab* 98: 2084–2089, 2013)



Vázlat

- A hyponatraemia okai
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- A hyponatraemia ellátása
- A hyponatraemia hátterében álló endokrin eltérések
- Stressz és hormonok

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STRESS AND THE GEN

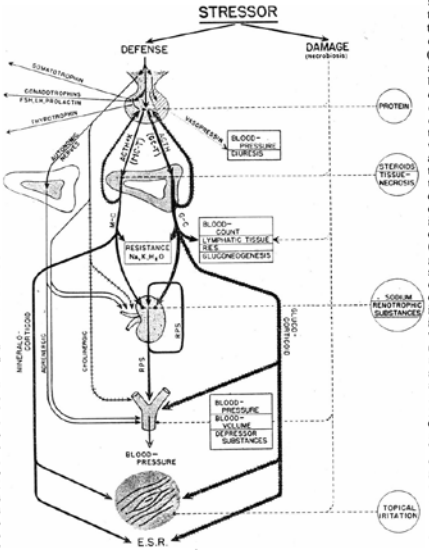
HANS SELYE,

Professor and Director of the Institute of Experim

syndrome. In the biological sense stress is the interaction between damage and defence, just as in physics tension or pressure represents the interplay between a force and the resistance offered to it.

In addition to damage and defence, every stressor also produces certain specific actions (e.g., anaesthetics act upon the nervous system, diuretics upon water metabolism, insulin upon the blood sugar) quite apart from their stressor effects. Hence the general adaptation syndrome never occurs in its pure form, but is always complicated by superimposed specific actions of the eliciting stressors. In contemplating any biologic response (e.g.,a spontaneous disease, an intoxication, a psychosomatic reaction), it is usually quite difficult to identify individual manifestations as being due respectively to damage, defence, or specific actions of the provocative agent. Only non-specific damage and defence are integral parts of the general adaptation syndrome, but the specific actions of the eliciting stressors modify the course of the resulting general adaptation syndrome (e.g., the glycaemic curve will deviate from the characteristic pattern if insulin is used as the stressor agent; the neurological manifestations will be atypical if the general adaptation syndrome is provoked by ether). In this sense they act as "conditioning factors." Certain circumstances, not directly related to the stress situation, are also prone to alter the course of the general adaptation syndrome. Among these heredity, pre-existent disease of certain organ systems, and the diet are especially important.

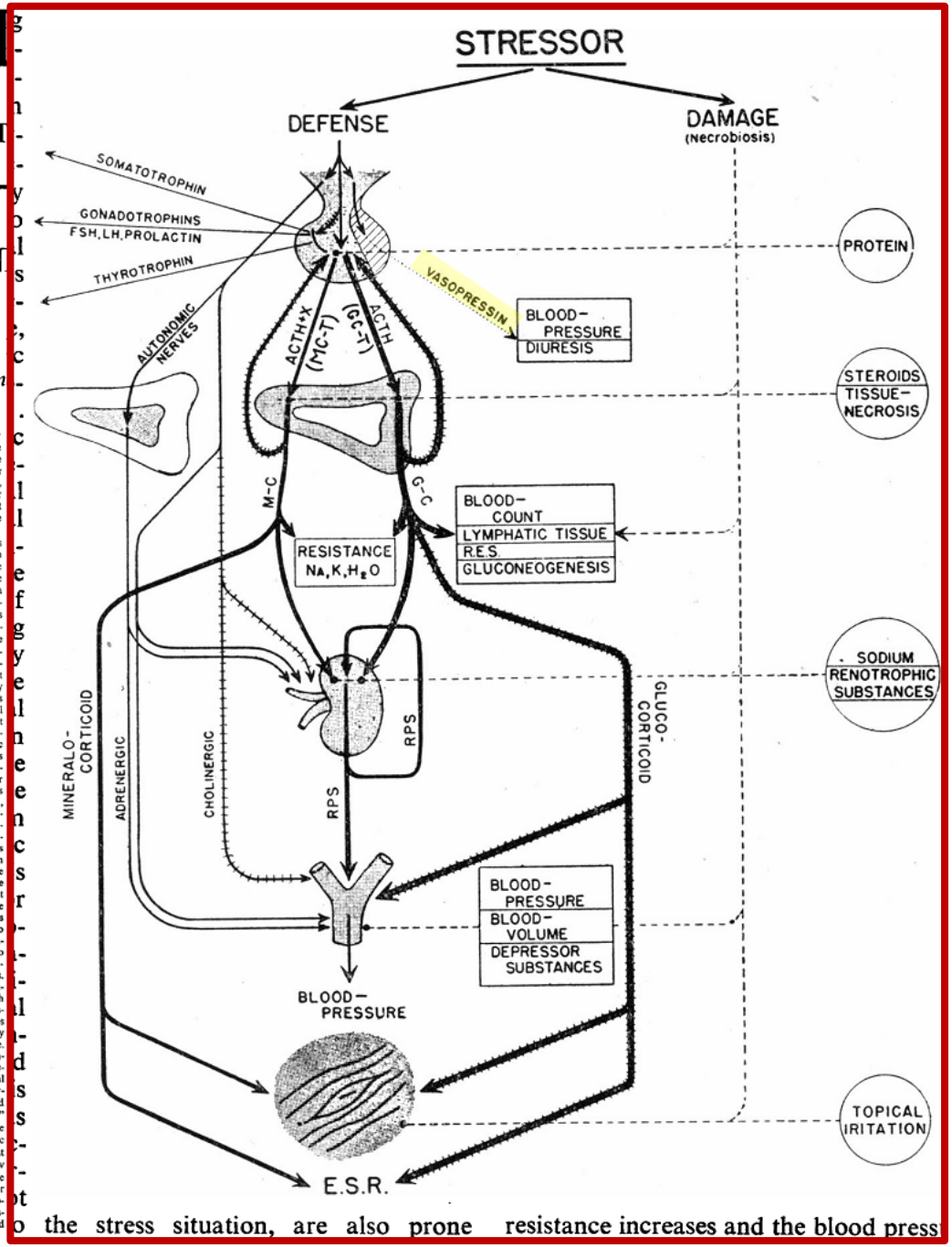
The accompanying schematic drawing disregards the specific actions of stressors, since they are not part of the general adaptation syndrome. It attempts to depict only the main pathways through which non-specific stress itself affects the organism and the manner in which such reactions are conditioned.



Defence

The systemic defence measures, against both general and localized (topical) injuries, are co-ordinated through the hypothalamic vegetative centres and the hypophysis. The initial pathways through which stressors act upon these centres are not yet known. Probably either humoral or nervous impulses, coming from the site of direct injury, can induce the hypothalamus-hypophysis system to gear the body for defence. Subsequently both of the two great integrating mechanisms, the nervous and the endocrine system, are alerted

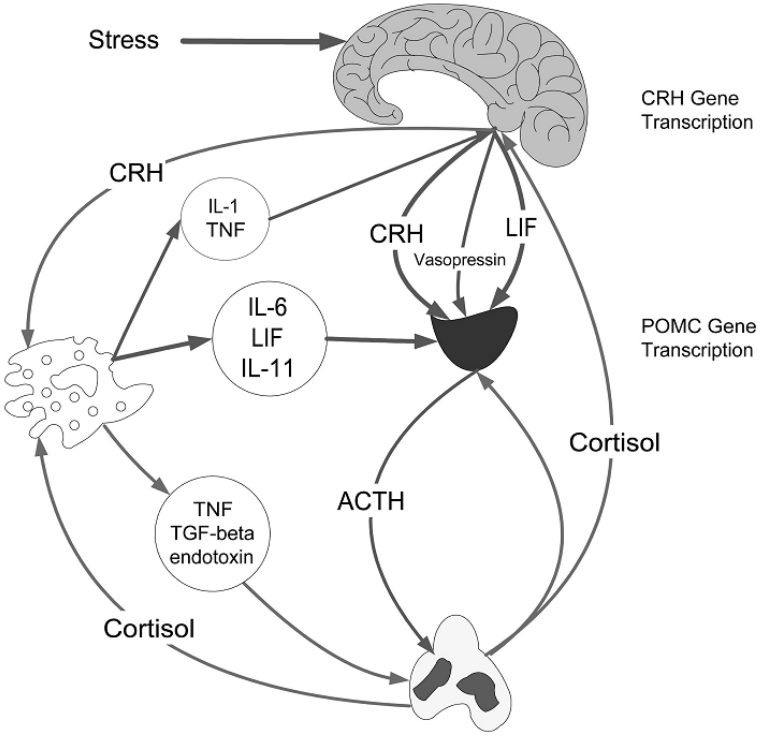
The Nervous Defence Mechanism.—Nervous impulses descend from the hypothalamic vegetative centres, through the autonomic nerves, to the peripheral organs. The splanchnics induce the adrenal medulla to discharge adrenergic hormones (adrenaline and *nor*-adrenaline) into the blood. Other adrenergic nerves influence their target organs directly through fibres which in the final analysis again act through the liberation of adrenergic compounds, in this case at their endings in the effector organs themselves (blood vessels, glands, etc.). Presumably the discharge of adrenergic hormones into the circulation is most effective when they are needed throughout the body, while the sympathetic nerves are better suited to impart similar impulses selectively to certain circumscribed territories. The most conspicuous results of such neuro-humoral discharges are changes in the contractility of smooth muscle. Owing to an adrenergic vasoconstriction, peripheral resistance increases and the blood pressure rises. This hypertensive response may be further accentuated by an increased cardiac volume and the opening of the "renal shunt," which deviates blood from the cortical glomeruli to the juxtamedullary region of the kidney. This neurogenic activation of the "shunt" is quite comparable to that induced by mechanical interference with the arterial inflow (Goldblatt clamp, "endocrine kidney" operation) into the kidney; hence it augments the production of renal pressor substances (R.P.S.). The latter also cause peripheral vasoconstriction; thus they further augment peripheral resistance in the cardiovascular system and hence the blood pressure rises (cf. below).



the stress situation, are also prone resistance increases and the blood press

Recommendations for the diagnosis and management of corticosteroid insufficiency in critically ill adult patients: Consensus statements from an international task force by the American College of Critical Care Medicine

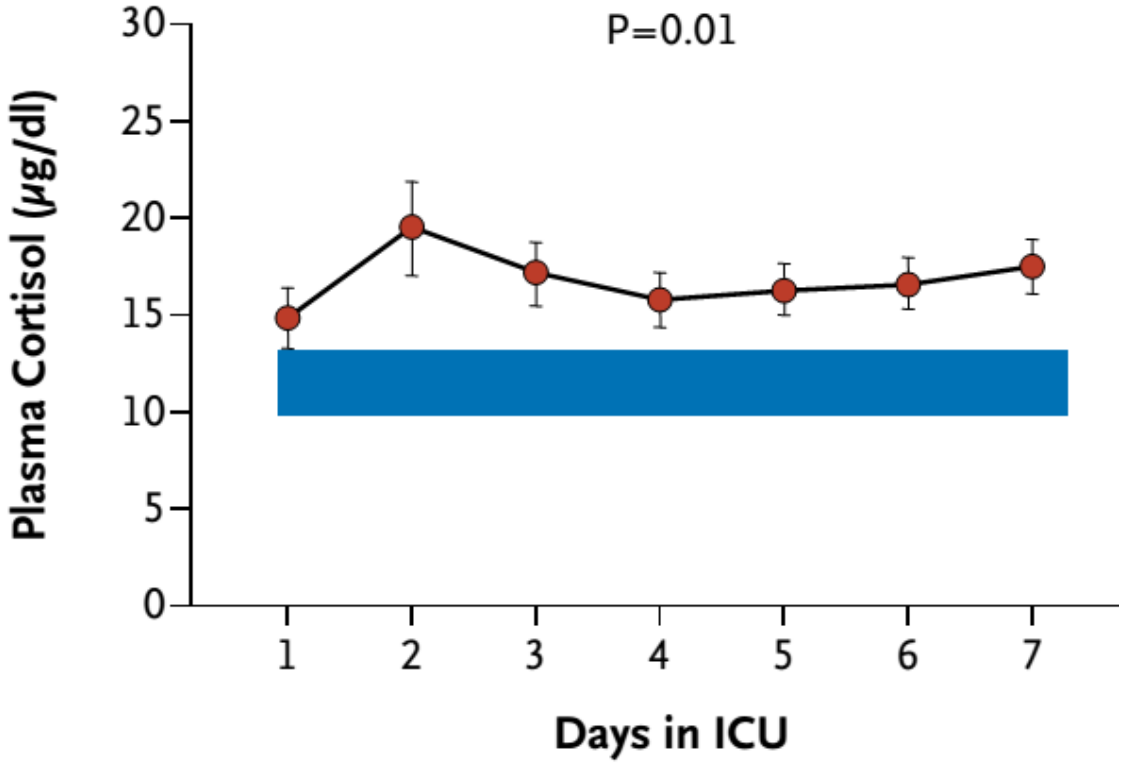
Paul E. Marik, MD, FCCM; Stephen M. Pastores, MD, FCCM; Djillali Annane, MD; G. Umberto Meduri, MD; Charles L. Sprung, MD, FCCM; Wiebke Arlt, MD; Didier Keh, MD; Josef Briegel, MD; Albertus Beishuizen, MD; Ioanna Dimopoulou, MD; Stylianos Tsagarakis, MD, PhD; Mervyn Singer, MD; George P. Chrousos, MD; Gary Zaloga, MD, FCCM; Faran Bokhari, MD, FACS; Michael Vogeser, MD



(Crit Care Med 2008; 36:1937–1949)

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 APRIL 18, 2013 VOL. 368 1477 1488



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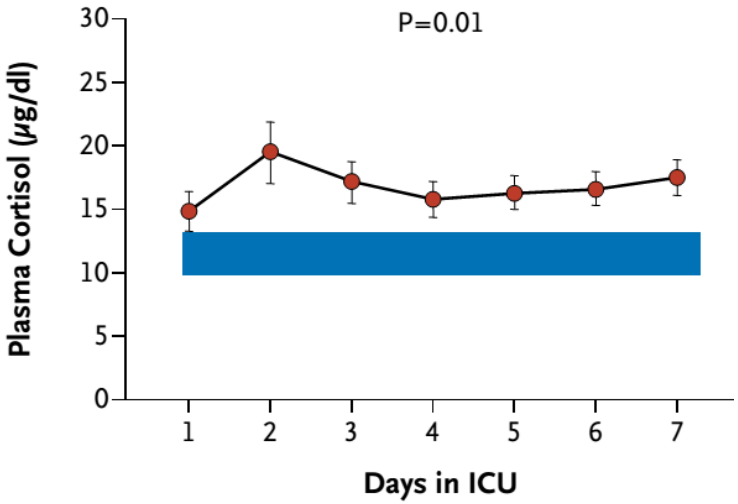

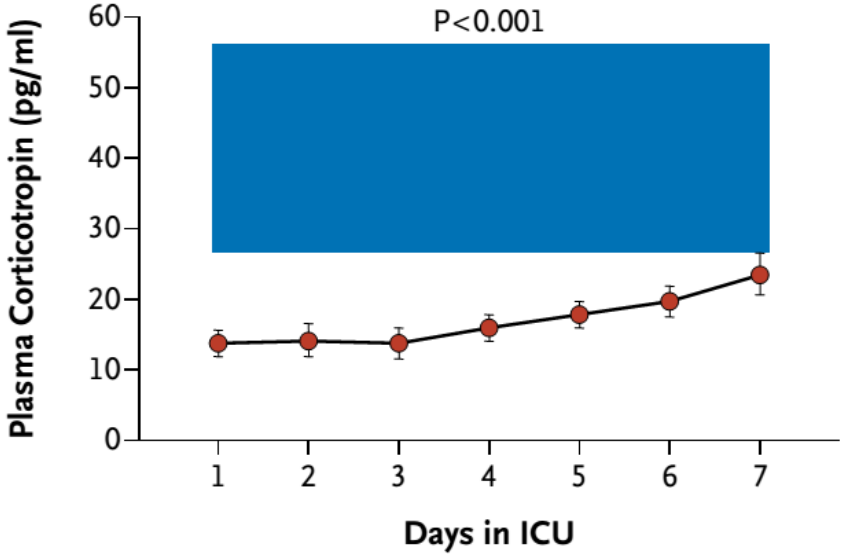

Vázlat

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- A hyponatraemia ellátása
- A hyponatraemia hátterében álló endokrin eltérések
- Stressz és hormonok
- Steroid szeptikus shockban és/vagy pneumoniában?

bajnok.laszlo@pte.hu

Recommendations for the diagnosis and management of corticosteroid insufficiency in critically ill adult patients: Consensus statements from an international task force by the American College of Critical Care Medicine

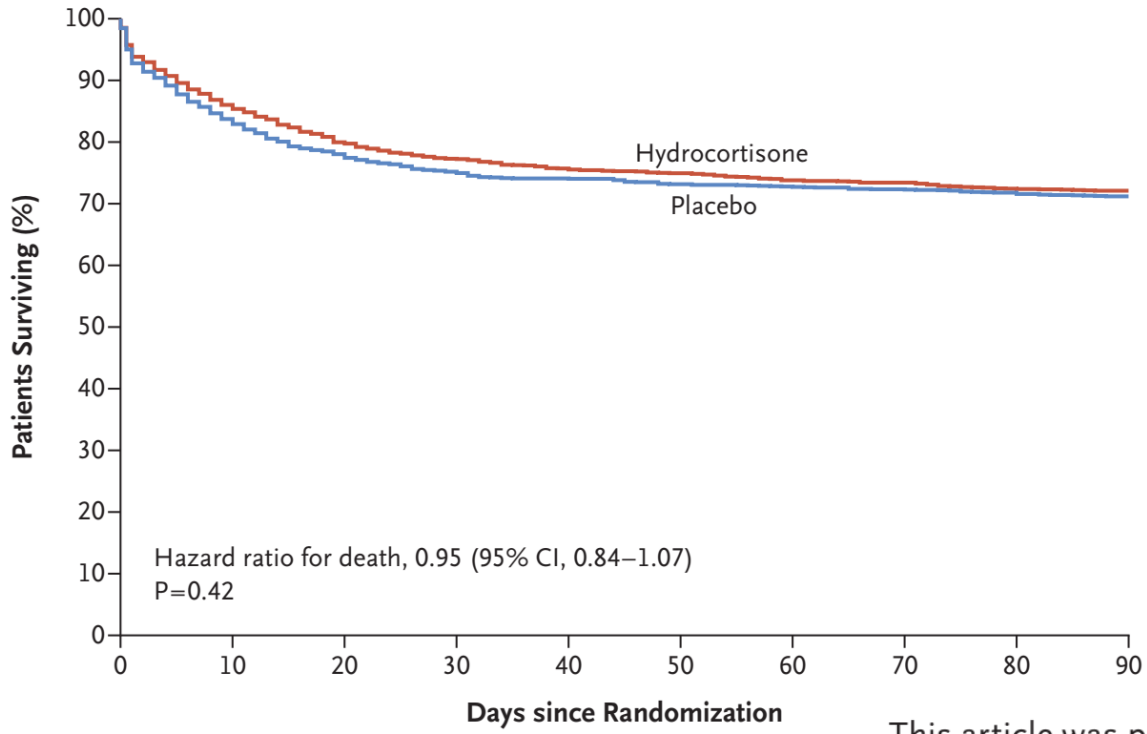
Paul E. Marik, MD, FCCM; Stephen M. Pastores, MD, FCCM; Djillali Annane, MD; G. Umberto Meduri, MD; Charles L. Sprung, MD, FCCM; Wiebke Arlt, MD; Didier Keh, MD; Josef Briegel, MD; Albertus Beishuizen, MD; Ioanna Dimopoulou, MD; Stylianos Tsagarakis, MD, PhD; Mervyn Singer, MD; George P. Chrousos, MD; Gary Zaloga, MD, FCCM; Faran Bokhari, MD, FACS; Michael Vogeser, MD

adrenocorticotrophic hormone stimulation test should not be used to identify those patients with septic shock or acute respiratory distress syndrome who should receive glucocorticoids. Hydrocortisone in a dose of 200 mg/day in four divided

(Crit Care Med 2008; 36:1937–1949)

Adjunctive Glucocorticoid Therapy in Patients with Septic Shock

B. Venkatesh, S. Finfer, J. Cohen, D. Rajbhandari, Y. Arabi, R. Bellomo, L. Billot, M. Correa, P. Glass, M. Harward, C. Joyce, Q. Li, C. McArthur, A. Perner, A. Rhodes, K. Thompson, S. Webb, and J. Myburgh, for the ADRENAL Trial Investigators and the Australian–New Zealand Intensive Care Society Clinical Trials Group*

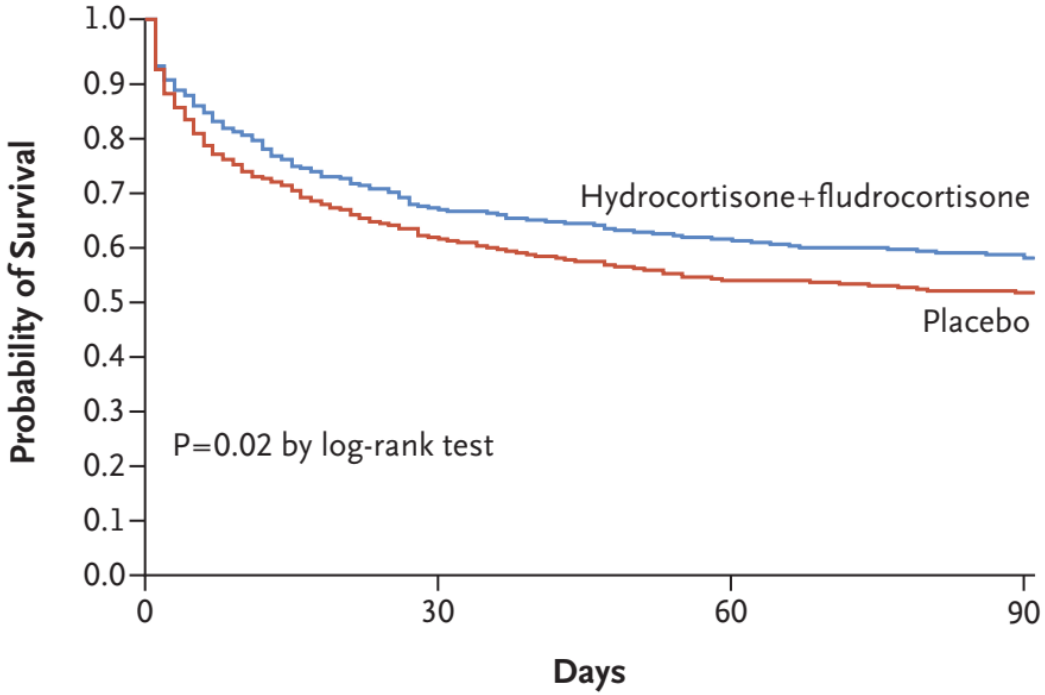


This article was published on January 19, 2018, at NEJM.org.

DOI: 10.1056/NEJMoa1705835

Hydrocortisone plus Fludrocortisone for Adults with Septic Shock

D. Annane, A. Renault, C. Brun-Buisson, B. Megarbane, J.-P. Quenot, S. Siami, A. Cariou, X. Forceville, C. Schwebel, C. Martin, J.-F. Timsit, B. Misset, M. Ali Benali, G. Colin, B. Souweine, K. Asehnoune, E. Mercier, L. Chimot, C. Charpentier, B. François, T. Boulain, F. Petitpas, J.-M. Constantin, G. Dhonneur, F. Baudin, A. Combes, J. Bohé, J.-F. Loriferne, R. Amathieu, F. Cook, M. Slama, O. Leroy, G. Capellier, A. Dargent, T. Hissem, V. Maxime, and E. Bellissant, for the CRICS-TRIGGERSEP Network*



*A complete list of investigators in the APROCCHSS trial is provided in the Supplementary Appendix, available at NEJM.org.

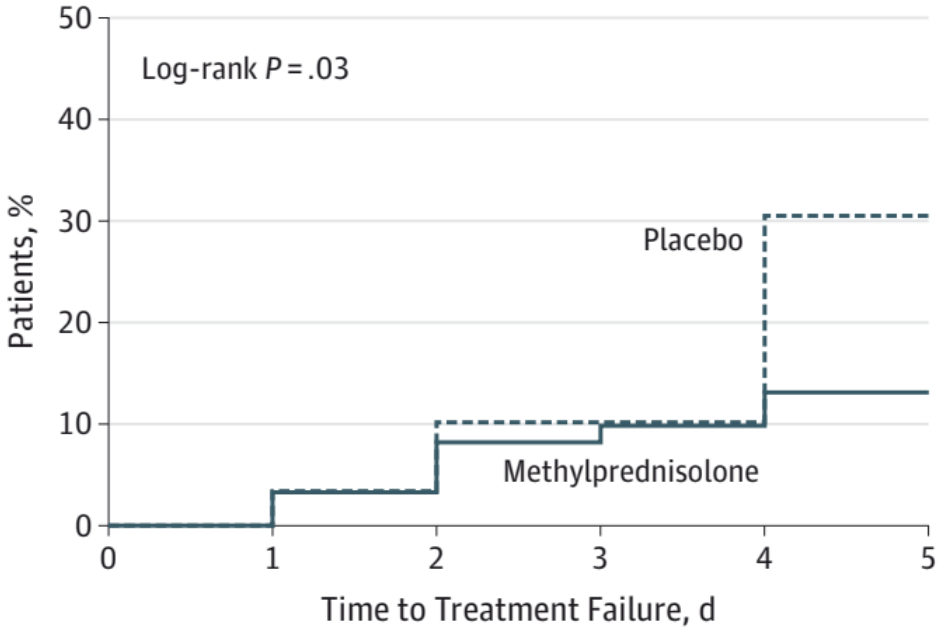
N Engl J Med 2018;378:809-18.
DOI: 10.1056/NEJMoa1705716

Effect of Corticosteroids on Treatment Failure Among Hospitalized Patients With Severe Community-Acquired Pneumonia and High Inflammatory Response

A Randomized Clinical Trial

Antoni Torres, MD, PhD; Oriol Sibila, MD, PhD; Miquel Ferrer, MD, PhD; Eva Polverino, MD, PhD; Rosario Menendez, MD, PhD; Josep Mensa, MD, PhD; Albert Gabarrús, MSc; Jacobo Sellarés, MD, PhD; Marcos I. Restrepo, MD, MSc; Antonio Anzueto, MD, PhD; Michael S. Niederman, MD; Carles Agustí, MD, PhD

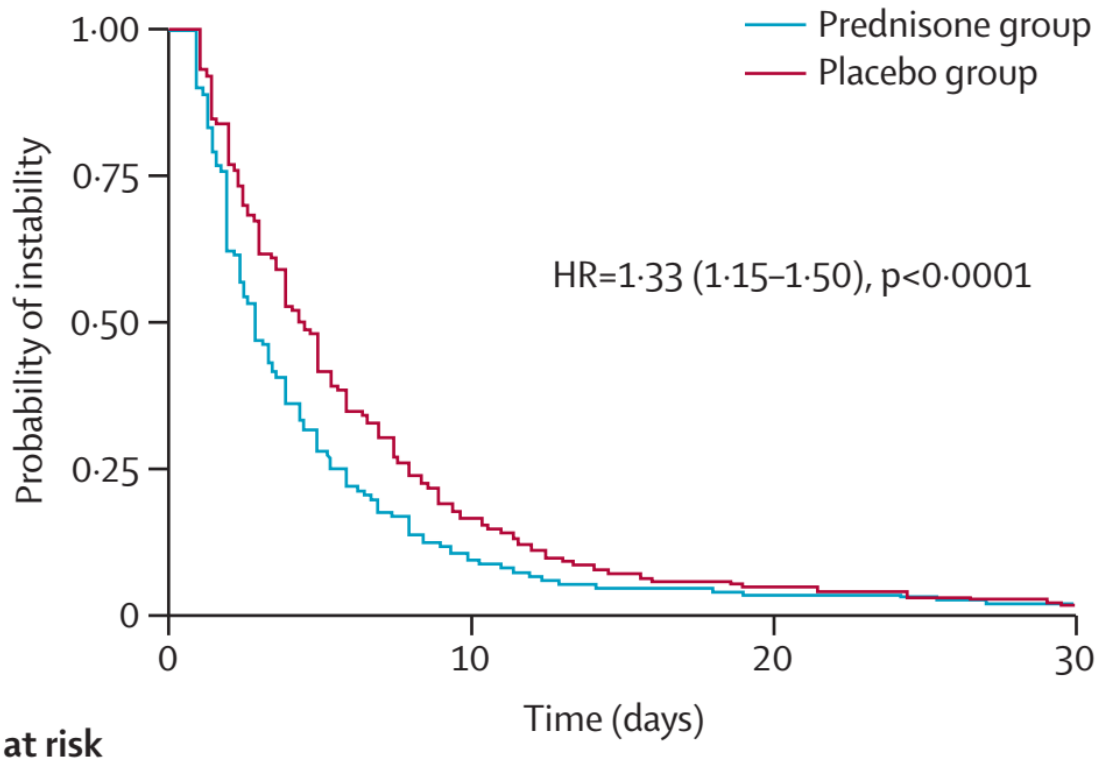
Figure 2. Kaplan-Meier Analysis of the Effect of Methylprednisolone on Time to Treatment Failure



JAMA. 2015;313(7):677-686.

Adjunct prednisone therapy for patients with community-acquired pneumonia: a multicentre, double-blind, randomised, placebo-controlled trial

Claudine Angela Blum, Nicole Nigro*, Matthias Briel, Philipp Schuetz, Elke Ullmer, Isabelle Suter-Widmer, Bettina Winzeler, Roland Bingisser, Hanno Elsaesser, Daniel Drozdov, Birsen Arici, Sandrine Andrea Urwyler, Julie Refardt, Philip Tarr, Sebastian Wirz, Robert Thomann, Christine Baumgartner, Hervé Duplain, Dieter Burki, Werner Zimmerli, Nicolas Rodondi, Beat Mueller, Mirjam Christ-Crain*



Number at risk

Prednisone group	392	37	12	6
Placebo group	393	63	16	5

Lancet 2015; 385: 1511–18

Vázlat

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- Stressz és hormonok
- Steroid szeptikus shockban és/vagy pneumoniában?
- Stressz, endokrin eltérések, prognózis

bajnok.laszlo@pte.hu

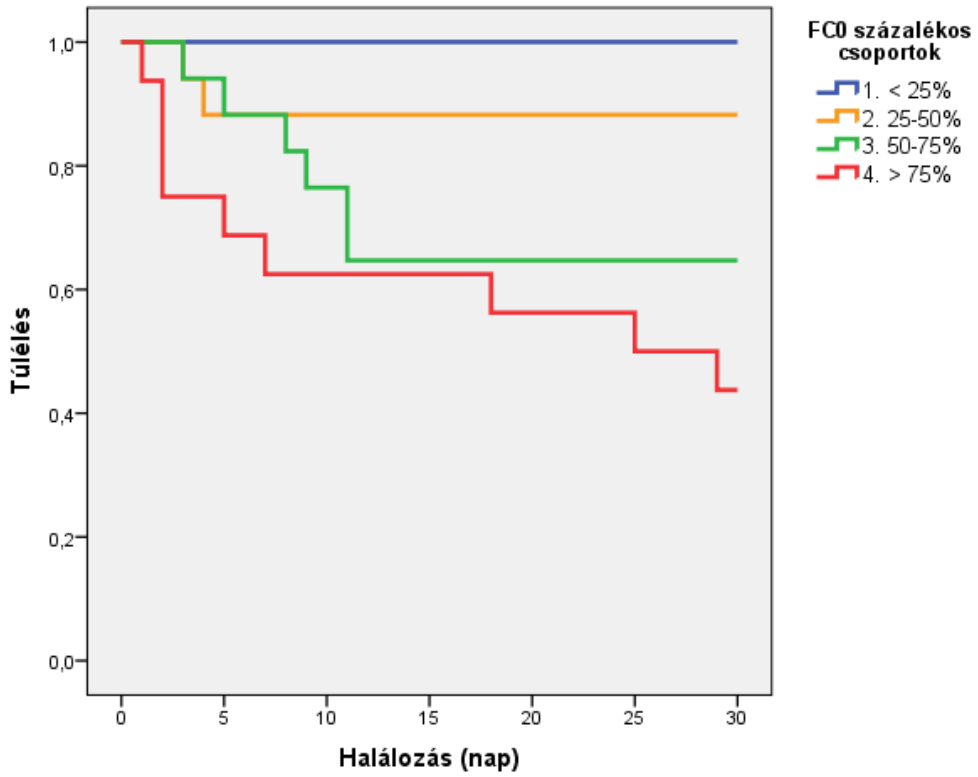
Free and total cortisol levels are useful prognostic markers in critically ill patients: a prospective observational study

Zita Tarjányi[1,2], Gergely Montskó[2,3], Péter Kenyeres[1], Zsolt Márton[1], Roland Hágendorn[1], Erna Gulyás[1], Orsolya Nemes[1], László Bajnok[1], Gábor L Kovács[2,3] and **Emese Mezősi[1]**

[1]First Department of Internal Medicine, Faculty of Medicine, University of Pécs, 13 Ifjúság, Pécs H-7624, Hungary, [2]Department of Laboratory Medicine, Faculty of Medicine, [3]Szentágothai Research Centre, University of Pécs, Pécs, Hungary

Correspondence should be addressed to E Mezősi
Email
emese.mezosi@aok.pte.hu

A túlélés Kaplan-Meier görbéi a szabad kortizol függvényében



European Journal of Endocrinology
(2014) **171**, 751–759

Az állapot súlyossága, klinikai adatok

APACHE II

12 változó:
- Testhőmérséklet
- Artériás középnyomás
- Szívfrekvencia
- GCS
- Légzésszám
- Fvs szám
- Hematokrit
- Kreatinin
- K
- Na
- pH
- PaO2/FiO2

Kor

Szervelégtelenség, immunszupr.

SAPS II

Kor

Felvétel oka: nem sebészi, sürgős/elektív műtét

Testhőmérséklet

Szisztolés vérnyomás

Szívfrekvencia

GCS

Diurézis

Fvs szám

Urea

K

Na

Bikarbonát

Bi

PaO2/FiO2

AIDS

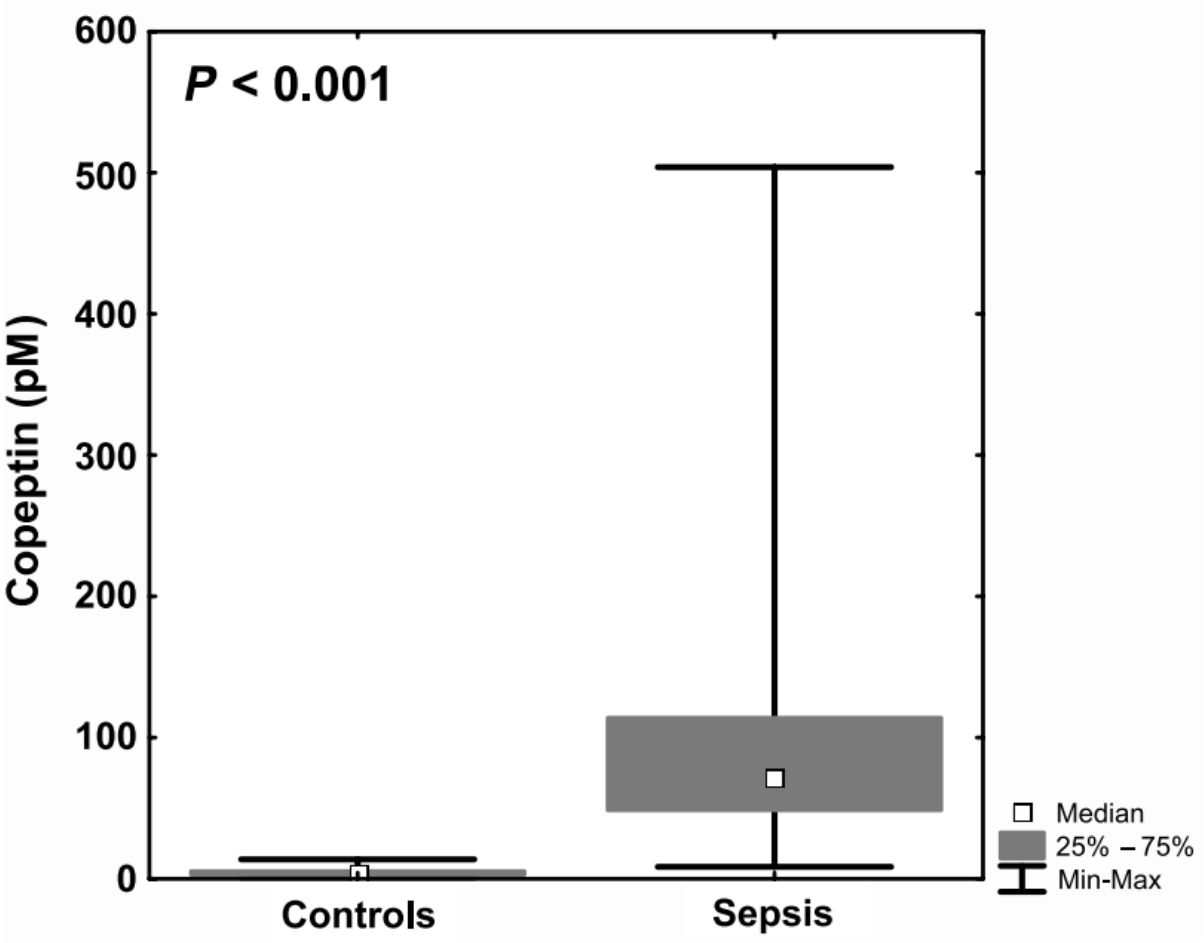
Daganat áttét

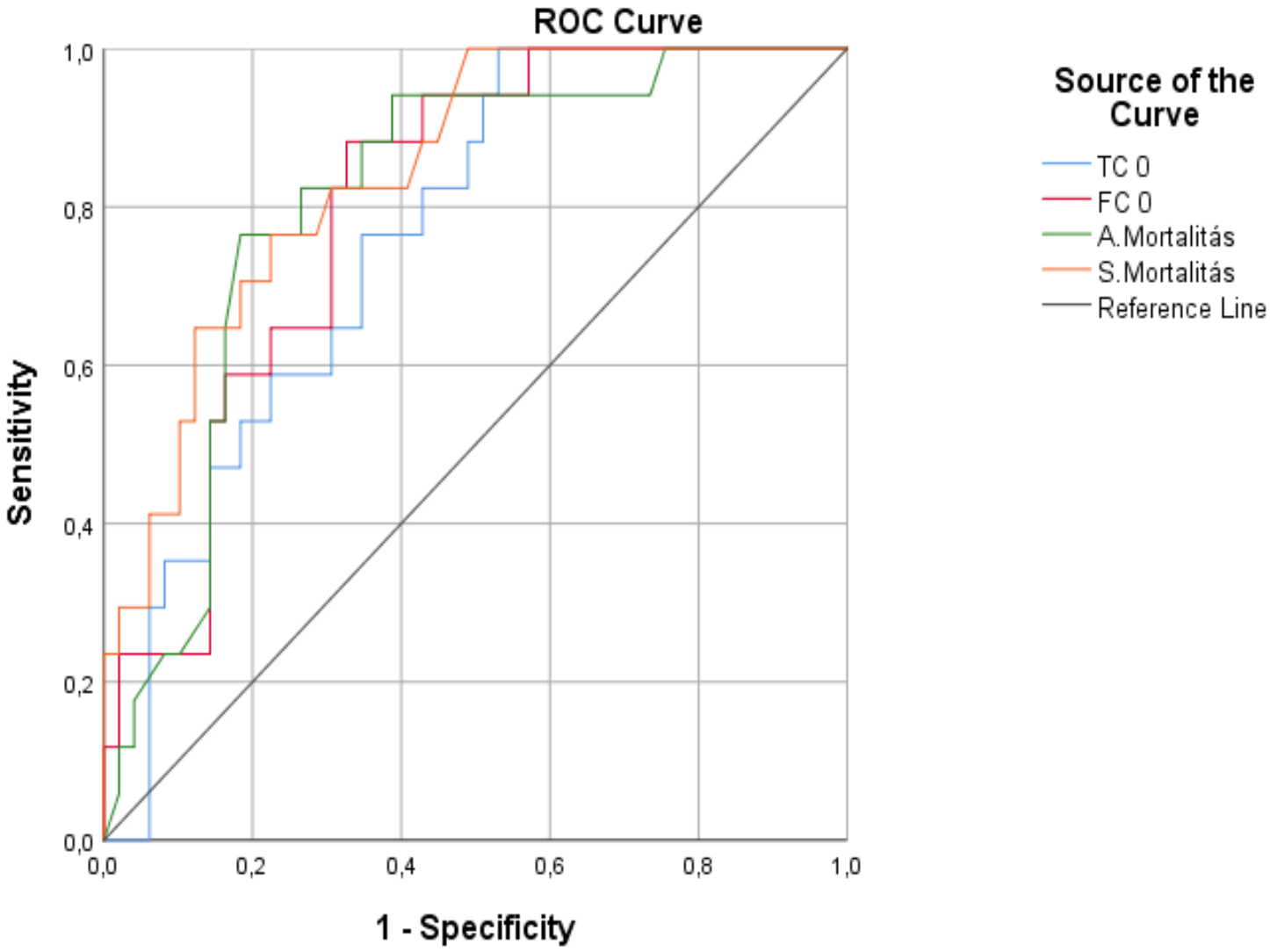
Hematológiai malignitás

SHOCK, Vol. 28, No. 2, pp. 219–226, 2007

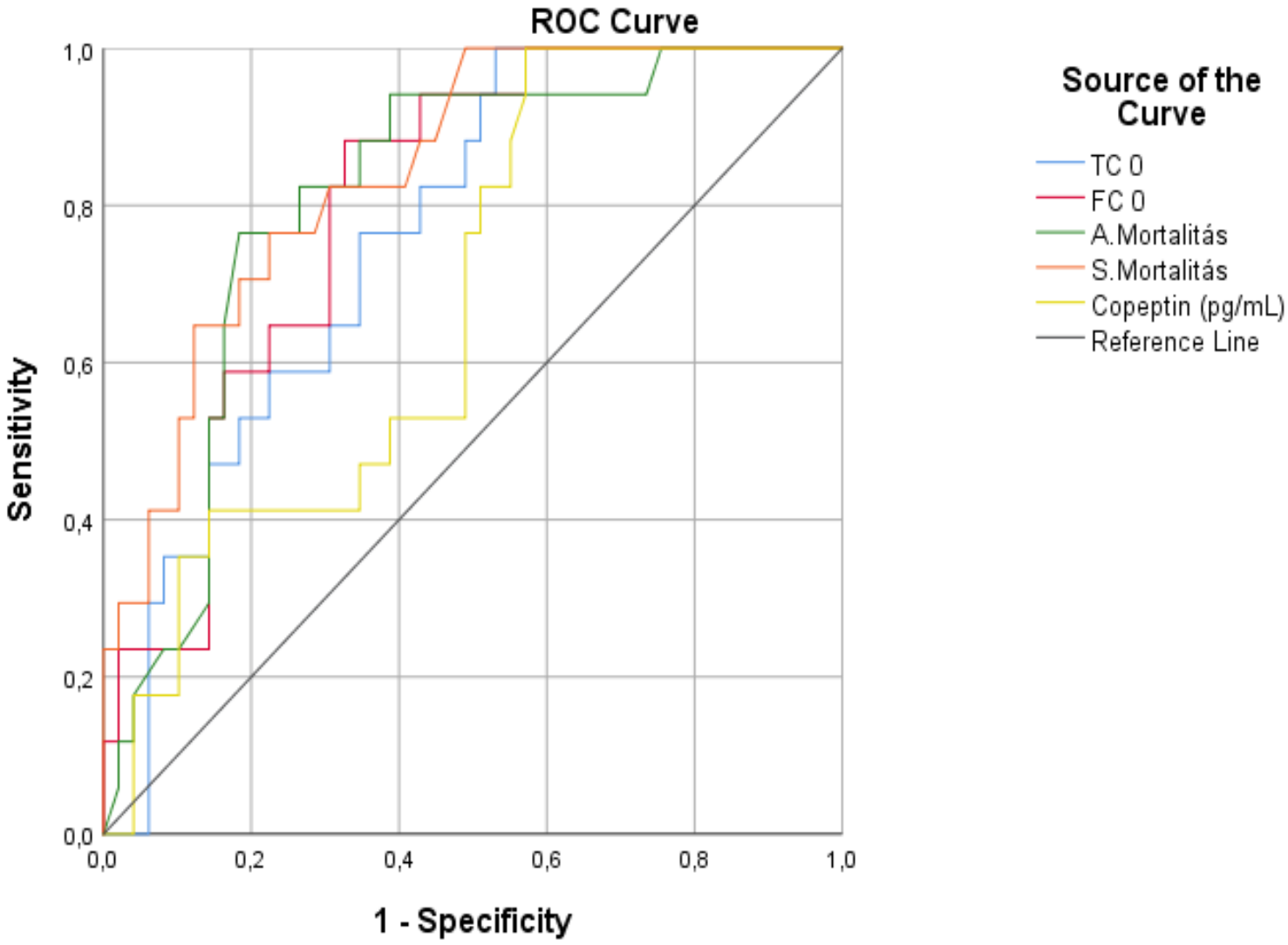
COPEPTIN, A STABLE PEPTIDE OF THE ARGININE VASOPRESSIN PRECURSOR, IS ELEVATED IN HEMORRHAGIC AND SEPTIC SHOCK

Nils G. Morgenthaler,* Beat Müller,† Joachim Struck,* Andreas Bergmann,* Heinz Redl,‡ and Mirjam Christ-Crain†





Diagonal segments are produced by ties.



Diagonal segments are produced by ties.

Vázlat

- A hyponatraemia okai
- A hyponatraemia a napi gyakorlatban
- A hyponatraemia ellátása
- A hyponatraemia hátterében álló endokrin eltérések
- Stressz és hormonok
- Steroid szeptikus shockban és/vagy pneumoniában?
- Stressz, endokrin eltérések, prognózis
- A CNET regiszter

bajnok.laszlo@pte.hu

TM Regiszterek
a jobb betegellátásért

Alapítvány | Központok | Munkacsoportok | Metaanalízis | Regiszterek | Vizsgálatok | A
Gasztroenterológia | Kardiológia | Neurológia | Endokrinológia | Traumatológia | Regisz

Akromegália
CNET
Belépés
Bevezetés
Űrlapok
Protokoll
Használati útmutató
Engedélyek
Kiegészítő tartalmak

CNET Regis

Kapcsolodó tart

FORM
CNET A adatlap
CNET B adatlap

Tetszik Megosztás

Tetszik Megosztás

Személyes adatok

TAJ szám:.....
Név:.....
Születési dátum:.....
A beteginterjú dátuma: ..... (csak dátum)

Kórház:

Orvos kód:

Radioterápia

Régió (többválasztós):

- mellkas
- agy (ha igen: Profilaktikus? igen / nem)
- csont
- egyéb:.....

(mindegyik mellé, ami igen) Dátum: (csak dátum)

Dózis: ..... mértékegység: Gy

Kísérő kemoterápia: igen / nem

ha igen: Kérem, töltse ki a C űrlapot.

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CLINICAL RESEARCH www.jasn.org

J Am Soc Nephrol 19: 1015–1024, 2008.

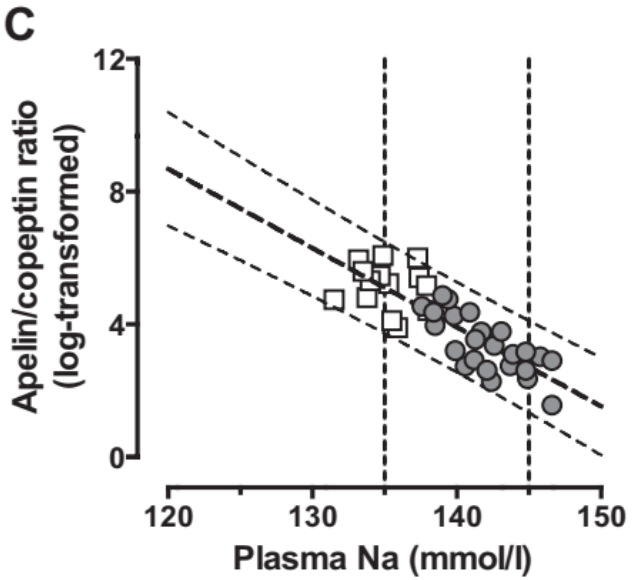
Reciprocal Regulation of Plasma Apelin and Vasopressin by Osmotic Stimuli

Michel Azizi,* Xavier Iturrioz,† Anne Blanchard,* Séverine Peyrard,* Nadia De Mota,† Nicolas Chartrel,‡ Hubert Vaudry,‡ Pierre Corvol,§ and Catherine Llorens-Cortes†

An Abnormal Apelin/Vasopressin Balance May Contribute to Water Retention in Patients With the Syndrome of Inappropriate Antidiuretic Hormone (SIADH) and Heart Failure

Anne Blanchard,* Olivier Steichen, Nadia De Mota, Emmanuel Curis, Cedric Gauci, Michael Frank, Grégoire Wuerzner, Peter Kamenicky, Amélie Passeron, Michel Azizi, and Catherine Llorens-Cortes

(*J Clin Endocrinol Metab* 98: 2084–2089, 2013)



Összegzés

- Romló általános állapot esetén gondoljunk hyponatraemiára!
- A háttérben rejtett gyógyszerhatás is lehet
- A hyponatraemia általában rendellenes ADH hatás következménye
- Ilyenkor az ADH szükséges, de nem elegendő feltétel
- Hyponatraemiában a folyadék-megszorítás általánosan indokolt, legalább átmenetileg
- A 10 mmol/l/24h-nál gyorsabb nátrium korrekció veszélye az osmotikus demyelinisatio

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