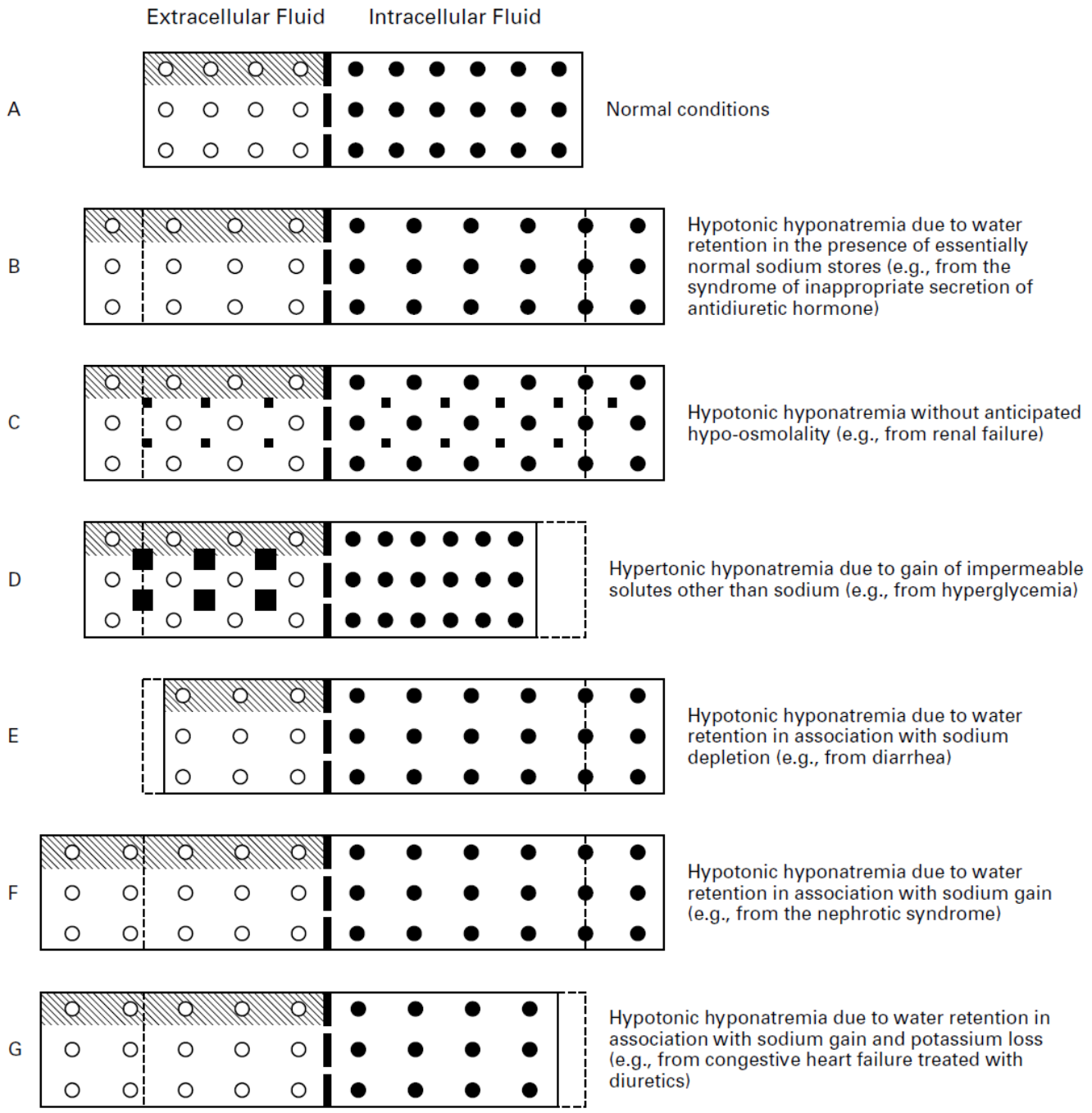
Υπονατριαιμική εγκεφαλοπάθεια

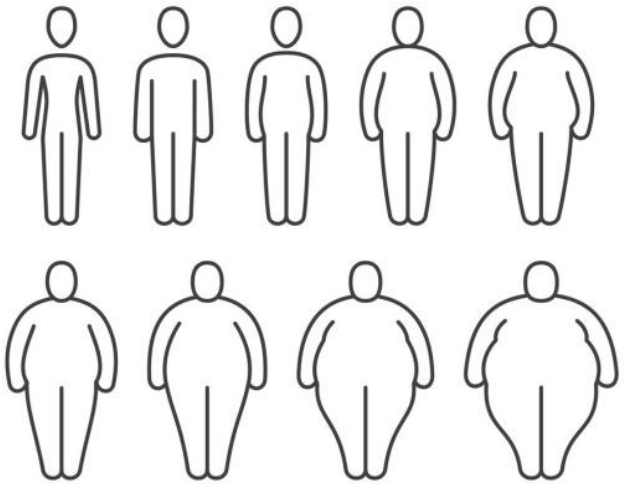
27° Πανελλήνιο Συνέδριο Νεφρολογίας

Χρήστος Πλέρος
Νεφρολόγος
ΠΑΓΝΗ

NO

Υπονατριαιμία - Ορισμός





V = ?

Adrogue' & Madias, NEJM 2000

2005 – 2018

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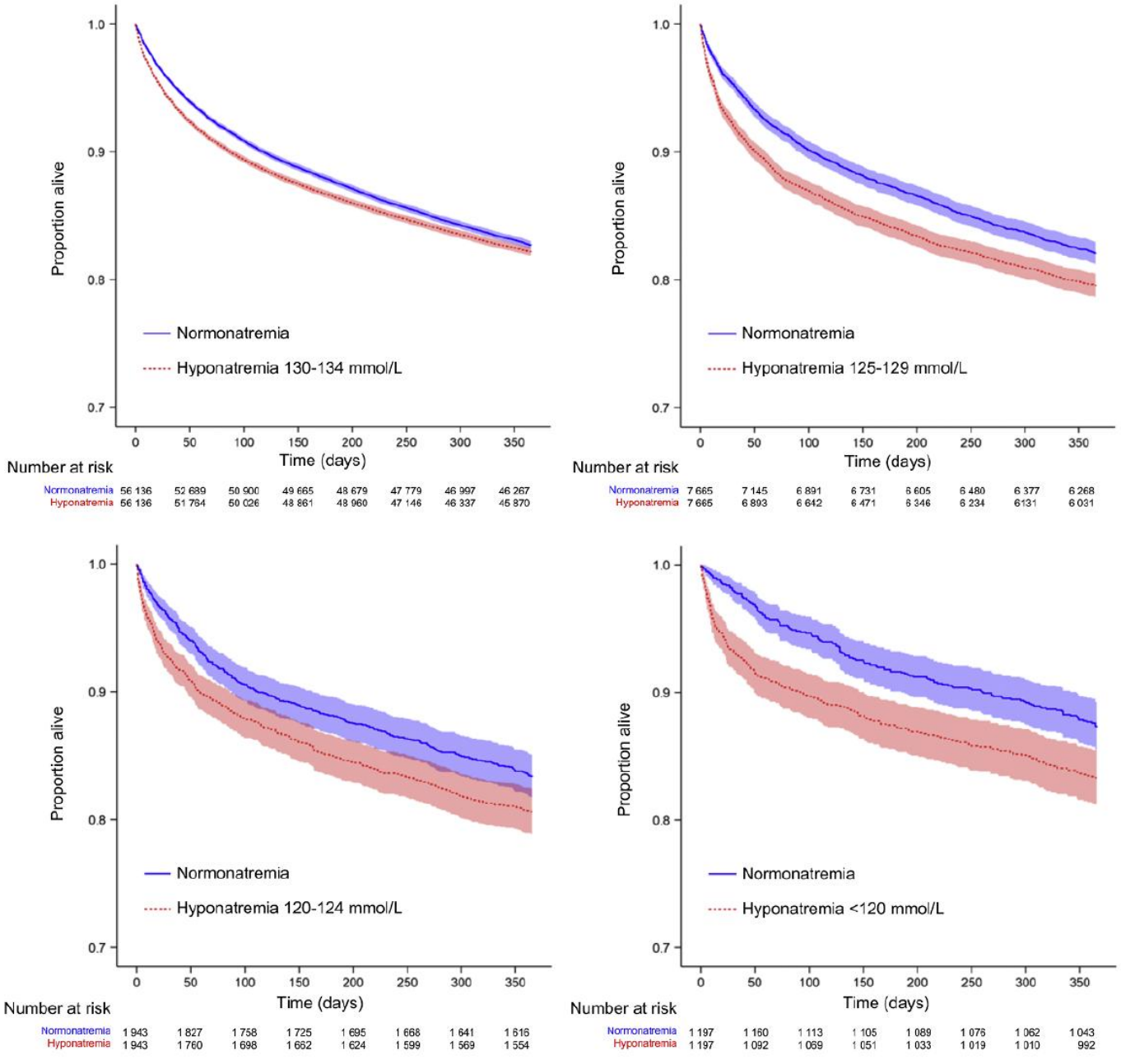
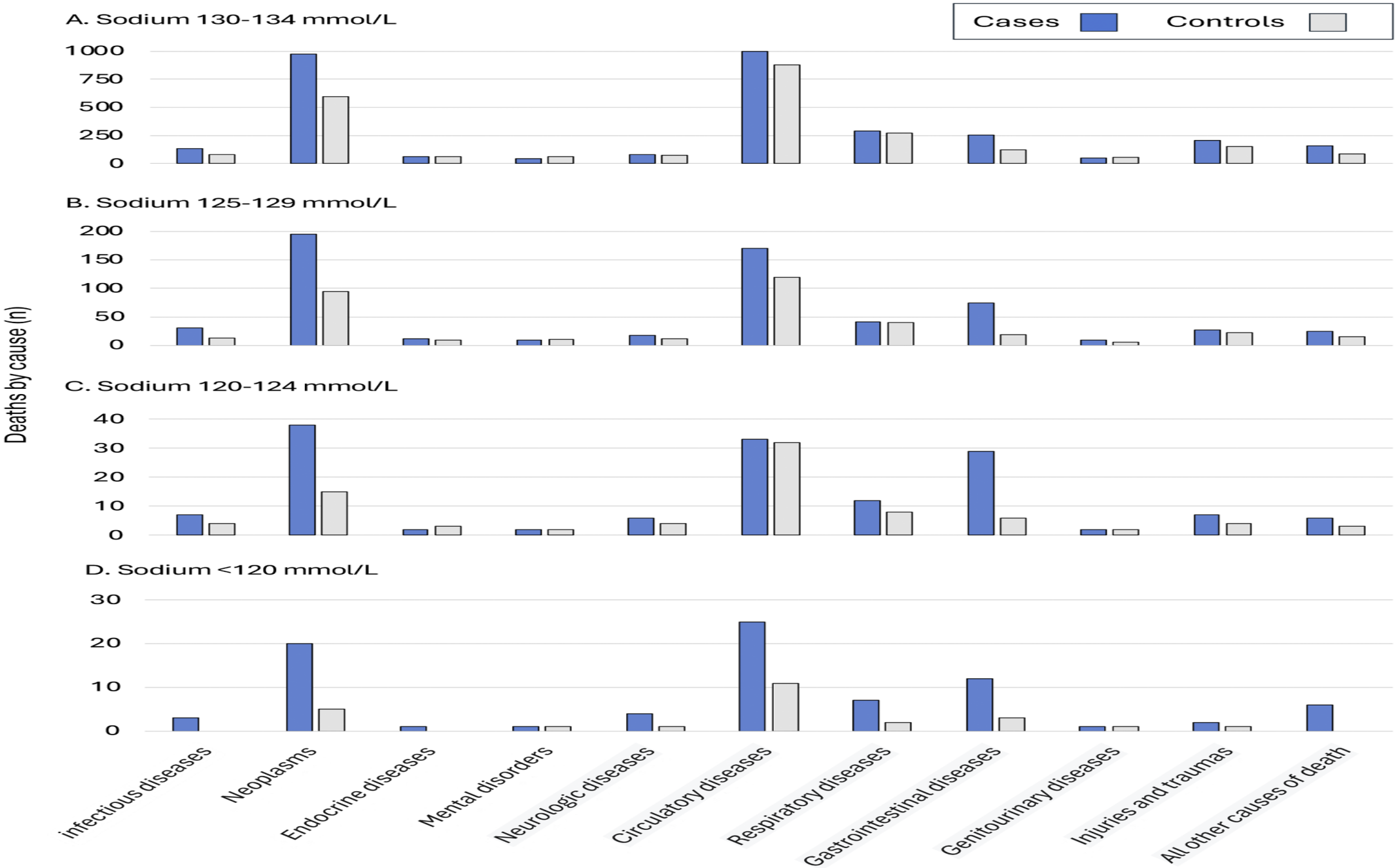
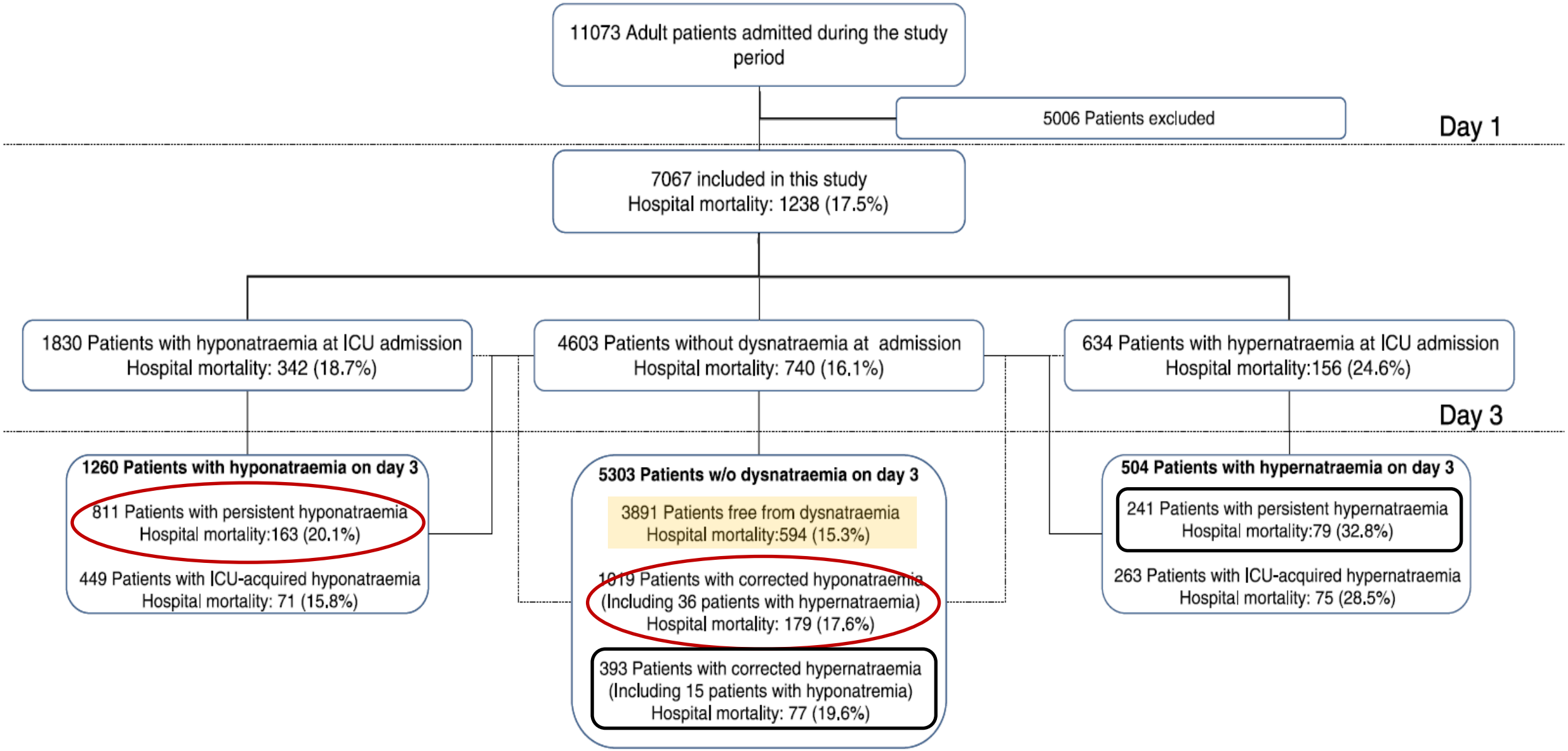


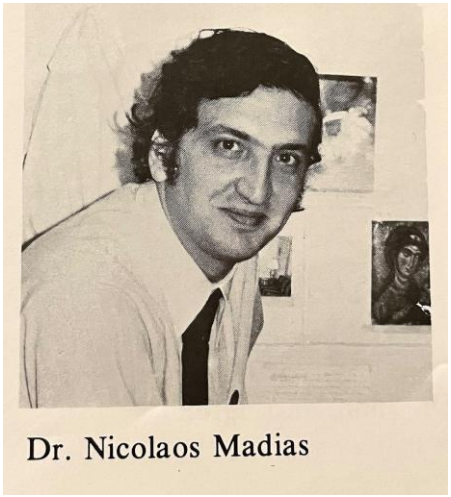
Mannheimer et al. Eur J Intern Med 2025



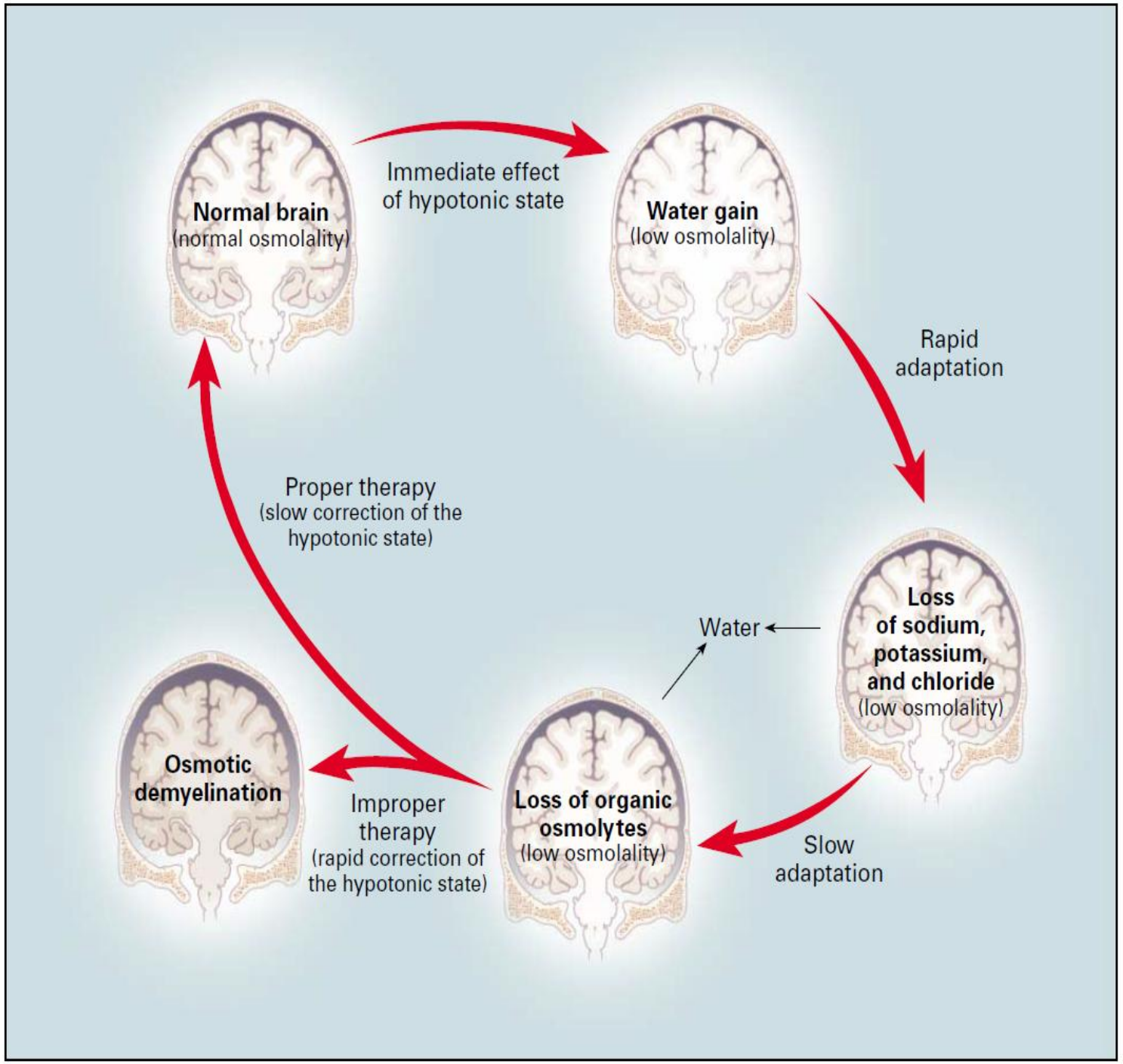
Mannheimer et al. Eur J Intern Med 2025



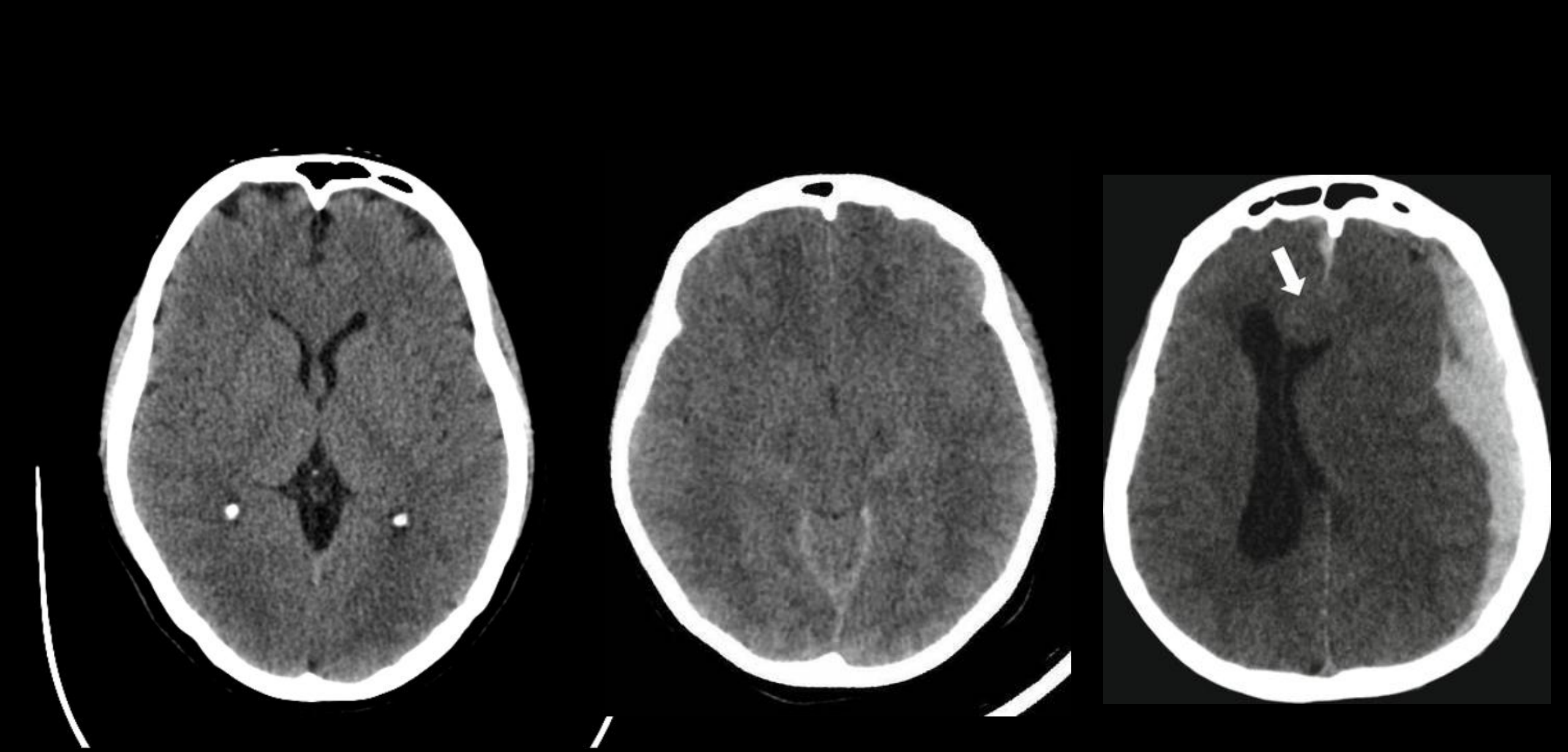
Darmon et al, Shock 2014

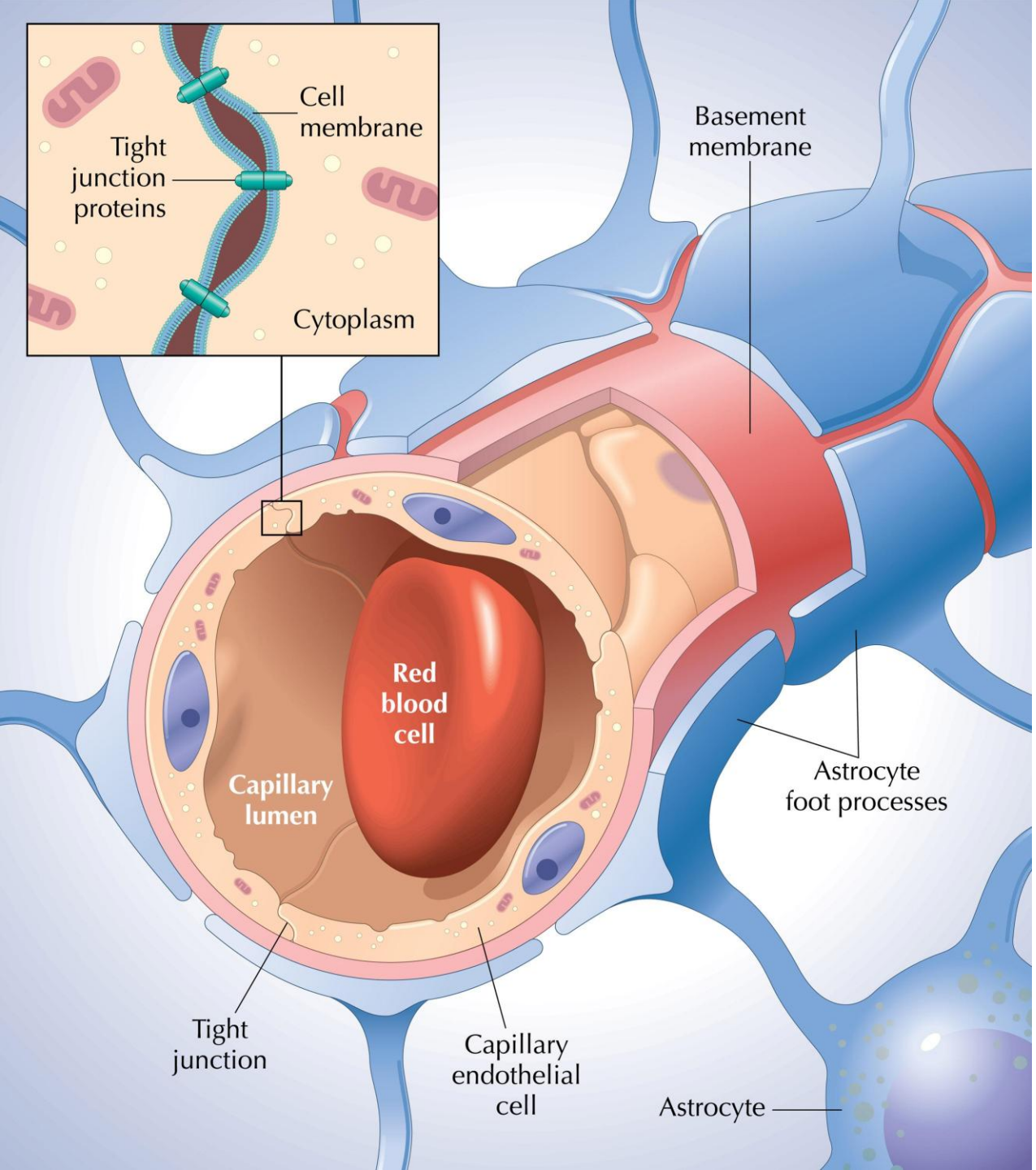


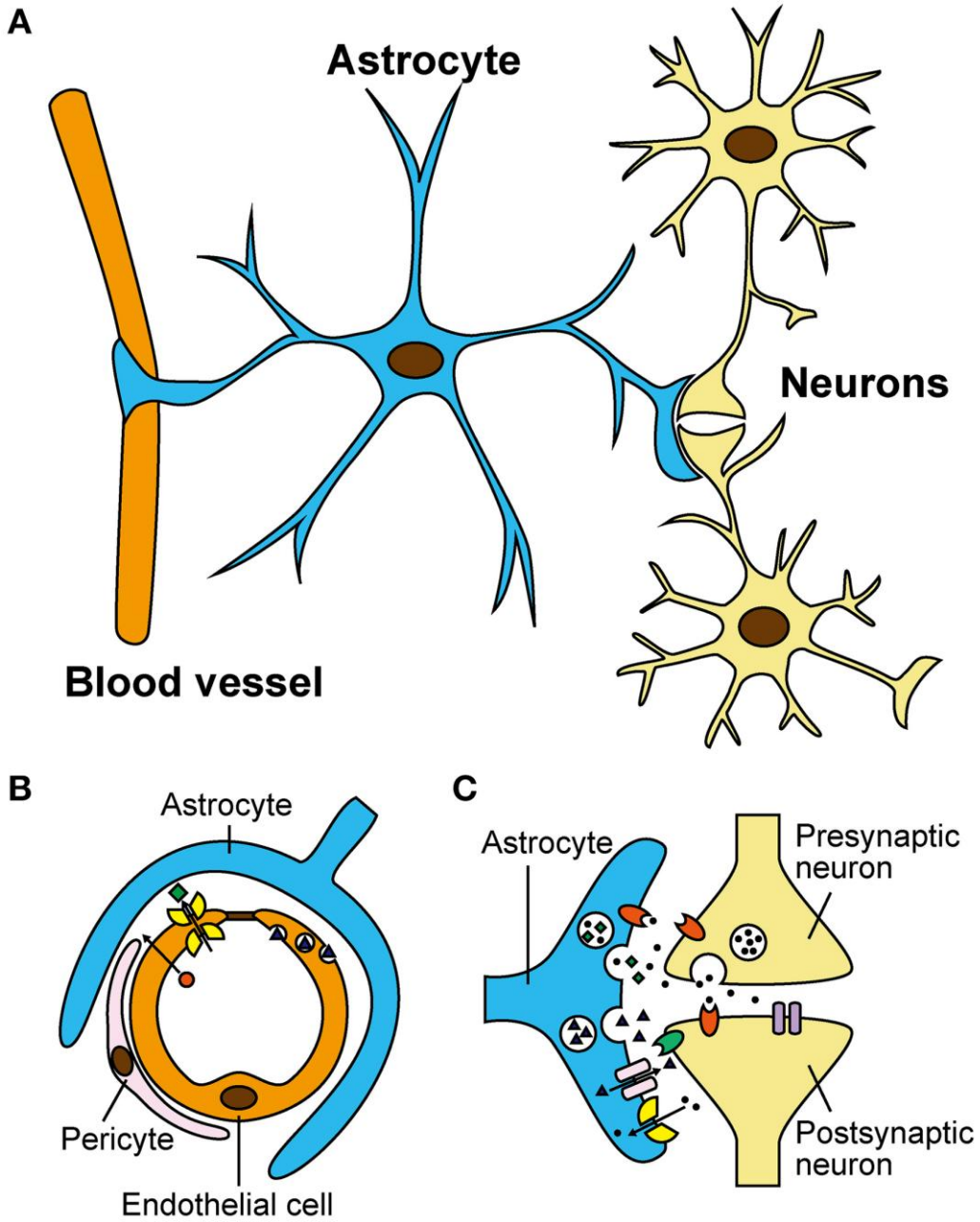
Dr. Nicolaos Madias



Adrogue & Madias, N Engl J Med 2000







Harada et al. Front Neurosci 2016

Alexander MONRO (secundus)

1733 – 1817

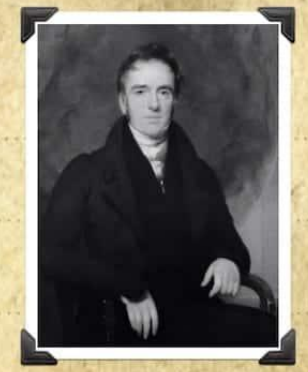
Monro-Kellie hypothesis

Foramen of Monro

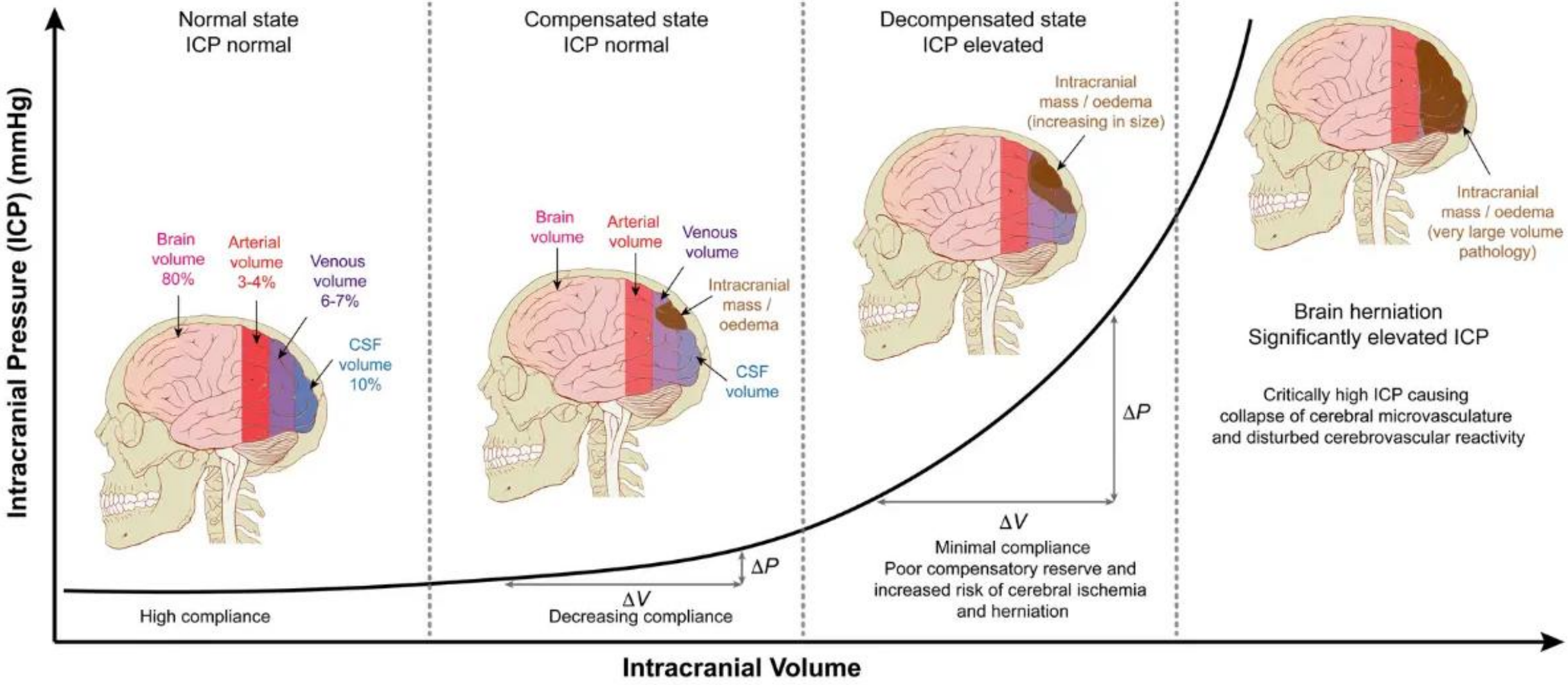
V = Tissue + Blood + CSF

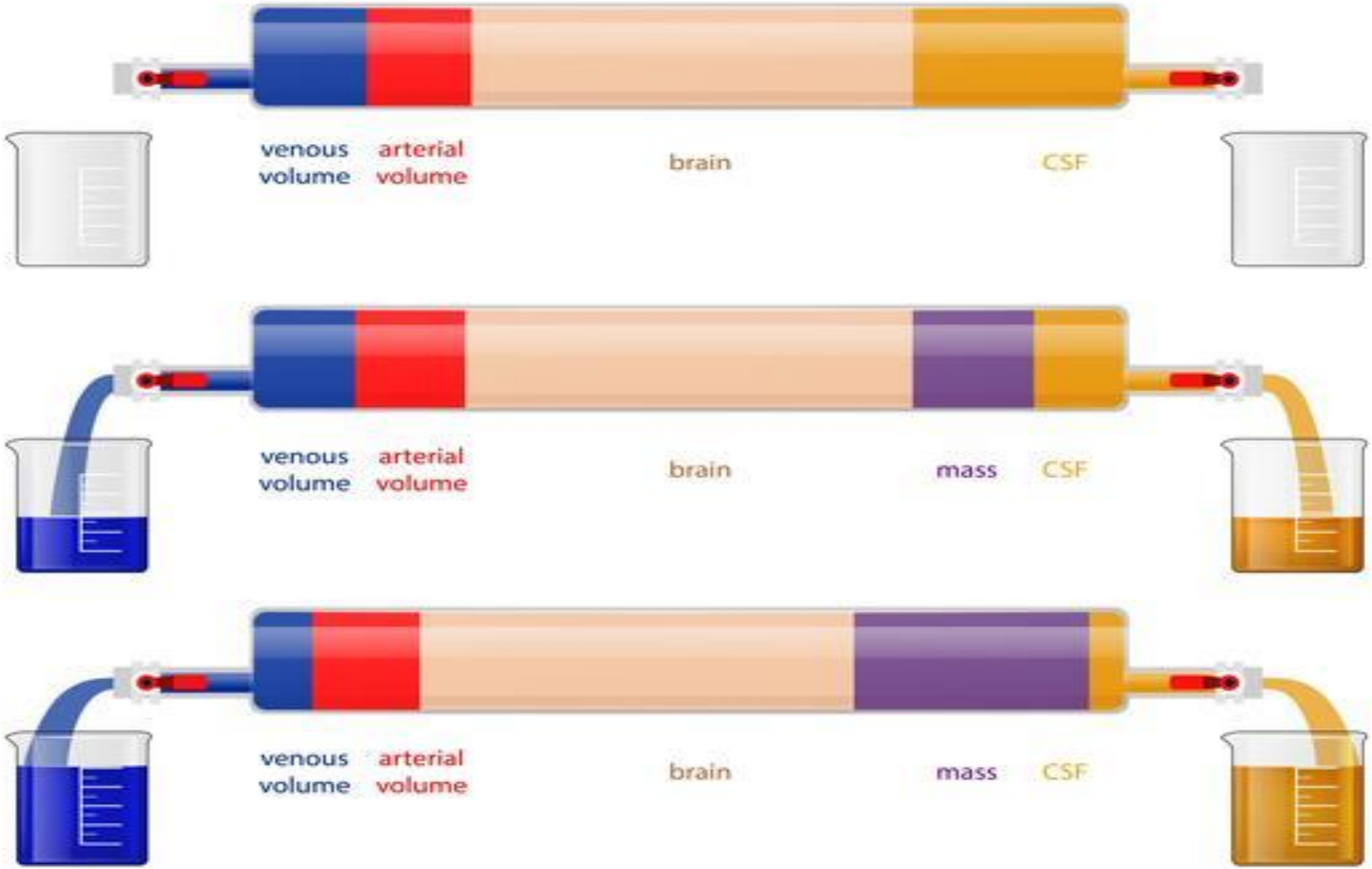
George KELLIE

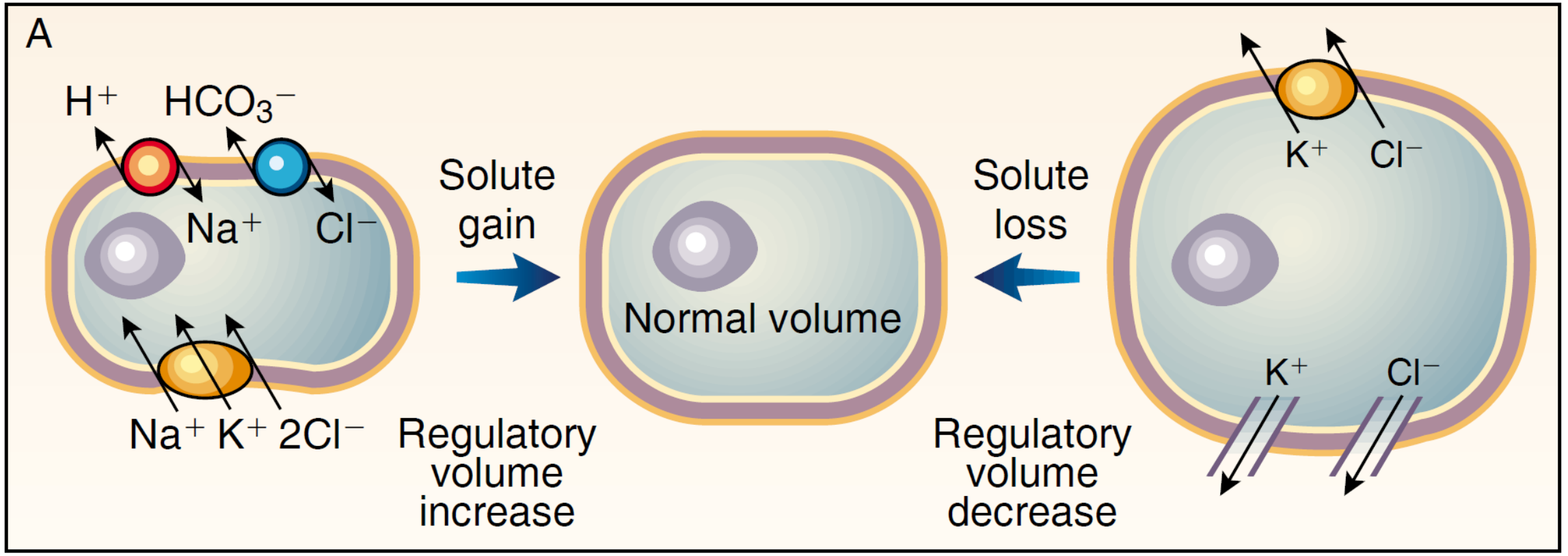
1770 – 1829



Monro-Kellie hypothesis


Normal state
ICP normal
Compensated state
ICP normal
Decompensated state
ICP elevated
Brain herniation
Significantly elevated ICP
Intracranial Pressure (ICP) (mmHg)
Intracranial Volume
Brain volume 80%
Arterial volume 3-4%
Venous volume 6-7%
CSF volume 10%
Brain volume
Arterial volume
Venous volume
Intracranial mass / oedema
CSF volume
Intracranial mass / oedema (increasing in size)
Intracranial mass / oedema (very large volume pathology)
ΔP
ΔV
High compliance
Decreasing compliance
Minimal compliance
Poor compensatory reserve and increased risk of cerebral ischemia and herniation
Critically high ICP causing collapse of cerebral microvasculature and disturbed cerebrovascular reactivity


venous volume
arterial volume
brain
CSF
venous volume
arterial volume
brain
mass
CSF
venous volume
arterial volume
brain
mass
CSF



McManus et al. N Engl J Med 1995

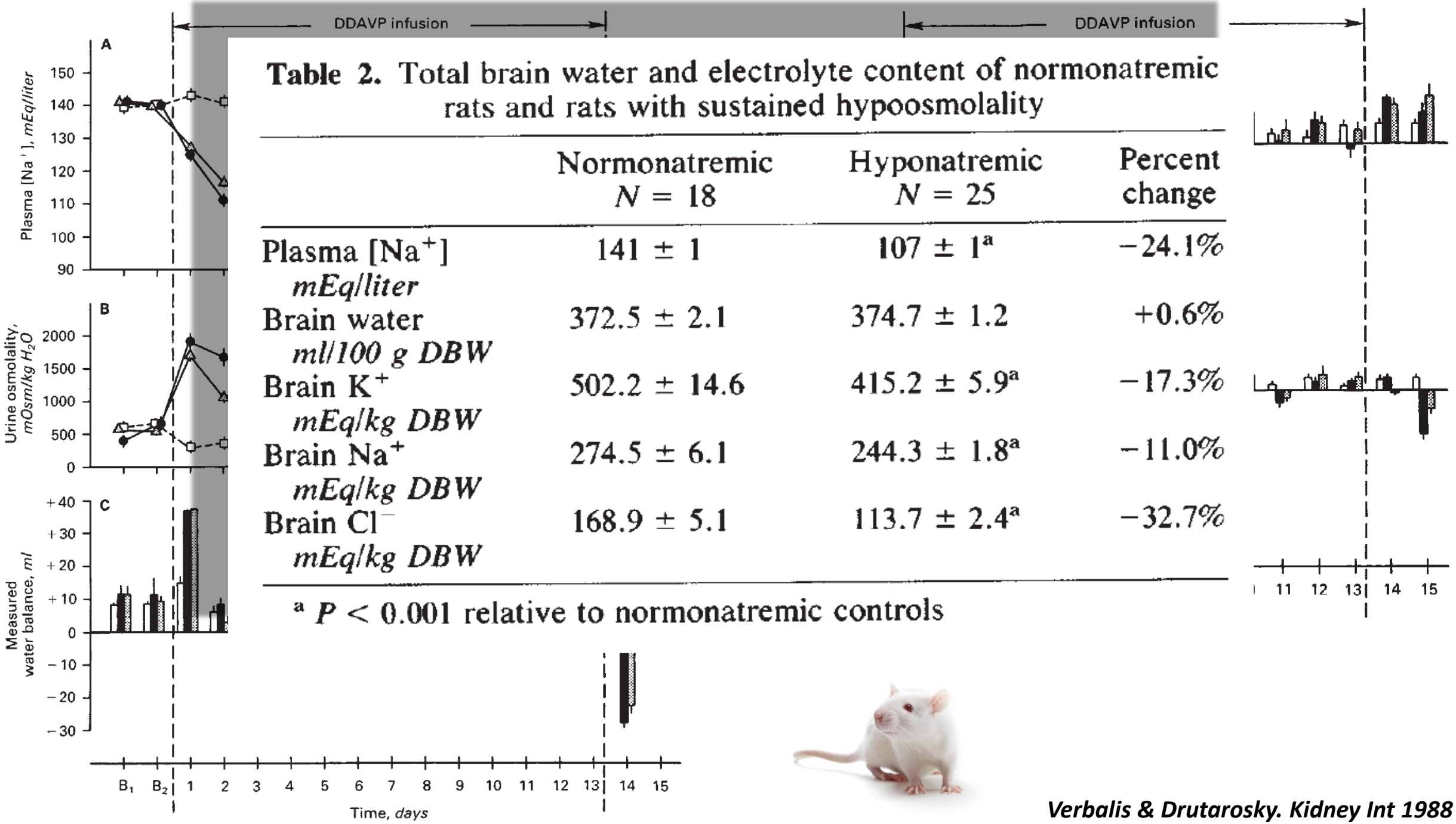


Table 2. Total brain water and electrolyte content of normonatremic rats and rats with sustained hypoosmolality

	Normonatremic $N = 18$	Hyponatremic $N = 25$	Percent change
Plasma [Na^+] *mEq/liter*	141 ± 1	107 ± 1[a]	−24.1%
Brain water *ml/100 g DBW*	372.5 ± 2.1	374.7 ± 1.2	+0.6%
Brain K^+ *mEq/kg DBW*	502.2 ± 14.6	415.2 ± 5.9[a]	−17.3%
Brain Na^+ *mEq/kg DBW*	274.5 ± 6.1	244.3 ± 1.8[a]	−11.0%
Brain Cl^- *mEq/kg DBW*	168.9 ± 5.1	113.7 ± 2.4[a]	−32.7%

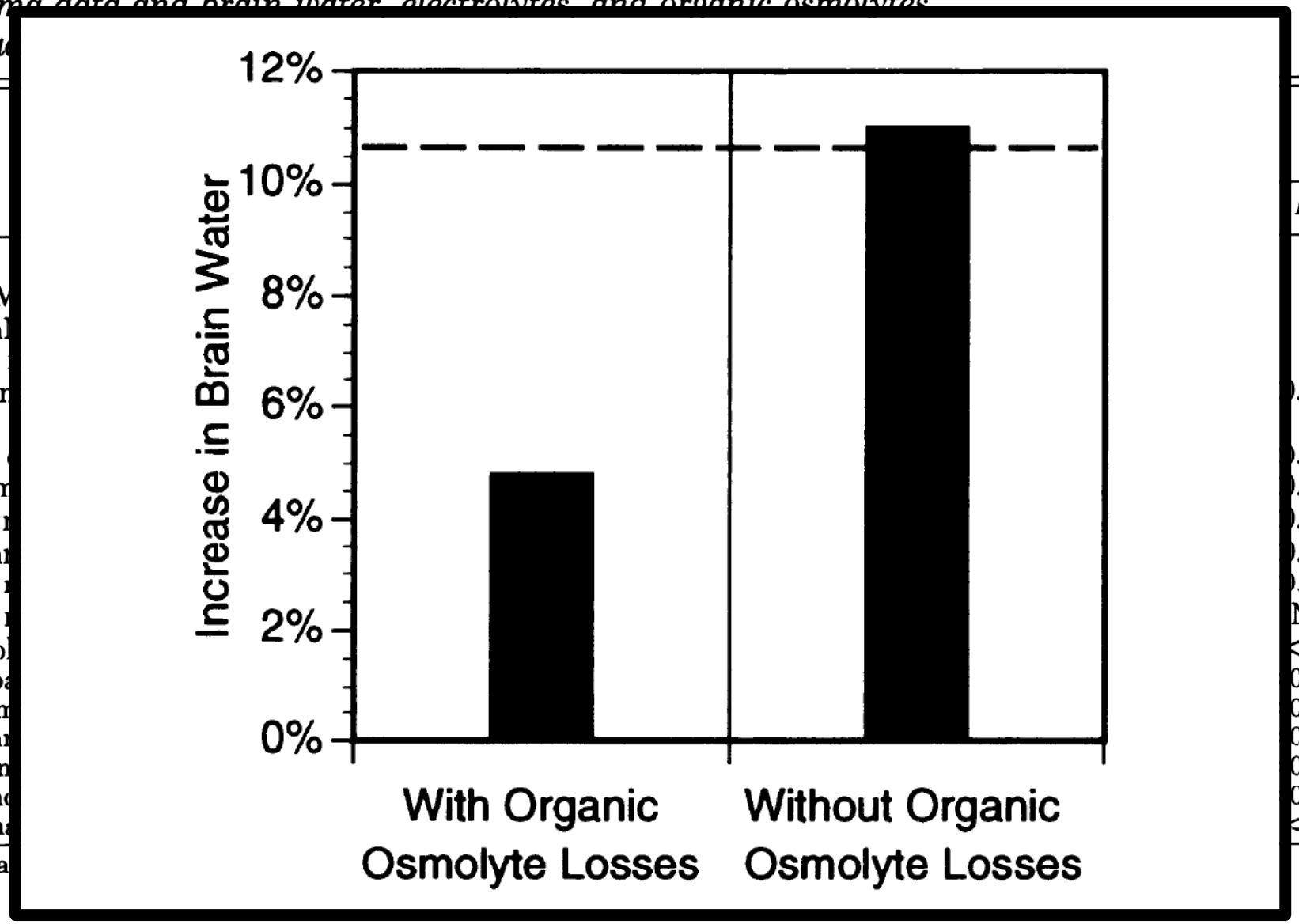
[a] $P < 0.001$ relative to normonatremic controls

Verbalis & Drutarosky. Kidney Int 1988

Table 1. *Plasma data and brain water, electrolytes, and organic osmolytes in animals ma*

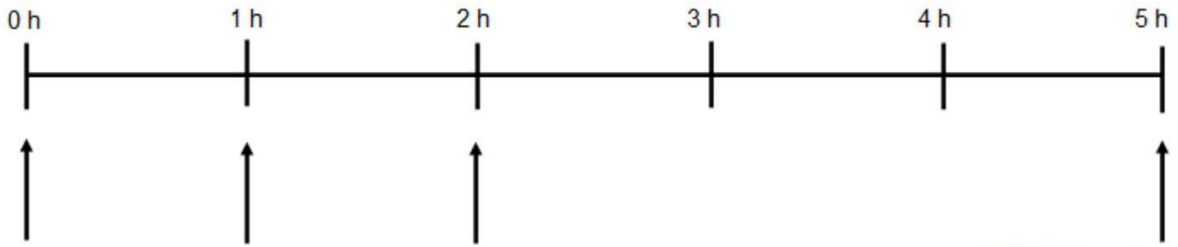
	Correlation With Plasma Sodium	
	P	*r*
Plasma		
Sodium, mM		
Chloride, m		
Osmolality,		
Weight char	.001	0.94
Brain		
Water, l/kg	.001	0.83
Sodium, mm	.001	0.98
Potassium,	.001	0.96
Chloride, m	.001	0.98
Glutamate,	.001	0.97
Glutamine,	NS	0.12
Myo-inositol	<0.04	0.49
N-acetylaspa	0.001	0.75
Aspartate, m	0.001	0.79
Creatine, m	0.001	0.92
Taurine, mn	0.006	0.63
GABA, mm	0.002	0.68
Phosphoeth	<0.05	0.63

Values are mea



Sterns et al. Am J Physiol 1993

A **Experimental protocol for acute hyponatremia studies**



Group 1 Sham injection

Group 2 dDAVP s.c. + water i.p. water i.p. dDAVP s.c. + water i.p.

Group 3 AVP s.c. + water i.p. water i.p. AVP s.c. + water i.p.

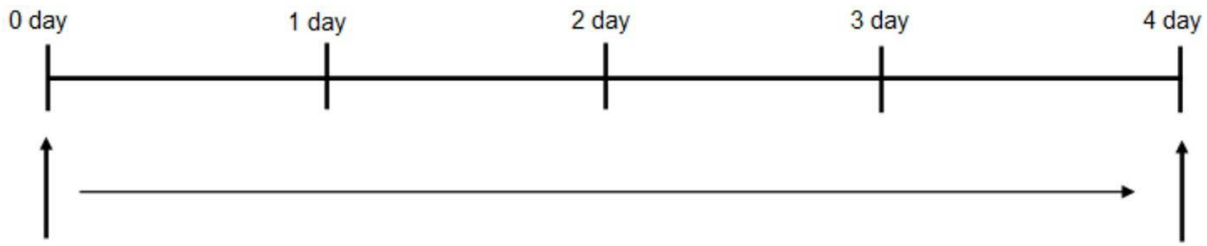
BBB determination (NaF), n=18

brain water content studies, n=18

tissue collection to RT-PCR, n=21

blood analyses, n=57

B **Experimental protocol for chronic hyponatremia studies**



Group 4 Sham standard rat chow + osmotic pump filled with saline

Group 5 liquid diet + osmotic pump filled with dDAVP

Group 6 liquid diet + osmotic pump filled with AVP

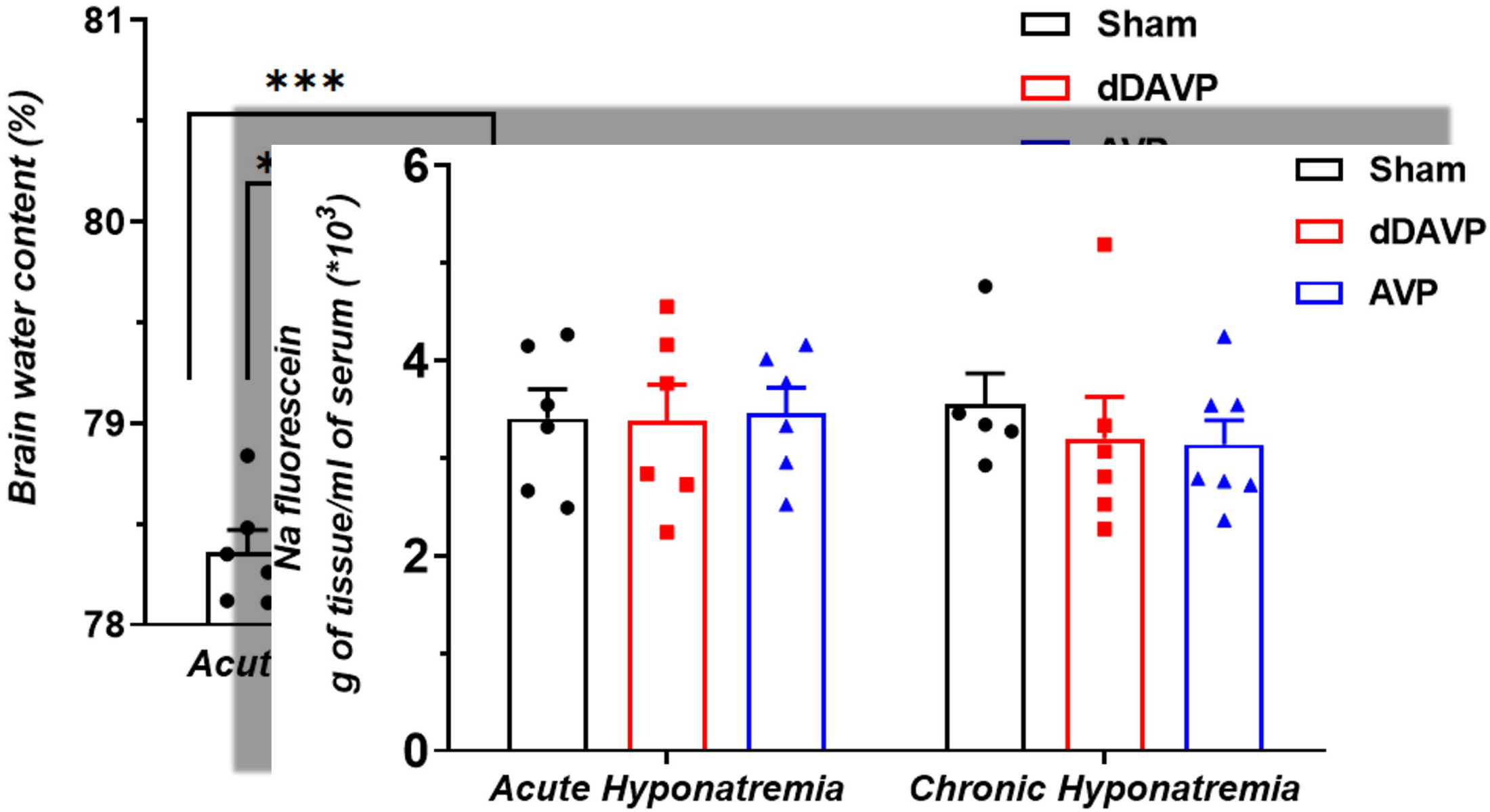
BBB determination (NaF), n=17

brain water content studies, n=18

tissue collection to RT-PCR, n=18

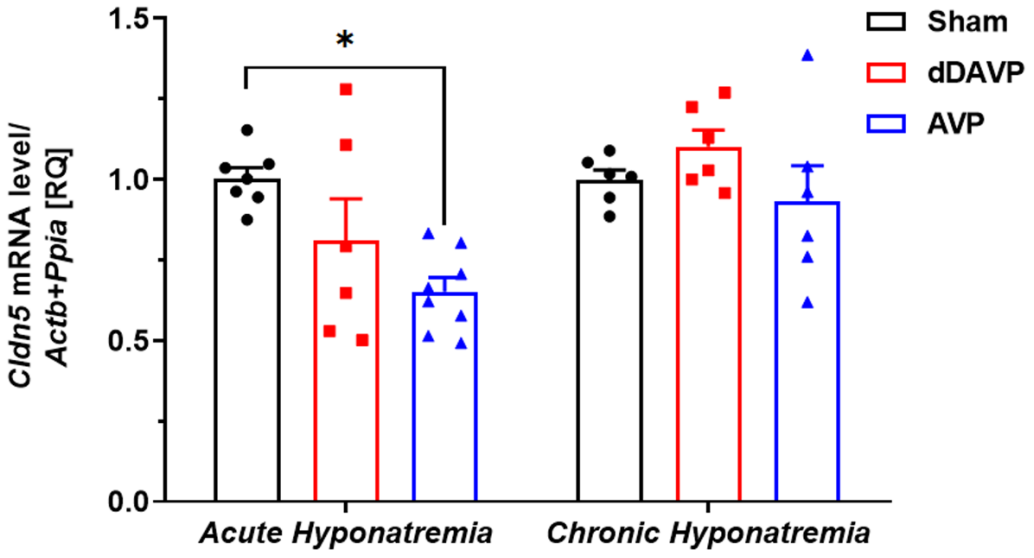
blood analyses, n=53

Aleksandrowicz et al. Sci Rep 2025

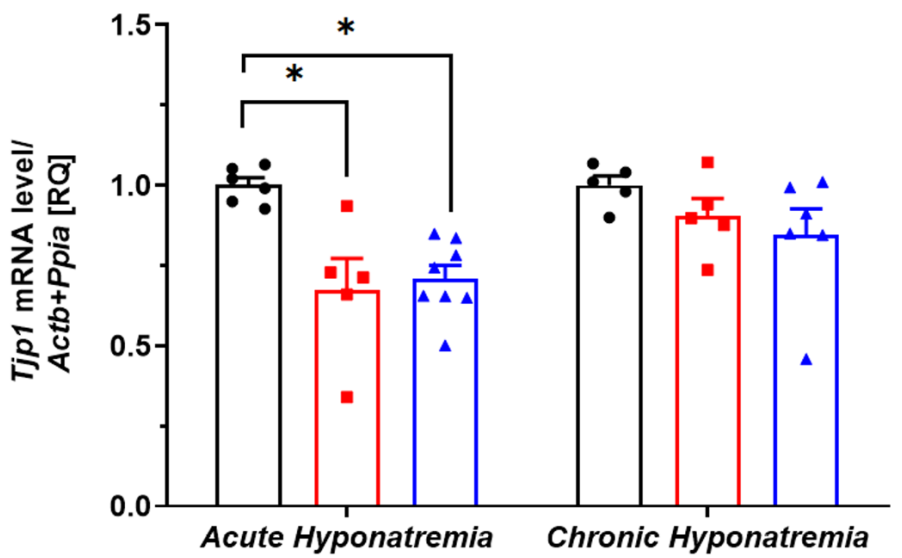


Aleksandrowicz et al. Sci Rep 2025

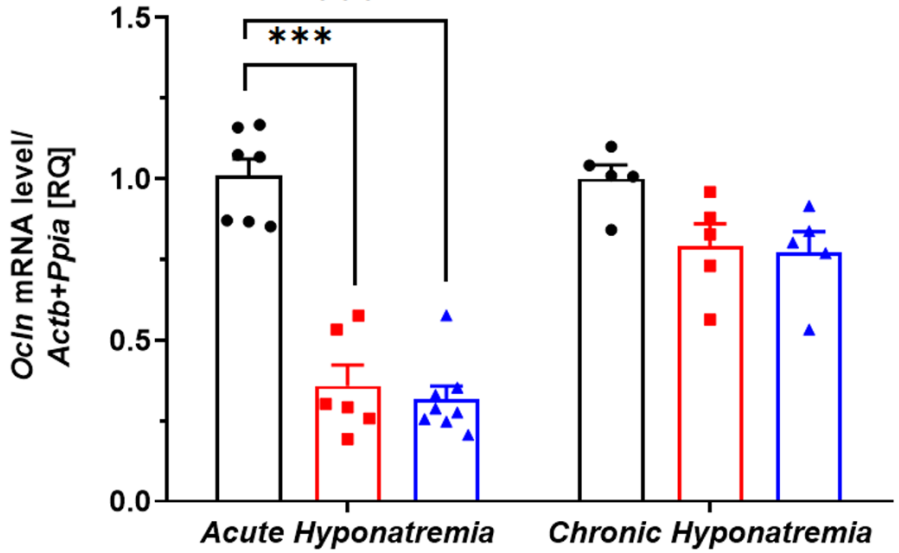
A

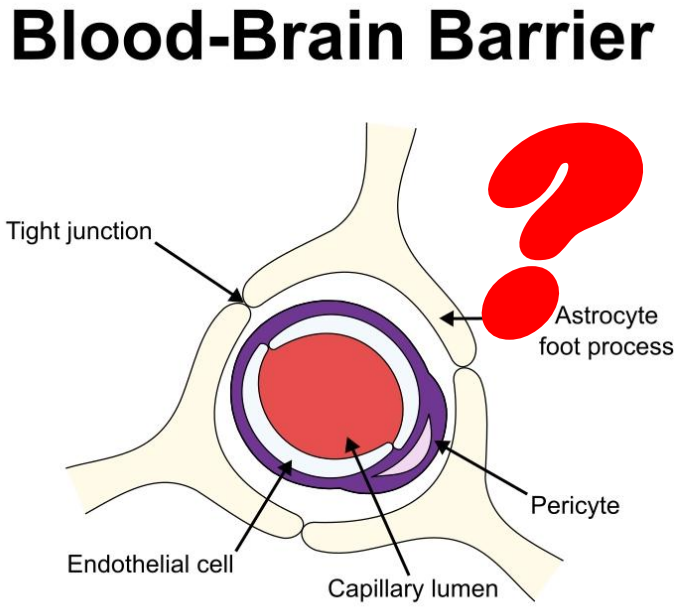

Sham
dDAVP
AVP
Cldn5 mRNA level/
Actb+Ppia [RQ]
1.5
1.0
0.5
0.0
*
Acute Hyponatremia
Chronic Hyponatremia

C

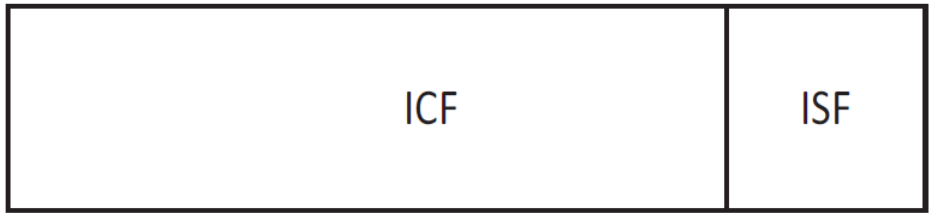

Tjp1 mRNA level/
Actb+Ppia [RQ]
1.5
1.0
0.5
0.0
*
*
Acute Hyponatremia
Chronic Hyponatremia

B


Ocln mRNA level/
Actb+Ppia [RQ]
1.5
1.0
0.5
0.0
***
***
Acute Hyponatremia
Chronic Hyponatremia


Blood-Brain Barrier
Tight junction
Astrocyte
foot process
Pericyte
Endothelial cell
Capillary lumen

Normal situation



Hyponatremia without adaptation
Increase in the ICF and the ISF from excess water due to hyponatremia.

Hyperacute adaptation
Decrease in the ISF fluid from bulk flow from ISF to systemic circulation.

Within minutes

Acute adaptation
Decrease in ICF fluid driven

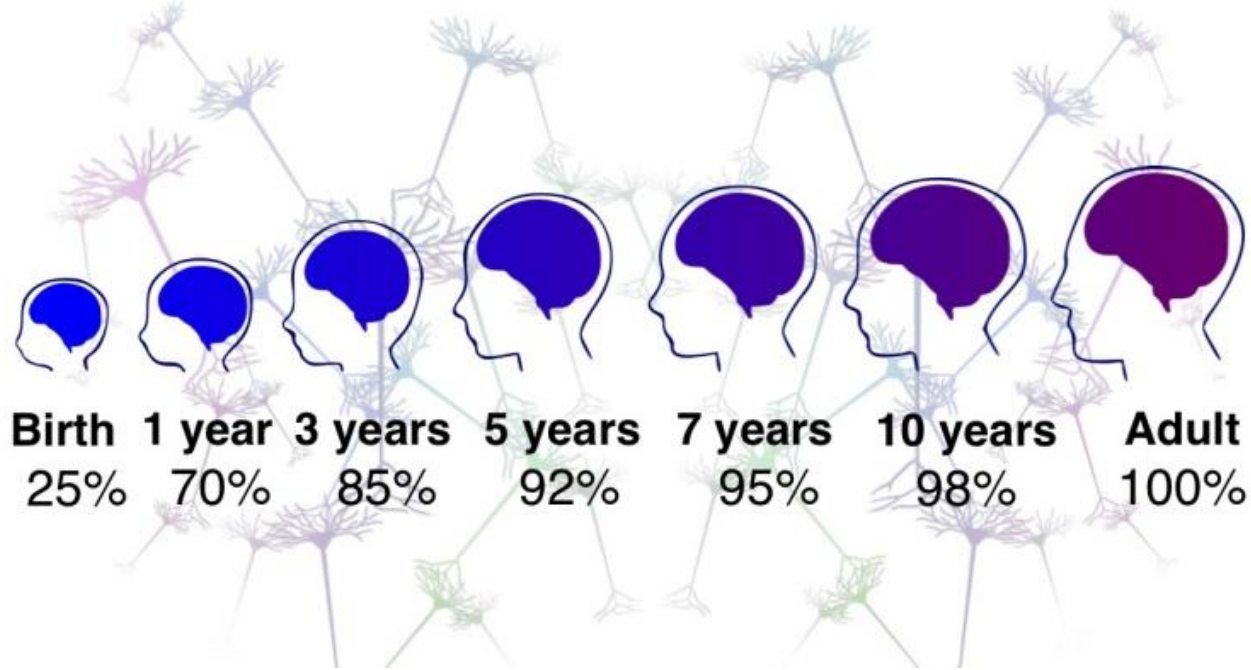
Within minutes, maximal after around 4 hours.

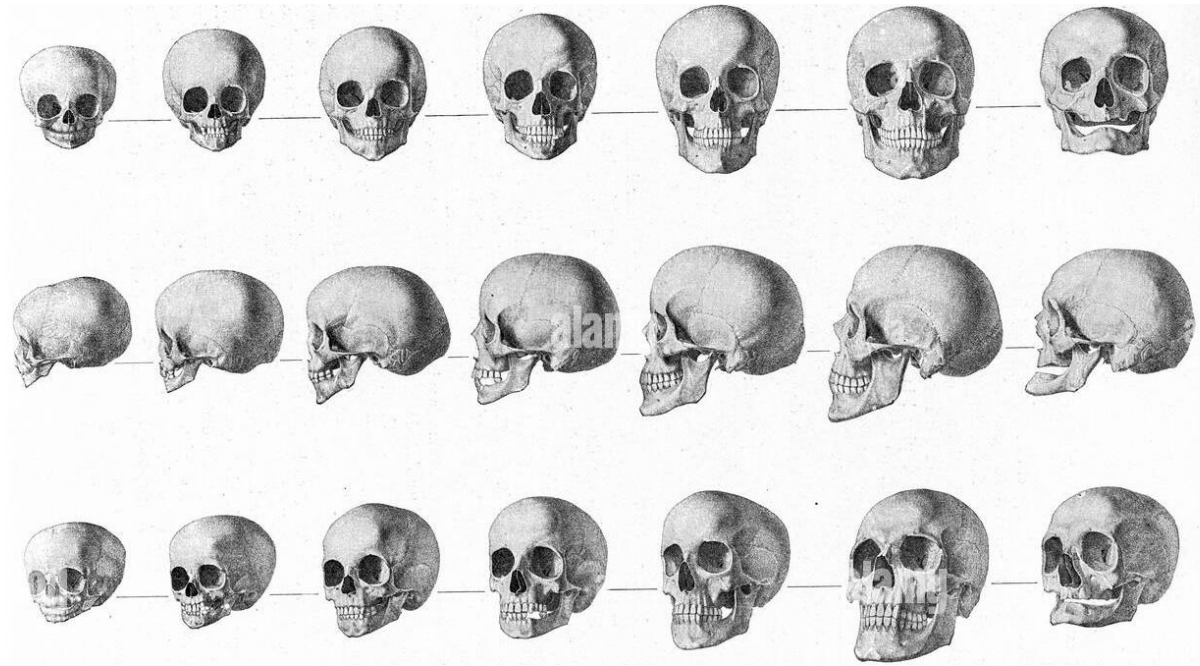
Chronic adaptation
Decrease in ICF fluid driven by brain organic osmolyte depletion.

Within hours, maximal after days

Sterns RH. Semin Nephrol 1990


MEDIUM
LOW
HIGH
RISK?


Birth 1 year 3 years 5 years 7 years 10 years Adult
25% 70% 85% 92% 95% 98% 100%

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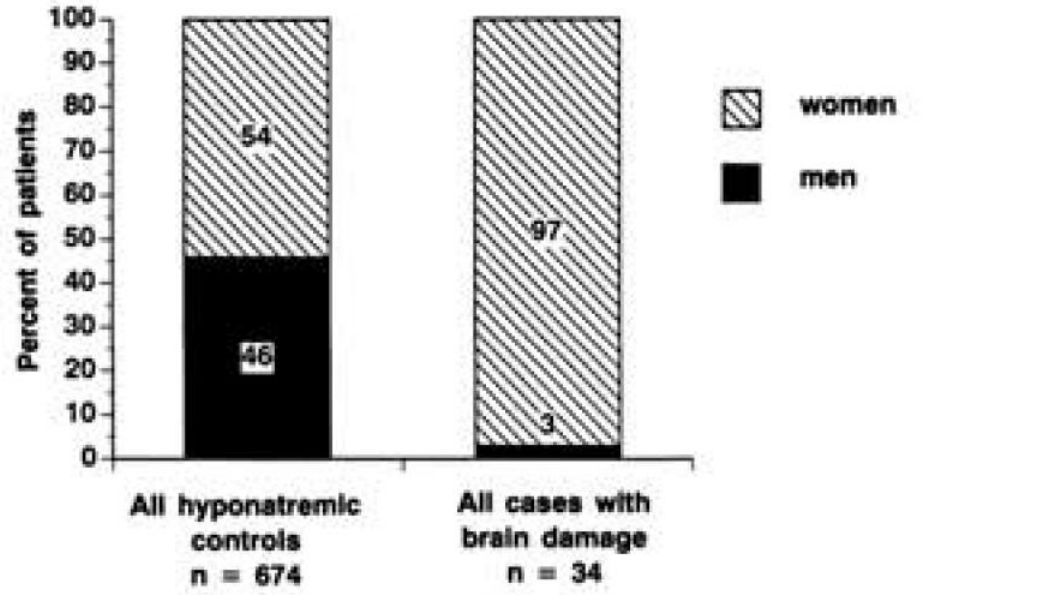
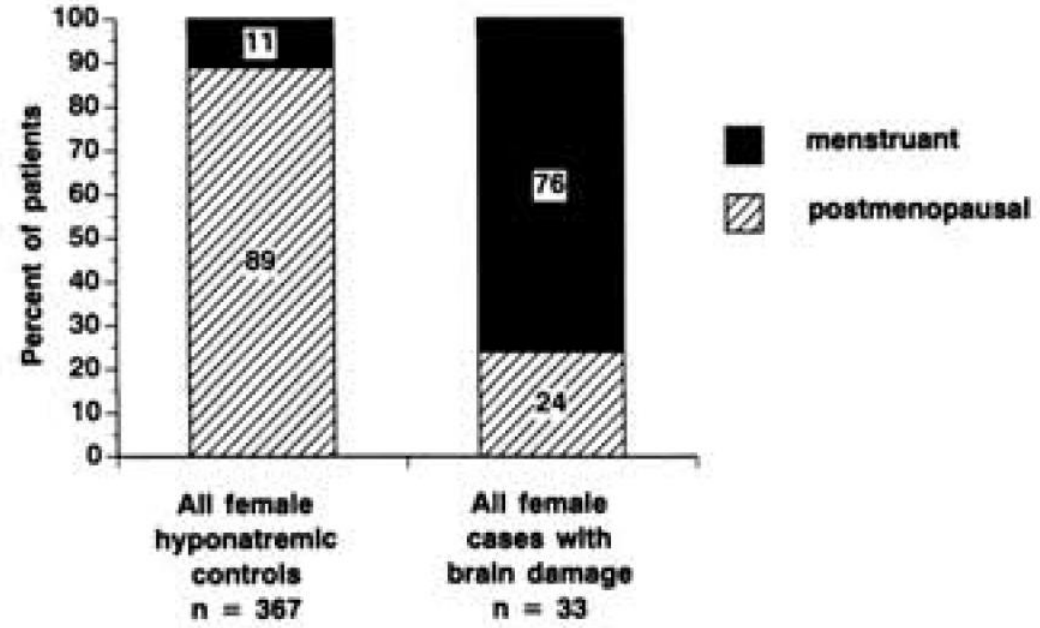
4 5 6 7 8 9 10
11 12 13 14 15 16 17
18 19 20 21 22 23 24
25 26 27 28 29 30 31





Ayus et al. Ann Intern Med 1992

Absence of Postoperative Hyponatremia Syndrome in Young, Healthy Females

Eelco F. M. Wijdicks, MD, and Timothy S. Larson, MD

Young and previously healthy females have been reported to develop severe postoperative hyponatremia with a fatal outcome. The clinical presentation is dramatic, with seizures, respiratory arrest, and permanent, often catastrophic, brain damage. The true incidence is unknown. We report a survey of 290,815 surgical procedures on females at the Mayo Clinic from 1976 to 1992. Postoperatively 1,498 females had cardiopulmonary arrest, 255 had a metabolic encephalopathy, 32 had new-onset seizures, and 6 had central pontine myelinolysis. We failed to identify any association of respiratory arrest with postoperative hyponatremia. Our findings indicate that the postoperative hyponatremia syndrome in young, healthy females with respiratory arrest is extremely uncommon.

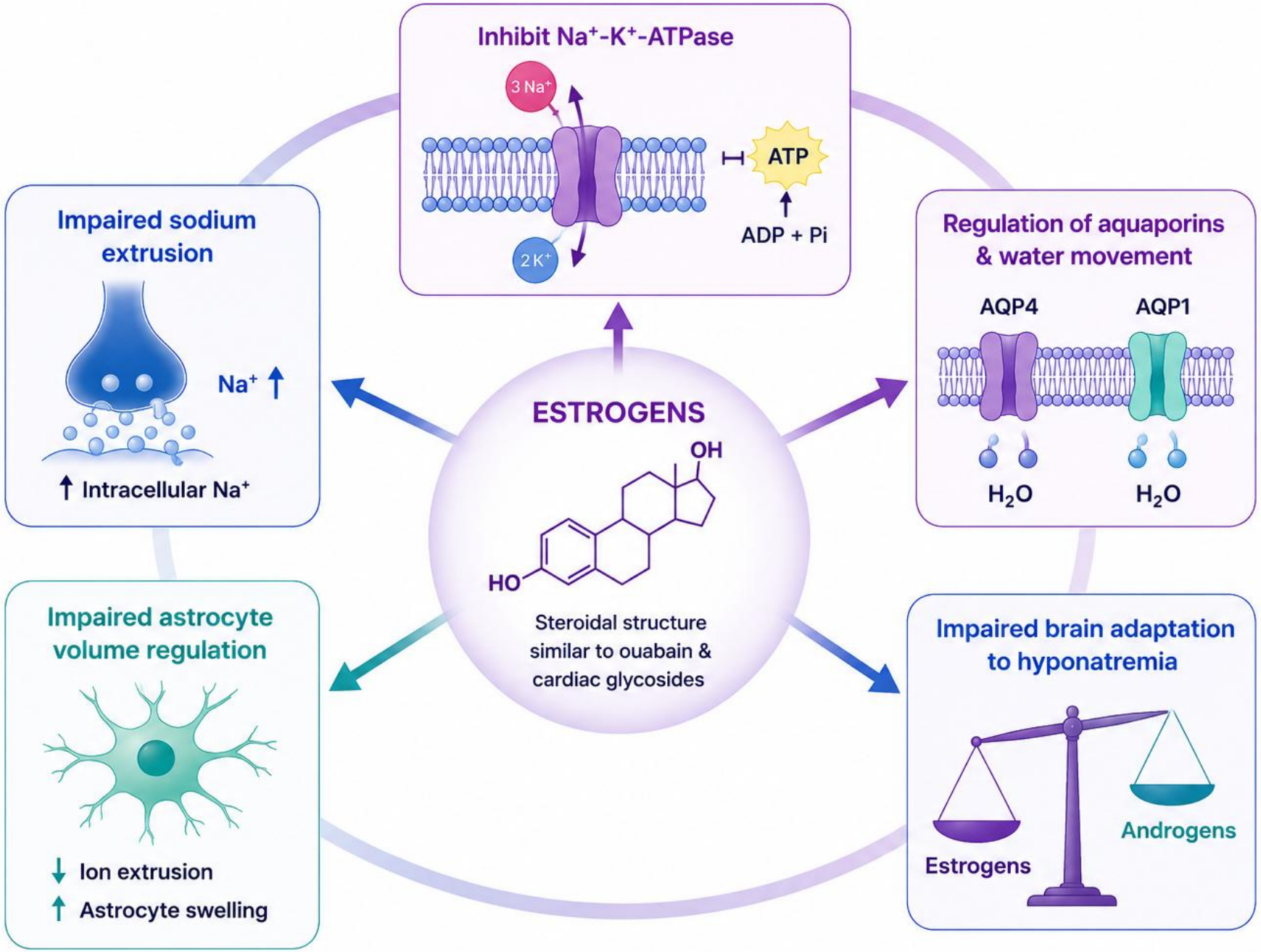
Wijdicks EFM, Larson TS. Absence of postoperative hyponatremia syndrome in young, healthy females. Ann Neurol 1994;35:626–628

Summary of Numbers of Postoperative Females Who Had a Cardiopulmonary Arrest or Developed Metabolic Encephalopathy, New-Onset Seizures, or Central Pontine Myelinolysis and Hyponatremia: 1976–1992[a]

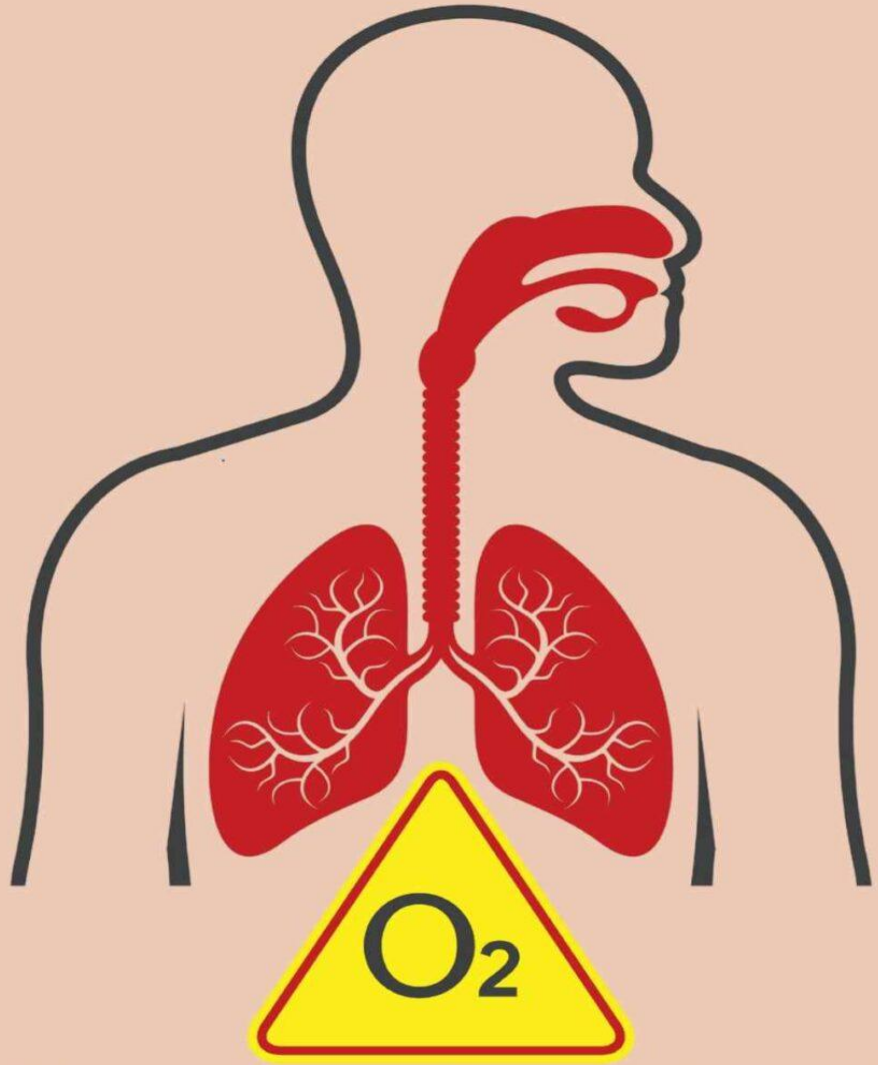
	No. with Post-operative Event	No. with Plasma Sodium <130 mmol/L
Cardiopulmonary arrest	1,498	0
Metabolic encephalopathy	255	2
New-onset seizures	32	3
Central pontine myelinolysis	6	6

[a]Total number of surgical procedures in females was 290,815.

ChatGPT



Auys et al. Am J Physiol Renal Physiol 2008

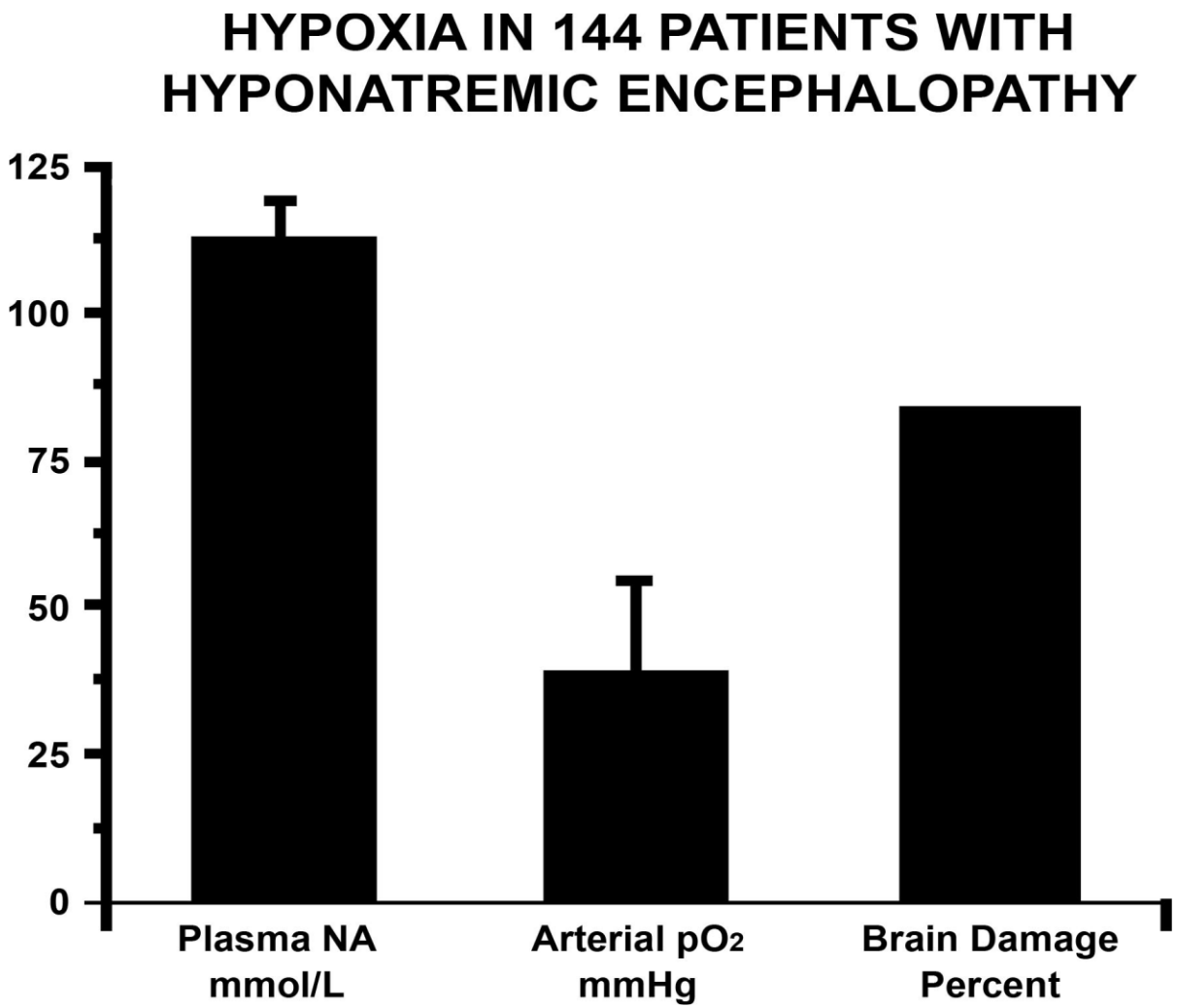


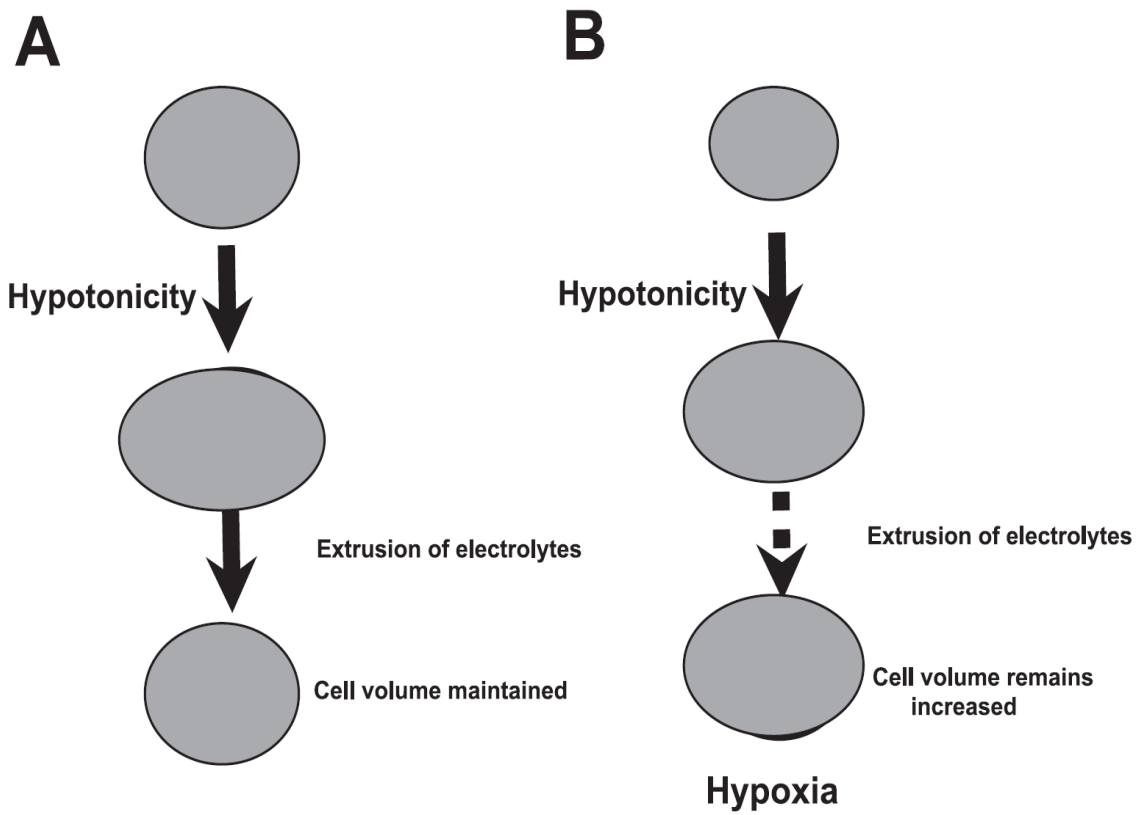
ΥΠΟΧΙΑ

ΔΕΝ ΕΧΩ ΟΞΥΓΟΝΟ

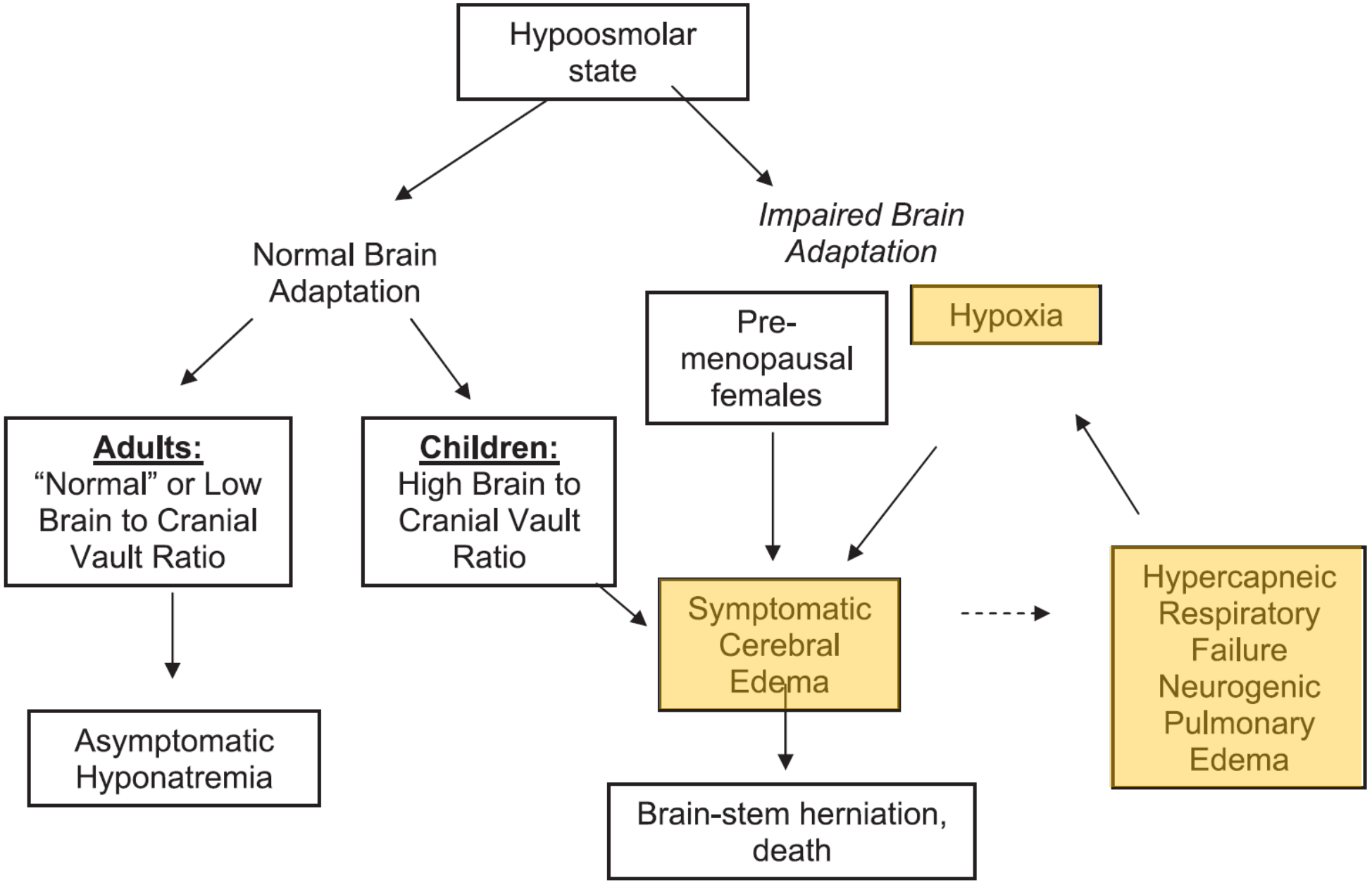
ΚΥΡΙΑΚΗ 26/01 12:00μ.μ.

Αθήνα, Πλατεία Συντάγματος, **Θεσσαλονίκη**-Καμάρα | **Λάρισα**, Κεντρική Πλατεία
Βόλος, Παραλία Άγαλμα | **Δράμα**, Πλατεία Ελευθερίας | **Αλεξανδρούπολη**, Δημαρχείο
Χανιά, Πλατεία Αγοράς | **Ηράκλειο**, Πλατεία Ελευθερίας |
Ρέθυμνο Πλατεία Αγνώστου Στρατιώτη | **Κοζάνη**, Κεντρική Πλατεία
Κατερίνη, Πλατεία Ελευθερίας | **Κέρκυρα**, Ανουτσιατα | **Πάτρα**, Πλατεία Γεωργίου
Πάρος, Πλατεία Μαντώ Μαυρογένους | **Γιάννενα**, Κεντρική Πλατεία, Περιφέρεια
Βέροια, Δημαρχείο | **Μυτιλήνη**, Πλατεία Σαπφούς | **Πέραμα**, Πλατεία Δημαρχείου
Κεφαλλονιά - Πλατεία Βαλλιάνου | **Αγρίνιο** - Κεντρικη Πλατεία | **Ορεστιάδα |**
Ρόδος Δημαρχείο **| Χίος** – Πλατεία Βουνακίου | **Θήβα** -Κεντρική Πλατεία | **Κομοτηνη**
Πύργος – Κεντρική Πλατεία | **Χαλκίδα** - Πλατεία Ταχυδρομείου | **Σέρρες** Πλ. Ελευθερίας
Αλιβέρι Άγαλμα Λιγνιτωρύχου | **Άνδρος**, Πλατεία Θεοφιλου Καΐρη
Τρίκαλα Θεσσαλίας, Καλαμπάκα | **Καλαμάτα** Πλατεία Β. Γεωργίου | **Σπάρτη**
Καστοριά - Δημαρχείο | **Αριδαία** - Πλατεία | **Γεράκι** Λακωνίας | **Σητεία |**
Κιλκίς – Πλατεία Ειρήνης | **Ιεράπετρα** – Πλατεία Ειρήνης | **Άνδρος**





Auys et al. Am J Physiol Renal Physiol 2008



Auys et al. Am J Physiol Renal Physiol 2008

Table 1. Manifestations of hyponatremic encephalopathy

Acute severe	Chronic
Nausea and vomiting	Nausea
Headaches	Fatigue
Seizures	Gait and attention deficit
Coma	Falls and bone fractures
Death	
Respiratory arrest	
Noncardiogenic pulmonary edema	

Gankam Kengne & Decaux. Kidney Int Rep 2018

Table 3. Summary of reported cases of ecstasy-associated hyponatremia[a]

Age	Gender	Sodium		Osmolality		Symptoms	Intervention	Outcome	Reference
		Serum	Urine	Serum	Urine				
18	F	120	186	242	568	A, C	H, F, R	Recovered	(78)
27	F	124	NA	267	NA	A, C	F	Death	(60)
26	M	101	NA	248	NA	C, S, P	F, N	Recovered	(51)
17	F	115	10	256	577	A, C, S	N	Recovered	(61)
19	F	115	162	253	522	S, P	H, F	Recovered	(49)
24	F	113	153	240	639	A, C, S	R	Recovered	(56)
20	F	119	145	263	491	A, C, S	R, N	Recovered	(79)
30	F	117	1	NA	38	A, C, S	NA	NA	(62)
15	F	125	NA	NA	NA	A, RA, P, C	N, R, F	Death	(54)
20	F	112	112	238	256	A, C	Mannitol	Recovered	(57)
15	F	119	6	256	655	A, C	R	Recovered	(53)
16	F	112	99	242	184	A, C	R	Recovered	(53)
24	F	120	68	258	365	A	H	Recovered	(50)
23	F	123	NA	NA	NA	A	Observation	Recovered	(64)
17	F	130	NA	NA	NA	A	NA	Recovered	(52)
17	F	118	NA	247	970	A, E	Conservative	Recovered	(52)
23	F	115	246	NA	NA	C	N, glucose	Death	(80)
19	F	121	111	242	485	A, C	H	Recovered	(81)
16	F	NA	NA	NA	NA	A	NA	Death	(82)
18	F	NA	NA	NA	NA	CA, HT	NA	Death	(82)
18	M	NA	NA	NA	NA	A, HT	NA	Death	(82)
14	F	NA	NA	NA	NA	CA	NA	Death	(82)
16	F	NA	NA	NA	NA	A, CA	NA	Death	(82)
29	F	NA	NA	NA	NA	CA	NA	Death	(82)
16	F	123	NA	NA	NA	S, LBP, C, P	NA	Death	(82)
20	F	119	NA	NA	NA	LBP, LT, RD	NA	Death	(82)
20	F	124	NA	NA	NA	A, C	NA	Death	(82)
19	M	122	NA	NA	NA	A, C	NA	Death	(82)
19	F	NA	NA	NA	NA	CA	NA	Death	(83)
19	M	NA	NA	NA	NA	HT, LBP, AKI	"Saline"	Death	(84)
20	F	117	NA	245	NA	CA	N, H, F	Death	(65)
18	W	130	101	264	335	HA	R	Recovered	(66)

[a]A, altered mental status; C, cerebral edema; CA, cardiac arrest; E, electrocardiogram abnormalities; F, furosemide; H, hypertonic saline; HA, headache; HT, hyperthermia; LBP, hypotension; LT, hypothermia; N, normal saline; NA, not available; P, pulmonary edema on chest x-ray; R, water restriction; RA, respiratory arrest; RD, respiratory depression; S, seizure.

Campbell & Rosner. Clin J Am Soc Nephrol 2008

JAMA Network | Open

Original Investigation | Diabetes and Endocrinology

Oxytocin and the Role of Fluid Restriction in MDMA-Induced Hyponatremia
A Secondary Analysis of 4 Randomized Clinical Trials

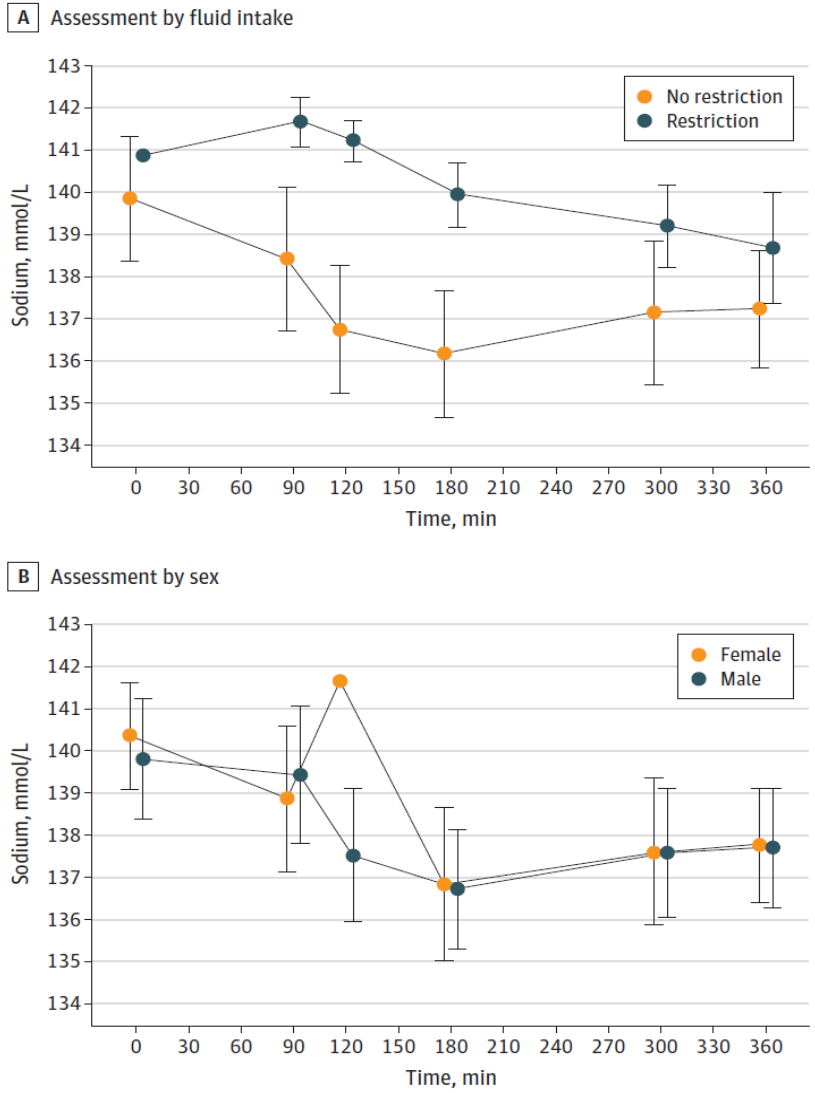
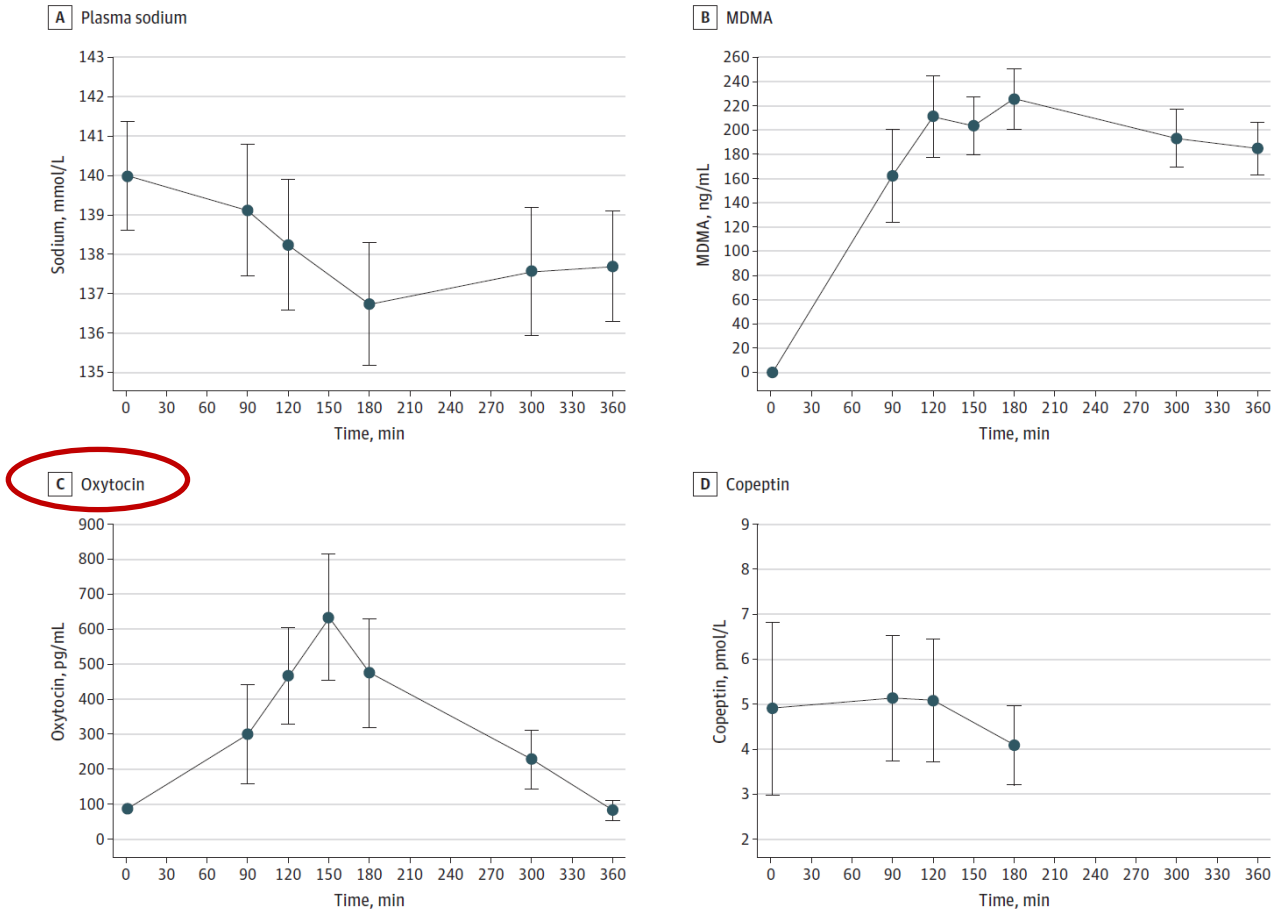


Figure 1. Changes in Laboratory Values Over 360 Minutes in Response to 3,4-Methylenedioxymethamphetamine (MDMA) Intake



Atila et al. JAMA Netw Open 2024

THERAPEUTIC GUIDELINES™

Table 2. Comparison of the United States and European guidelines

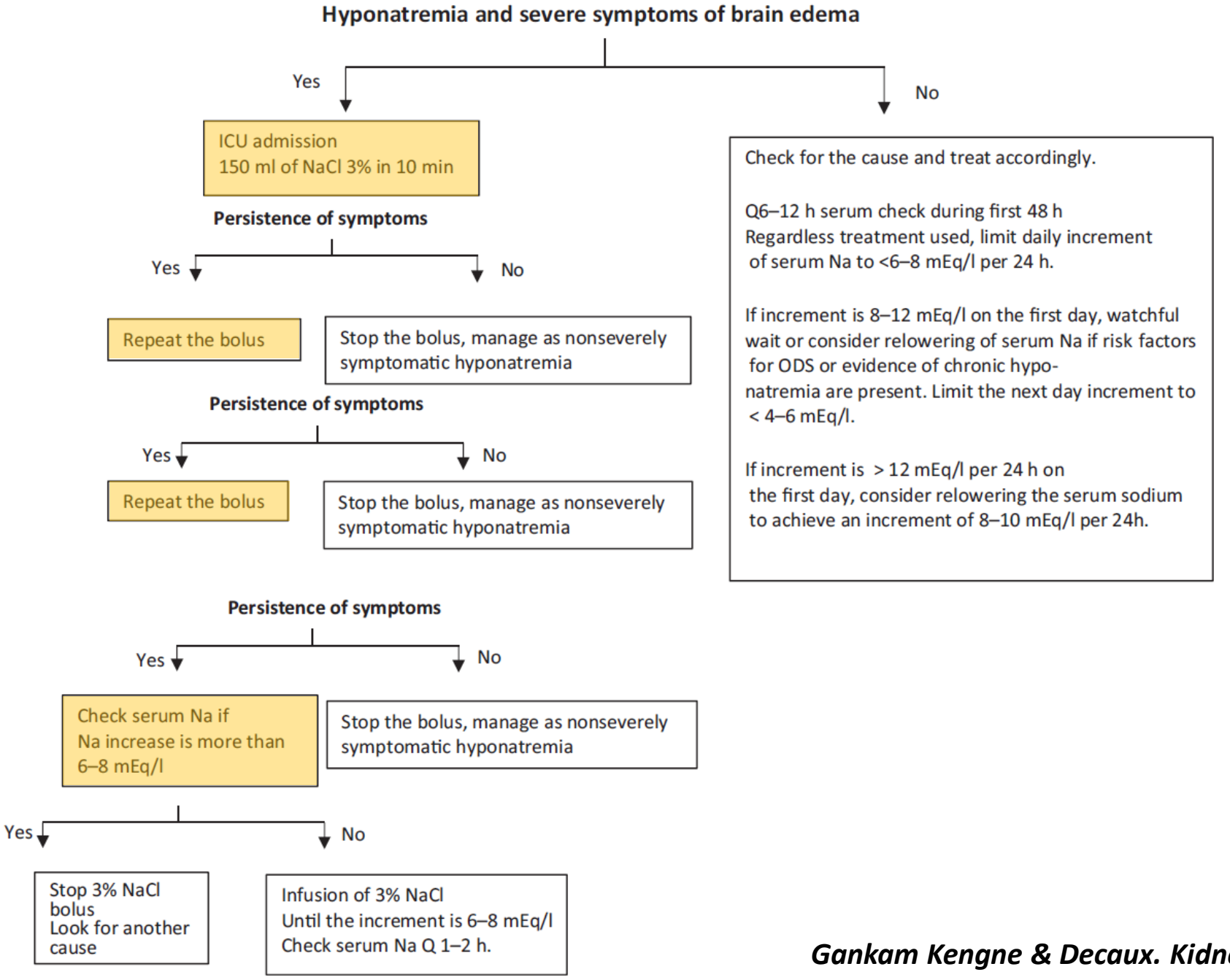
Subject	United States Guideline	European Guideline
Acute or symptomatic hyponatremia	Severe symptoms: Bolus 3% NaCl (100 ml over 10 min × 3 as needed) Moderate symptoms: Continuous infusion 3% NaCl (0.5–2 ml/kg per h)	Severe symptoms: Bolus 3% NaCl (150 ml over 20 min 2–3 times as needed) Moderate symptoms: Bolus 3% NaCl (150 ml 3% over 20 min once)
Chronic hyponatremia SIAD	Fluid restriction (first line) Demeclocycline, urea, or vaptan (second line)	Fluid restriction (first line) Urea or loop diuretics + oral NaCl (second line) Do not recommend or recommend against vaptan[a] Recommend against lithium or demeclocycline
Hypovolemic hyponatremia	Isotonic saline	Isotonic saline or balanced crystalloid solution
Hypervolemic hyponatremia	Fluid restriction Vaptans[b]	Fluid restriction Recommend against vaptan
Correction rates	Minimum: 4–8 mmol/L per d, 4–6 mmol/L per d (high risk of ODS) Limits: 10–12 mmol/L per d, 8 mmol/L per d (high risk of ODS)	No minimum Limit: 10 mmol/L per d
Management of overcorrection	Baseline $S_{Na} \geq 120$ mmol/L: probably unnecessary Baseline $S_{Na} < 120$ mmol/L: start relowering with electrolyte-free water or desmopressin after correction exceeds 6–8 mmol/L per d	Start once limit is exceeded Consult an expert to discuss infusion containing electrolyte-free water (10 ml/kg) with or without 2 μg desmopressin iv

[a]"Do not recommend" when $S_{Na} < 130$ mmol/L, "recommend against" when $S_{Na} < 125$ mmol/L.
[b]In liver cirrhosis, restrict to patients where potential benefit outweighs risk of worsened liver function.[9]

Hoorn & Zietse. J Am Soc Nephrol 2017

+

120 ml διάλυμα 3.17% NaCl



Gankam Kengne & Decaux. Kidney Int Rep 2018

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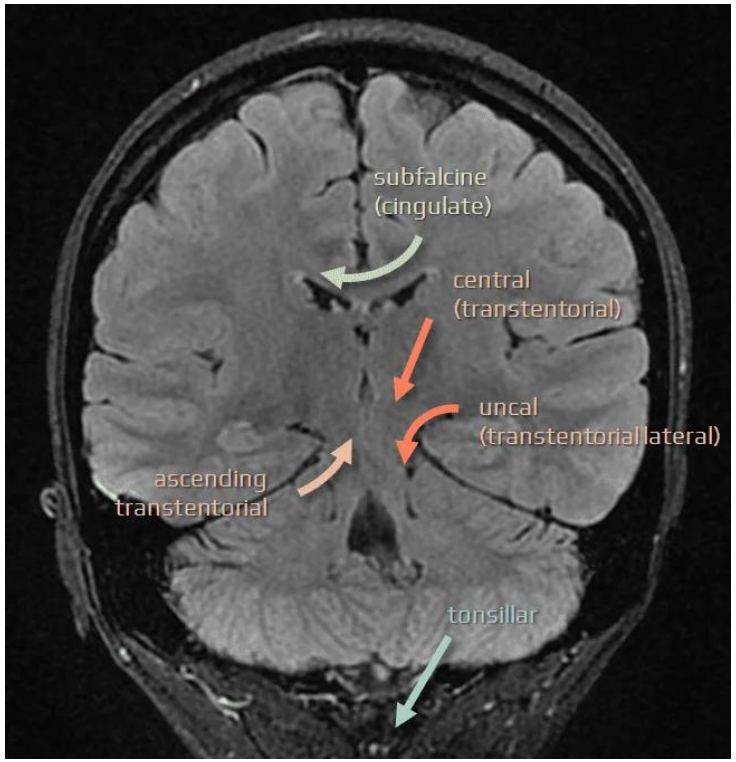


Figure Changes in physiologic variables before and after administration of 23.4% saline bolus

N = 68

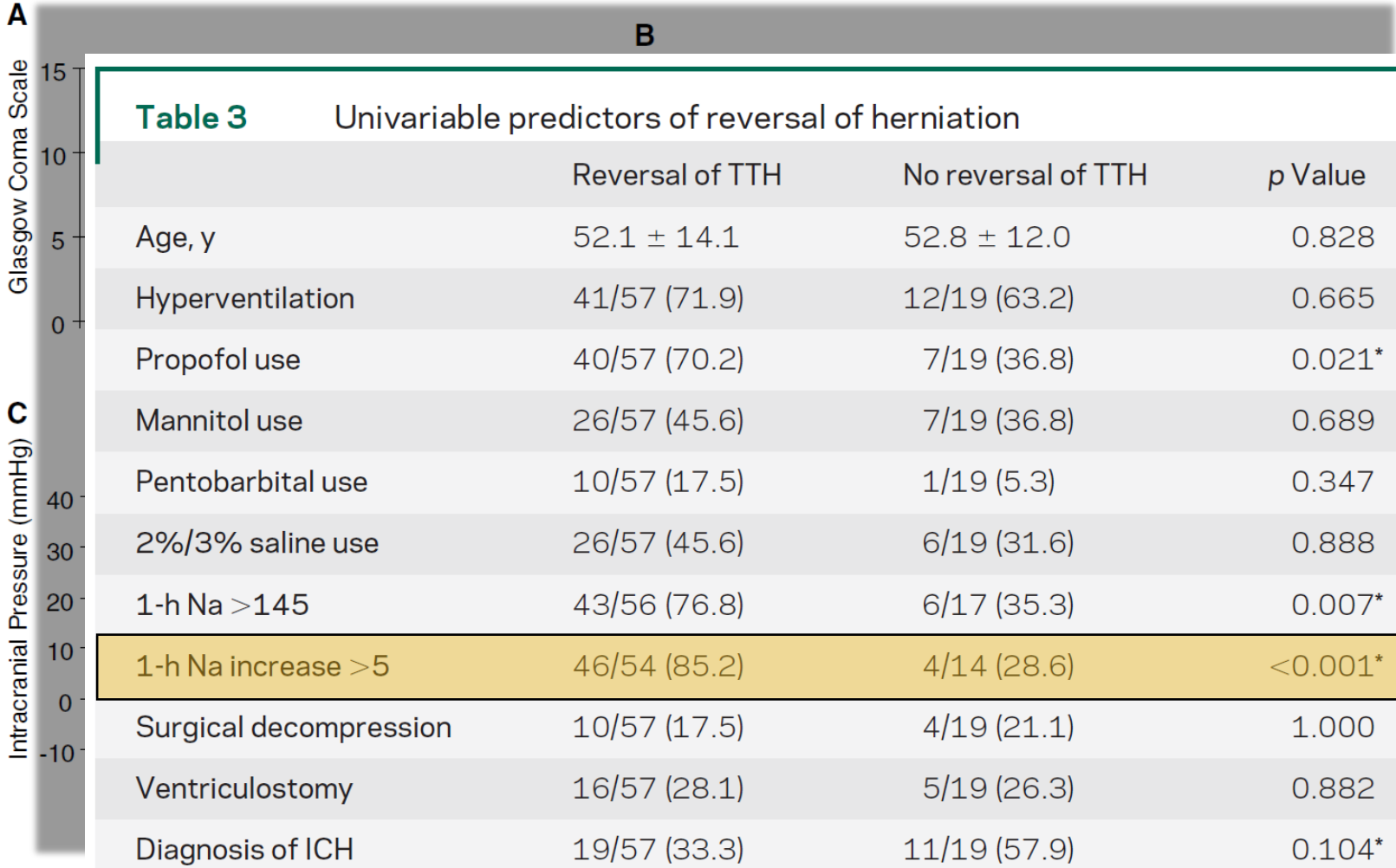


Table 3 Univariable predictors of reversal of herniation

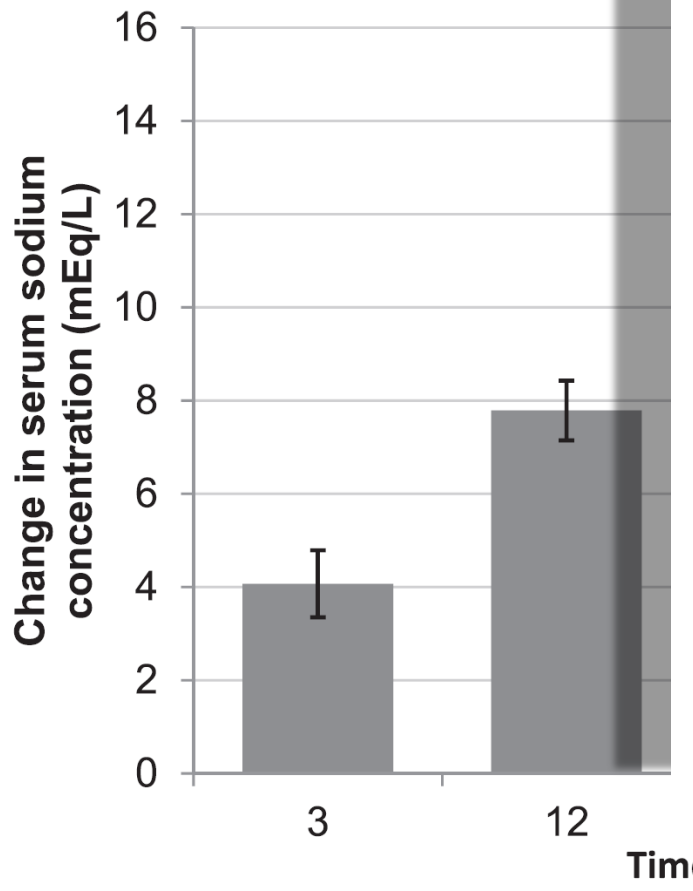
	Reversal of TTH	No reversal of TTH	*p* Value
Age, y	52.1 ± 14.1	52.8 ± 12.0	0.828
Hyperventilation	41/57 (71.9)	12/19 (63.2)	0.665
Propofol use	40/57 (70.2)	7/19 (36.8)	0.021*
Mannitol use	26/57 (45.6)	7/19 (36.8)	0.689
Pentobarbital use	10/57 (17.5)	1/19 (5.3)	0.347
2%/3% saline use	26/57 (45.6)	6/19 (31.6)	0.888
1-h Na >145	43/56 (76.8)	6/17 (35.3)	0.007*
1-h Na increase >5	46/54 (85.2)	4/14 (28.6)	<0.001*
Surgical decompression	10/57 (17.5)	4/19 (21.1)	1.000
Ventriculostomy	16/57 (28.1)	5/19 (26.3)	0.882
Diagnosis of ICH	19/57 (33.3)	11/19 (57.9)	0.104*

Koenig et al. Neurology 2008

AJKD
Original Investigation

Treatment of Hyponatre… Chloride

Juan Carlos Ayus, MD,[1,2]
Ricardo Heguilen, MD,[5] Clau…



AJKD
Correspondence

LETTERS TO THE EDITOR

Treatment of Hyponatremic Encephalopathy

To the Editor:

In the March issue of *AJKD*, Ayus et al[1] describe their experience treating hyponatremic encephalopathy with 500 mL of 3% sodium chloride solution over 6 hours. This provocative report raises at least 2 important questions.

First, the authors point out that "symptoms of hyponatremic encephalopathy are largely related to cerebral edema" and therefore this condition "constitutes a medical emergency."[1(p435)] Given this context, why do the investigators support a slow infusion (83 mL/h) rather than the recently recommended bolus therapy of 100 to 150 mL of 3% sodium chloride solution given over 10 to 20 minutes (up to 3 doses or until serum sodium concentration has increased by 5 mEq/L), which can prevent brain herniation and stop seizure activity quickly?[2,3]

Second, why did the authors' continuous hypertonic saline solution protocol not include an adjustment for body weight, as has been recommended for bolus therapy?[2,3] Because all patients received the same "uniform treatment protocol," a 50-kg patient would be expected to have had twice the increase in serum sodium concentration as would a 100-kg patient (assuming similar percentages of water weight). Thus, applying this protocol without considering a patient's total-body water risks overcorrection in small people, who are found frequently among patients with diuretic-induced hyponatremia,[4] and undercorrection in very large ones, as may have occurred in 2 of the authors' cases.

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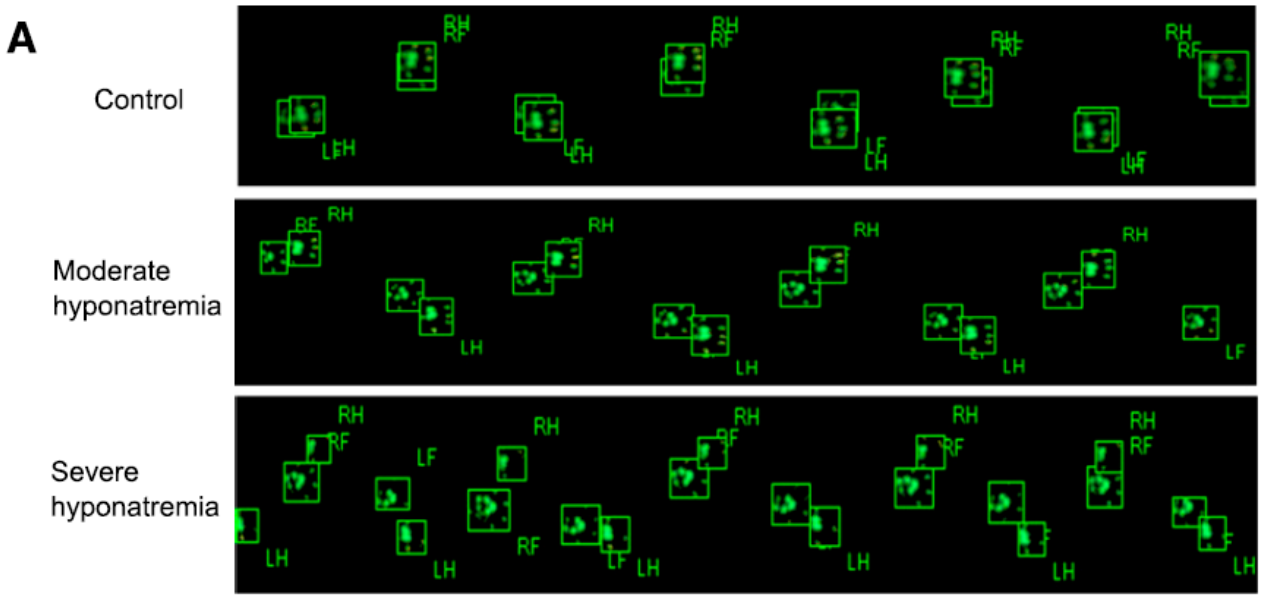
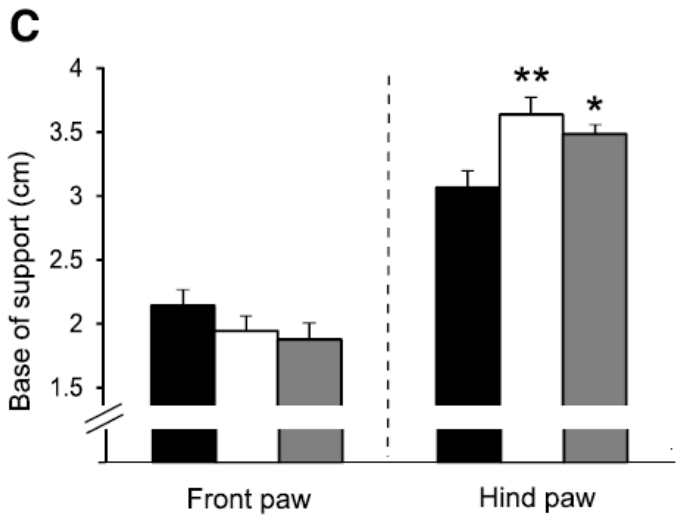
Mt. Sinai St. Luke's Roosevelt Hospital, New York, New York

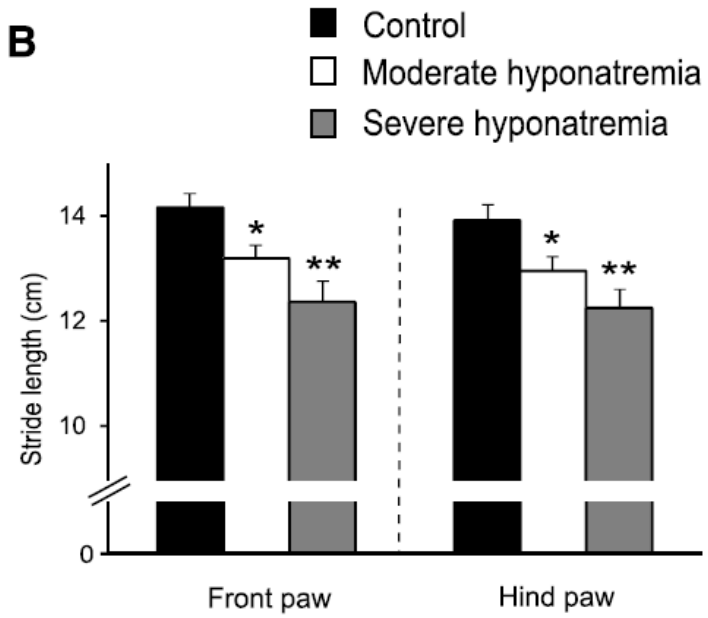
Acknowledgements

Financial Disclosure: The author declares that he has no relevant financial interests.

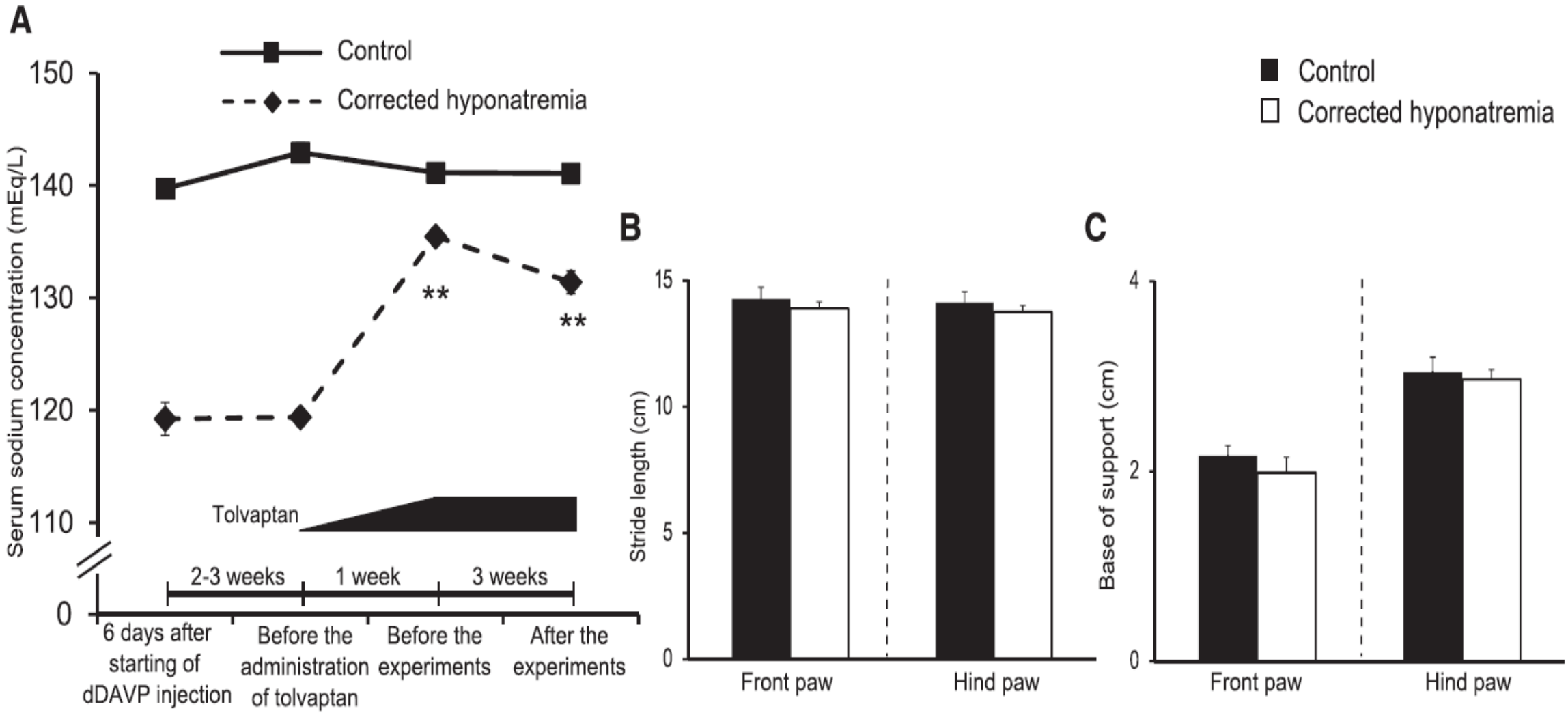
Ayus et al. Am J Kidney Dis 2014







Fujisawa et al. J Am Soc Nephrol 2015



Fujisawa et al. J Am Soc Nephrol 2015

Chronic Hyponatremia and Brain Structure and Function Before and After Treatment

Setting & Participants

Prospective cohort study

Single-center in Cologne, Germany

N = 26 patients with presumed chronic hyponatremia [Na^+]
- Treated in ED
- No signs of hypo- or hypervolemia

April 2018-January 2022

Analysis

Standardized neuropsychological testing (NPT) + MRI

Baseline — Normonatremia

N = 21 patients with complete data

Mean [Na^+], mmol/L

Pre-treatment:	Post-treatment:
118.4	135.5

Results

After Resolution of Hyponatremia:

Improved cognition in most tests

- Decreased brain tissue volumes, especially in the hippocampus
- Reduced neuronal activity & neuronal synchronization across all gray matter

During Hyponatremia:

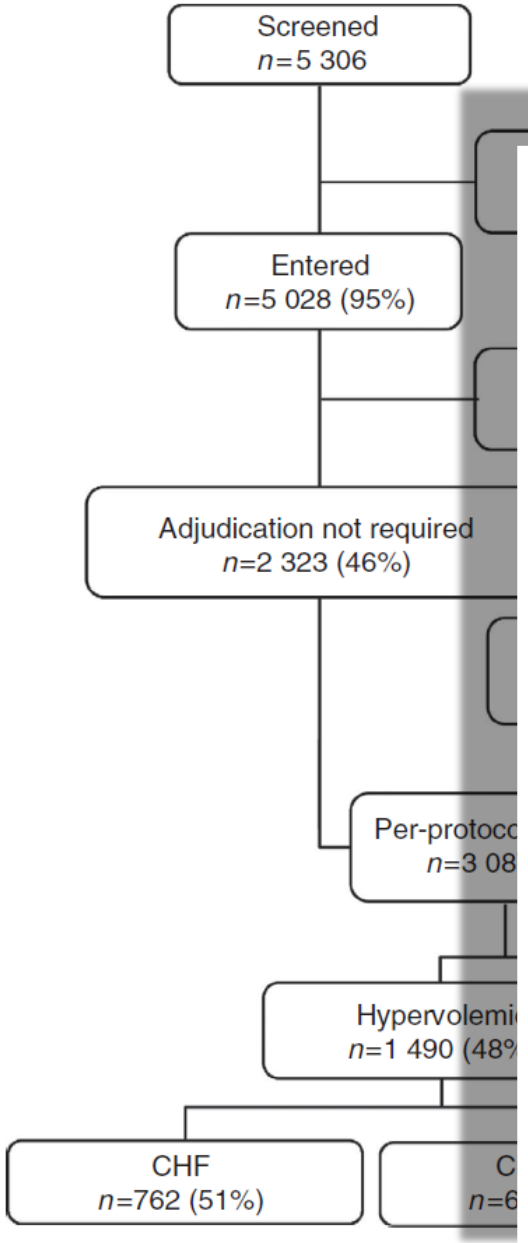
Negative correlation of neuronal synchronization with [Na+] and cognitive function

CONCLUSION: Resolution of hyponatremia was associated with improved cognition and reductions in brain volumes and neuronal activity. Impaired cognition during hyponatremia is closely linked to increased neuronal activity rather than to tissue volumes. Furthermore, the hippocampus appears to be particularly susceptible to hyponatremia, exhibiting pronounced changes in tissue volume.

Victor Suárez, Rosanne Picotin, Ronja Faßbender, et al

@AJKDonline | DOI: 10.1053/j.ajkd.2023.11.007

AJKD
AMERICAN JOURNAL OF KIDNEY DISEASES

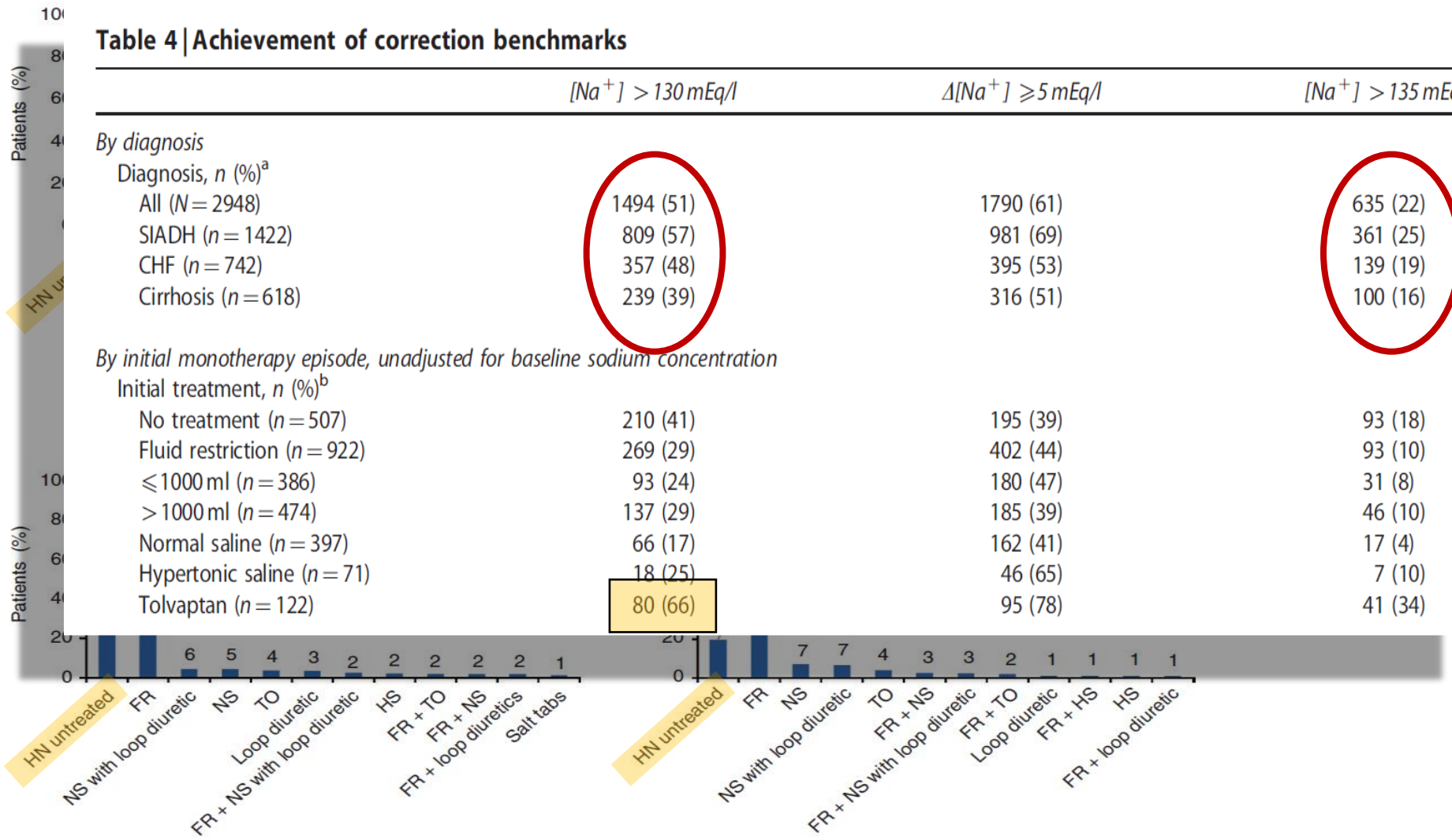


All (n = 3 087)[a,b]

SIADH (n = 1 524)[a,c]

Table 4 | Achievement of correction benchmarks

	$[Na^+] > 130$ mEq/l	$\Delta[Na^+] \geqslant 5$ mEq/l	$[Na^+] > 135$ mEq/l
By diagnosis			
Diagnosis, n (%)[a]			
All ($N = 2948$)	1494 (51)	1790 (61)	635 (22)
SIADH ($n = 1422$)	809 (57)	981 (69)	361 (25)
CHF ($n = 742$)	357 (48)	395 (53)	139 (19)
Cirrhosis ($n = 618$)	239 (39)	316 (51)	100 (16)
By initial monotherapy episode, unadjusted for baseline sodium concentration			
Initial treatment, n (%)[b]			
No treatment ($n = 507$)	210 (41)	195 (39)	93 (18)
Fluid restriction ($n = 922$)	269 (29)	402 (44)	93 (10)
$\leqslant$1000 ml ($n = 386$)	93 (24)	180 (47)	31 (8)
>1000 ml ($n = 474$)	137 (29)	185 (39)	46 (10)
Normal saline ($n = 397$)	66 (17)	162 (41)	17 (4)
Hypertonic saline ($n = 71$)	18 (25)	46 (65)	7 (10)
Tolvaptan ($n = 122$)	80 (66)	95 (78)	41 (34)



Greenberg et al. Kidney Int 2015

OPEN ACCESS

Multicentre study of investigation and management of inpatient hyponatraemia in the UK

Ploutarchos Tzoulis,[1] Rhys Evans,[2] Agnieszka Falinska,[3] Maria Barnard,[4] Tricia Tan,[3]

Table 1 Investigation of patients with hyponatraemia

Investigation	Total (N=100) (%)	Centre 1 (N=38) (%)	Centre 2 (N=30) (%)	Centre 3 (N=32) (%)
Volume status	62	71.0	53.4	59.4
Serum osmolality	39	39.5	33.3	43.8
Urine osmolality	33	39.5	30.0	28.1
Urine Na	29	34.2	36.6	15.6
Paired osmolality–Na	23	26.3	26.7	15.6
Serum TSH	61	71.0	63.3	46.9
Serum cortisol	31	34.2	26.6	31.2
Full work-up	18	23.7	20.0	9.4
Expert input	16	13.1	13.3	21.8

TSH, thyroid-stimulating hormone.

Table 3 Serum sodium (sNa) concentration at hospital discharge

SNa at discharge	Overall N=84
Patients with sNa <125 mmol/L (%)	4.8
Patients with sNa 125–129 mmol/L (%)	19.0
Patients with sNa 130–134 mmol/L (%)	39.3
Patients with sNa ≥135 mmol/L (%)	36.9
Mean±SD sNa (mmol/L)	132.8±4.7

Tzoulis et al. Postgrad Med J 2014

THE AMERICAN JOURNAL of MEDICINE®

Utility and Limitations of the Traditional Diagnostic Approach to Hyponatremia: A Diagnostic Study

Wiebke Fenske,[a] Sebastian K. G. Maier,[b] Anne Blechschmidt,[a] Bruno Allolio,[a,*] Stefan Störk[c,*]

[a]Department of Endocrinology and Diabetes, [b]Division of Intensive Care Medicine, and [c]Cardiology Unit, Department of Medicine I, University of Würzburg, Germany.

Table 2 Etiologic Diagnosis – Performance of *Senior Physician*

	Senior Physician							
Reference Standard	Primary Polydipsia	Hypervolemia	Hypovolemia	SIADH	Diuretic-induced	Adrenal Insufficiency	Total	Delta per Category
Primary polydipsia	1	0	0	1	3	0	5	0.20
Hypervolemia	0							
Hypovolemia	0							
SIADH	0							
Diuretic-induced	0							
Adrenal insufficiency	0							
Total	1							

SIADH = syndrome of inappr
Overall kappa (SE) = 0.20 (0.06)
Overall delta (SE) = 0.19 (0.06).

Table 4 Etiologic Diagnosis – Performance of *Algorithm*

	Algorithm							
Reference Standard	Primary Polydipsia	Hypervolemia	Hypovolemia	SIADH	Diuretic-induced	Adrenal Insufficiency	Total	Delta per Category
Primary polydipsia	0	0	0	5	0	0	5	—*
Hypervolemia	0	14	1	9	0	0	24	0.50
Hypovolemia	0	1	29	9	0	0	39	0.66
SIADH	0	0	1	41	0	0	42	0.78
Diuretic-induced	0	0	1	4	3	0	8	0.28
Adrenal insufficiency	0	0	0	2	0	1	3	0.21
Total	0	15	32	70	3	1	121	

SIADH = syndrome of inappropriate antidiuretic hormone secretion.
Overall kappa (SE) = 0.64 (0.06).
Overall delta (SE) = 0.70 (0.06).
*Not computed because "Algorithm" failed to diagnose "Primary polydipsia."

SALT trial

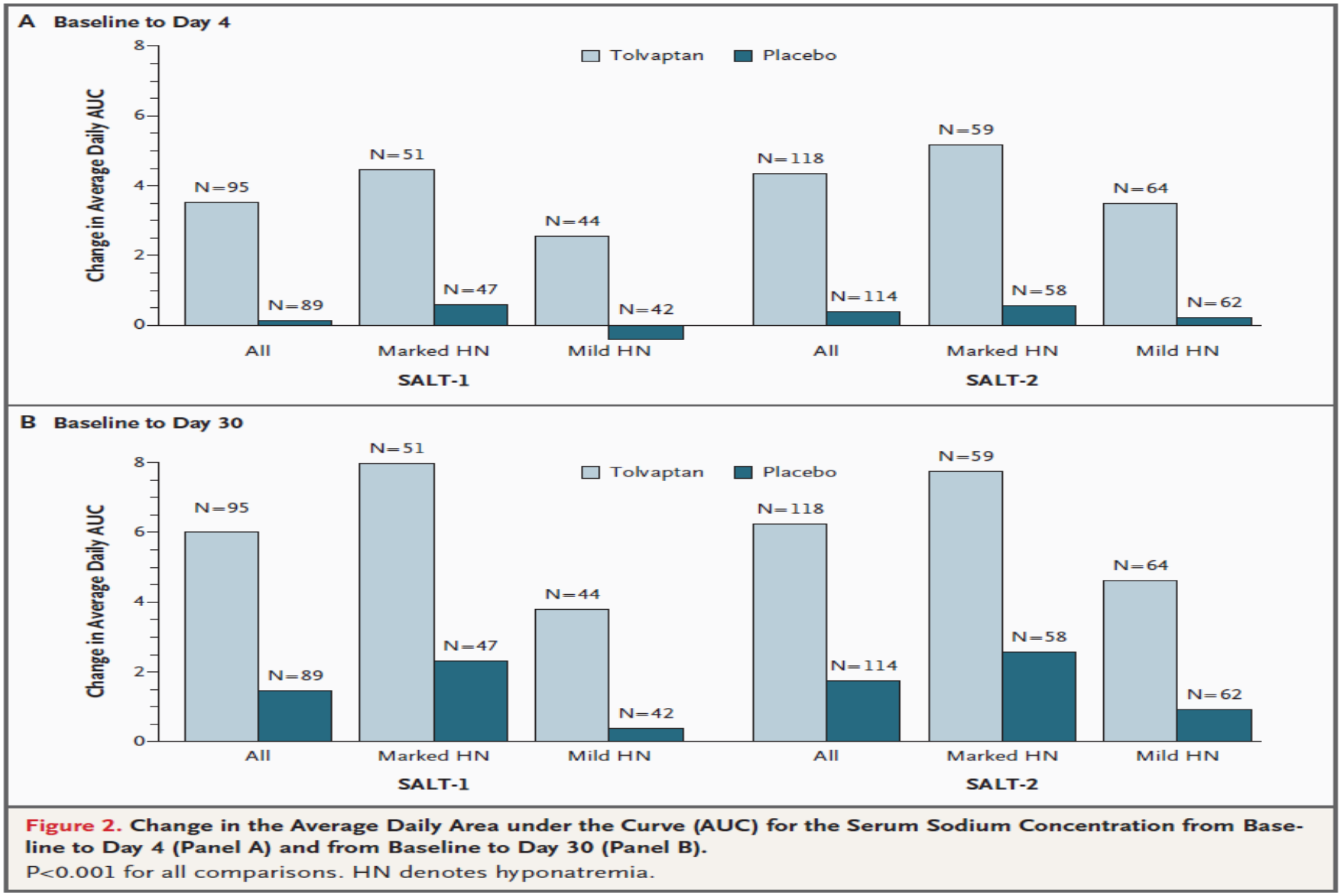


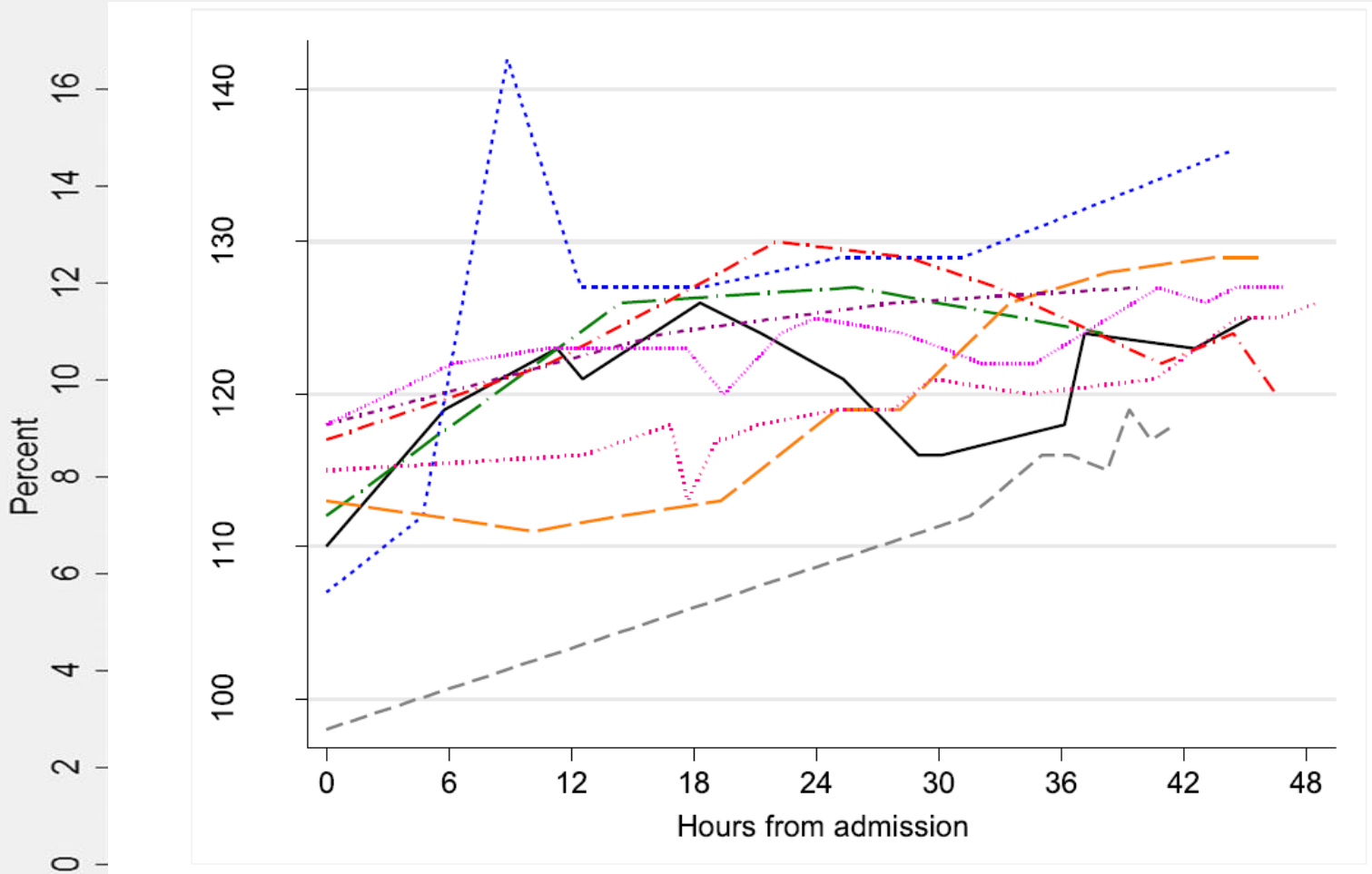
Figure 2. Change in the Average Daily Area under the Curve (AUC) for the Serum Sodium Concentration from Baseline to Day 4 (Panel A) and from Baseline to Day 30 (Panel B).
P<0.001 for all comparisons. HN denotes hyponatremia.

Schrier et al. N Engl J Med 2006

What are the risk factors for rapid Na^+ correction and osmotic demye

Method

Conclusi
correction occ
a documented



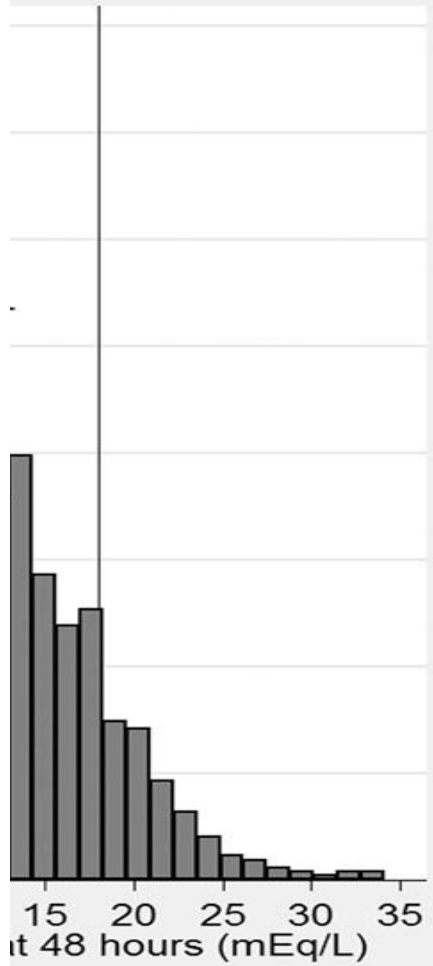
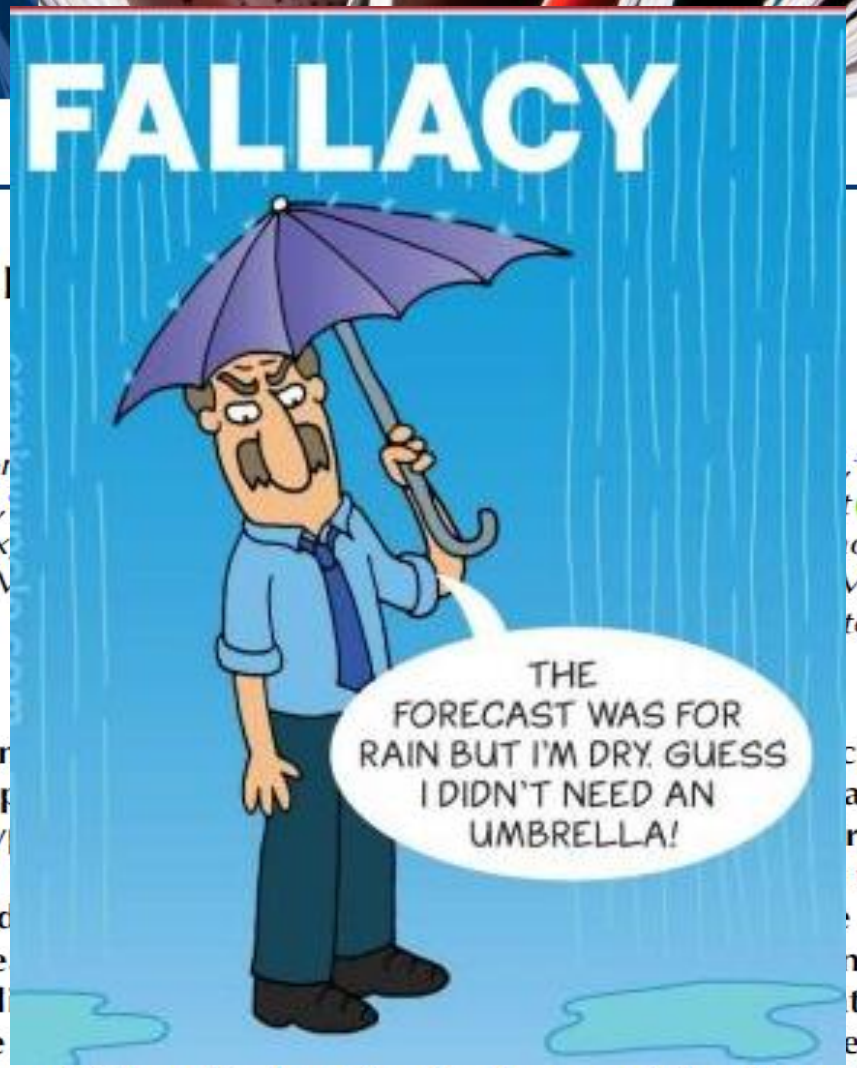


Figure 2. | Serum sodium trends during the first 24 and 48 hours of admission in patients with radiologic evidence of osmotic demyelination.

George et al, CJASN 2018

REVIEW



OPEN

Treatment Guideli
Stay the Course

Richard H. Sterns[1,2] *Helbert Rondor*[5] *Volker Burst*[6]
David M. Cohen,[7] *Mirjam Christ-Crain,*[11] *Aoife Garrahy*[12]
Fabrice Gankam-Kengne,[13] *John K. Hix*os E. Madias,[16]
Alessandro Peri[17] *Julie Refardt*[8] *M*M. Silver,[2] *Alain Soupart*[10]
Chris J. Thompson[19] *and Joseph G.* tors*

Abstract

International guidelines designed to r cur when correcting severe hyponatremia have been widely accep a recent large retrospective study of patients hospitalized with hy remia guidelines have gone too far in limiting the rate of rise of therapeutic caution and frequent monitoring of the serum sod assertions are reminiscent of a controversy that began many ye ntroversy, the evidence supporting the guidelines, and the val t current safeguards should not be abandoned. To do so would be emained dry in a rainstorm. The authors of this review, who represent 20 medical centers in nine countries, have all contributed significantly to the literature on the subject. We urge clinicians to continue to treat severe hyponatremia cautiously and to wait for better evidence before adopting less stringent therapeutic limits.

CJASN 19: 129–135, 2024. doi: https://doi.org/10.2215/CJN.0000000000000244

Due to the number of contributing authors, the affiliations are listed at the end of this article.

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