OSPEDALE NIGUARDA
CA' GRANDA

Sistema Sanitario Regione Lombardia

Traumatologia cranica: Terapia osmotica

Neurorianimazione
arturo.chieregato@ospedaleniguarda.it

10/03/2017

Immaginando la risposta filogenetica al trauma cranico

Trauma cranico “moderate”

tSAH

RBC che si lisano

lattato

Acidosi liquorale

iperventilazione

ipocapnia

vasocostrizione

Riduzione CBV

Danno diffuso/depolarizzazione

Riduzione CMRO2

Riduzione CBF

Riduzione CBV

Trasduzione genica

Proteine protettive

Digiuno

Riduzione input acqua (e Na+) e calorico

Riduzione sintesi albumina

Aumento sintesi proteine fase acuta

Iperaldosteronismo secondario

ipersodiemia

Iposodiemia

Iperattivazione adreno-ortosimpatico

Ipertensione arteriosa

iperglicemia

Deficit di diffusione glucosio

Riduzione ICP

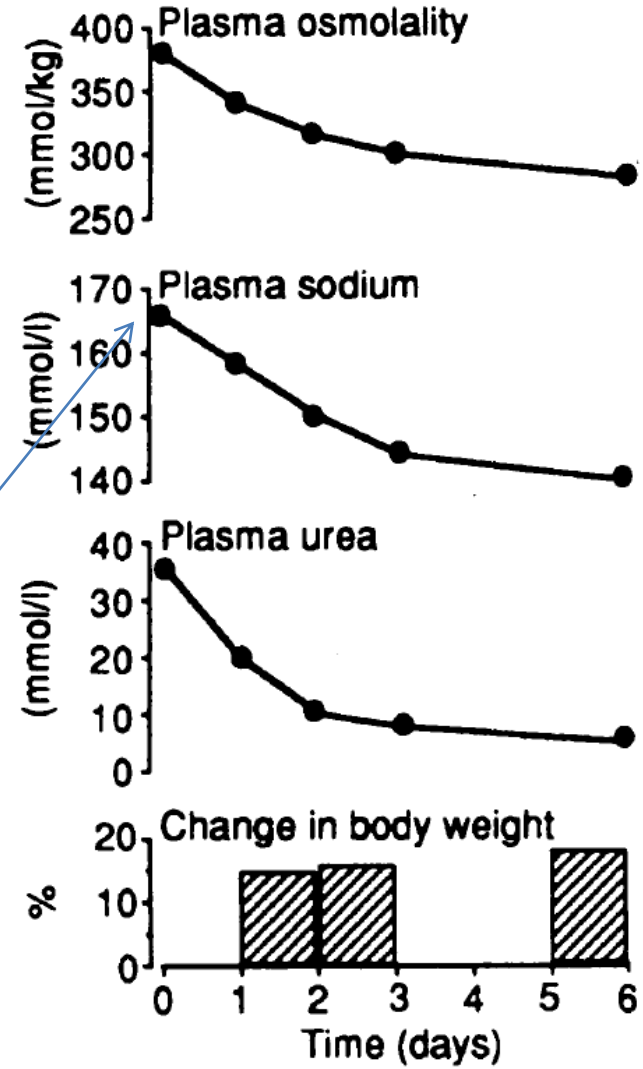
“neuroprotezione da ischemia”

“neuroprotezione da deficit glucidico”

Ipersodiemia nel digiuno

"Thirst strike": hypernatraemia and acute prerenal failure in a prisoner who refused to drink

BMJ 1992;**304**:1352



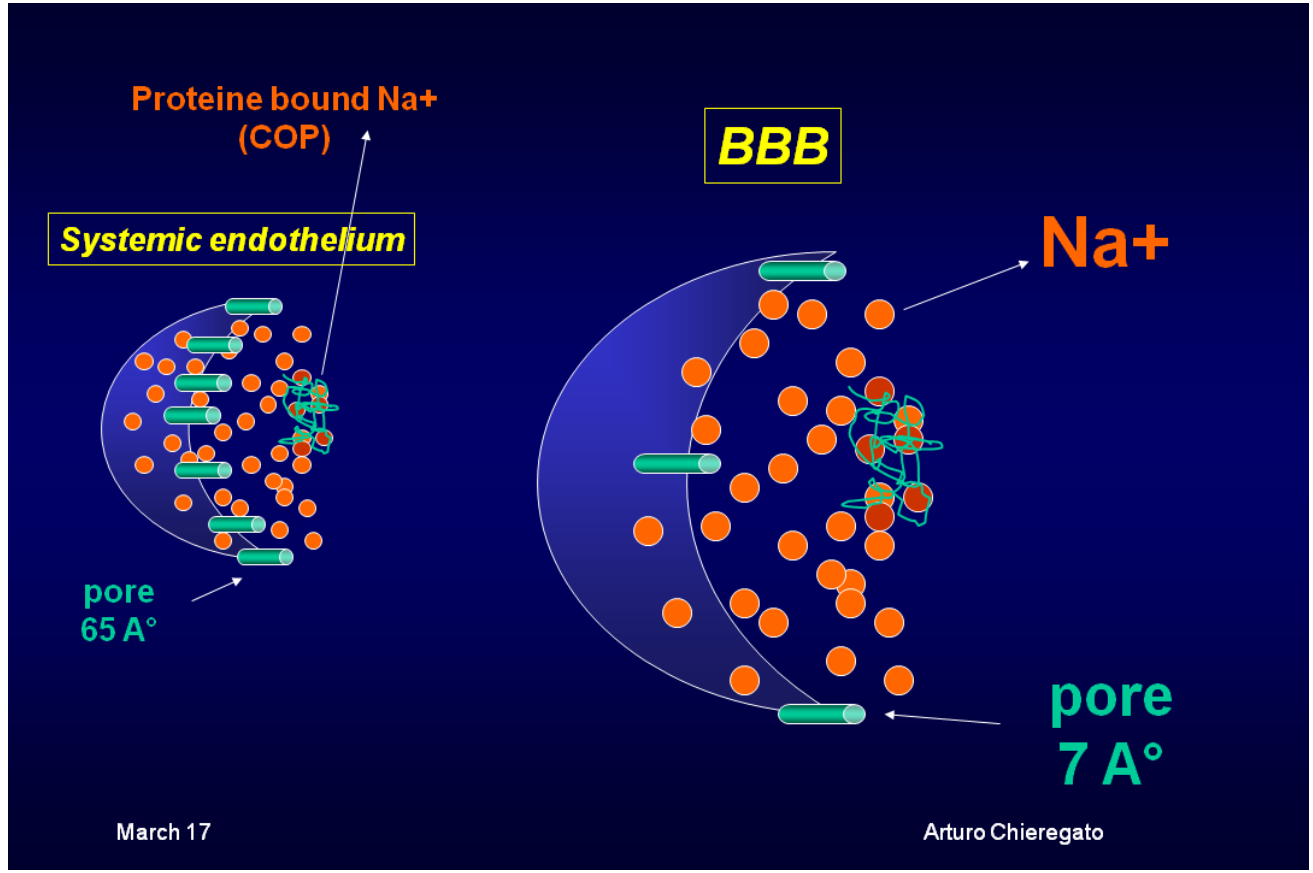
Changes in body weight (from value of 56 kg on admission), plasma urea concentration, sodium concentration, and osmolality during oral and parenteral rehydration

Paziente iperventilante, tachicardico, iperteso, iperglicemico, secco e *ipernatriemico*

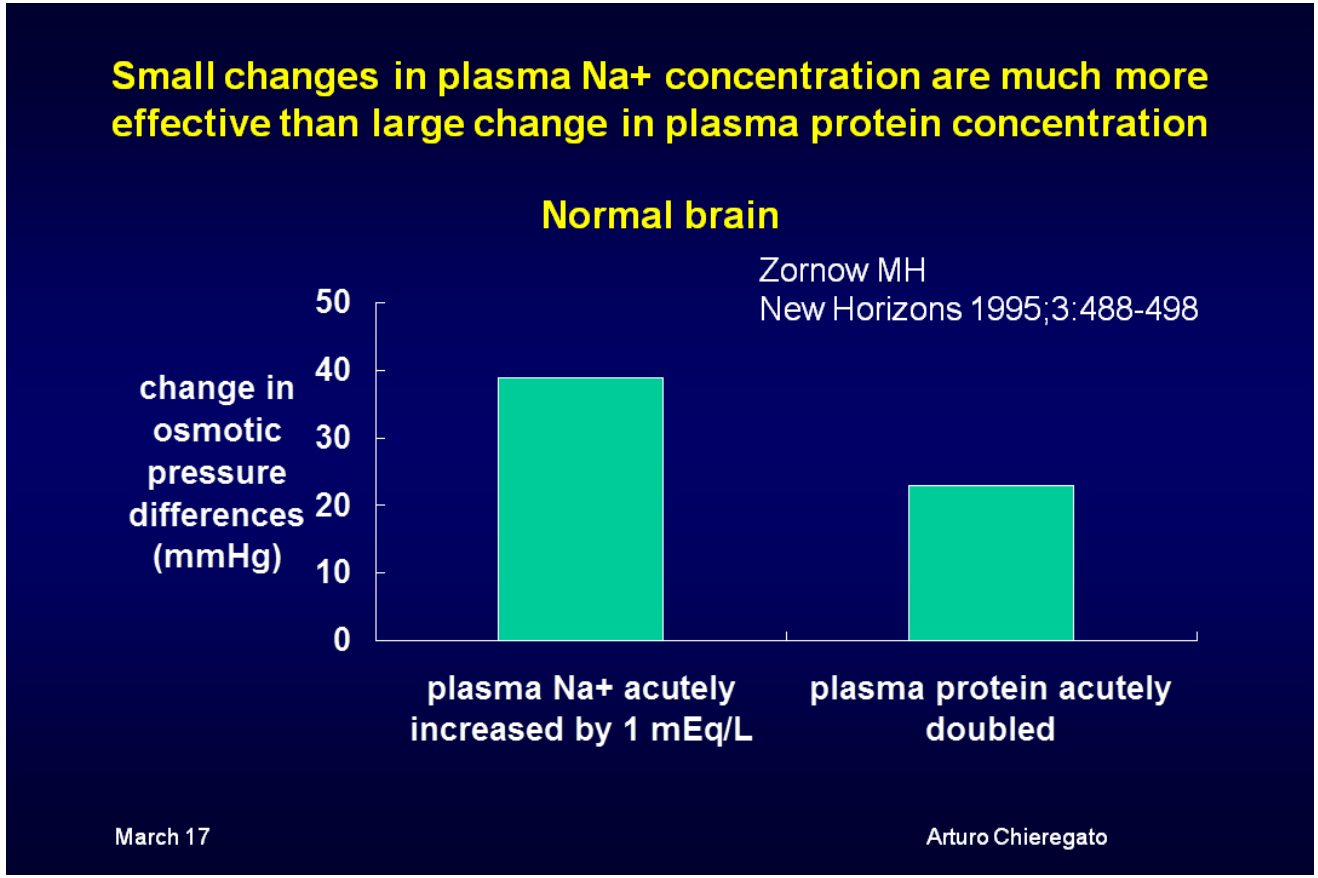
TBI moderate to severe

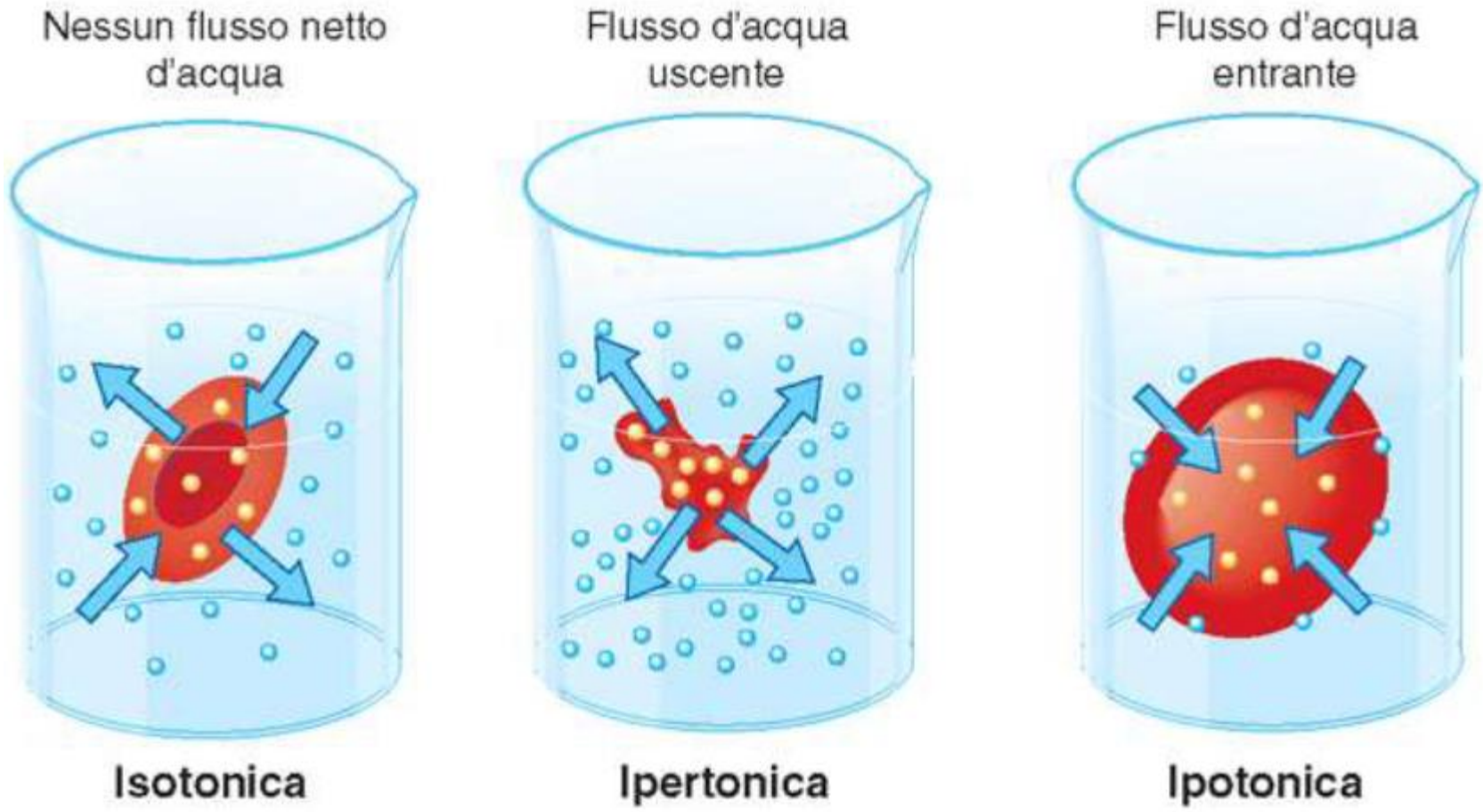
Nozioni di base

Semipermeabile per il Na+



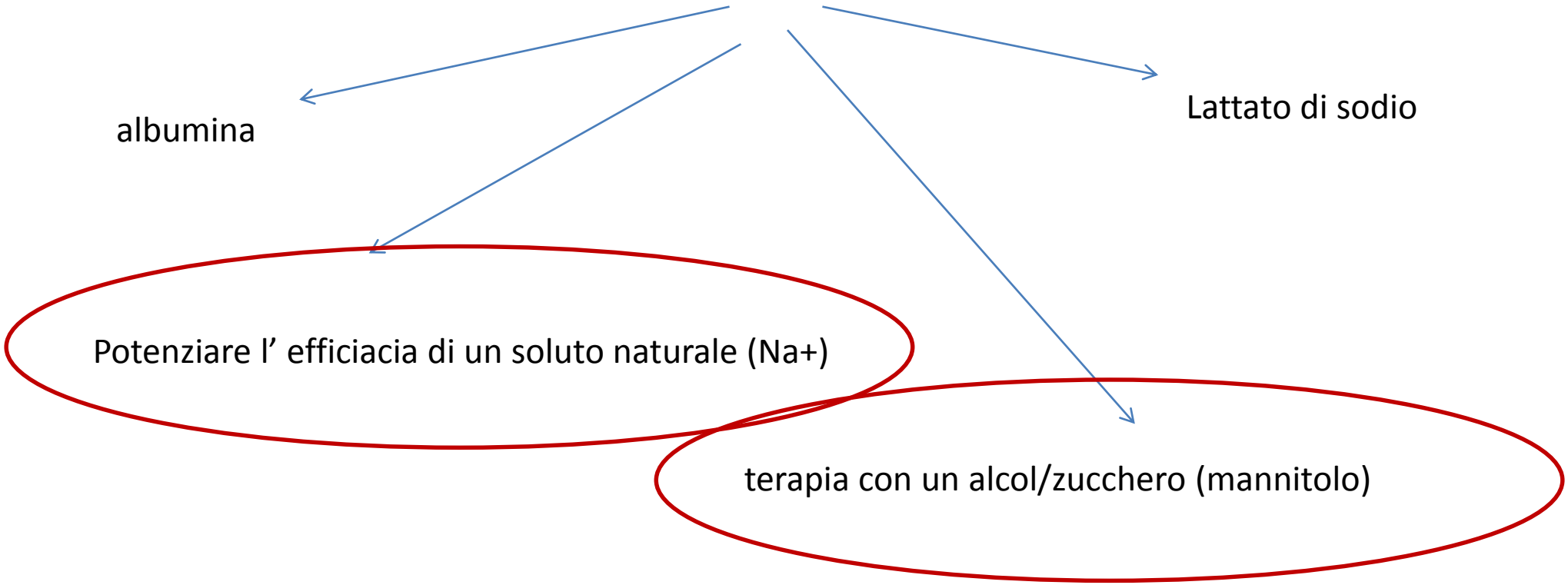
Pochissimo effetto albumina dipendente (BBB integra)





Dal web, Prof Davide Cervia

Gestione terapeutica



effetto vasocostrittivo

Gli osmotici

meccanismo

Richiama acqua dalla BBB

emodiluizione

Riduzione viscosità

Riduzione resistenze al flusso

aumenta CBF

Carico volemico

aumenta cardiac index

aumenta CBF

Autoregolazione pressoria

Vasocostrizione miogena

riduzione CBF

riduzione CBV

Hemodilution causes size-dependent constriction of pial arterioles in the cat

MARK L. HUDAK, M. DOUGLAS JONES, JR., ALEKSANDER S. POPEL, RAYMOND C. KOEHLER, RICHARD J. TRAYSTMAN, AND SCOTT L. ZEGER

Departments of Pediatrics (Eudowood Neonatal Pulmonary Division), Biomedical Engineering, Anesthesiology and Critical Care Medicine, and Biostatistics, The Johns Hopkins Medical Institutions, Baltimore, Maryland 21205

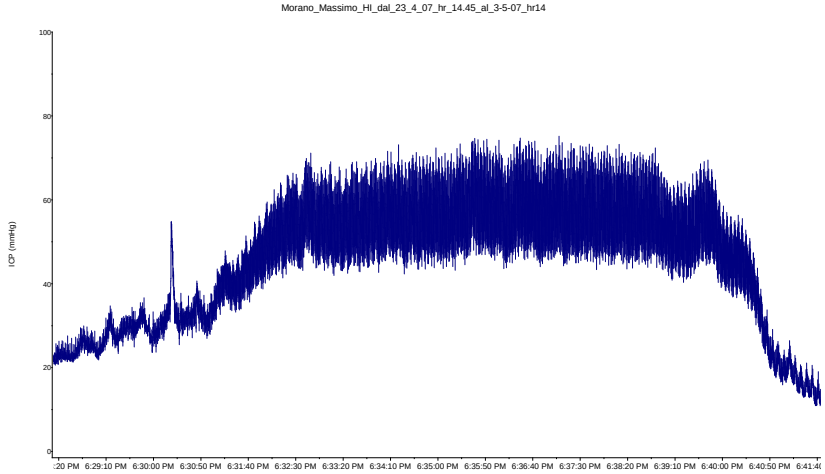
J Neurosurg 59:822–828, 1983

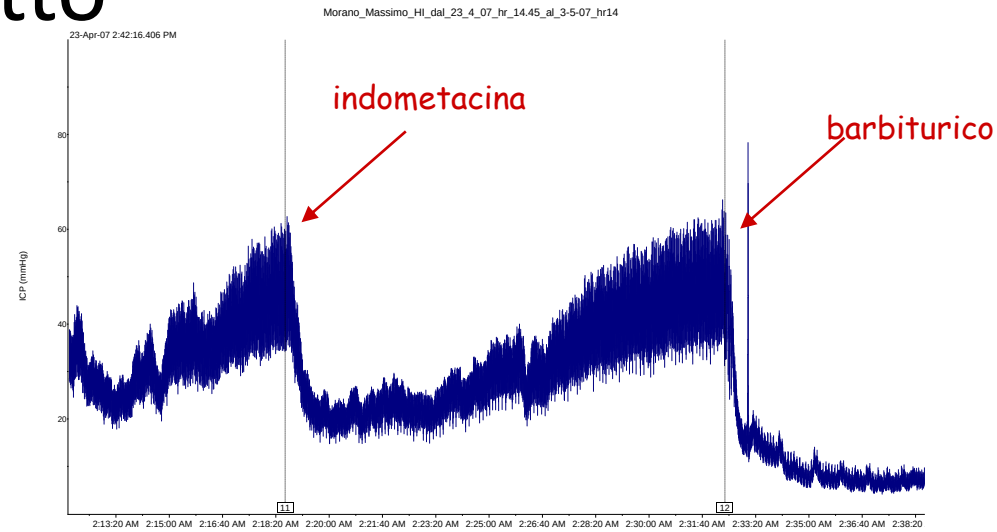
Mannitol causes compensatory cerebral vasoconstriction and vasodilation in response to blood viscosity changes

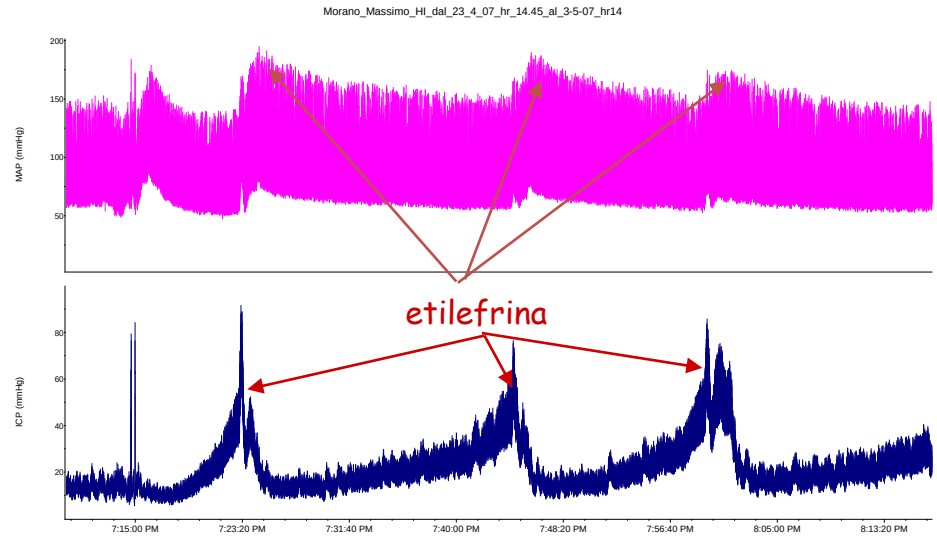
J. PAUL MUIZELAAR, M.D., PH.D., ENOCH P. WEI, PH.D., HERMES A. KONTOS, M.D., PH.D., AND DONALD P. BECKER, M.D.

Division of Neurosurgery and Department of Medicine, Medical College of Virginia, Richmond, Virginia, and Department of Neurosurgery, Wilhelmina Gasthuis, University of Amsterdam, Amsterdam, The Netherlands

Chi controlla la ICP istantanemente è un vasocostrittore indiretto







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effetto di sottrazione acqua, se BBB integra

Gli osmotici

BBB integra

Mannitol bolus preferentially shrinks non-infarcted brain in patients with ischemic stroke

Article abstract—Changes in brain tissue volume in six patients who had acute complete middle cerebral artery (MCA) infarctions and CT evidence of midline shift were measured using the brain boundary shift integral (BBSI) on sequential T1-weighted MR images acquired before and after a 1.5-g/kg bolus infusion of mannitol. At 50 to 55 minutes after the baseline scan, total brain volume decreased by 8.1 ± 2.8 mL (0.6%, $p < 0.005$). Brain in the noninfarcted hemisphere shrank more (0.8 ± 0.4%) than in the infarcted hemisphere (0.0 ± 0.5%, $p < 0.05$).

NEUROLOGY 2001;57:2120–2122

T.O. Videen, PhD; A.R. Zazulia, MD; E.M. Manno, MD; C.P. Derdeyn, MD; R.E. Adams, MD; M.N. Diringer, MD; and W.J. Powers, MD

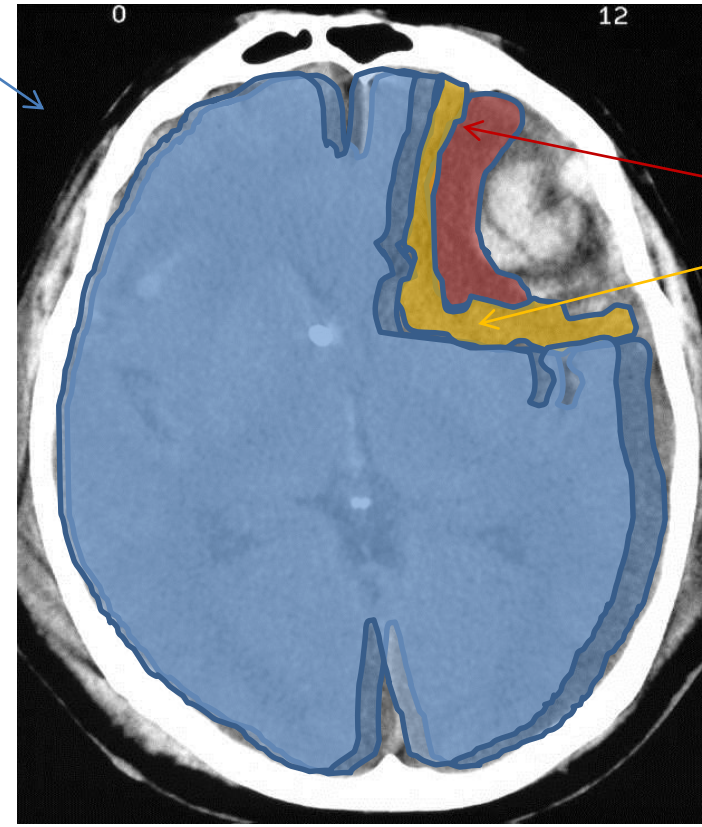
CASE REPORT

Determination of cerebral water content by magnetic resonance imaging after small volume infusion of 18% hypertonic saline solution in a patient with refractory intracranial hypertension

M. SALTARINI[1]*, D. MASSARUTTI[1], M. BALDASSARRE[1], G. NARDI[2], C. DE COLLE[3] and G. FABRIS[3]

BBB integra

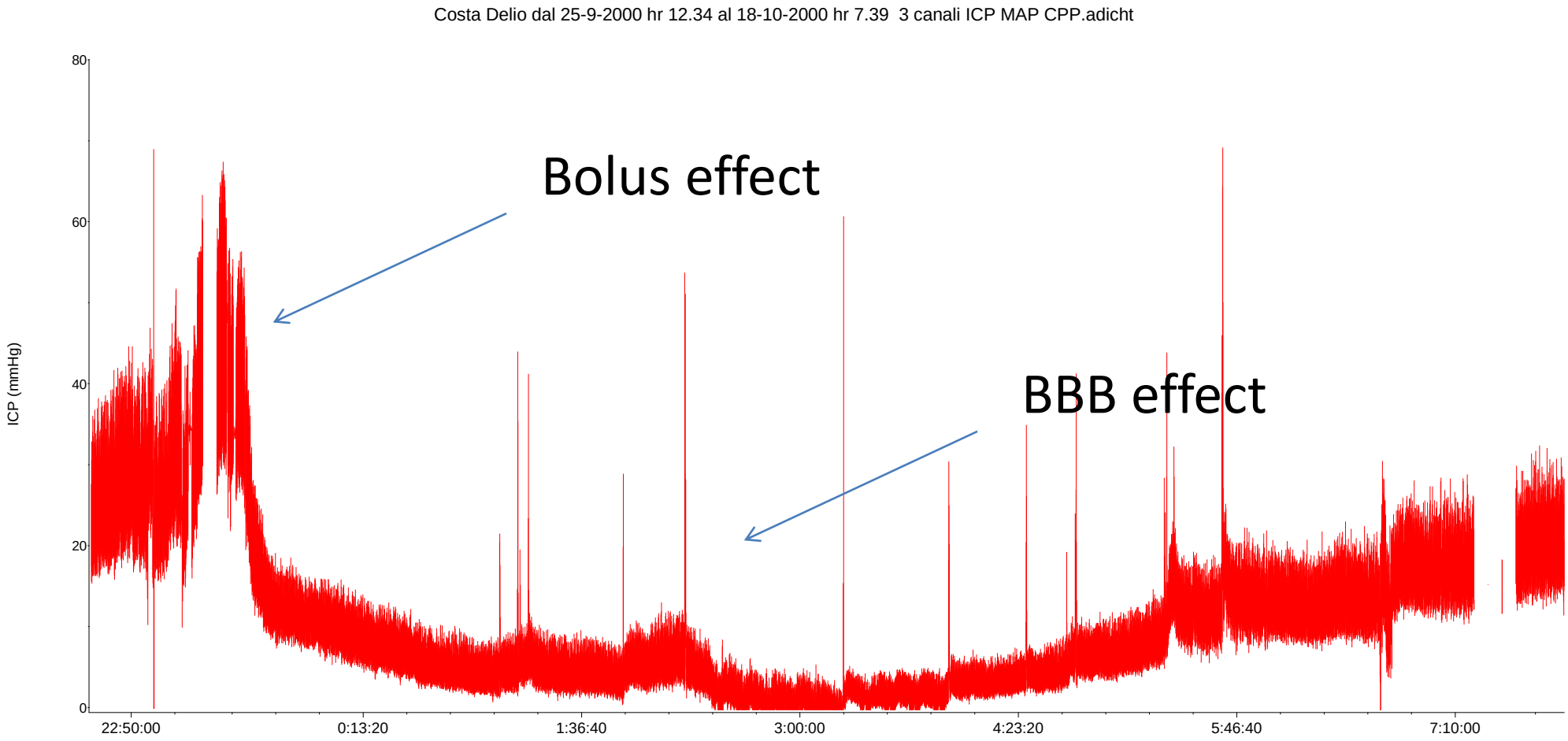
Cervello tipo 1



Cervello tipo 2

arturo.chieregato@ospedaleniguarda.it

Per riassumere



Esperienza personale

- professo dal 1990,
  - scoperto il Na+ nel 1996
- Esperienza diretta su mannitolo
  - Indiretta su saline
- Mai rimpiazzo di ipoalbuminemia

Saline ipertoniche

equipollenza:
la maneggevolezza della HS rende maggiore la dose di ipertonica e quindi l' efficacia?

JNS
CLINICAL ARTICLE
J Neurosurg 122:202–210, 2015

Hypertonic saline reduces cumulative and daily intracranial pressure burdens after severe traumatic brain injury

Halinder S. Mangat, MD,[1,2,3] Ya-Lin Chiu, MS,[3] Linda M. Gerber, PhD,[3,4] Marjan Alimi, MD,[2,3] Jamshid Ghajar, MD, PhD,[2,4] and Roger Härtl, MD[2,3]

Mannitol Dose (ml)

0 - 300 ml
300 - 600 ml
600 - 1200 ml
1200 - 2400 ml

TABLE 1. Equiosmolar dose-range calculation for mannitol and HTS

Mannitol Dose (g)	3% HTS Dose (ml)	23.4% HTS Dose (ml)	Osmolality (mOsm)
0–50	0–268	0–34	0–275
51–100	269–536	35–68	276–550
101–200	537–1072	69–136	551–1100
201–600	1073–3216	137–408	1101–3300

Ipertonica induce meno diuresi

■ CLINICAL INVESTIGATIONS

Anesthesiology 2007; 107:697–704

Effect of Equiosmolar Solutions of Mannitol* versus *Hypertonic Saline on Intraoperative Brain Relaxation and Electrolyte Balance

Irene Rozet, M.D., Nuj Tontisirin, M.D.,† Saipin Muangman, M.D.,† Monica S. Vavilala, M.D.,‡ Michael J. Souter, M.B., Ch.B., F.R.C.A.,§ Lorri A. Lee, M.D.,§ M. Sean Kincaid, M.D.,‖ Gavin W. Britz, M.D., M.P.H.,# Arthur M. Lam, M.D., F.R.C.P.C.***

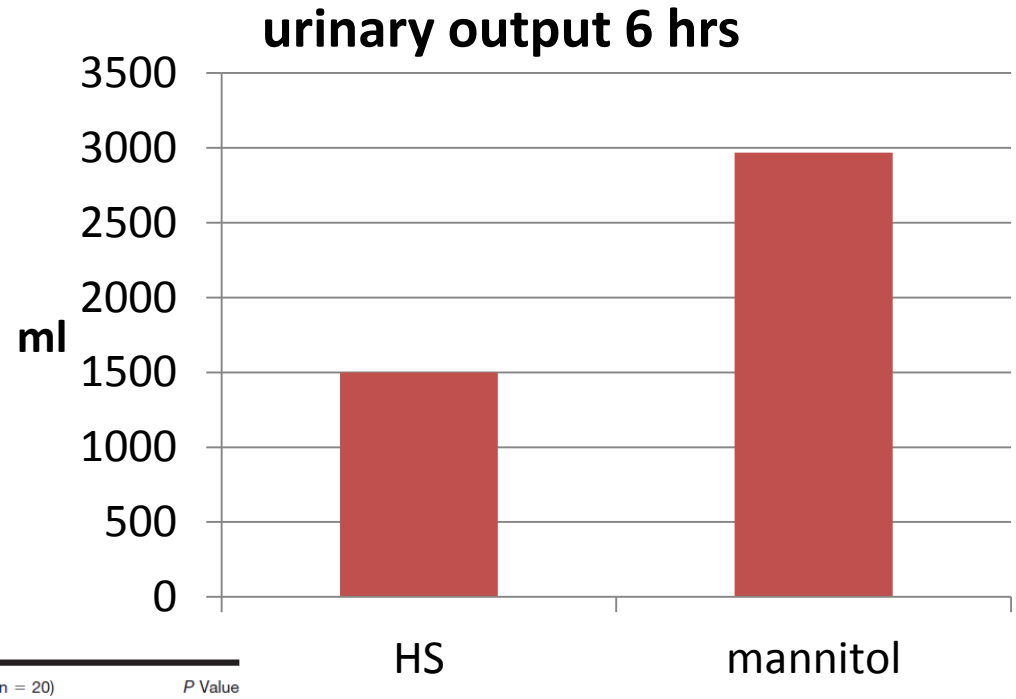


Table 3. Brain Relaxation and Fluid Balance in Hypertonic Saline and Mannitol Groups

	HS Group (n = 20)	Mannitol Group (n = 20)	*P* Value
Brain relaxation (n = 40)			
Number of patients with grade 1/2/3/4 (total)	6/8/6/0 (20)	5/9/4/2 (20)	0.86
Median grade (range)	2 (1–3)	2 (1–4)	
Non-SAH (n = 20)			
Number of patients with grade 1/2/3/4 (total)	6/4/0/0 (10)	5/5/0/0 (10)	0.66
Median grade (range)	1 (1–2)	1.5 (1–2)	
SAH (n = 20)			
Number of patients with grade 1/2/3/4 (total)	0/4/6/0 (10)	0/4/4/2 (10)	0.6
Median grade (range)	3 (2–3)	3 (2–4)	
Blood loss, ml	309 ± 45	305 ± 107	0.97
Fluid input, ml	4,335 ± 581	6,648 ± 1,346	0.16
Urine output 3 h, ml	908 ± 829	2,248 ± 1,443	0.001
Urine output 6 h, ml	1,500 ± 509	2,969 ± 1,600	0.06
Fluid balance 3 h, positive, ml	3,230 ± 1,543	1,638 ± 1,620	0.004
Fluid balance 6 h, positive, ml	2,850 ± 2,654	2,519 ± 1,330	0.7
Number of patients given two boluses (%)	6 (30)	6 (30)	1

Four-point scale score of brain relaxation: 1 = perfectly relaxed, 2 = satisfactorily relaxed, 3 = firm (leveled) brain, 4 = bulging brain. Intravenous fluids included crystalloids and colloids. There was no difference between groups.

HS = hypertonic saline; SAH = subarachnoid hemorrhage.

L’ ipertonica potrebbe esitare in un cambiamento della sodiemia basale

CLINICAL STUDIES

Marcus L. Ware, M.D., Ph.D.
Department of Neurological Surgery, University of California, San Francisco, San Francisco, California

Effects of 23.4% Sodium Chloride Solution in Reducing Intracranial Pressure in Patients with Traumatic Brain Injury: A Preliminary Study

TABLE 4. Effect of 23.4% saline administration on intracranial pressure, mean arterial pressure, and serum sodium values*

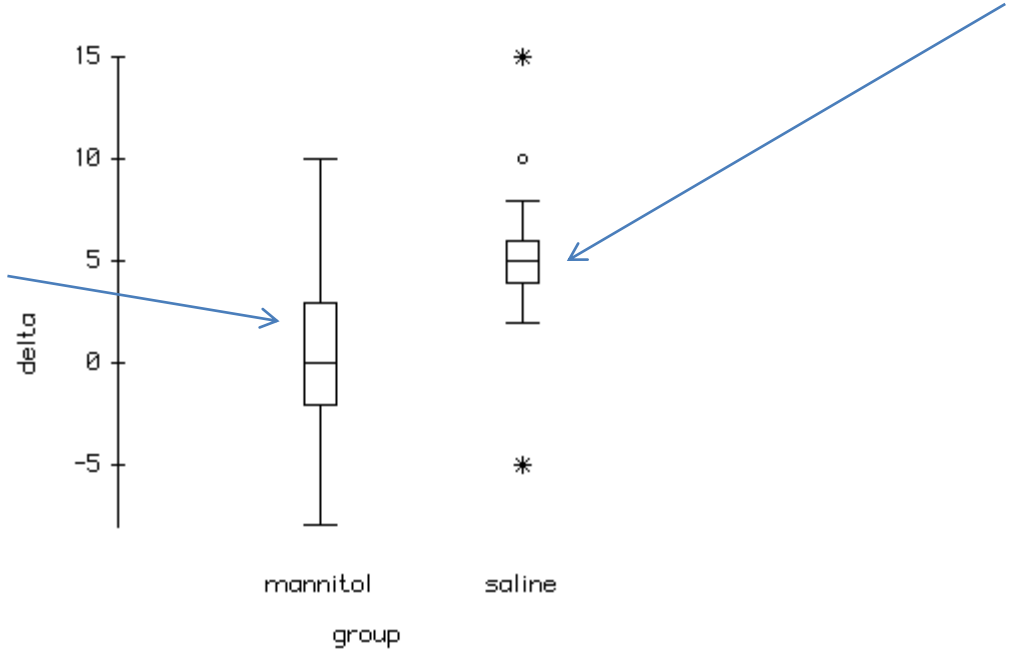
Patient no.	ICP (mmHg) peak/nadir (% reduction)	Duration of effect (min)	MAP (mmHg) before/after	Serum sodium (mEq/L) before/after
1	24/15 (38%)	90	125/122	137/140
2	35/6 (83%)	240	89/94	127/137
3	26/15 (42%)	180	122/96	139/144
4.1	38/6 (84%)	120	106/50	150/152
4.2	63/55 (13%)	No effect	78/83	152/NA
5.1	30/11 (63%)	60	87/90	128/143
5.2	33/7 (79%)	60	98/105	150/154
5.3	37/7 (81%)	120	121/112	151/156
5.4	27/7 (74%)	180	104/112	151/154
5.5	28/7 (75%)	180	118/116	151/157
5.6	37/10 (73%)	60	123/118	150/158
6.1	32/6 (81%)	60	121/92	139/144
6.2	33/4 (88%)	240	123/116	141/146
7	26/13 (50%)	90	96/113	137/140
8	56/37 (34%)	5	106/117	141/156
9	88/66 (25%)	No effect	103/121	144/150
10.1	20/10 (50%)	60	109/94	136/143
10.2	21/19 (10%)	15	128/133	143/146
11	29/22 (24%)	15	NA/NA	135/140
12	22/18 (18%)	60	85/86	144/149
13.1	21/16 (24%)	180	93/107	140/135
13.2	24/14 (42%)	240	104/122	134/139
Mean	36/20 (44%)	96	105/105	140/145
Median	29/15 (38%)	90	105/111	140/145
SD	19/16	76	13.9/16.7	5.9/6.4

*ICP, intracranial pressure; MAP, mean arterial pressure; NA, not available; SD, standard deviation. There were 22 treatments with 23.4% saline solution in the 13 patients. Repeat treatments were given as indicated to the patients listed with decimals (e.g., 4.1, 4.2).

TABLE 3. Effect of mannitol administration on intracranial pressure, mean arterial pressure, and serum sodium values*

Patient no.	Mannitol dosage in grams (g/kg)	ICP (mm Hg) peak/nadir (% reduction)	Duration of effect (min)	MAP (mm Hg) before/after	Serum sodium (mEq/L) before/after
1	50 (0.85)	45/16 (64%)	150	102/114	134/138
2	60 (1.02)	48/2 (96%)	15	100/92	127/137
3	100 (0.68)	28/11 (61%)	15	99/105	136/139
4	50 (0.85)	40/7 (83%)	225	88/104	149/150
5.1	50 (0.66)	25/10 (60%)	30	87/84	145/142
5.2	35 (0.46)	31/7 (77%)	75	103/97	149/150
5.3	35 (0.46)	43/7 (84%)	30	133/132	151/151
5.4	50 (0.66)	25/8 (68%)	45	111/114	156/151
5.5	35 (0.46)	38/11 (71%)	30	115/118	159/151
5.6	35 (0.46)	34/8 (76%)	45	105/90	157/156
6	100 (1.94)	38/11 (71%)	60	117/117	143/143
7	50 (0.68)	40/3 (93%)	90	111/114	143/142
8	100 (1.68)	54/48 (11%)	No effect	110/106	136/141
9	50 (0.79)	68/65 (4%)	No effect	94/103	145/144
10	100 (1.02)	31/10 (68%)	60	84/98	143/144
11	100 (1.13)	25/21 (16%)	No effect	105/103	137/135
12	100 (1.12)	24/15 (38%)	105	86/87	144/144
13.1	100 (0.92)	22/12 (45%)	60	87/82	139/136
13.2	50 (0.46)	24/14 (42%)	150	99/104	134/139
Mean	75 (0.86)	38/18 (56%)	59	100/103	147/145
Median	75 (0.79)	39/11 (66%)	74	101/105	143/142
SD	24 (0.40)	13/19	22	11/9	6/5

*ICP, intracranial pressure; MAP, mean arterial pressure; SD, standard deviation. There were 19 treatments with mannitol in the 13 patients. Repeat treatments were given as indicated to the patients listed with decimal figures (e.g., 5.1, 5.2).



Da qui nasce dubbio/pregiudizio di metodo sulla HS…

Nephrol Dial Transplant (2006) 21: 70–76
doi:10.1093/ndt/gfi082
Advance Access publication 2 September 2005

Original Article

Nephrology Dialysis Transplantation

Development of severe hyponatraemia in hospitalized patients: treatment-related risk factors and inadequate management

Ewout J. Hoorn[1], Jan Lindemans[2] and Robert Zietse[1]

Diagnosis and management of hyponatremia in acute illness

Robert W. Schrier and Shweta Bansal

Current Opinion in Critical Care 2008, 14:627–634

REVIEW

Disturbances of Sodium in Critically Ill Adult Neurologic Patients

A Clinical Review

Martin Tisdall, MRCS, Matthew Crocker, MRCS, Jonathan Watkiss, FRCA, and Martin Smith, FRCA

nephron Physiology

Minireview

Nephron Physiol 2008;108:p46–p59 Published online: March 5, 2008
DOI: 10.1159/000119709

Hyponatremia Revisited: Translating Physiology to Practice

Ewout J. Hoorn Robert Zietse

Q J Med 2005; **98**:529–540
doi:10.1093/qjmed/hci081

Advance Access publication 13 June 2005

Commentary

QJM

Diagnostic approach to a patient with hyponatraemia: traditional versus physiology-based options

E.J. HOORN[1], M.L. HALPERIN[2] and R. ZIETSE[1]

REVIEW

HYPONATREMIA IN NEUROSURGICAL PATIENTS: CLINICAL GUIDELINES DEVELOPMENT

Maryam Rahman, M.D.
Department of Neurosurgery,
University of Florida,
Gainesville, Florida

OBJECTIVE: Neurosurgical patients have a high risk of hyponatremia and associated complications. We critically evaluated the existing literature to identify the determinants for the development of hyponatremia and which management strategies provided the best outcomes.

Neurosurgery 65:925–936, 2009

Review Article

Cerebral salt wasting: Truths, fallacies, theories, and challenges

Sheila Singh, MD; Desmond Bohn, MB; Ana P. C. P. Carlotti; Michael Cusimano, MD; James T. Rutka; Mitchell L. Halperin, MD

the intensive care unit, hyponatremia should not develop. (Crit Care Med 2002; 30:2575–2579)

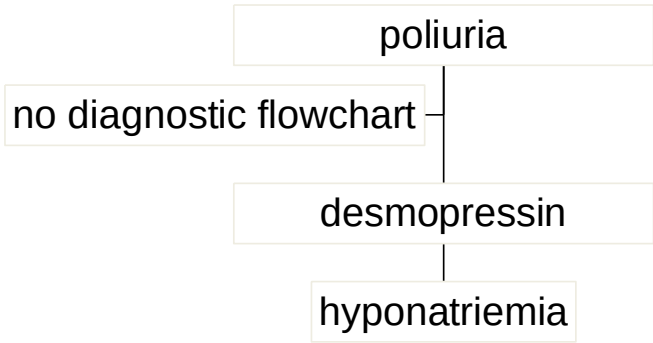
Clinical Endocrinology (2007) **66**, 367–372 doi: 10.1111/j.1365-2265.2007.02741.x

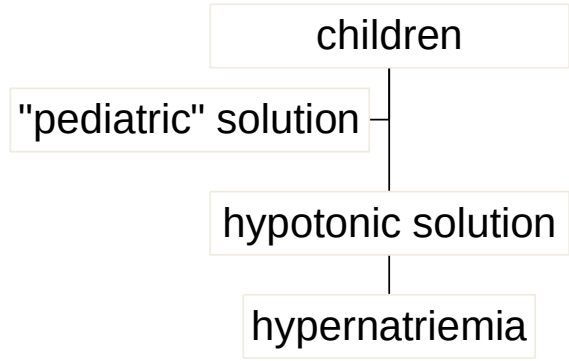
ORIGINAL ARTICLE

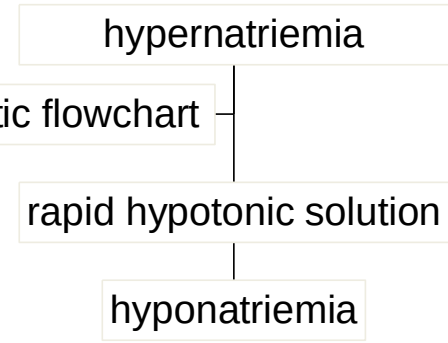
Novel risk factors for hospital-acquired hyponatraemia: a matched case–control study

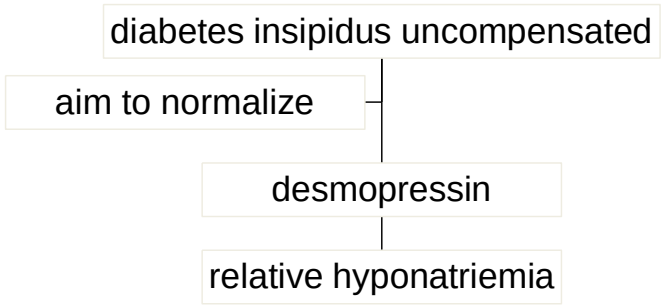
Carolien M. Beukhof*‡, Ewout J. Hoorn*‡, Jan Lindemans† and Robert Zietse*

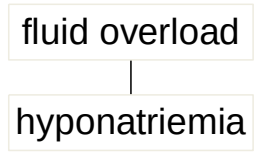
Errori medici frequenti

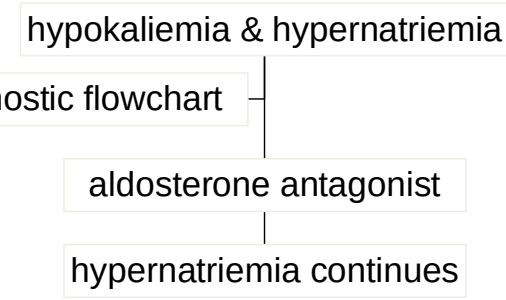


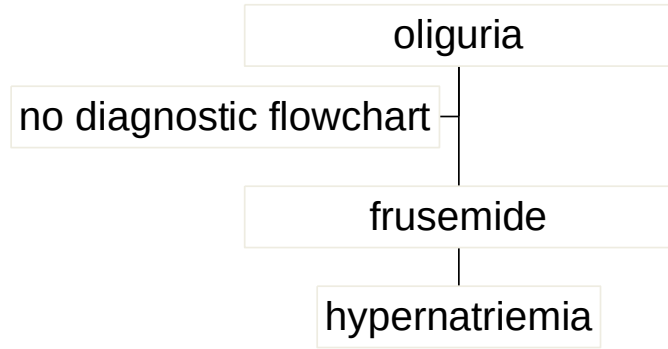


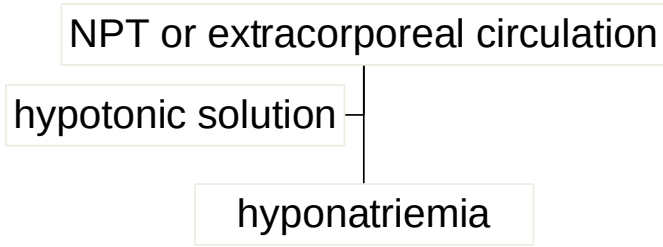












mannitolo

1961-1963

THE VALUE OF HYPERTONIC MANNITOL SOLUTION IN DECREASING BRAIN MASS AND LOWERING CEREBROSPINAL-FLUID PRESSURE*

BURTON L. WISE, M.D., AND NORMAN CHATER, M.D.
Division of Neurological Surgery, University of California School of Medicine, San Francisco, California

(Received for publication June 11, 1962)

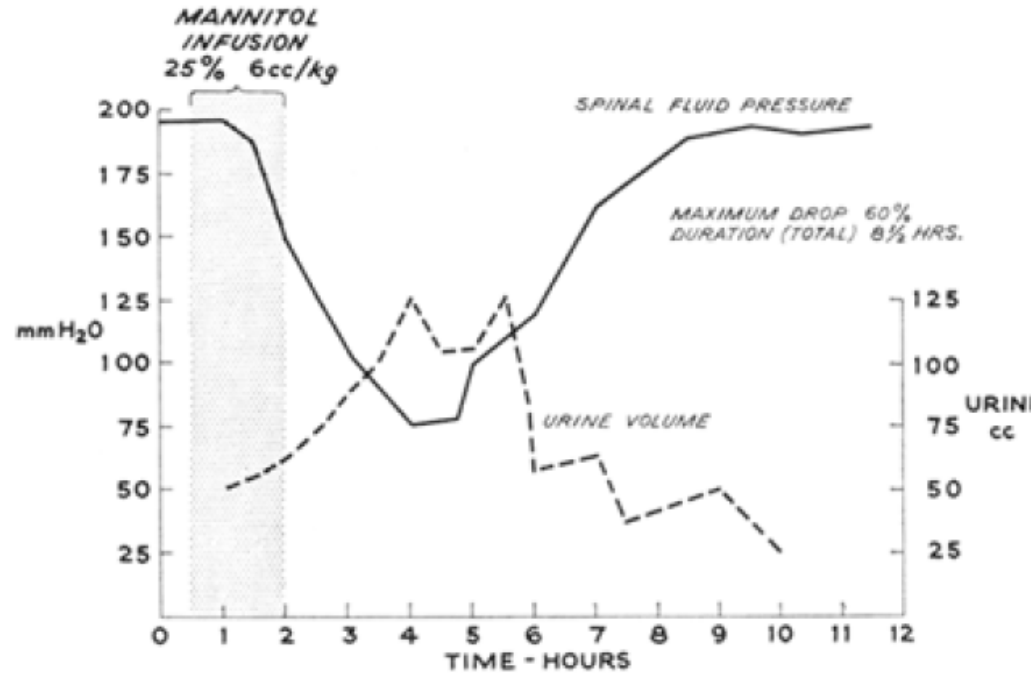
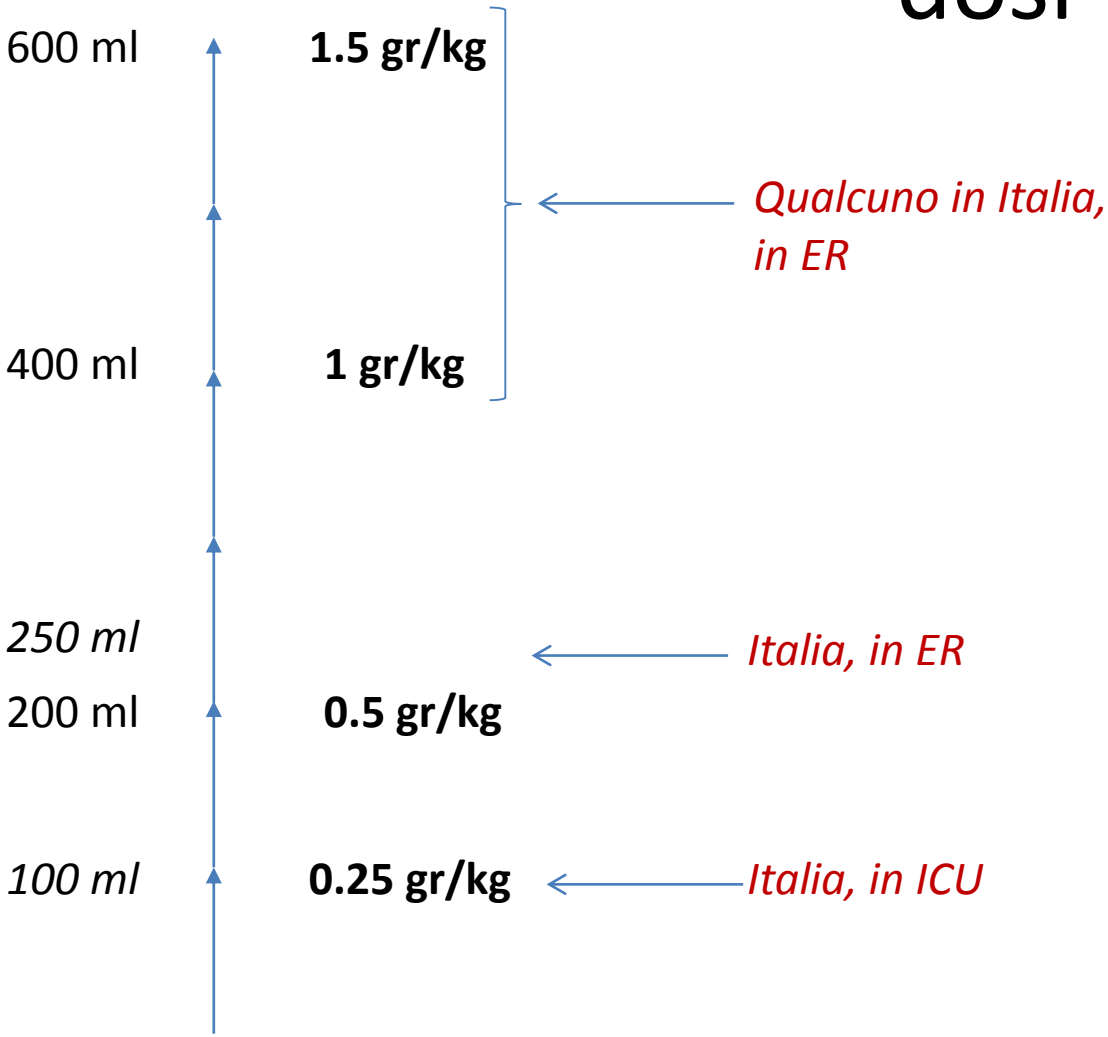


FIG. 1. Chart of cerebrospinal-fluid pressure of patient with glioma of thalamus, during and following intravenous infusion of 25 per cent mannitol solution, 1.5 gm./kg. Urinary volumes indicated by dotted lines.

Mannitolo (18%) dosi



Mannitolo 18%

High-Dose Mannitol

TO THE EDITOR: I was pleased to read the article by Cruz, et al. (Cruz J, Minoja G, Okuchi K, et al: Successful use of the new high-dose mannitol treatment in patients with Glasgow Coma Scale scores of 3 and bilateral abnormal pupillary widening: a randomized trial. *J Neurosurg 100:* 376–383, March, 2004), as well as the accompanying editorial by Marshall (Marshall LF: Editorial. High-Dose Mannitol. *J Neurosurg 100:*367–368, March, 2004). I was surprised, however, to find that a dose of 1.4 g/kg of mannitol was considered to be a "high dose."

tion into neurological surgery in 1961,[2] we used a dose of 1.5 to 2.5 g/kg. In 1962[3] we described its use in 70 patients. In that publication we described the usual dosage as 2.5 to 3.5 g/kg but we had used as much as 4.25 g/kg. There were no significant side effects or complications, except for one instance of hypovolemia. In fact, we found that the use of hypertonic mannitol during surgical procedures tended to counteract the usual mild hyponatremia that occurs on the first postoperative day.[1] Repeated use of mannitol, of course, requires management of fluid and electrolyte balance.

BURTON L. WISE, M.D.
Diplomatic American Board of Neurological Surgery
San Francisco, California

J. Neurosurg. / Volume 101 / September, 2004

Mannitolo
come

- A) injection
- B) infusione lenta per aumento ICP
- C) infusione lenta ad orari fissi

Modo A)
injection

Modo B)
Slow infusion su ICP target

THE VALUE OF HYPERTONIC MANNITOL SOLUTION IN DECREASING BRAIN MASS AND LOWERING CEREBROSPINAL-FLUID PRESSURE*

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Division of Neurological Surgery, University of California School of Medicine, San Francisco, California

(Received for publication June 11, 1962)

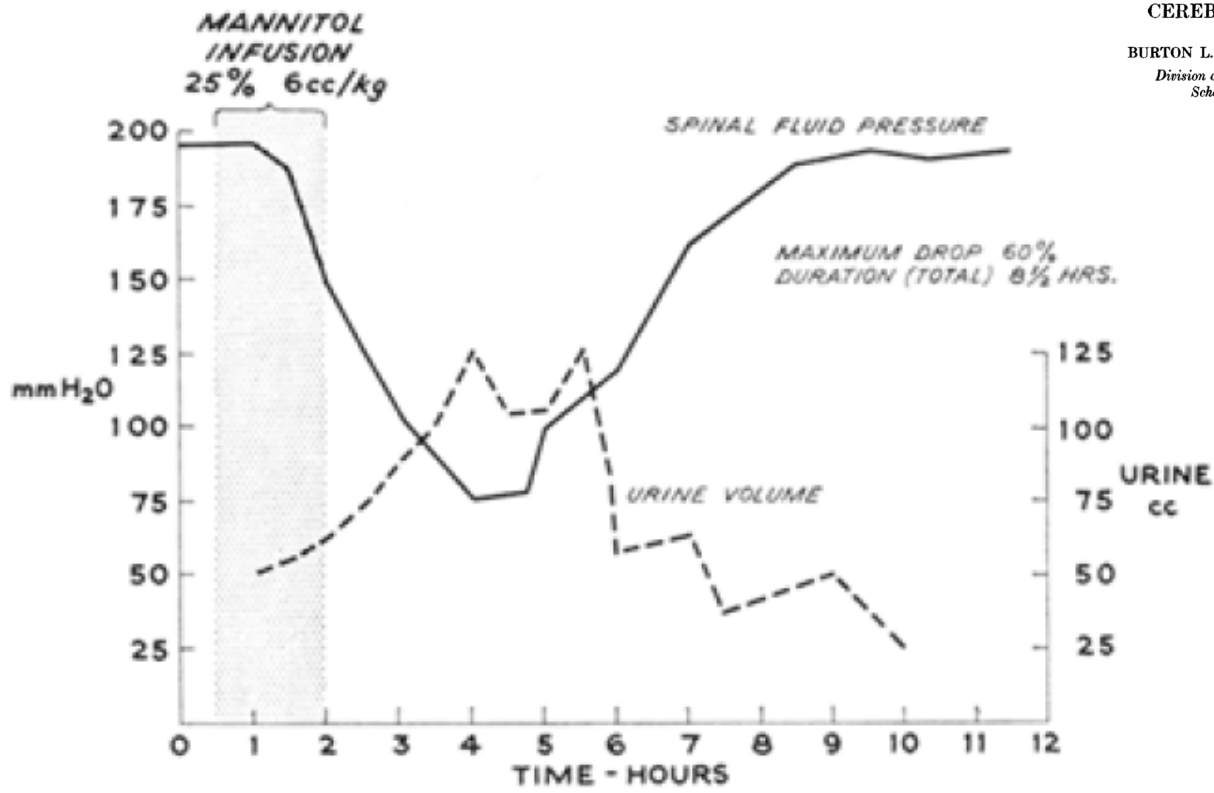


FIG. 1. Chart of cerebrospinal-fluid pressure of patient with glioma of thalamus, during and following intravenous infusion of 25 per cent mannitol solution, 1.5 gm./kg. Urinary volumes indicated by dotted lines.

Modo C)

Terapia standard (scheduled dosage) indipendente da ICP

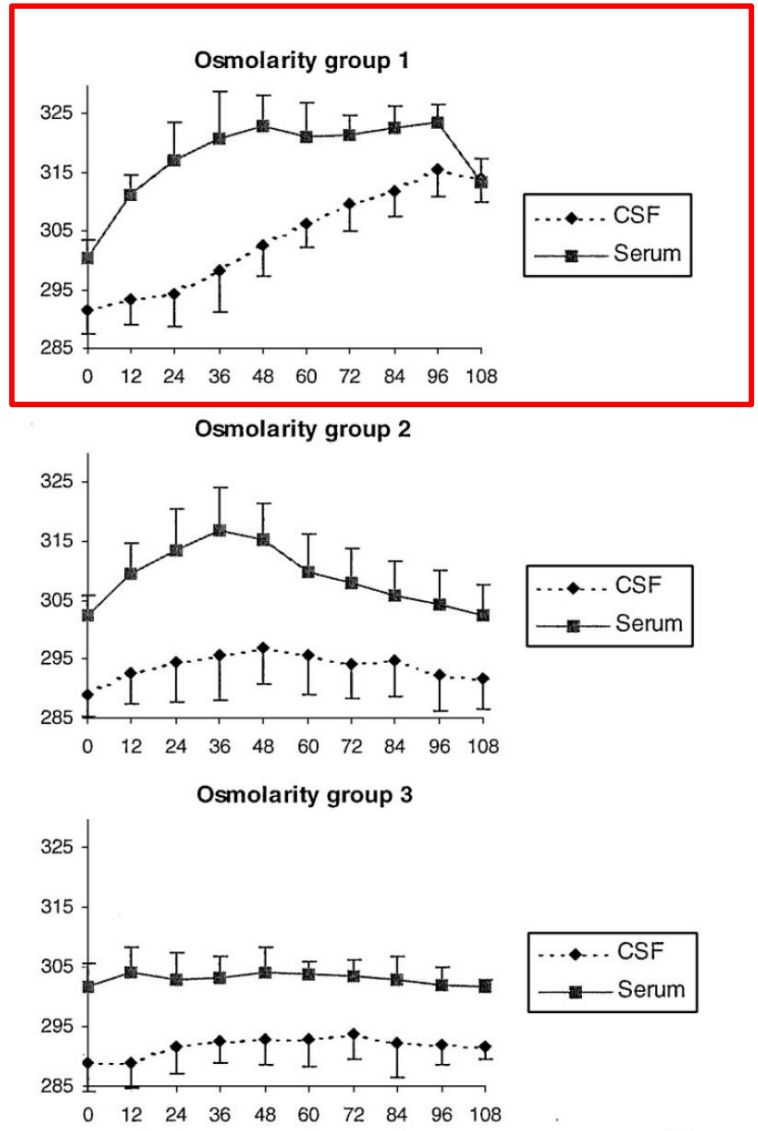
Neurologic Critical Care

Increases in spinal fluid osmolarity induced by mannitol

K. H. Polderman, MD, PhD; G. van de Kraats, MD; J. M. Dixon, MD; W. P. Vandertop, MD, PhD; A. R. J. Girbes, MD, PhD

Crit Care Med 2003 Vol. 31, No. 2

Figure 2. Changes in serum and cerebrospinal fluid (*CSF*) osmolarity in groups 1–3. *Top panel*, osmolarity in serum and cerebrospinal fluid during long-term mannitol administration. Initially, serum osmolarity increased whereas cerebrospinal fluid osmolarity remained unchanged; this is the effect that is desired, theoretically enabling extraction of water (i.e., edema) from the edematous brain. After 48 to 60 hrs, plasma osmolarity stabilized at a higher level, but cerebrospinal fluid osmolarity also began to increase. After halving the dose of mannitol at t = 96 hrs, serum osmolarity decreased, but cerebrospinal fluid osmolarity remained unchanged, causing a temporary equilibration of the two values. *Middle panel*, osmolarity in serum and cerebrospinal fluid during short-term (24 to 48 hrs) mannitol administration. There was an increase in serum osmolarity, and the gap between serum and cerebrospinal fluid osmolarity widened although cerebrospinal fluid osmolarity also increased. The dose of mannitol was tapered after 24 to 48 hrs, leading to a decrease in both serum and cerebrospinal fluid osmolarity. *Bottom panel*, osmolarity in serum and cerebrospinal fluid in patients not receiving mannitol. Serum and cerebrospinal fluid osmolarity remained unchanged.

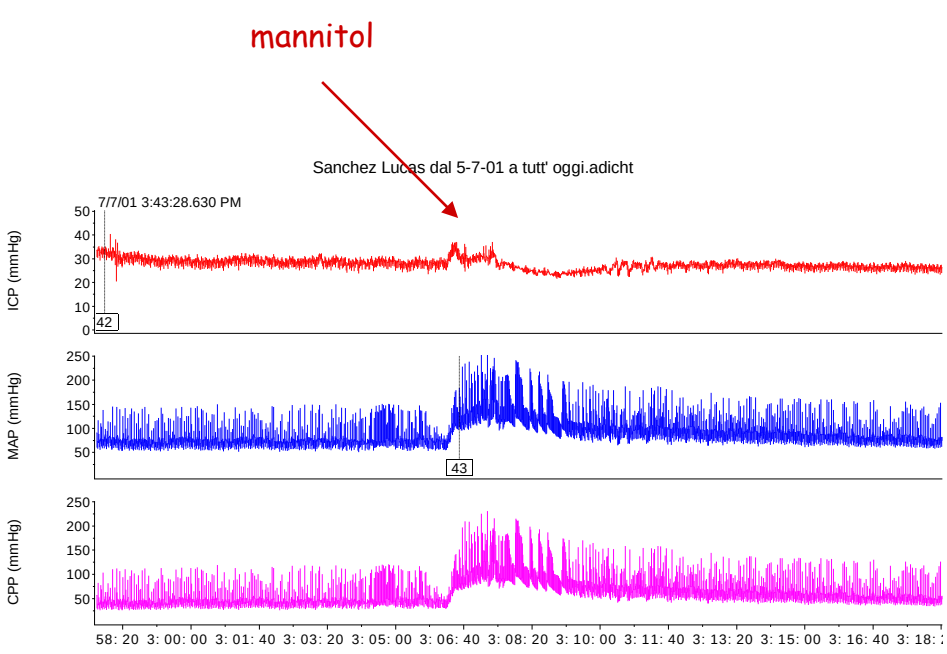


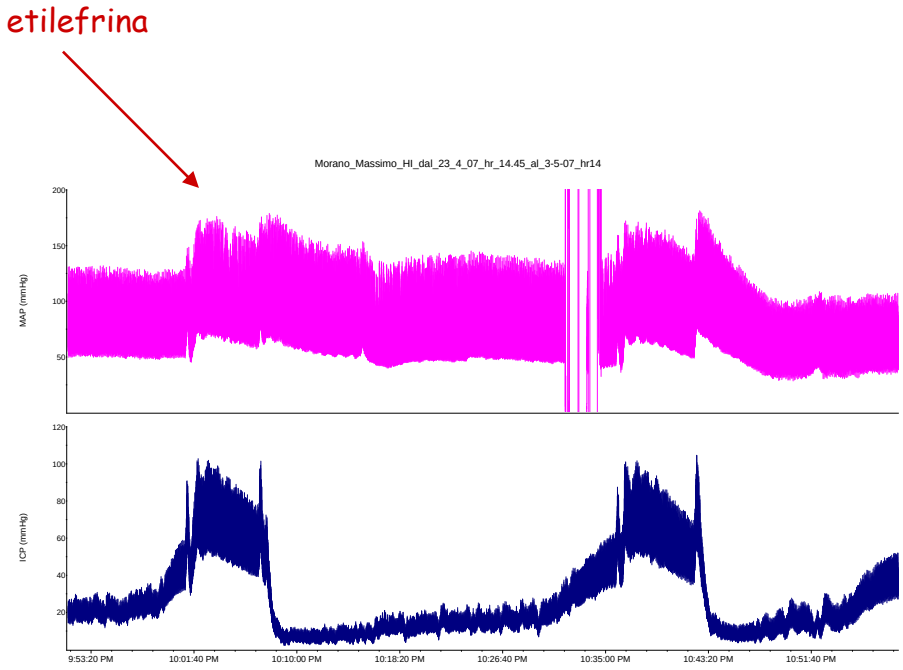
Mannitolo o salina, effetti paradossi

Mannitolo o salina, effetti paradossi

Aumento immediato della ICP

non presente autoregolazione





Mannitolo o salina, effetti paradossi

Aumento tardivo della ICP

Scarse tracce in letteratura

Neurosurgery 1992-98
January 1993, Volume 32, Number 1
17 Long-Term Observations of Intracranial Pressure after Severe Head Injury. The Phenomenon of Secondary Rise of Intracranial Pressure
Clinical Study

AUTHOR(S): Unterberg, Andreas, M.D.; Kiening, Karl, M.D.; Schmiedek, Peter, M.D.; Lanksch, Wolfgang, M.D.

Tipi di edema

Neurosurg Focus 22 (5):E5, 2007

Tissue hyperosmolality and brain edema in cerebral contusion

Tatsuro Kawamata, M.D., Tatsuro Mori, M.D., Shoshi Sato, M.D., and Yoichi Katayama, M.D.

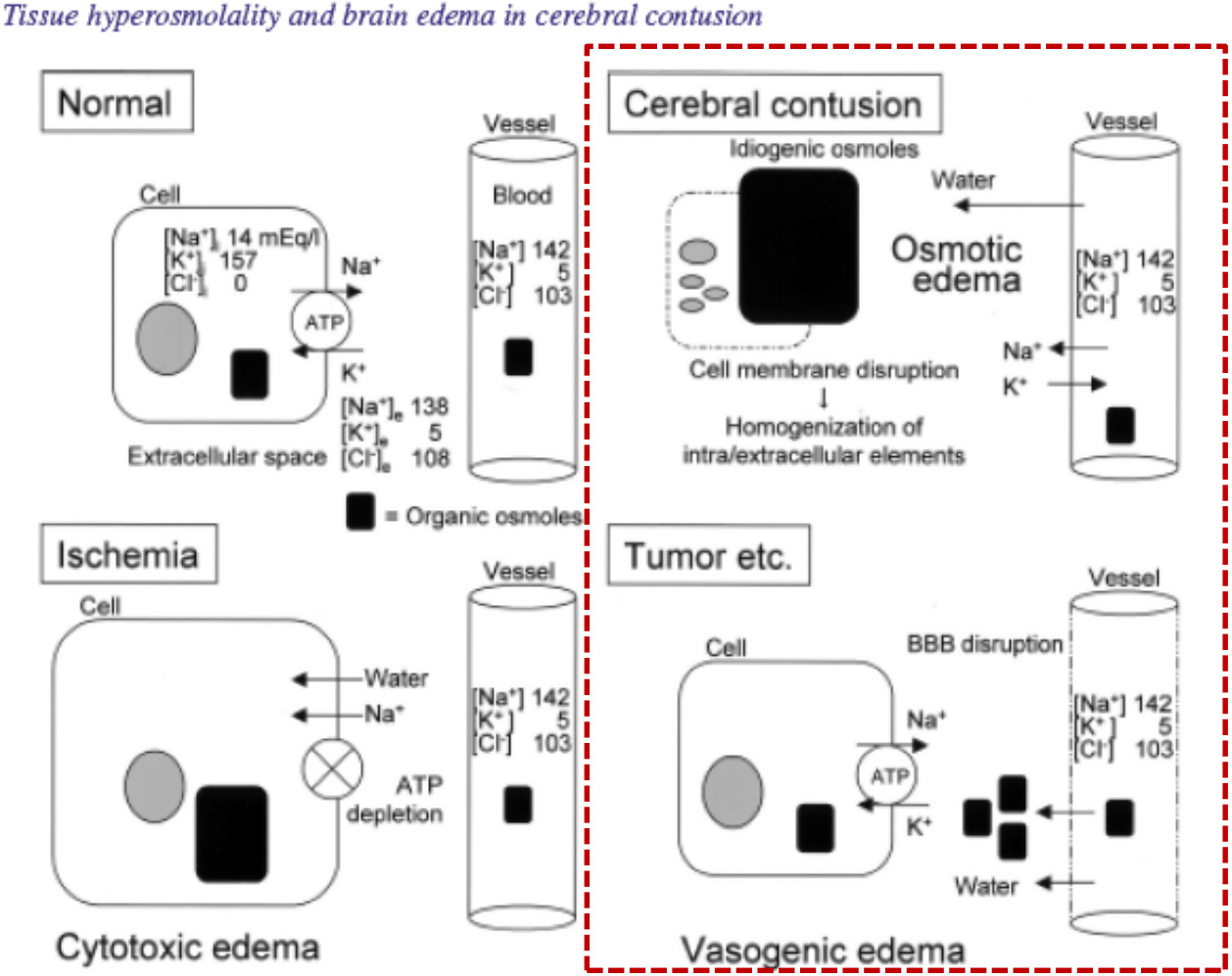


FIG. 4. Schematic showing the concentration distributions of organic and inorganic osmoles, which constitute tissue osmolality in normal brain *(upper left)*, and in pathological conditions such as cerebral contusion *(upper right)*, ischemia *(lower left)*, and tumor *(lower right)*. Ischemia induces energy depletion and ion pump failure, resulting in cellular swelling, that is, cytotoxic edema. Brain tumors, infectious diseases, or other pathological conditions disrupt vascular wall integrity and increase vascular permeability, leading to leakage of large molecules from plasma to the extracellular space and subsequent vasogenic edema. In contused brain, the formation of idiogenic osmoles can produce osmotic potential and attract a large amount of water from the blood or surrounding extracellular space, resulting in contusion edema with mass effect. ATP = adenosine triphosphate; BBB = blood-brain barrier; $[Cl^-]_e$, $[K^+]_e$, and $[Na^+]_e$ = extracellular concentration of Cl^-, K^+, and Na^+; K^+; $[Cl^-]_i$, $[K^+]_i$, and $[Na^+]_i$ = intracellular concentrations of Cl^-, K^+, and Na^+.

Pathobiology of ischaemic stroke: an integrated view

Ulrich Dirnagl, Costantino Iadecola and Michael A. Moskowitz

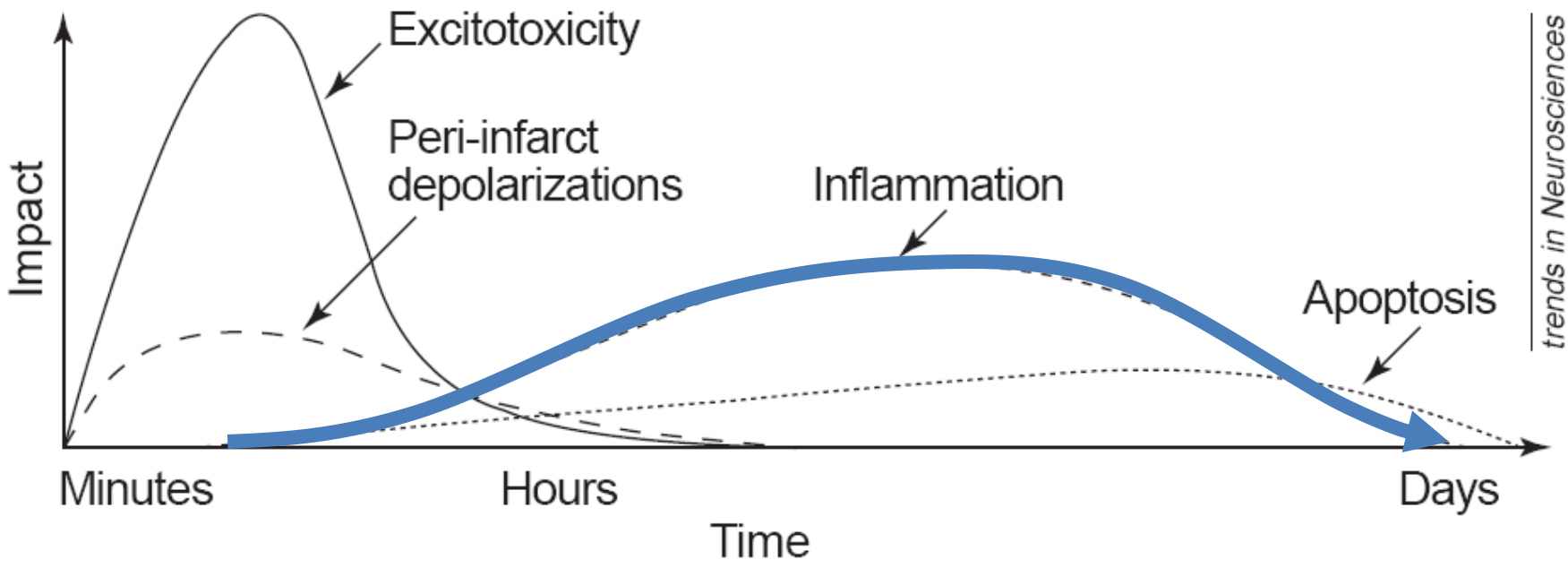
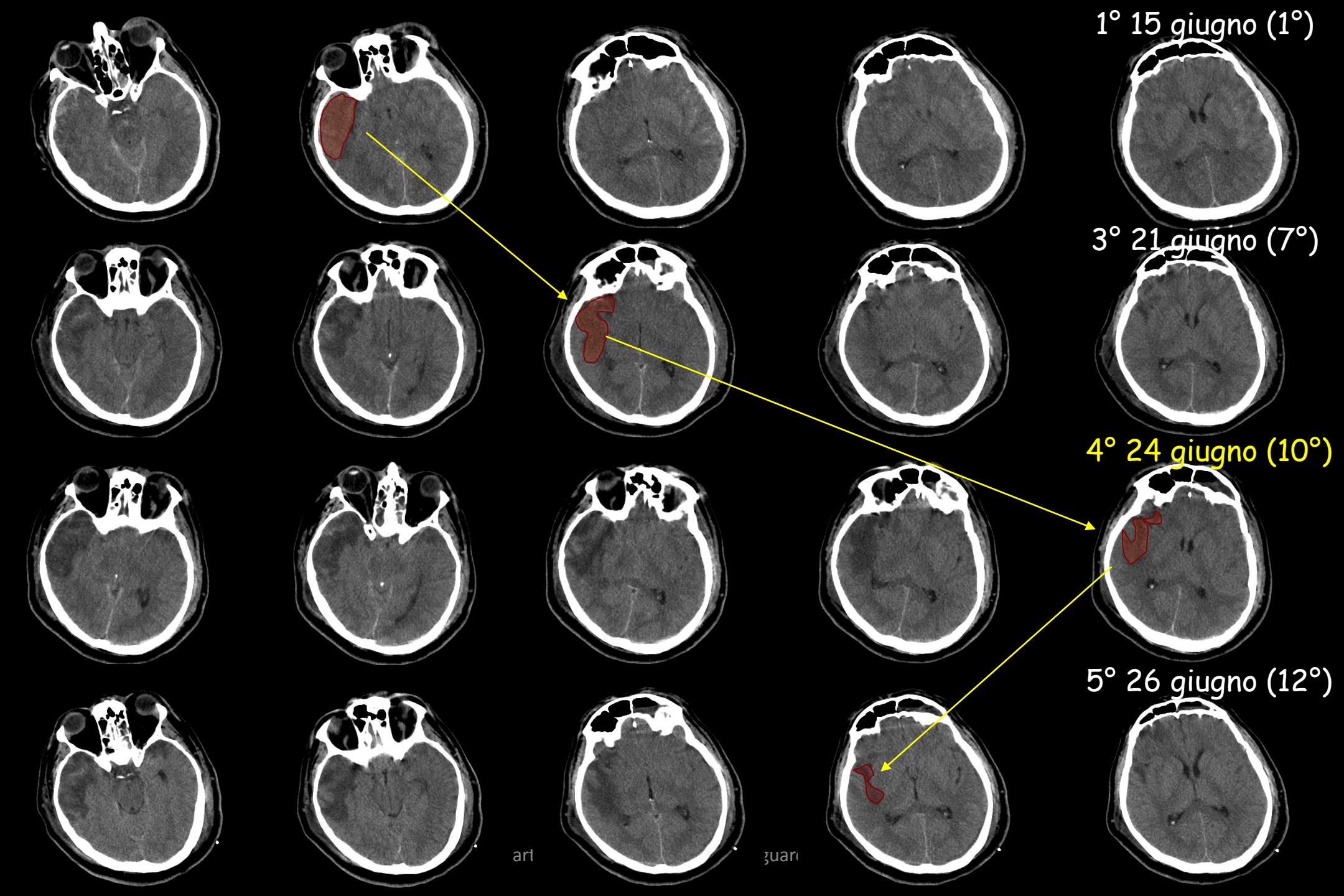
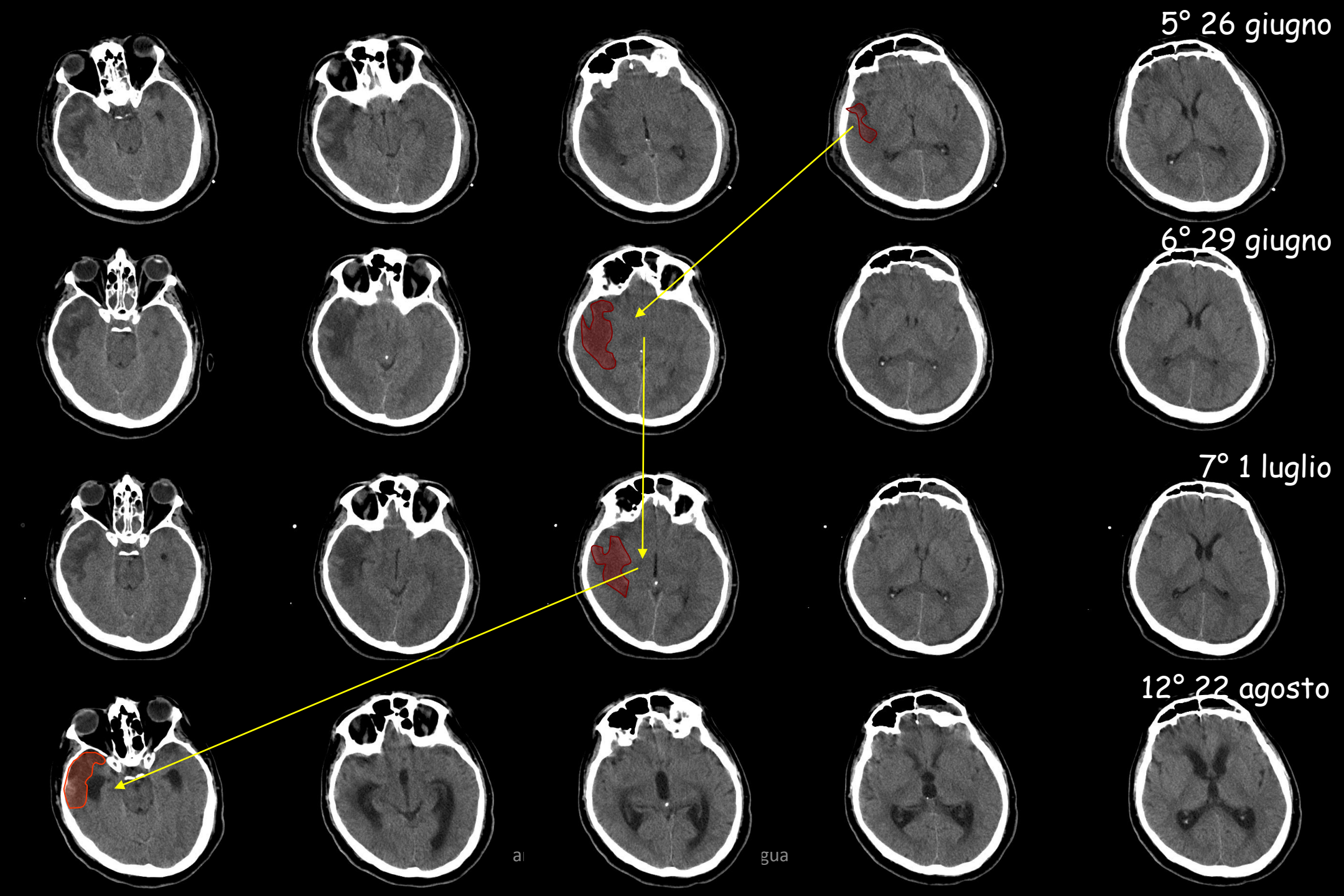
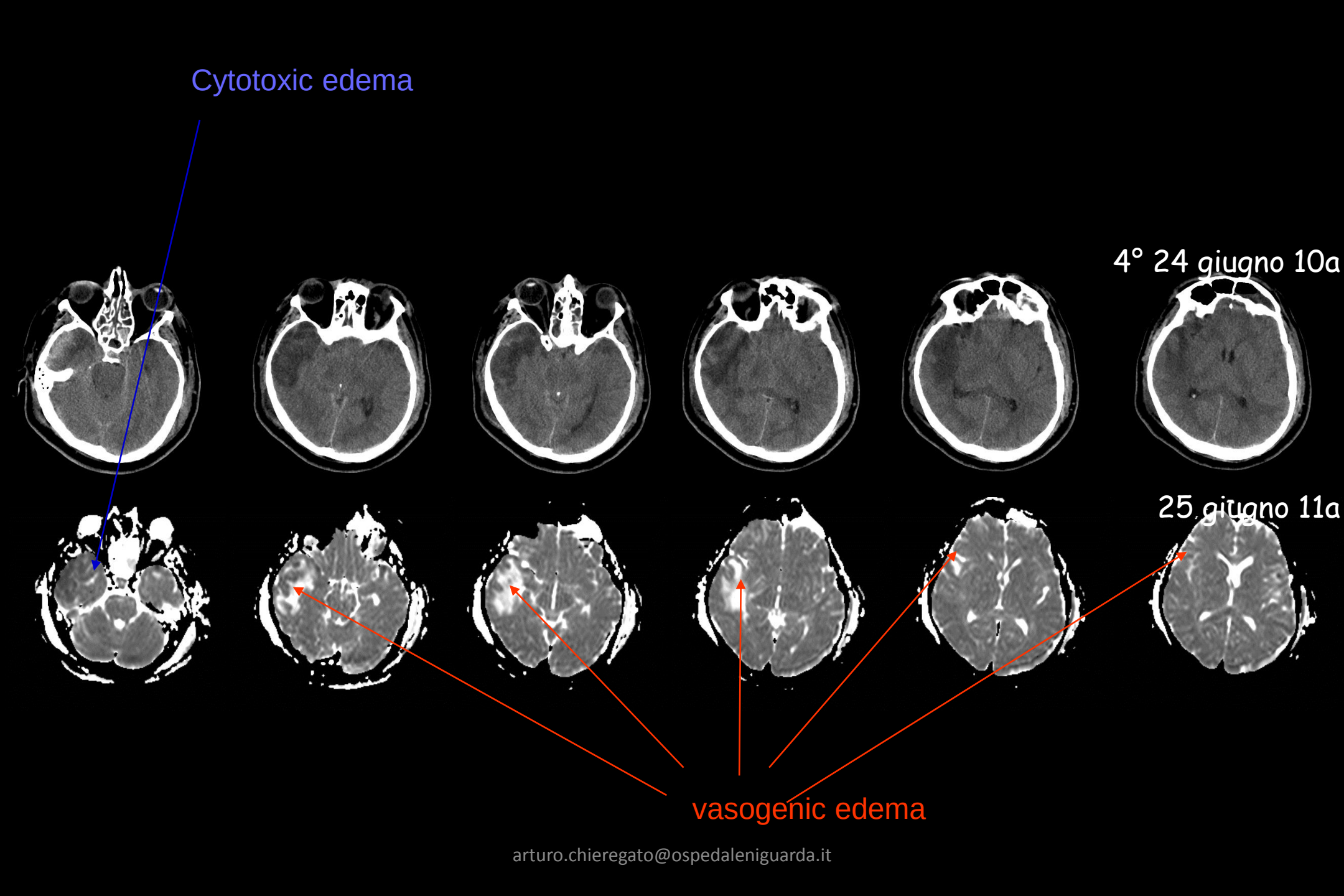


Fig. 1. ***Putative cascade of damaging events in focal cerebral ischaemia.*** *Very early after the onset of the focal perfusion deficit, excitotoxic mechanisms can damage neurones and glia lethally. In addition, excitotoxicity triggers a number of events that can further contribute to the demise of the tissue. Such events include peri-infarct depolarizations and the more-delayed mechanisms of inflammation and programmed cell death. The x-axis reflects the evolution of the cascade over time, while the y-axis aims to illustrate the impact of each element of the cascade on final outcome.*


1° 15 giugno (1°)
3° 21 giugno (7°)
4° 24 giugno (10°)
5° 26 giugno (12°)


5° 26 giugno
6° 29 giugno
7° 1 luglio
12° 22 agosto



arturo.chieregato@ospedaleniguarda.it

BBB non integra

Opposed effects of hypertonic saline on contusions and noncontused brain tissue in patients with severe traumatic brain injury*

Thomas Lescot, MD; Vincent Degos, MD; Abderrezak Zouaoui, MD, PhD; Françoise Préteux, PhD; Pierre Coriat, MD; Louis Puybasset, MD, PhD

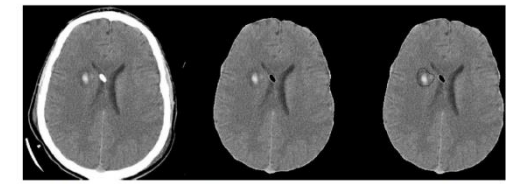


Figure 1. Original computed tomography scan image (*left panel*). Automatic isolation of the intracranial compartment by the software (*middle panel*). Isolation of the contused and noncontused tissue (*right panel*). Note that the intraventricular derivation catheter is excluded from intracranial compartment.

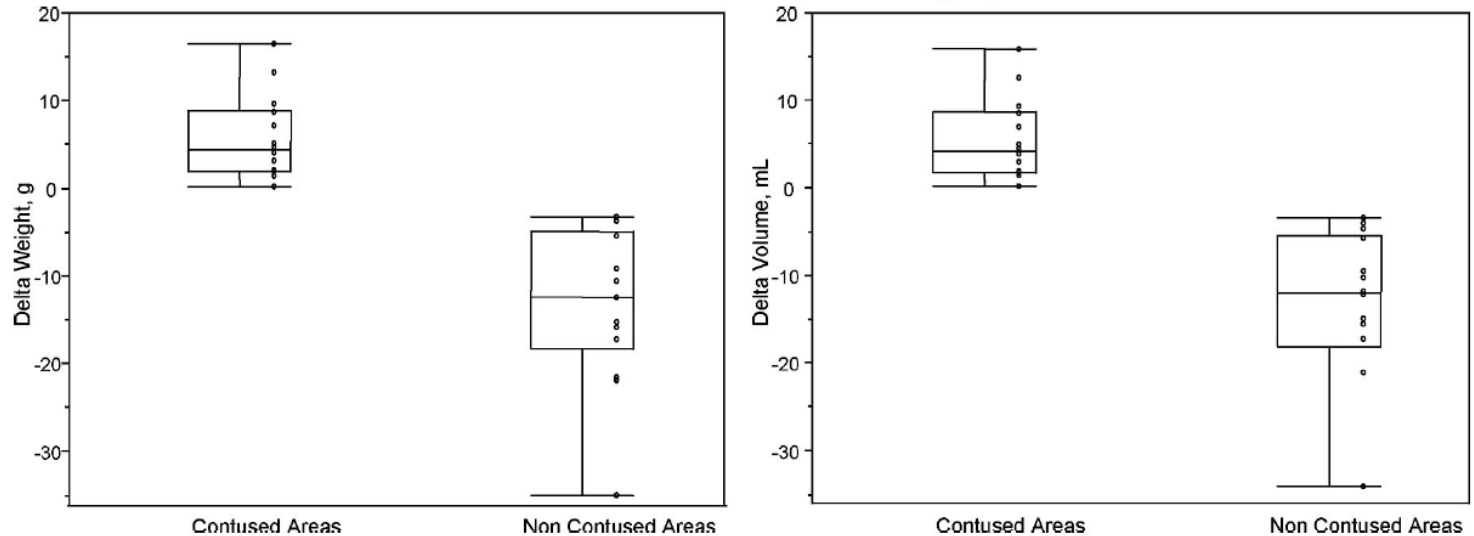
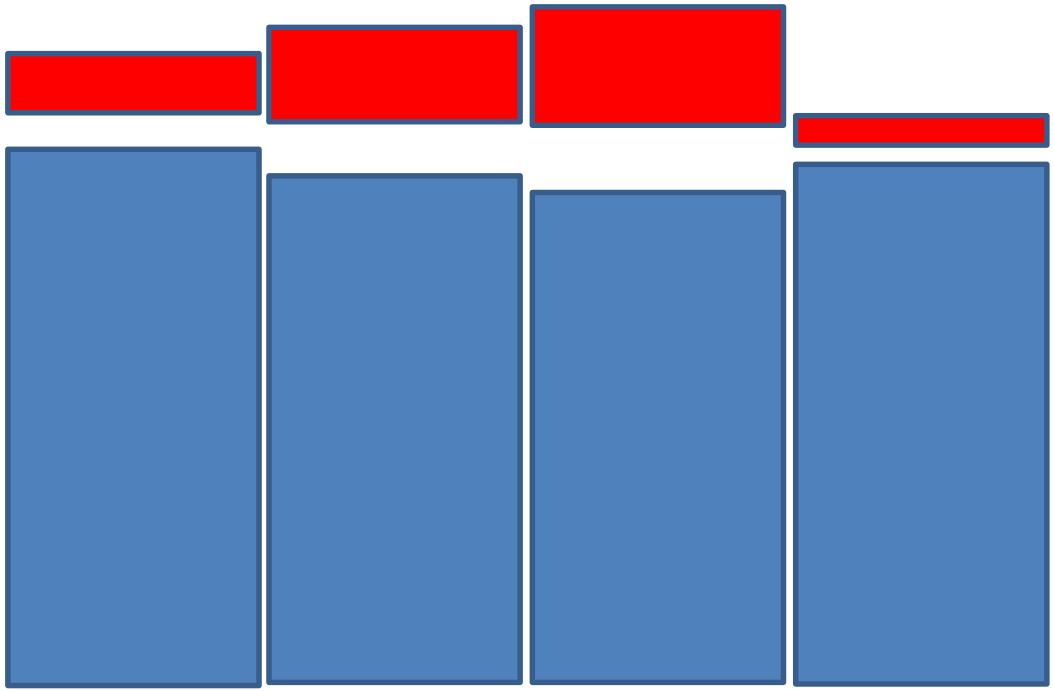


Figure 2. Mean effect of hypertonic saline on the weight and volume of contused and noncontused areas. The box plots summarize the distribution of points at each factor level. The ends of the box are the 25th and 75th quartiles. The line across the middle of the box identifies the median sample value. The whiskers extend from the ends of the box to the outermost data point that falls within the distances computed.

Rosso=tessuto contuso

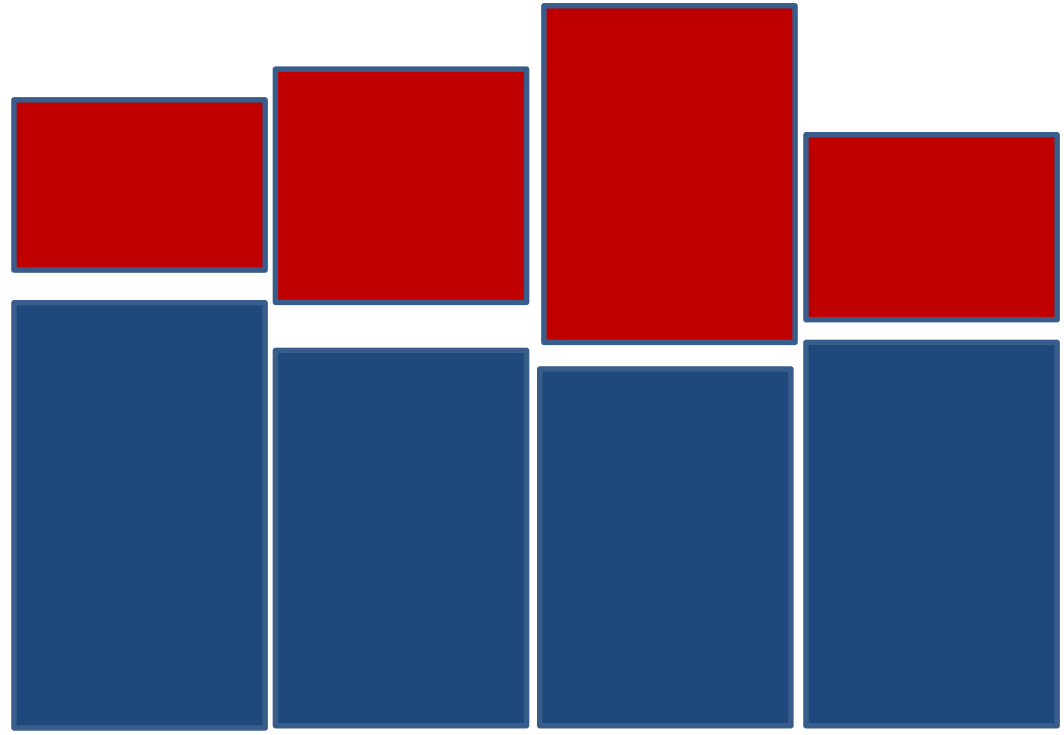
Blu=tessuto apparentemente normale

Caso A



Inizia mannitolo

Caso B



Inizia mannitolo

riassunto

Utilizzo terapia osmotica

Primo obiettivo

Evitare instabilità sodiemia

- Capire iposodiemia/ipersodiemia ed evitarla

La relazione (pooled) punto su punto Na+/ICP non c'è

Wells *et al. Critical Care* 2012, **16**:R193
http://ccforum.com/content/16/5/R193

CRITICAL CARE

RESEARCH Open Access

The relationship between serum sodium and intracranial pressure when using hypertonic saline to target mild hypernatremia in patients with head trauma

Diana L Wells[1*], Joseph M Swanson[2], G Christopher Wood[2], Louis J Magnotti[3], Bradley A Boucher[2], Martin A Croce[3], Charles G Harrison[2], Michael S Muhlbauer[4] and Timothy C Fabian[3]

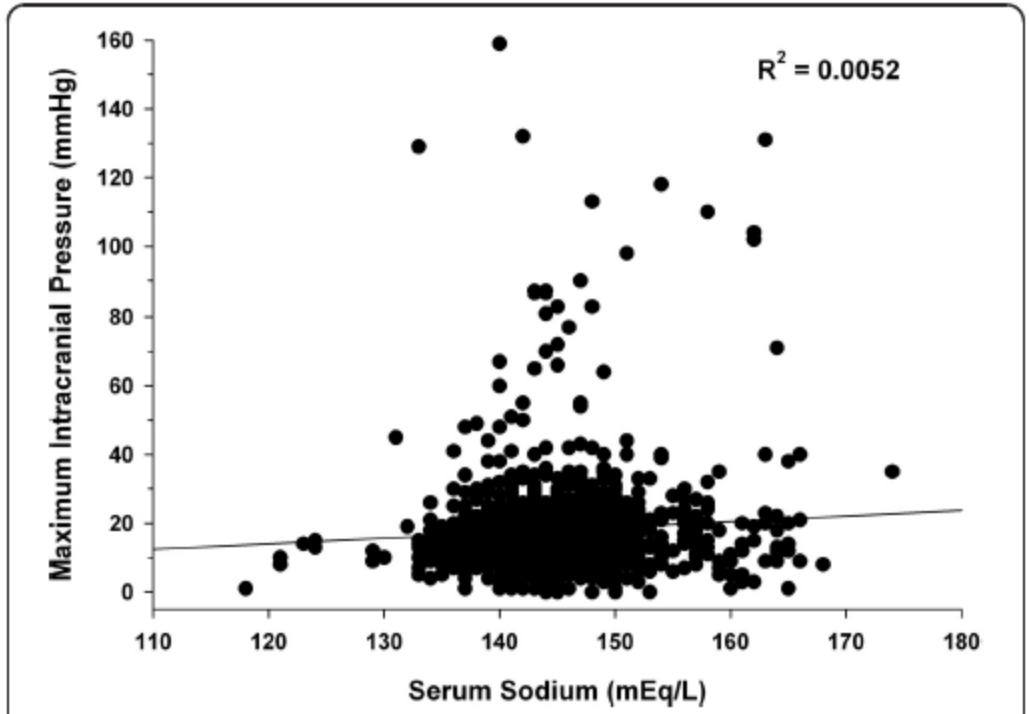
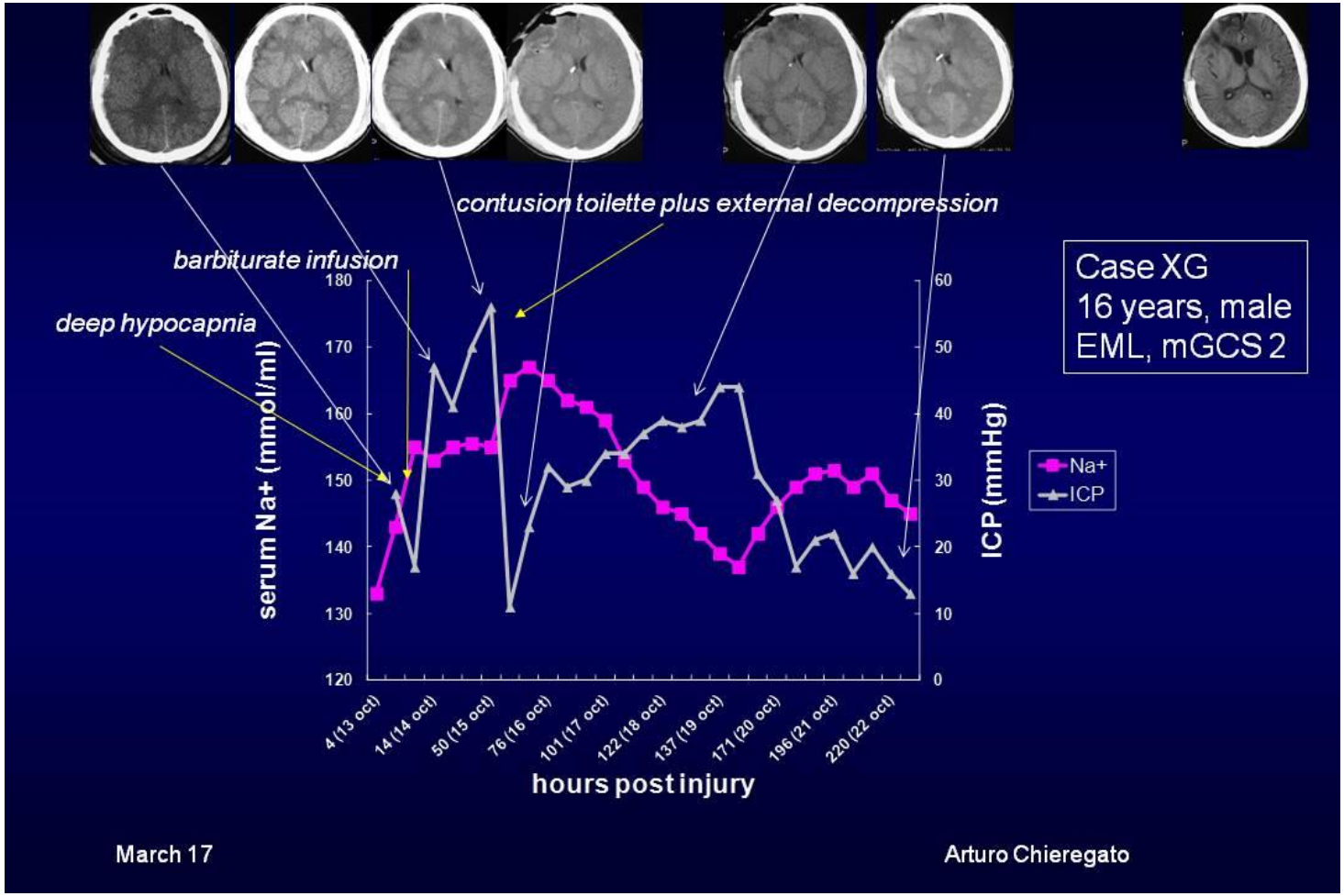
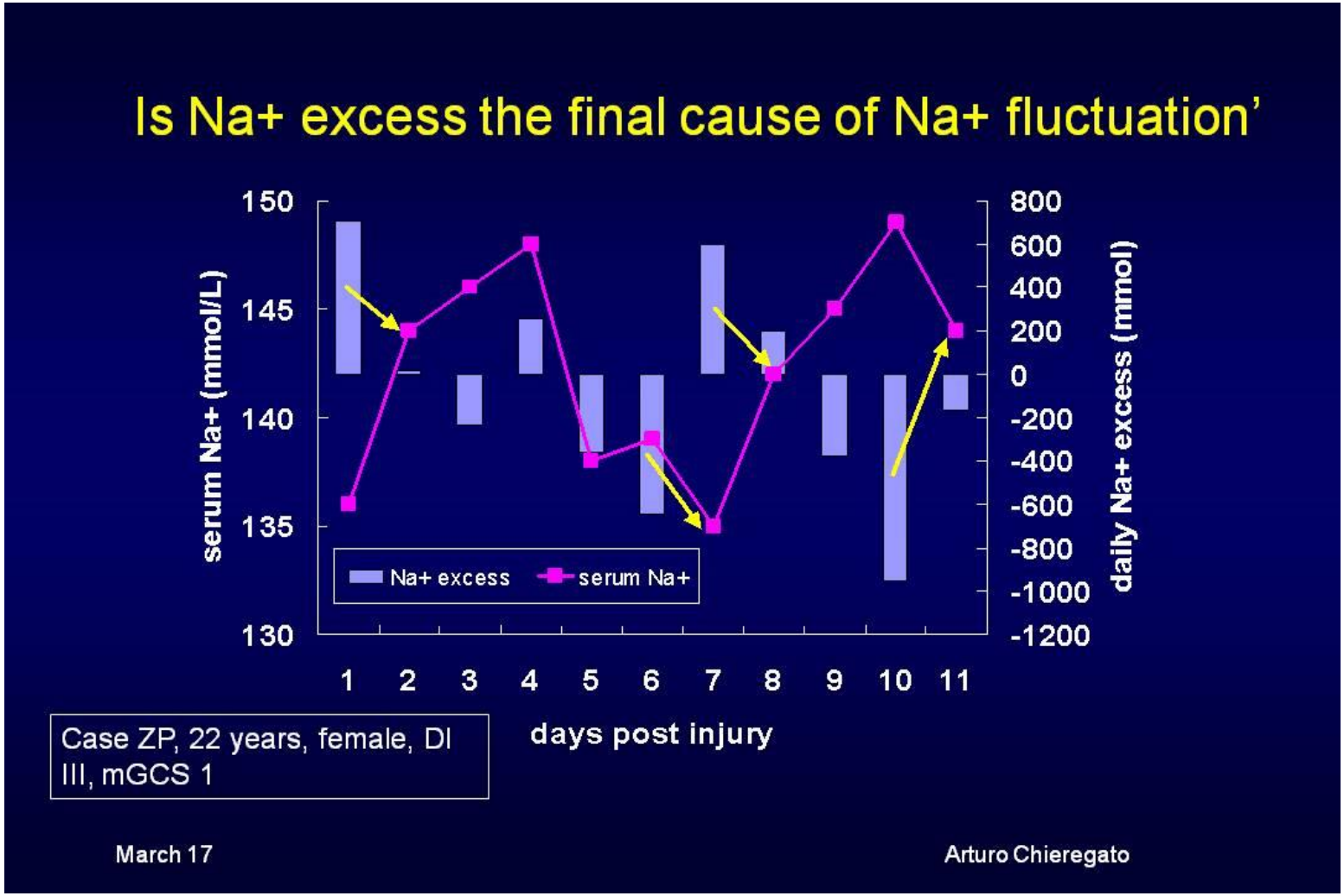


Figure 1 Scatter plot for correlation between serum sodium and maximum intracranial pressure for all patients.

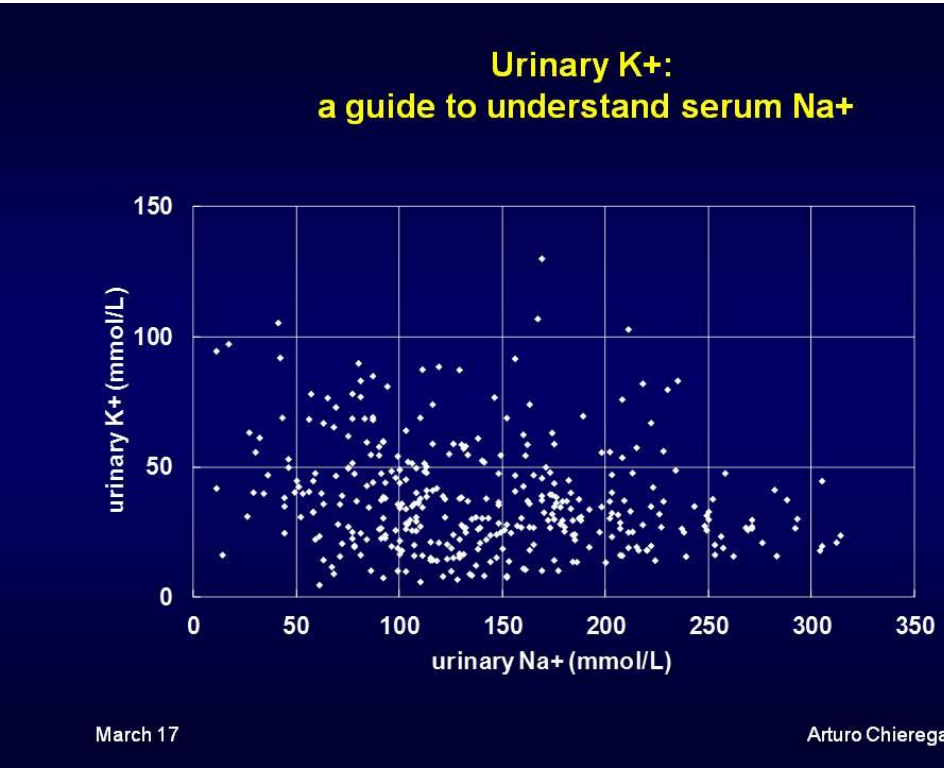
Cervello osmometro che sente i delta

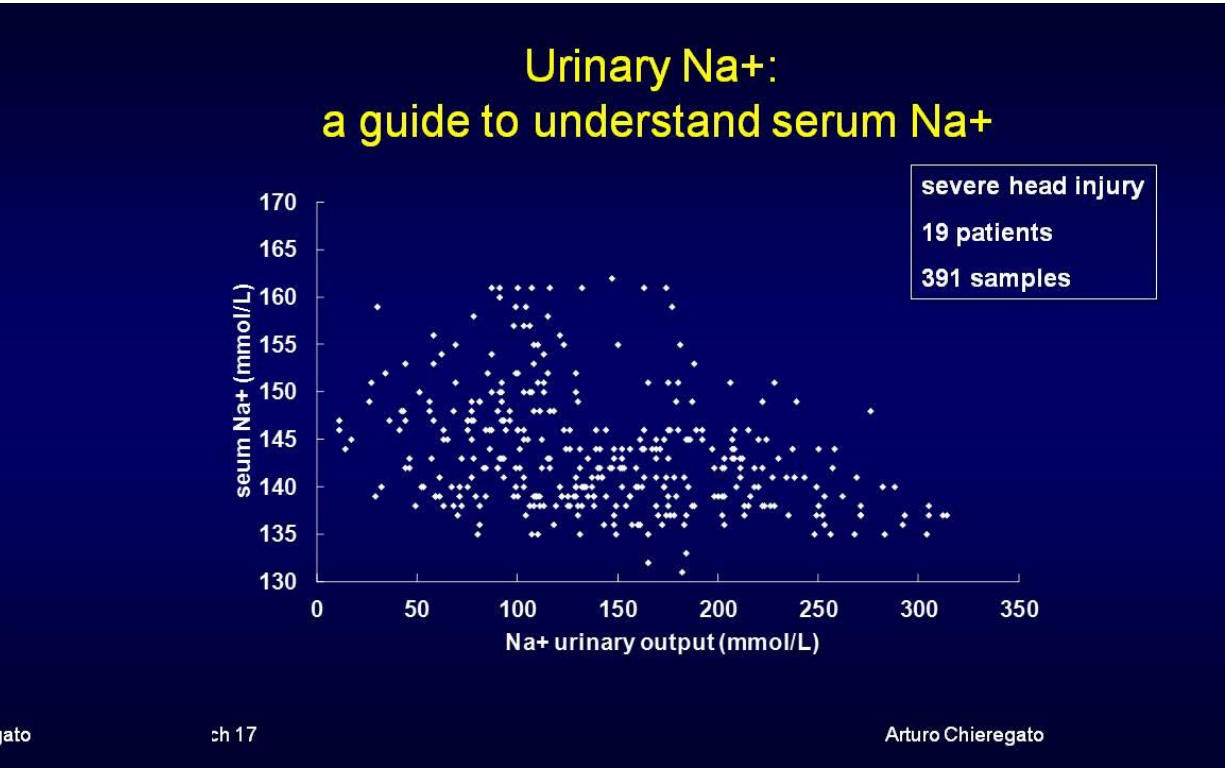


Bilancio del Na+ e dell' H_20



Na+/K+ urinari


Urinary K+:
a guide to understand serum Na+
urinary K+ (mmol/L)
150
100
50
0
0
50
100
150
200
250
300
350
urinary Na+ (mmol/L)
March 17
Arturo Chieregato


Urinary Na+:
a guide to understand serum Na+
severe head injury
19 patients
391 samples
seum Na+ (mmol/L)
170
165
160
155
150
145
140
135
130
0
50
100
150
200
250
300
350
Na+ urinary output (mmol/L)
ch 17
Arturo Chieregato

Sodio per sondino

Journal of the Royal Society of Medicine Volume 78 December 1985 1009

Changes in plasma solutes after food[1]

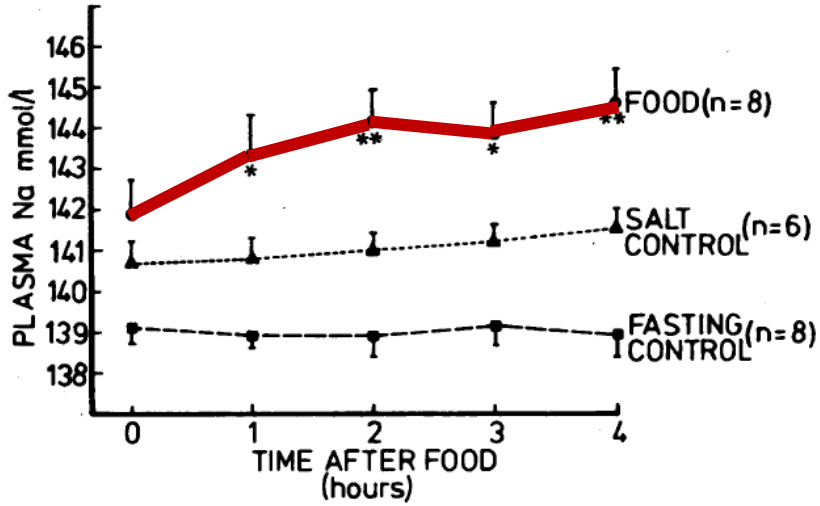


Figure 1. Plasma Na and osmolality changes (mean ± s.e. mean) in subjects after food, compared to fasting and after ingestion of salt (same amount as in test meal). Compared to basal values (time 0), the increases in plasma Na and plasma osmolality after food were significant (* = $P<0.05$, ** = $P<0.01$, *** = $P<0.001$). The triangle denotes thirst onset

In preH o in PS
coma + anisocoria o midriasi

Se shock

- salina

No shock

- Salina (Koening)
- Mannitolo > 1gr /kg (400-600 ml per 70 kg)
  - Alta velocità (iniezione)
    - Compensare poliuria

Neurology® 2008;70:1023-1029

Reversal of transtentorial herniation with hypertonic saline

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M. Bryan, MS
J.L. Lewin III, PharmD
M.A. Mirski, MD, PhD
R.G. Geocadin, MD
R.D. Stevens, MD

ABSTRACT

Objective: To evaluate the role of 23.4% saline in the management of transtentorial herniation (TTH) in patients with supratentorial lesions.

Methods: Consecutive patients with clinically defined TTH treated with 23.4% saline (30 to 60 mL) were included in a retrospective cohort. Factors associated with successful reversal of TTH were determined.

Results: Seventy-six TTH events occurred in 68 patients admitted with intracerebral hemorrhage (n = 29), subarachnoid hemorrhage (n = 16), stroke (n = 8), brain tumor (n = 8), subdural hema-

In ICU
Aumento ICP slow

1. Mannitolo iniettato partendo da dose minima (100 ml) o equivalente Salina
2. Se iposodiemia capire (restrizione o sottocorrezione del sodio possibilmente SNG) + mannitolo a boli

In ICU
Aumento ICP, plateau waves

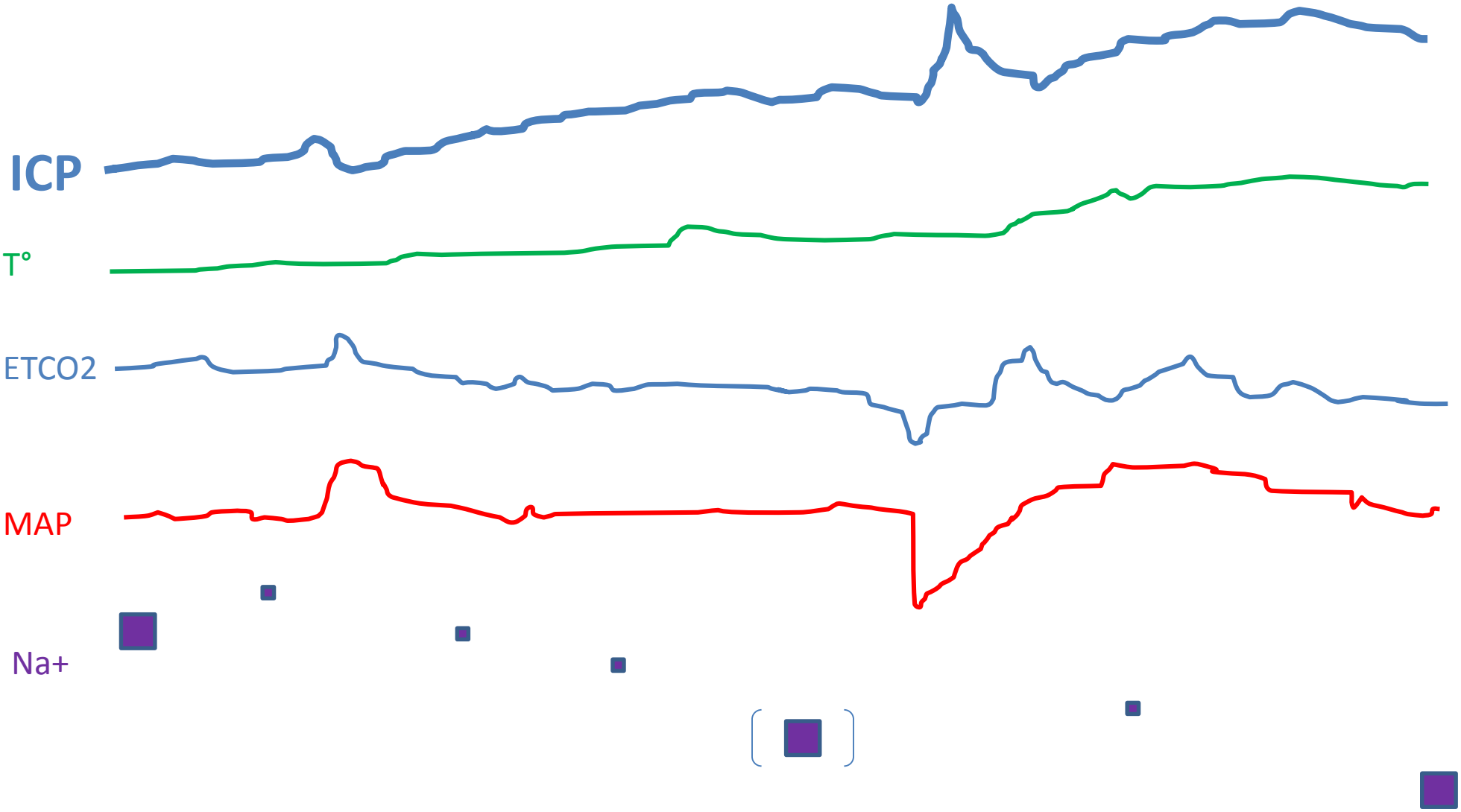
- mannitolo iniettato (già pronto) partendo da dose minima (100 ml)
  - Lentezza operativa
    - Bolus effect se in ritardo
  - Utile in rotazione con altre terapie

In ICU
Aumento ICP tardivo

- Capire se aree ipodense si stanno allargando
- Capire se si associa ad ipersodiemia persistente o mannitolo ripetuto
- Trattamento (TRIAL and ERROR)
  - “Ingannare” con mannitolo e ridurre input di sodio (ipotesi BBB malata ancora aperta)
  - Mantenere ipersodiemia e usare sedazione maggiore a ponte (ipotesi BBB malata si è chiusa e si tiene il sodio)

Il vero problema del sodio è…

Peggiora l’ ICP



Na+/K+ tre volte die salvo diverse indicazioni

Terapia osmotica?

meglio una cultura “osmotica”