

# Prevence iatrogenních hypo/hybernatriemií pomocí sodného protokolu v neurointenzivní péči

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NEUROCENTRUM JIP, LIBEREC, ČESKÁ REPUBLIKA

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18<sup>th</sup>, Colours of Sepsis, Ostrava

# DYSNATREMIE V NEUROINTENZIVNÍ PÉČI

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**1. Akutní poškození mozku**

**2. Následek terapeutických postupů**

**3. Iatrogenní příčiny**

# DYSNATREMIE V NEUROINTENZIVNÍ PÉČI

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**1. Akutní poškození mozku**

**2. Následek terapeutických postupů**

**3. Iatrogenní příčiny**



# Management hypo/hyponatremií

v neurointenzivní péči

# DYSNATREMIE V NEUROINTENZIVNÍ PÉČI

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**Cíl neurointenzivní péče**

**Prevence iatrogenních dysnatremií**

**Sodný protokol**

# DYSNATREMIE V NEUROINTENZIVNÍ PÉČI

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**Sodný protokol**

**Prevence iatrogenních dysnatremií**

**Diagnostika & terapie**

# DYSNATREMIE V NEUROINTENZIVNÍ PÉČI

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## HYPONATREMIE & HYPERNATREMIE

### Časté a vážné komplikace v neurointenzivní péči

*Qureshi AI, Suri MF, Sung GY, Straw RN, Yahia AM, Saad M et al. Prognostic significance of hypernatremia and hyponatremia among patients with aneurysmal subarachnoid hemorrhage. Neurosurgery 2002; 50: 749-55.*

*Diringer MN, Zazulia AR. Hyponatremia in neurologic patients: consequences and approaches to treatment. Neurologist 2006; 12: 117-26.*

*Beties MG. Hyponatremia in acute brain disease: the cerebral salt wasting syndrome. Eur J Intern Med 2002; 13: 9-14.*

*Aiyagari V, Deibert E, Diringer M. Hypernatremia in the neurologic intensive care unit: how high is too high? J Crit Care 2006; 21: 163-72.*

*Fraser JF, Stieg PE. Hyponatremia in the neurosurgical patient: epidemiology, pathophysiology, diagnosis, and management. Neurosurgery 2006; 59: 222-9.*

# DYSNATREMIE V NEUROINTENZIVNÍ PÉČI

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## Hyponatremie

častější

## Hypernatremie

závažnější

*Qureshi AI, Suri MF, Sung GY, Straw RN, Yahia AM, Saad M et al. Prognostic significance of hypernatremia and hyponatremia among patients with aneurysmal subarachnoid hemorrhage. Neurosurgery 2002; 50: 749-55.*

**Spatenkova V, Bradac O, Skrabalek P.** *The impact of a Standardized Sodium Protocol on incidence and Outcome of Dysnatremias in Neurocritical care.* J Neurol Surg A Cent Eur Neurosurg. 2015 Jul;76(4):279-90. doi: 10.1055/s-0034-1393927. Epub 2014 Dec 24. PMID: 25539069



## Outcome and frequency of sodium disturbances in neurocritically ill patients

Vera Spatenkova · Ondrej Bradac ·  
Pavel Skrabalek

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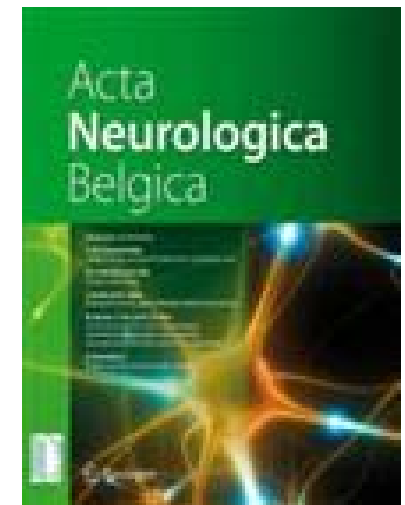
**Abstract** Sodium disturbances are frequent and serious complications in neurocritically ill patients. Hyponatremia is more common than hypernatremia, which is, however, prognostically worse. The aim of this study was to analyse outcome and frequency of sodium disturbances in relation to measured serum osmolality in neurologic neurosurgical critically ill patients. A 5-year retrospective collection of patients (pts) and laboratory data were made from the Laboratory Information System database in the Clinical Biochemistry Department. The criteria for patients' inclusion was acute brain disease and serum sodium ( $\text{SNa}^+$ )  $\leq 135$  mmol/l (hyponatremia) or  $\text{SNa}^+ \geq 150$  mmol/l (hypernatremia). Hypoosmolality was defined as measured serum osmolality ( $\text{SOsm}$ )  $\leq 275$  mmol/kg, hyperosmolality as  $\text{SOsm} \geq 295$  mmol/kg. We performed analysis of differences between hyponatremia and hypernatremia and subanalysis of differences between hypoosmolal hyponatremia and hypernatremia. From 1,440 pts with acute brain diseases there were 251 (17 %) pts with hyponatremia (mean  $\text{SNa}^+$   $131.78 \pm 2.89$  mmol/l,  $\text{SOsm}$   $279.46 \pm 11.84$  mmol/kg) and 75 (5 %) pts with hypernatremia (mean  $\text{SNa}^+$   $154.38 \pm 3.76$  mmol/l,  $\text{SOsm}$   $326.07 \pm 15.93$  mmol/kg). Hypoosmolal hyponatremia occurred in

50 (20 % of hyponatremic patients) pts (mean  $\text{SNa}^+$   $129.62 \pm 4.15$  mmol/l; mean  $\text{SOsm}$   $267.35 \pm 6.28$  mmol/kg). Multiple logistic regression analysis showed that hypernatremia is a significant predictor of mortality during neurologic-neurosurgical intensive care unit (NNICU) stay (OR 5.3,  $p = 0.002$ ) but not a predictor of bad outcome upon discharge from NNICU, defined as Glasgow Coma Scale 1–3. These results showed that hypernatremia occurred less frequently than all hyponatremias, but more often than hypoosmolal hyponatremia. Hypernatremia was shown to be a significant predictor of NNICU mortality compared to hyponatremia.

**Keywords** Hyponatremia · Hypernatremia · Neurointensive care · Outcome

### Introduction

Sodium disturbances are frequent and serious complications in neurointensive care [1–6]. Both hyponatremia and hypernatremia cause brain injury, primary in patients without brain damage and secondary in patients with



# The Impact of a Standardized Sodium Protocol on Incidence and Outcome of Dysnatremias in Neurocritical Care

Vera Spatenkova<sup>1</sup> Ondrej Bradac<sup>2</sup> Pavel Skrabalek<sup>3</sup>

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<sup>2</sup>Department of Neurosurgery, Military University Hospital and First Medical School, Charles University, Prague, Czech Republic

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J Neurol Surg A

## Abstract

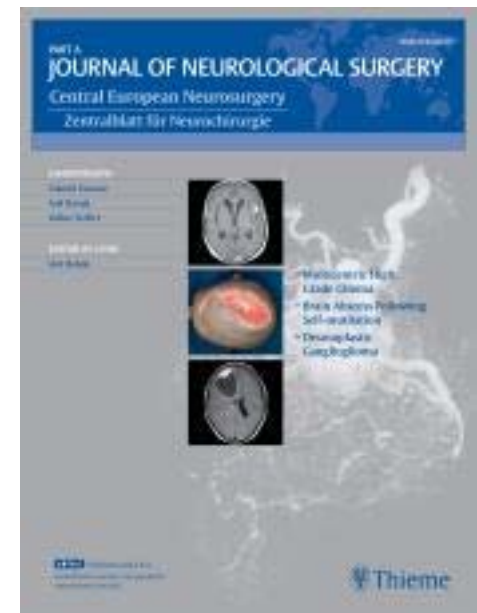
**Background** Dysnatremias are common and prognostically serious in neurocritical care. We studied whether a standardized sodium protocol would improve our neurocritical care of dysnatremias.

**Methods** A 5-year prospective study of a standardized sodium protocol for 1,560 patients admitted with various brain diseases in an adult neurologic-neurosurgical intensive care unit (NNICU) was compared with a 5-year retrospective analysis of 1,440 patients without the sodium protocol. Hyponatremia was defined as serum sodium ( $\text{SNa}^+$ ) < 135 mmol/L and hypernatremia  $\text{SNa}^+$  > 150 mmol/L. The sodium protocol involved measuring  $\text{SNa}^+$ , serum, and urine osmolality, measured and calculated renal function parameters, fluid intake 40 mL/kg weight/day without hypotonic saline, thiazide, and desmopressin acetate in all normonatremic NNICU patients.

**Results** In the protocol study, hyponatremia occurred slightly less often (15.7 versus 16.3% of patients;  $p = 0.684$ ), hypernatremia was significantly higher (respectively 8.5% versus 5.2% of patients;  $p < 0.001$ ), and no differences were noted in hypo/hypernatremia ( $p = 0.483$ ). There were no differences in the incidence of hypo-osmolal hyponatremia (respectively 3.5% versus 3.5% of patients;  $p = 0.987$ ), cerebral salt wasting (CSW; respectively 1.7% versus 1.7% of patients;  $p = 0.883$ ), syndrome of inappropriate secretion of antidiuretic hormone (SIADH; respectively 0.1% versus 0.3% of patients;  $p = 0.152$ ), central diabetes insipidus (CDI; respectively 1.0% versus 0.6% of patients;  $p = 0.149$ ). In hyponatremia there were no differences in the Glasgow Coma Scale (GCS) score upon onset of hyponatremia ( $p = 0.294$ ), NNICU mortality (respectively 1.0% versus 0.4% patients;  $p = 0.074$ ), and bad outcome upon discharge from NNICU (respectively 5.1% versus 6.5% of patients;  $p = 0.101$ ), but in hypernatremia GCS score upon onset ( $p < 0.001$ ), mortality (respectively 2.8% versus 1.0%;  $p < 0.001$ ), and bad outcome from NNICU (respectively 6.7% versus 2.7% patients;  $p < 0.001$ ) were significantly higher. Multivariate logistic regression analysis showed that hypernatremia, compared with hyponatremia, was a significant predictor of mortality during NNICU stay (respectively odds ratio [OR]: 1.14;  $p = 0.003$  versus OR: 5.3;  $p = 0.002$ ).

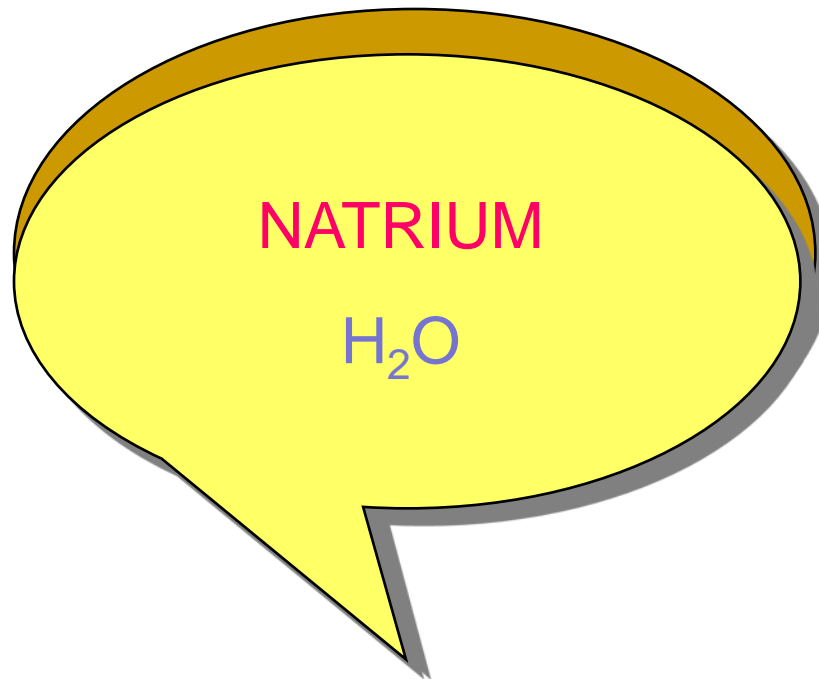
## Keywords

- ▶ central diabetes insipidus
- ▶ CSW
- ▶ hypernatremia
- ▶ hyponatremia
- ▶ SIADH



**Conclusions** The standard sodium protocol lowered the frequency of SIADH, which was encountered in only one patient over 5 years. However, it did not significantly reduce the incidence and improve the outcome of hyponatremia. Hypernatremia occurred more often and had a higher mortality and worse outcome than hyponatremia, but these patients were neurologically worse upon its onset. The prospective study confirmed that CSW, SIADH, and CDI were not common in our neurocritical care.

## SEKUNDÁRNÍ POŠKOZENÍ MOZKU



V neurointenzivní péči  
je nutné se jim věnovat a aktivně vyhledávat.



Hyponatremie  
Hypernatremie

Poruchy efektivní osmolality

# NATRIUM

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Hlavní extracelulární kationt

Největší podíl na efektivní osmolalitě ECT



# NATRIUM

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## Změna v ECT

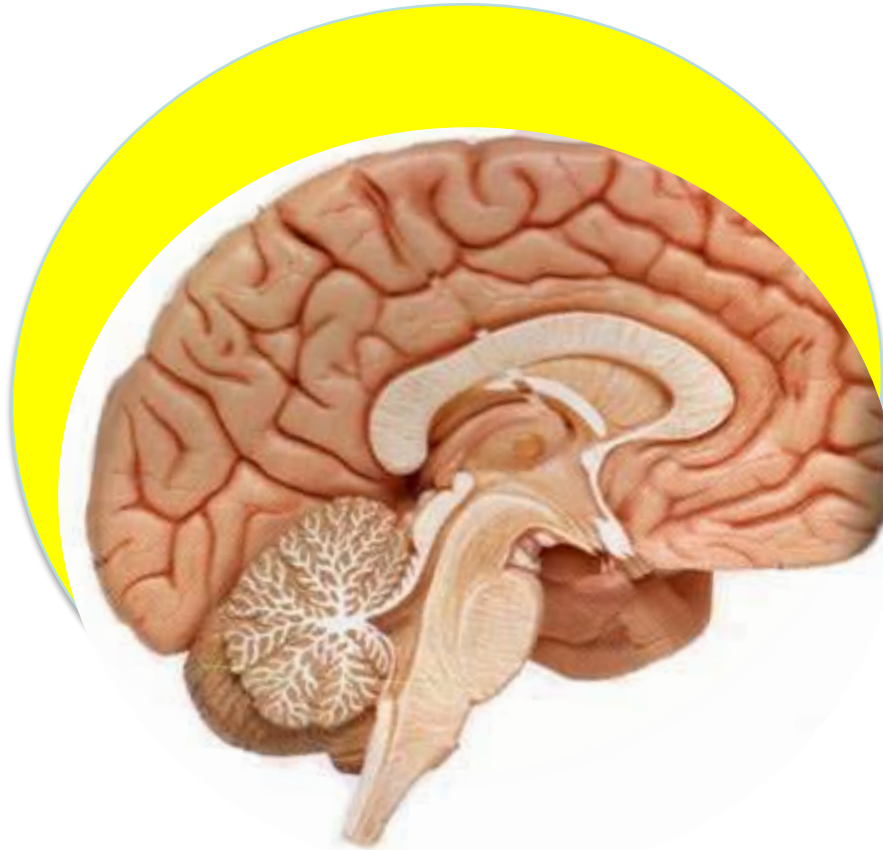
vytváří osmotický gradient mezi ECT a ICT

vyrovnáván přesunem vody

edém nebo dehydratace buněk



## NITROLEBNÍ PROSTOR



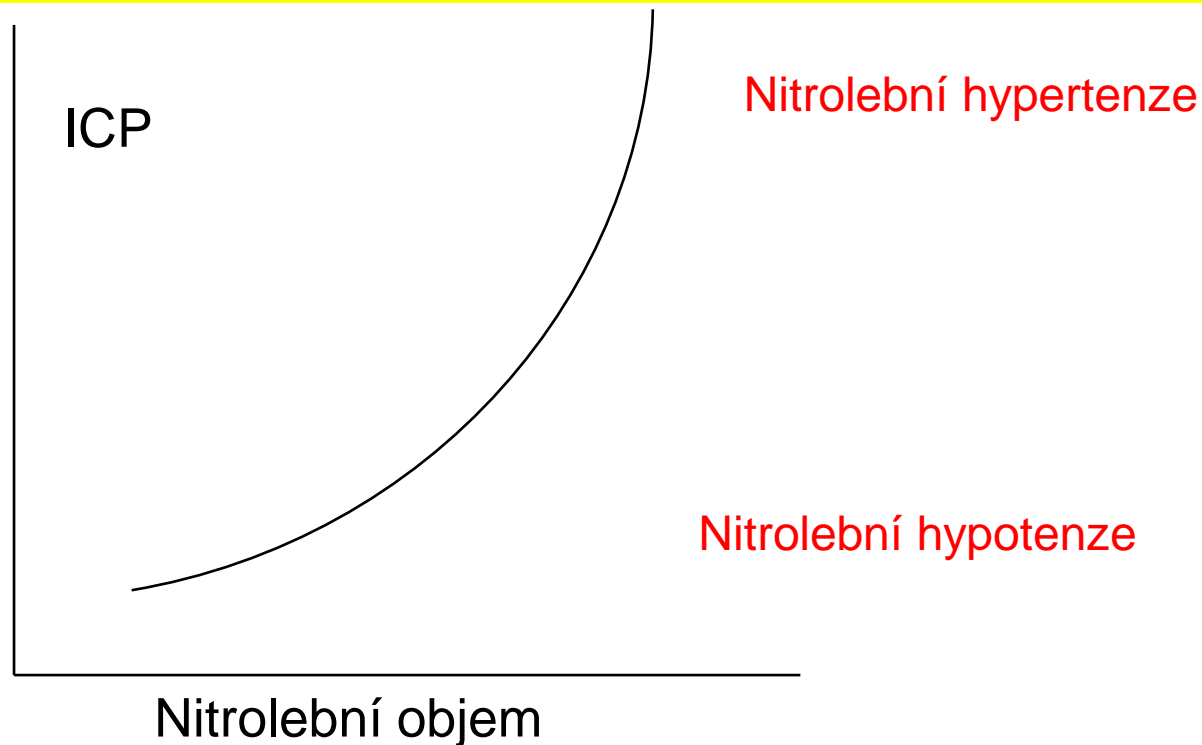
Uzavřený nitrolební prostor limituje nárůst objemu

# MOZEK

## Monroova – Kellieho doktrína

V uzavřeném prostoru intrakrania

$$V \text{ mozku} + V \text{ krve} + V \text{ likvoru} = \text{konst.}$$







# Management hypo/hyponatremií

## v neurointenzivní péči

Sodný protokol má svá specifika v neurointenzivní péči.

SPATENKOVA V. Protokol diagnostiky a léčby hyponatremie a hypernatremie v neurointenzivní péči.

**Cesk Slov Neurol N 2015;78:34-37**

# HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

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HYPONATREMIE

## Hyponatremie

SNa < 135 mmol/l

Lehká 130 – 134 mmol/l

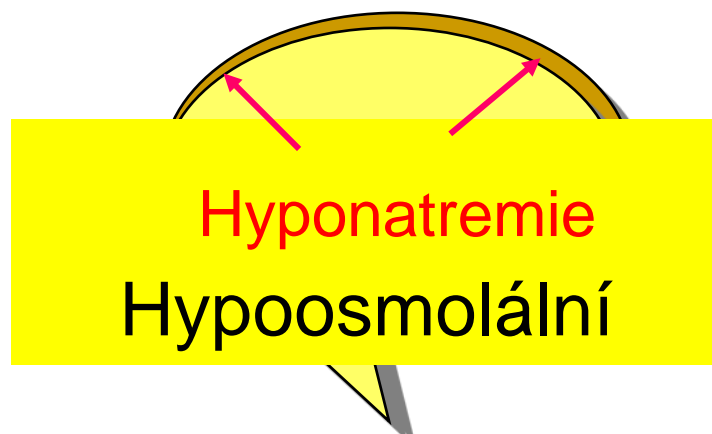
Střední 129 – 125 mmol/l

Těžká < 125 mmol/l

# HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

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## NEUROLOGICKÉ PŘÍZNAKY



Edém mozku

Nitrolební hypertenze

**EFEKTIVNĚ OSMOLÁLNÍ DYSNATREMIE**



# Dysnatraemia is frequently a poor prognostic indicator in patients with acute subarachnoid hemorrhage having targeted sodium management

Spatenkova V. <sup>1</sup>, Bradac O. <sup>2</sup>, de Lacy P. <sup>3</sup>, Skrabalek P. <sup>4</sup>, Suchomel P. <sup>5</sup>

<sup>1</sup> Neurocenter, Neurointensive Care Unit, Regional Hospital, Liberec, Czech Republic; <sup>2</sup> Department of Neurosurgery, Central Military Hospital, Charles University, Prague, Czech Republic; <sup>3</sup> Department of Neurosurgery, Royal Hallamshire Hospital, Sheffield, United Kingdom; <sup>4</sup> Department of Clinical Biochemistry, Regional Hospital, Liberec, Czech Republic; <sup>5</sup> Neurocenter, Department of Neurosurgery, Regional Hospital, Liberec, Czech Republic

**BACKGROUND:** Dysnatraemias are common and carry a risk of poor prognosis in acute subarachnoid hemorrhage (SAH) patients. The aim of this study was to determine the frequency and outcome of dysnatraemias in 344 SAH patients treated by a targeted sodium management regimen. **METHODS:** We performed a 10-year observational dysnatraemia study. Hyponatraemia was defined as serum sodium (SNa) below 135 mmol/l, hypernatraemia SNa above 150 mmol/l.

**RESULTS:** Dysnatraemia occurred in 35.8% patients (pts); this was more frequently hyponatraemia (19.8%) with a mean SNa  $132.23 \pm 2.09$  mmol/l, (16.0% mild, 3.2% moderate, 0.6% severe). Hypernatraemia occurred less commonly in 11.9%,  $p < 0.001$  with a mean SNa  $154.21 \pm 3.72$  mmol/l, (6.1% mild, 2.9% moderate, 2.9% severe). In 4.8% of pts there were episodes of both dysnatraemias. The incidence of hypo-osmolar hyponatraemia was 6.4%, Cerebral salt wasting (CSW) 3.5%, syndrome of inappropriate secretion of antidiuretic hormone (SIADH) 0.3% and Central diabetes insipidus 1.7%. The hypernatraemic pts had a higher inpatient mortality rate ( $p = 0.001$ ) and a worse overall outcome ( $p < 0.001$ ) than those hyponatraemic or normotraemic patients. Multivariate logistic regression showed that hypernatraemia was an independent risk factor for increased inpatient mortality and poor outcome in patients with SAH. **CONCLUSIONS:** Our 10-year targeted sodium management regimen in acute SAH patients showed that dysnatraemias were frequent, predominantly hyponatraemia of which the more usual causes were CSW and not SIADH. Hypernatraemia was shown to be an independent risk factor for inpatient mortality and poor outcome.

# OSMOLALITA

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**Měření serové osmolality (SOsm)**

**Osmometr**



# HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

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## 1. krok v diagnostice hyponatrémii

**Hodnota měřené sérové osmolality**



# HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

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## **1. Akutní poškození mozku**

**CSWS, SIADH**

## **2. Následek terapeutických postupů**

**Thiazidy**

## **3. Iatrogenní příčiny**

**Hypotonický roztok**

**Iatrogenní SIADH (normonatremie a desmopressin)**



# HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

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## 1. Akutní poškození mozku

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Hypotonický roztok

**Iatrogenní SIADH (normonatremie a desmopressin)**

# HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

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**Diagnostický management**

**fyziologická odpověď organismu**

**ADH – ledviny**

# PORUCHY EFEKTIVNÍ OSMOLALITY U AKUTNÍHO POŠKOZENÍ MOZKU

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diagnóza

renální funkční parametry

**Lolin Y, Jackowski A.** Hyponatraemia in neurosurgical patients: diagnosis using derived parameters of sodium and water homeostasis. *Br J Neurosurg* 1992; 6: 457-66.

**Shoker AS.** Application of the clearance concept to hyponatremic and hypernatremic disorders: a phenomenological analysis. *Clin Chem* 1994; 40: 1220-7.

**Kazda A.** Vnitřní prostředí. In: Zima T. *Laboratorní diagnostika*. Galén, 2002, 265-96.

**Kazda A, Balik M.** Osmolální dysbalance v intenzivní péči a možnosti jejich monitorování. *Klin Biochem Metab* 1995; 4: 223-7.

**Kazda A, Balik M, Jabor A.** Efektivní osmolalita a její poruchy. *Anesteziologie a neodkladná péče* 1999; 4: 142-6.

**Jabor A.** Voda, ionty a modelování poruch vnitřního prostředí. *STAPRO, Pardubice*, 1999.

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**Jabor A, Kazda A.** Výukové možnosti u poruch metabolismu vody a iontů. *Anesteziologie a neodkladná péče* 1999; 4: 157-61.

**Jabor A.** Clearance bezelektrolytové vody u selhávajících ledvin, při hypernatrémii a hyponatrémii. *Klin Biochem Metab* 1997; 4: 248-50.

# HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

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## Assessment of axis ADH-kidneys

Hypoosmolality, hypotonicity – serum osmolality < 280 mmol/kg

EWC > 0,116 ml/s (10 l/day).....normal response ADH-kidneys

EWC 0,006 – 0,116 ml/s.....impaired response ADH-kidneys

EWC < 0,006 ml/s (0,5 l/day).....abnormal response ADH-kidneys

## SIADH

Hyperosmolality, hypertonicity – serum osmolality > 295 mmol/kg

EWC < 0,005 ml/s (0,4 l/day).....normal response ADH-kidneys

EWC ≥ 0,005 ml/s.....abnormal response ADH-kidneys

*Shoker AS. Application of the clearance concept to hyponatremic and hypernatremic disorders: a phenomenological analysis. Clin Chem 1994; 40: 1220-7.*

# Kazuistika

# CASE REPORT KAZUISTIKA

## Polyurie v neurointenzivní péči – kazuistika

### Polyuria in Neurocritical Care – a Case Report

V. Špatenková<sup>1</sup>, P. Skrabálek<sup>2</sup> Krajská nemocnice Liberec, a.s.:

1 Neurocentrum, Neurointenzivní jednotka

2 Oddělení klinické biochemie

Cesk Slov Neurol N 2014; 77/110(5): 647–647

- 34-letá pacientka
- Akutní subarachnoidální krvácení z ruptury aneurysmatu na arteria carotis interna (coiling)
- WFNS (World Federation of Neurological Surgeons) skóre I, Fisher skóre 2

# CASE REPORT KAZUISTIKA

## Polyurie v neurointenzivní péči – kazuistika

### Polyuria in Neurocritical Care – a Case Report

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Cesk Slov Neurol N 2014; 77/110(5): 647–647

Den NJIP	SNa mmol/l	SOsm mmol/kg	Diuréza ml/den	EWC ml/s	Desmopressin
1.	138	294			
3.	135	286	4 500	0.016	10 ug/den
4.	130	265			

	P	P	S	P	P
3LU	5,6	5,7	-	6,0	6,3 +
3BNP	-	371 +	-	-	-
<b>rály</b>					
Na...	123 --	124 --	-	124 --	126 --
K...	4,4	4,3	-	4,2	4,3
Cl...	96 -	95 -	-	95 -	98
Ca...	2,23	-	-	-	-
Mg...	1,00	-	-	-	-
P...	1,32	-	-	-	-
OSMV	255---	-	-	-	-
OSME	252!!!	254!!!	-	254!!!	258!!!
BBS.	31,4 --	33,3 --	-	33,2 --	32,3 --
AGAP	7,6 --	-	-	-	-
OSM.	262 --	265 -	-	264 --	270 -
dCP	2,41	-	-	-	-
dCA1	8,21	-	-	-	-
dUA	0,69---	-	-	-	-
dCl.	109,3	-	-	-	-
SIDa	35,11 --	-	-	-	-
SIDe	34,42 --	-	-	-	-
AGPc	10,6	-	-	-	-
Ogap	7	-	-	-	-
<b>met.</b>					
UREA	3,3	-	-	-	-
KREA	48	-	-	-	-
GFR	1,821	-	-	-	-
ALB.	29,1 -	-	-	-	-
CRP.	2,5	-	-	-	-
KREV Arterial	-	-	-	-	-
TAKT	36,8	-	-	-	-
FIO2	0,210	-	-	-	-
HB...	9,5---	-	-	-	-
PH...	7,428	-	-	-	-
PCO2	4,88	-	-	-	-
SBC	24,5	-	-	-	-
SBE	0,0	-	-	-	-
PO2	9,66	-	-	-	-
SO2	0,950	-	-	-	-
ABE	0,1	-	-	-	-
TCO2	24,9	-	-	-	-
AKTB	23,8	-	-	-	-
VO2.	5,6---	-	-	-	-
AaDO	3,78	-	-	-	-
OXHB	0,938 -	-	-	-	-
MeHB	0,004	-	-	-	-
COHB	0,009 +	-	-	-	-
p50	3,33	-	-	-	-
px	3,74 --	-	-	-	-
SHUN	8,1 +	-	-	-	-

MOC		Odpady								
fU_NVYP	7,60 --	-	-	-	-	-	-	*	10,0-30,0	g
U_OBJ.	3870 ++	-	-	-	-	-	-	*	1000-1500	ml
U_CAS.	24	-	-	-	-	-	-			hod.
U_UREA	67	-	-	-	-	-	-			mmo1/1
fU_UREA	259 -	-	-	-	-	-	-	*	330 - 580	mmo1/1
U_KREA	1,966	-	-	-	-	-	-			mmo1/1
fU_KREA	7,608	-	-	-	-	-	-	*	4,50-18,0	mmo1
U_Na...	203	-	-	-	-	-	-			mmo1/1
fU_Na...	786!!!	-	-	-	-	-	-	*	100 - 260	mmo1/1
U_K...	28,3	-	-	-	-	-	-			mmo1/1
fU_K...	109,5 +	-	-	-	-	-	-	*	40,0-90,0	mmo1
U_Cl...	217	-	-	-	-	-	-	*	120 - 260	mmo1/1
fU_Cl...	840!!!	-	-	-	-	-	-	*	120 - 260	mmo1
U_Ca...	5,73	-	-	-	-	-	-			mmo1/1
fU_Ca...	22,18!!!	-	-	-	-	-	-	*	2,50-7,50	mmo1
U_P...	4,01	-	-	-	-	-	-			mmo1/1
fU_P...	15,52	-	-	-	-	-	-	*	15,0-90,0	mmo1
U_Mg...	10,40	-	-	-	-	-	-			mmo1/1
fU_Mg...	40,25!!!	-	-	-	-	-	-	*	1,20-8,20	mmo1
U_OSM.	544	501	-	-	544	490	627	*	50 - 796	mmo1/kg
U_OSMo	575	-	-	-	-	-	-			mmo1/kg
fU_OSM.	2225+++	-	-	-	-	-	-	*	430 -1150	mmo1/d
q_OSMq	2,076	1,891	-	-	2,061	1,815	2,255			-
<b>Clearenc</b>										
Pt_VYSK	166	-	-	-	-	-	-			cm
Pt_VAHA	82	-	-	-	-	-	-			kg
Pt_POVR	1,90	-	-	-	-	-	-			m2
Pt_BMI.	29,76 +	-	-	-	-	-	-	*	18,5-25,0	kg/m2
C_KREA	1,670	-	-	-	-	-	-	*	1,15-2,00	ml/s
C_Na...	0,074!!!	-	-	-	-	-	-	*	,008-.016	ml/s
C_K...	0,288 +	-	-	-	-	-	-	*	,083-0,25	ml/s
C_UREA	0,828	-	-	-	-	-	-	*	,683-1,65	ml/s
C_OSM.	0,098 ++	-	-	-	-	-	-	*	,005-0,05	ml/s
C_H2O.	-0,053 --	-	-	-	-	-	-	*	-.02--0,0	ml/s
C_Elek	0,081!!!	-	-	-	-	-	-	*	,011-.023	ml/s
C_EWC	-0,037 --	-	-	-	-	-	-	*	-0,0-.006	ml/s
C_RESO	0,973 --	-	-	-	-	-	-	*	0,99-1,00	jedn.
Fe_Na...	0,040 +	-	-	-	-	-	-	*	,004-.012	1
Fe_K...	0,157	-	-	-	-	-	-	*	0,04-0,19	1
Fe_OSM.	0,054 +	-	-	-	-	-	-	*	0,01-.035	1
Fe_H2O.	0,027 +	-	-	-	-	-	-	*	0,01-0,02	1
C_KT/V	20,71	-	-	-	-	-	-			

Renální funkční parametry součástí  
biochemického souboru z OKB



# HYPERNATREMIE V NEUROINTENZIVNÍ PÉČI

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HYPERNATREMIE

## Hypernatremie

SNa > 145 mmol/l

Lehká 151 – 155 mmol/l

Střední 156 – 160 mmol/l

Těžká > 160 mmol/l

[J Crit Care.](#) 2006 Jun;21(2):163-72.

## **Hypernatremia in the neurologic intensive care unit: how high is too high?**

[Aiyagari V](#), [Deibert E](#), [Diringner MN](#).

### **Source**

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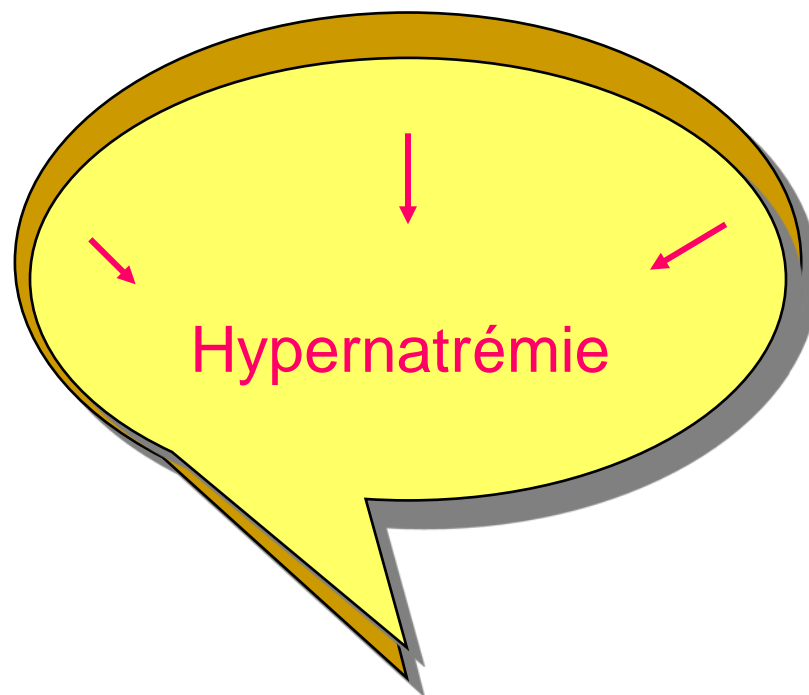
### **Abstract**

Hypernatremia is associated with increased mortality in hospitalized patients and in medical/surgical intensive care units. This relationship has not been studied in neurologic/neurosurgical intensive care units (NNICUs), where hypernatremia is often a component of treatment of cerebral edema. We performed a retrospective analysis of prospectively collected data in patients admitted to the NNICU over a 6.5-year period. Hypernatremia (serum sodium >150 mEq/L) was seen in 339 patients (7.9%) and was more common (24.3%) in patients who were treated with mannitol. Hypernatremic patients had a lower median admission Glasgow Coma Scale score (8 vs 14,  $P < .001$ ), higher initial Acute Physiology and Chronic Health Evaluation II probability of death (34.9% vs 19.1%,  $P < .001$ ), higher incidence of mechanical ventilation (80.5% vs 41.15%,  $P < .001$ ), higher mortality (30.1% vs 10.2%,  $P < .001$ ), and higher incidence of renal failure (10.3% vs 0.9%,  $P < .001$ ). Mortality increased with increasing hypernatremia; however, only severe hypernatremia (serum sodium >160 mEq/L) was independently associated with increased mortality. Other factors independently associated with mortality were age, mechanical ventilation, initial Acute Physiology and Chronic Health Evaluation II probability of death or low admission Glasgow Coma Scale score, and a diagnosis of cerebrovascular disease. **In conclusion**, hypernatremia is common in the NNICU, more so in patients treated with mannitol. In this population, **severe (but not mild or moderate) hypernatremia is independently associated with increased mortality.**



# HYPERNATREMIE V NEUROINTENZIVNÍ PÉČI

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Dehydratace mozku

Nitrolební hypotenze

**EFEKTIVNĚ OSMOLÁLNÍ DYSNATREMIE**

# HYPERNATREMIE V NEUROINTENZIVNÍ PÉČI

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## **1. Akutní poškození mozku**

**Centrální diabetes insipidus (CDI)**

## **2. Následek terapeutických postupů**

**Osmoterapie – Manitol, NaCl, Furosemid**

## **3. Iatrogenní příčiny**

**Zvýšený příjem soli, profuzní pocení**

# HYPERNATREMIE V NEUROINTENZIVNÍ PÉČI

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- **Centrální diabetes insipidus**

Není nejčastěji se vyskytující hypernatrémie

- Multifaktoriální  
osmoterapie, manitol, renální selhán

*Wong MF, Chin NM, Lew TW. Diabetes insipidus in neurosurgical patients. Ann Acad Med Singapore 1998; 27: 340-3.*

*Tisdall M, Crocker M, Watkiss J, Smith J, Smith M. Disturbances of sodium in critically ill adult neurologic patients: a clinical review. J Neurosurg Anesthesiol 2006; 18: 57-63.*

*Aiyagari V, Deibert E, Diringer M. Hyponatremia in the neurologic intensive care unit: how high is too high? J Crit Care 2006; 21: 163-72.*

# Central diabetes insipidus is not a common and prognostically worse type of hypernatremia in neurointensive care

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NNICU. **RESULTS:** We found cDI in 8 pts (mean SNa<sup>+</sup> 154.8 ± 5.4 mmol/l). Most pts (67) were classified as “non cDI” hypernatremias (mean SNa<sup>+</sup> 154.3 ± 3.6 mmol/l). There were no differences in serum sodium ( $p=0.682$ ), serum osmolality ( $p=0.476$ ) between the two groups, however patients with cDI indicated low urine osmolality ( $p=0.001$ ) and positive EWC ( $p=0.049$ ). We did not find any differences in GCS score on onset of hypernatremia ( $p=0.395$ ), incidence of cerebral complications ( $p=0.705$ ), GOS score upon discharge from NNICU ( $p=0.61$ ) and mortality in NNICU ( $p=0.638$ ). More patients in the “non cDI” group received antiedematous therapy ( $p=0.028$ ) and diuretic furosemide ( $p=0.026$ ). Multivariate logistic regression analysis showed that independent predictors of NNICU mortality was the highest level of serum sodium (Odds ratio, OR 1.13, per 1 mmol/l increase in maximal hypernatremia during NNICU stay, 95% confidence interval, CI 1.01–1.26,  $p=0.027$ ), and GCS on admission of less than 9 (OR 2.61, 95% CI 1.41–5.44,  $p=0.003$ ). **CONCLUSIONS:** Central diabetes insipidus is not a frequent type of hypernatremia in neurointensive care. Prognosis is connected with serum sodium level, not with type of hypernatremia.

# DYSNATREMIE V NEUROINTENZIVNÍ PÉČI

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Závěr



## DYSNATREMIE V NEUROINTENZIVNÍ PÉČI

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- Sodný management má své místo v neurointenzivní péči. Dysnatremiím je nutné se v neurointenzivní péči věnovat a aktivně vyhledávat.
- Cílem neurointenzivní péče je prevence hypo/hyponatremií z iatrogenních příčin.
- Sodný protokol má svá specifika v neurointenzivní péči.

# NCSİM

NEUROINTENZIVNÍ  
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ČESKÉ SPOLEČNOSTI  
INTENZIVNÍ MEDICÍNY



## VÁŽENÉ KOLEGYNĚ, VÁŽENÍ KOLEGOVĚ,

je mi velkým potěšením informovat Vás o založení Neurointenzivní sekce ČSİM (NCSİM) a přivzvat Vás ke spolupráci na jejím rozšíření a rozvoji. Péče o pacienty s onemocněním nervového systému vyžaduje specializované vybavení a má také specifické postupy a priority. V současné době jsme svědky rapidního, celosvětového rozvoje neurointenzivní péče a tento trend lze v naší zemi sledovat, udržet a případně k němu přispět pouze skrze fungující společenství odborníků. Cílem Neurointenzivní sekce je poskytnout pevnou platformu pro takovou spolupráci.

Prvním cílem sekce je registrace lékařů a pracovišť poskytujících neurointenzivní péči, dále připravujeme organizaci odborných akcí, formulaci doporučených postupů a spolupráci více pracovišť na výzkumných projektech. Samozřejmostí je navázání oficiální spolupráce s mezinárodní společností NCS (Neurocritical Care Society) a Neurointenzivní sekcí ESICM (European Society of Intensive Care Medicine). Bližší informace naleznete na našich webových stránkách: [www.ncsim.cz](http://www.ncsim.cz).

MUDr. Věra Špatenková, PhD.  
Předsedkyně výboru NCSİM - Neurocentrum, Neurointenzivní jednotka, Liberec

V Liberci 10. ledna 2016

**Děkuji za pozornost**

