PACIENT
PO NEUROCHIRURGICKÉM VÝKONU
-
CO JE PODSTATNÉ

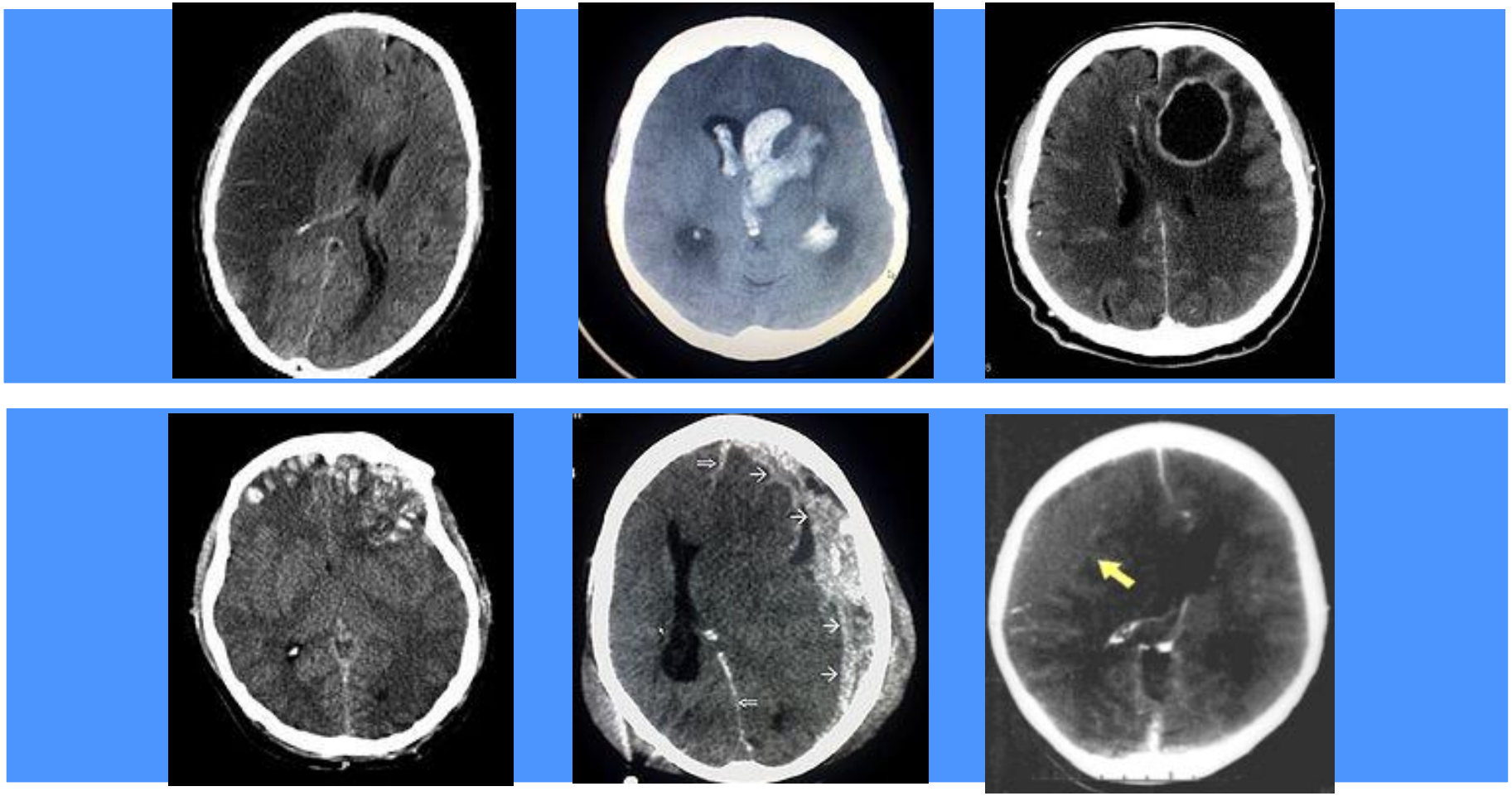
V. ŠPATENKOVÁ

JIP neurocentra, Krajská nemocnice Liberec

Fyziologický ústav, 1. LF UK Praha

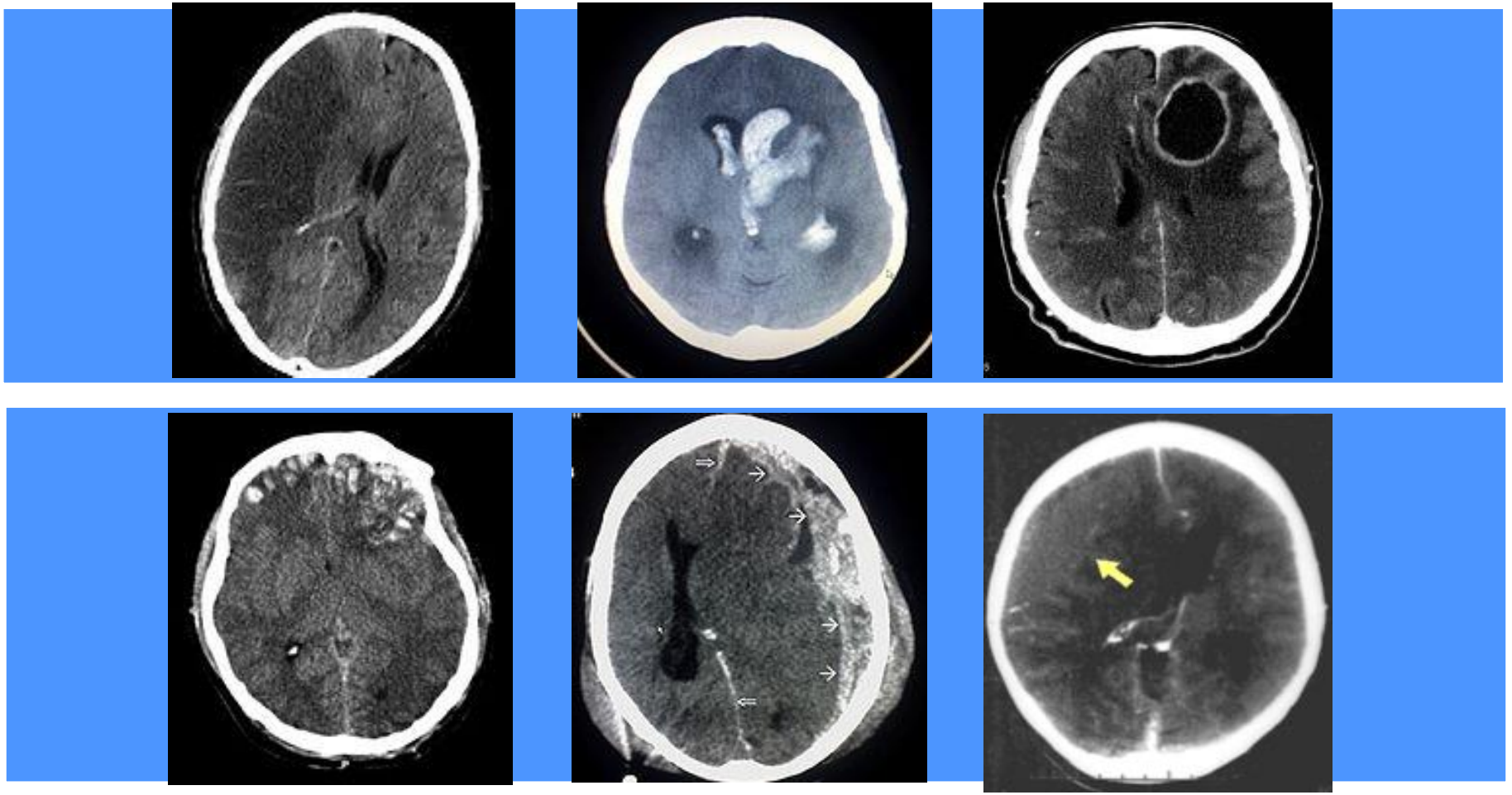
XXVI. kongres ČSARIM, Brno, 2019

Pacient po neurochirurgickém výkonu



NEUROINTENZIVNÍ PÉČE

MANAGEMENT



NEUROINTENZIVNÍ PÉČE

XXVI. kongres ČSARIM, Brno, 2019

CO JE PODSTATNÉ

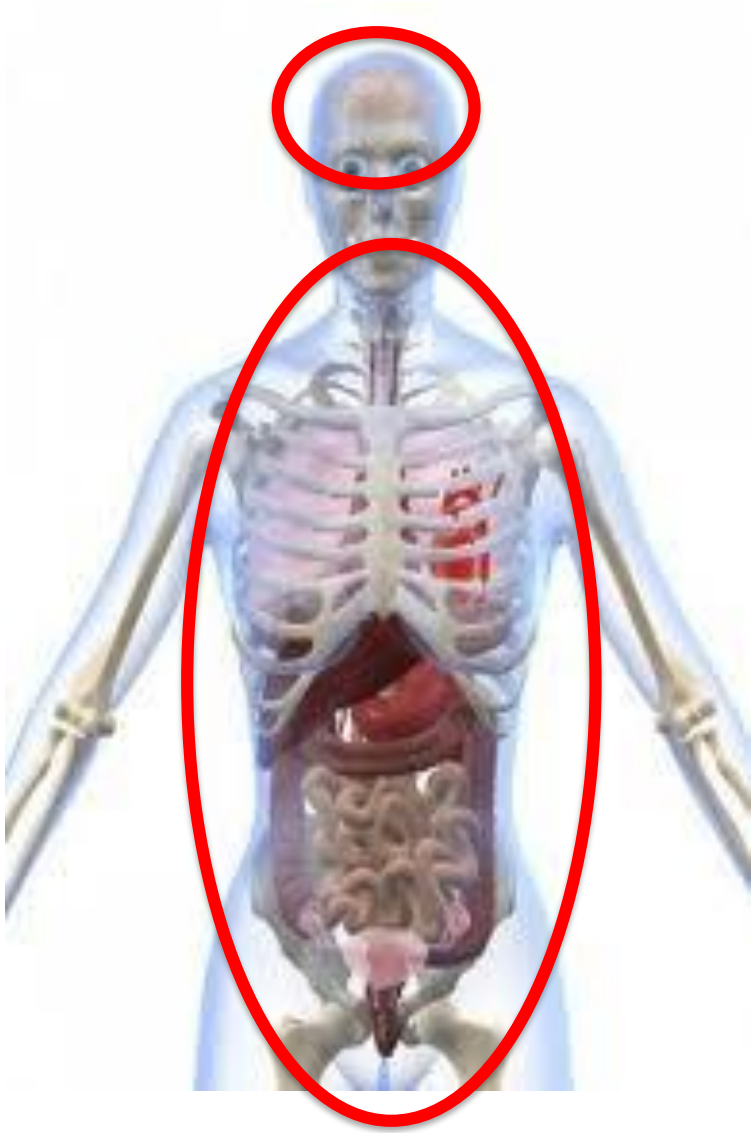
Postoperative care of neurosurgical patients: general principles

Gahan Bose, Astri M.V. Luoma

Anaesthesia and Intensive Care Medicine
June 2017Volume 18, Issue 6, Pages 296–303

The primary aim of care immediately after neurosurgery is to detect and prevent neurological deterioration while supporting systemic and neurological homoeostasis. Surgical-, anaesthetic- or disease- related factors may contribute to a slow return or failure to regain a patient's preoperative status. A period of specific monitoring and observation by nursing and medical staff accustomed to neurosurgical and neurocritical care procedures should be planned for the immediate postoperative period. In many neurosurgical centres the period of postoperative observation may be relatively short (e.g. limited uneventful craniotomies); however, if complicating factors such as cerebral oedema, intracranial haemorrhage, seizures or significant premorbid conditions are present, a period of higher dependency care over several days may be anticipated. In common with all postoperative care safe management of the airway, weaning of ventilatory support, control of circulation and fluid balance, feeding, sedation and analgesia are the mainstays of care. Meticulous attention to each of these is essential in the post neurosurgical patient as poor management can profoundly affect neurological outcome. Thus a robust perioperative plan is mandatory for management of the airway, control of blood pressure, and to ensure continuation of preoperative medication. Furthermore, the plan may entail elective creation of tracheostomy and percutaneous endoscopic gastrostomy. The early postoperative neurosurgical patient continues to require a high degree of clinical vigilance.

Sekundární poškození mozku



NITROLEBNÍ PŘÍČINY

Edém, Krvácení, Ischemie
Epileptický záchvat, Infekce

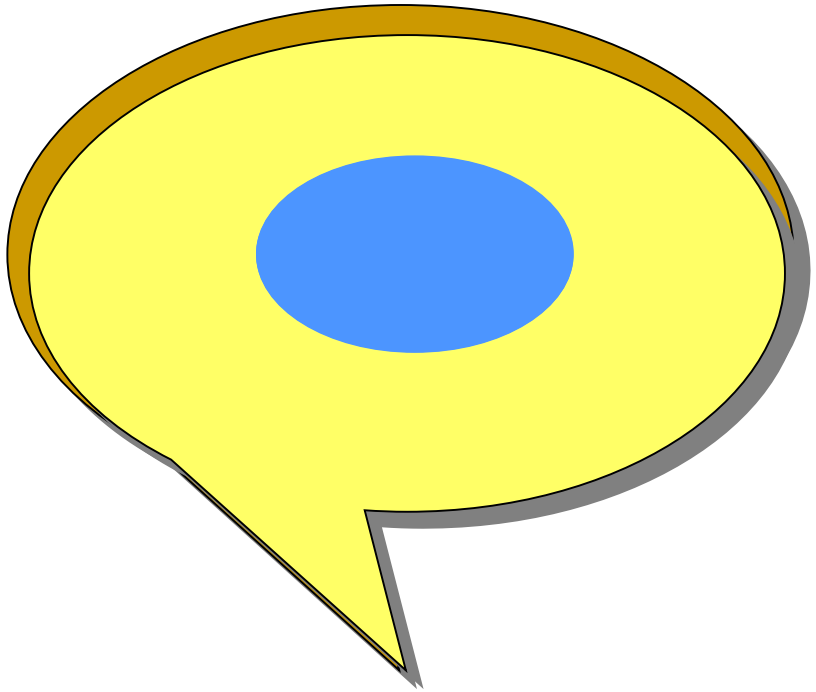
SYSTÉMOVÉ PŘÍČINY

Hypoxemie
Hypokapnie
Hypotenze
Anémie
Hypoglykemie
Hyponatremie
Hypomagnezemie
Hypotyreoza
Panhypopituitarismus
Hepatální encefalopatie

Hypertermie
Hyperkapnie
Hypertenze
Koagulopatie
Hyperglykemie
Hypernatremie
Hypofosfatemie
Hypokortikalismus
Urémie
Sepse

NEUROINTENZIVNÍ PÉČE

PRIMÁRNÍ POŠKOZENÍ MOZKU



SEKUNDÁRNÍ POŠKOZENÍ MOZKU

XXVI. kongres ČSARIM, Brno, 2019

CO JE PODSTATNÉ
NEUROINTENZIVNÍ PÉČE

The economic and clinical benefits of portable head/neck CT imaging in the intensive care unit

Masaryk T, Kolonick R, Painter T, Weinreb DB.

Cleveland Clinic, USA.

There is a 13% morbidity associated with transporting critically-ill patients outside of the ICU. The incidence of adverse events during transport specifically for CT imaging is as high as 71%. The objective of this study was to assess the feasibility and cost-effectiveness of a portable CT scanner designated to perform bedside imaging in the ICU. * A fully mobile 8-slice head/neck CT scanner was evaluated for efficiency and personnel allocation. The return-on-investment for the purchase of the portable scanner was calculated. * Data demonstrates that the introduction of a portable CT scanner in the ICU is feasible and cost-effective. At the Cleveland Clinic in Mayfield Heights, Ohio, the portable scanner provided a full return-on-investment within the first 6.9 months of its operation, an internal rate of return of 169%, and a 5 year expected economic benefit of $2,619,290.

Radiol Manage. 2008 Mar-Apr;30(2):50-4.

CO JE PODSTATNÉ

NEUROINTENZIVNÍ PÉČE

Začíná transportem z operačního sálu

The economic and clinical benefits of portable head/neck CT imaging in the intensive care unit

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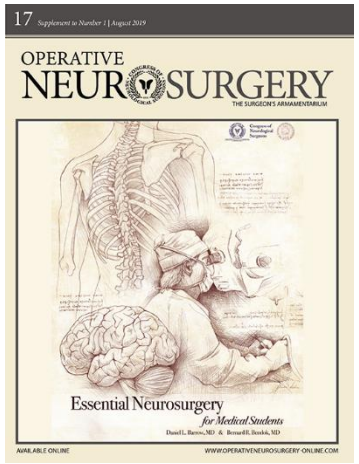


Neurosurgical Physiology and Neurocritical Management of the Acute Neurosurgical Patient
Sanjay Konakondla, MD, Cameron J Brimley, MD, Shelly D Timmons, MD, PhD
Operative Neurosurgery, Volume 17, Issue Supplement_1, August 2019, Pages S17–S44,

Understanding cerebral physiology is paramount in the management of neurosurgical patients. A thorough grounding is required to treat neurosurgical and neurological diseases and injury in the intensive care unit to prevent secondary insults and injury, to promote healing, and ultimately to bring about better outcomes. All brain catastrophes are subject to numerous physiological cascades over time after the initial event, with each having typical timelines of recovery. Such phenomena as cerebral edema, intracranial pressure (ICP) elevation (intracranial hypertension), low cerebral perfusion, hyperthermia, hypoxia, and herniation are regularly observed in the intensive care unit (ICU) and practitioners must not only be able to recognize them early, but also intervene to correct them in the most expeditious and targeted manner to avoid...

TIME IS BRAIN

NEURON

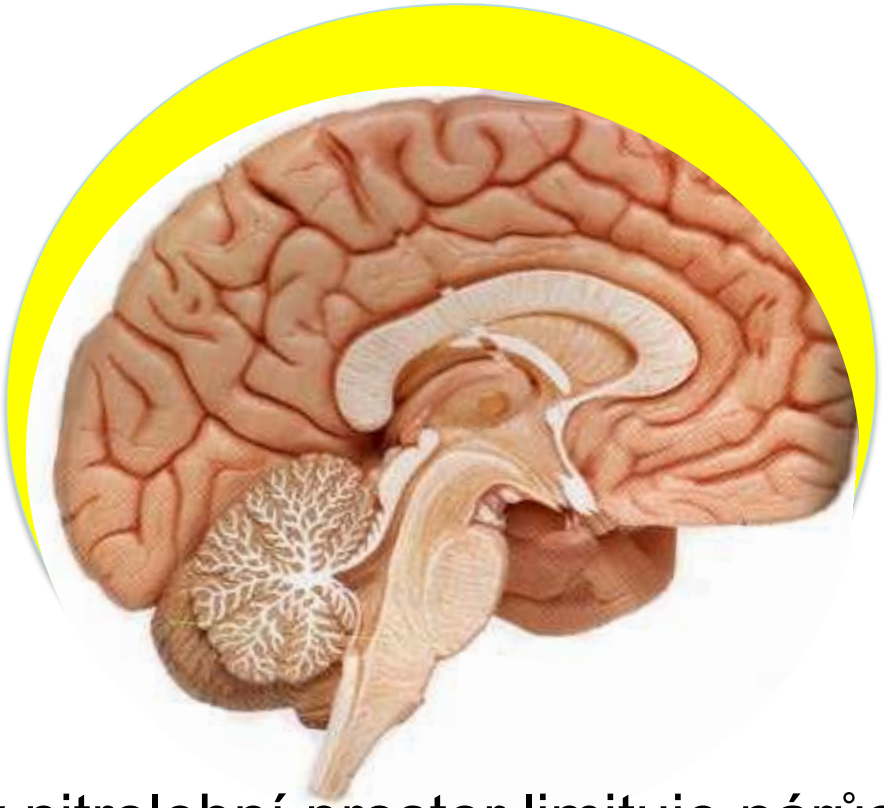
Nejcitlivější buňky k hypoxii

NITROLEBNÍ PROSTOR

Uzavřený – limituje nárůst objemu

TIME IS BRAIN

NITROLEBNÍ PROSTOR



Uzavřený nitrolební prostor limituje nárůst objemu

TIME IS BRAIN

NITROLEBNÍ PROSTOR

Uzavřený nitrolební prostor limituje nárůst objemu

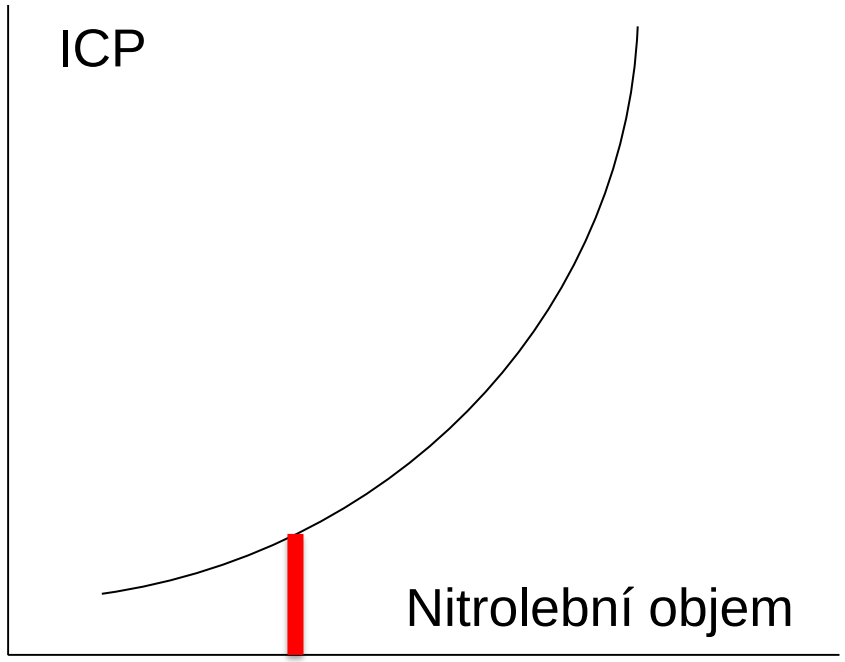
TIME IS BRAIN

MONROOVA – KELLIEHO DOKTRÍNA

V mozku + V krvi + V likvoru = konstatní

TIME IS BRAIN

MONROOVA – KELLIEHO DOKTRÍNA



Nitrolební hypertenze

Nitrolební hypotenze

V mozku + V krve + V likvoru = konstatní

MOZKOVÝ KREVNÍ PRŮTOK

NITROLEBNÍ PROSTOR

Uzavřený nitrolební prostor limituje nárůst objemu

Fyziologické ukazatele optimální hodnoty krevního tlaku pacienta s inzultem mozku

Pavla Mlčková

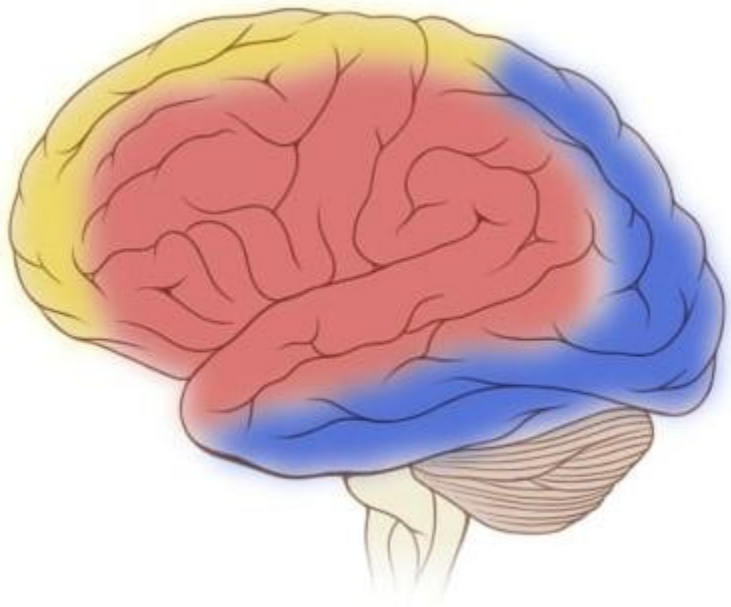
Fyziologický ústav 1. LF UK

ZÁKLADNÍ OTÁZKA

Jak mám řídit hodnotu arteriálního tlaku krve

u pacientů s primárním postižením mozku, aby nedošlo k jeho sekundárnímu poškození?

DODÁVKA O_2 DO MOZKU



ml O_2/min

A-V diference O_2 (ml/l) x V krve (l) /čas

DODÁVKA O_2 DO MOZKU

Který z následujících parametrů umožní nejlépe kvantifikovat dodávku O2 do mozku?

A. Arteriální krevní tlak (MAP, [mmHg])
B. Perfuzní tlak mozku (CPP, [mmHg])
C. Objem krve v mozku (V, [ml/100g])
D. Rychlost krevního toku arterií (FV, např. a.cerebri media - vyšetření TCD, [cm/s])
E. Průtok krve mozkem (CBF, [ml/100g/min])

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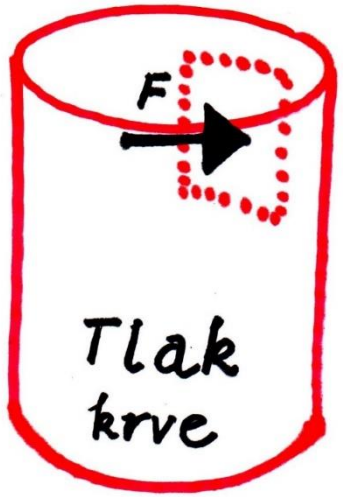

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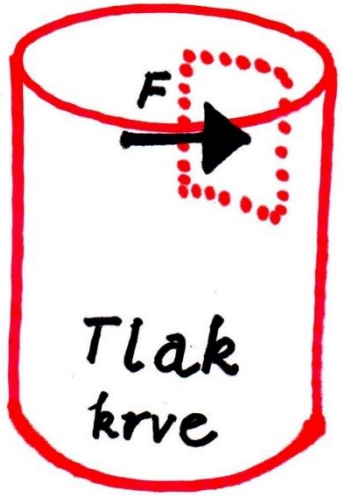

-10 (-20)
(mmHg)

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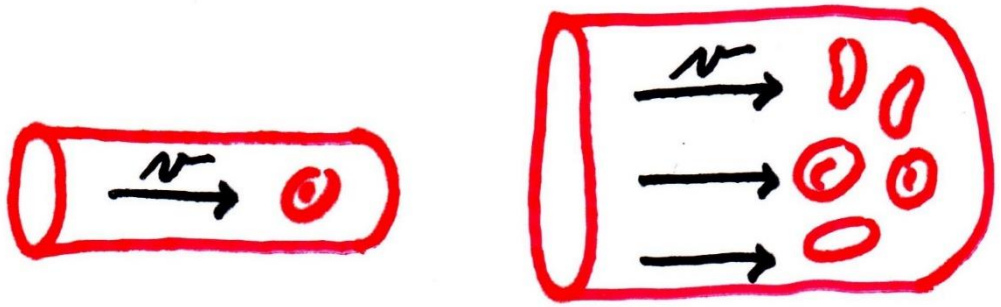

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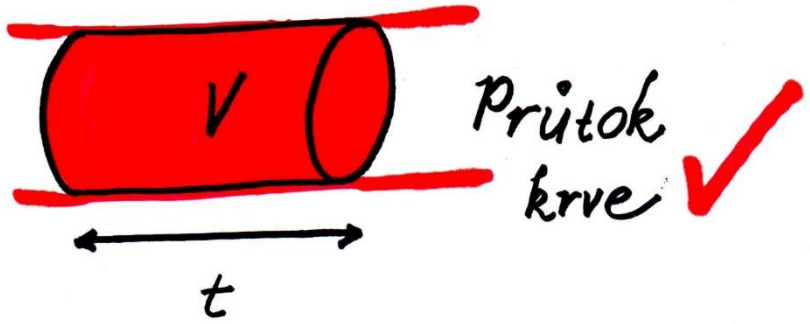


PRŮTOK KRVE MOZKEM

Není snadné změřit: PET, $CMRO_2$

Kontinuálně, to je nereálné

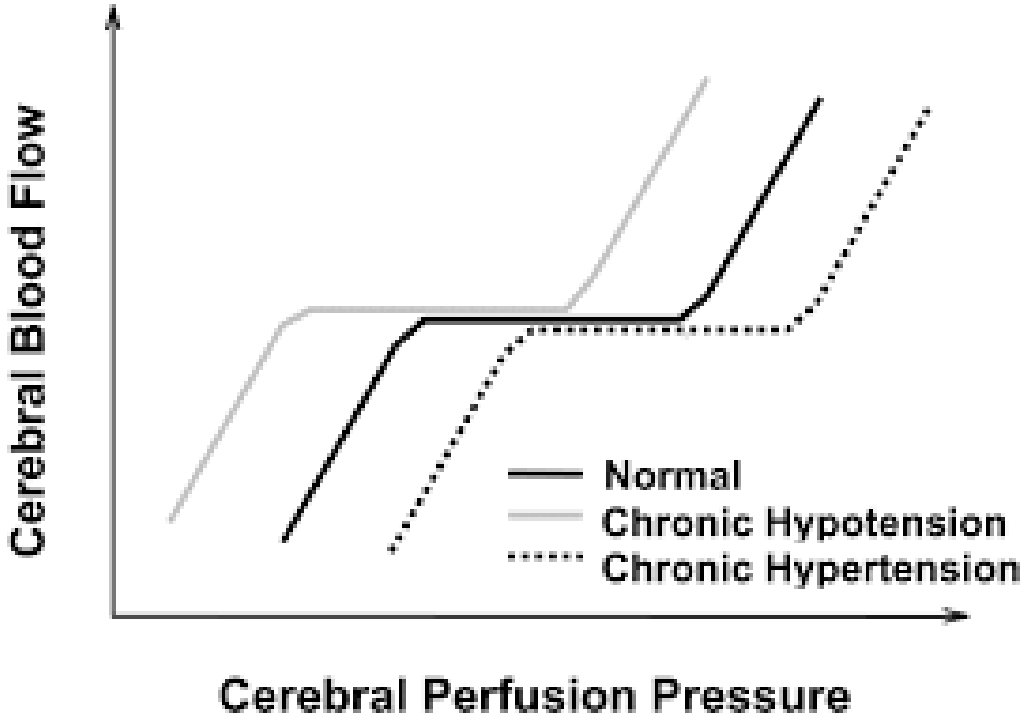
Co s tím??

Hledám náhradní parametr za průtok krve mozkem

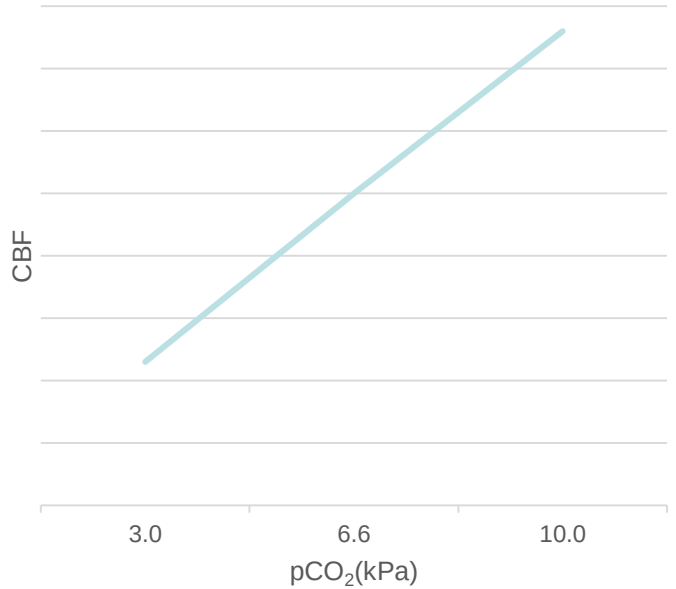
PRŮTOK KRVE MOZKEM

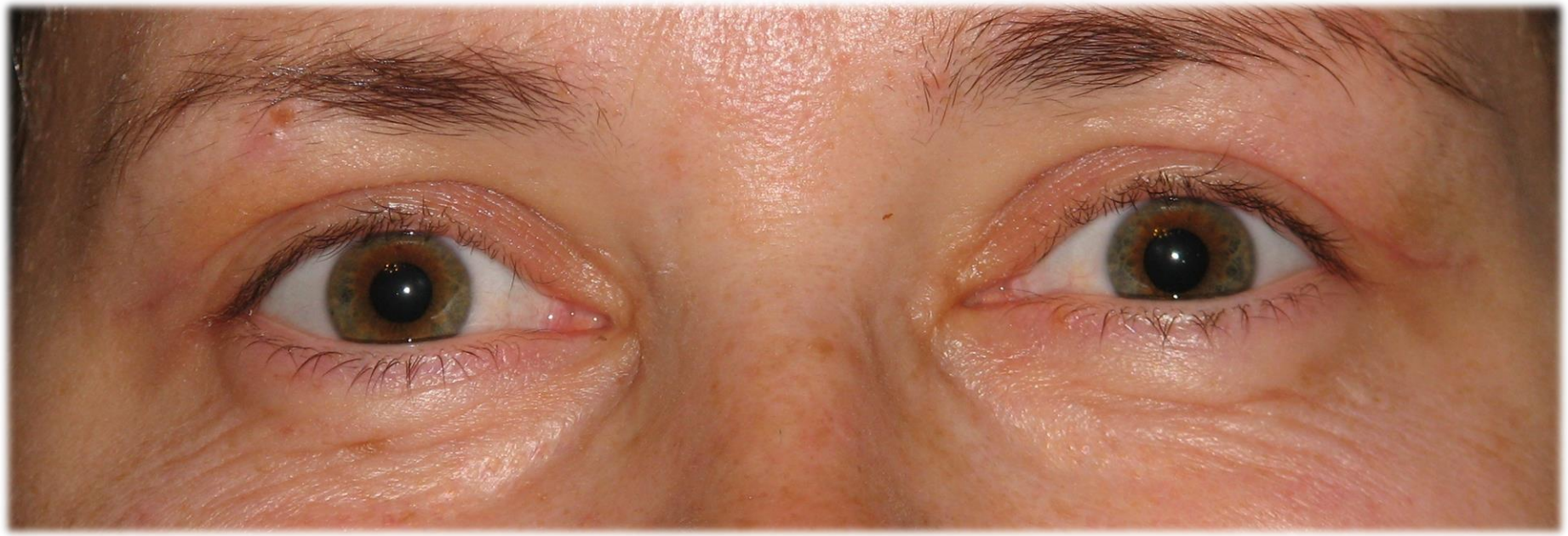
AUTOREGULACE

Myogenní



Metabolická

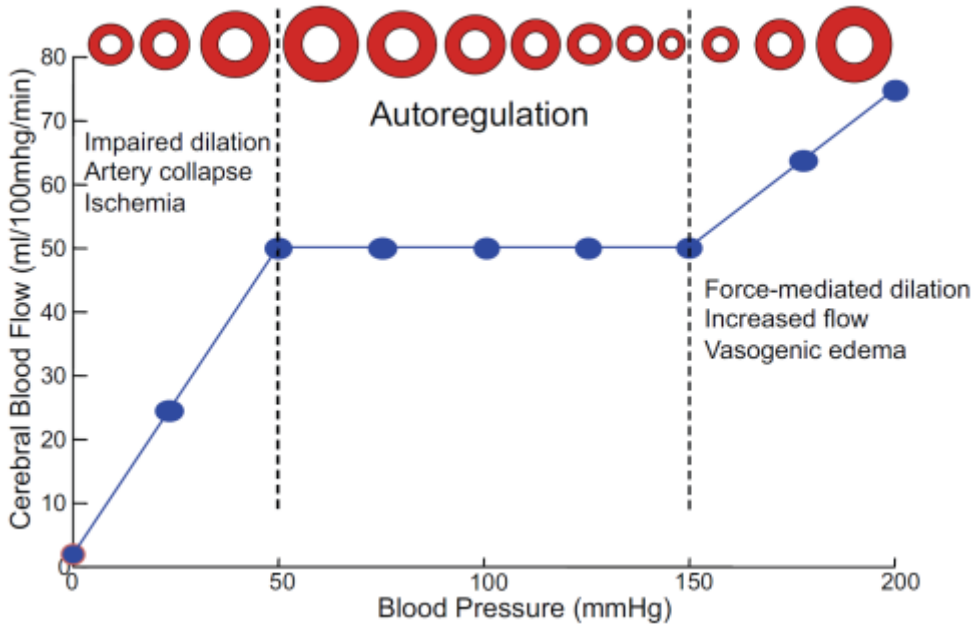




PRŮTOK KRVE MOZKEM

NÁHRADNÍ PARAMETR ZA CBF?

SCHOPNOST AUTOREGULACE?



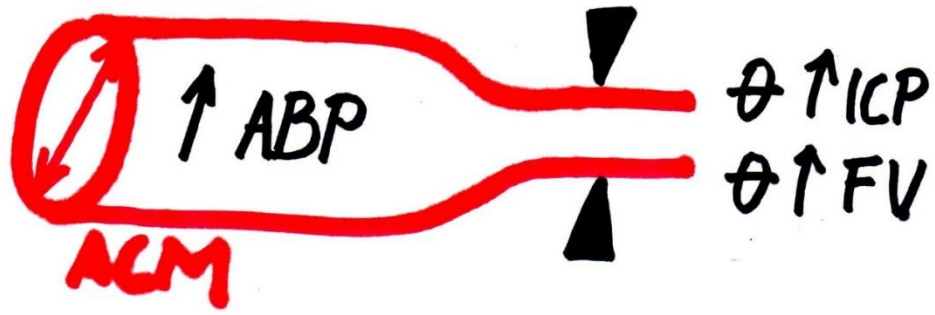
Essays, UK. (November 2018). Cerebral Autoregulation Mechanism

PRŮTOK KRVE MOZKEM

AUTOREGULACE - test

Je-li autoregulace mozkového průtoku zachována:

Projeví se zvýšení MAP o 20 mmHg zvýšením ICP nebo FV?



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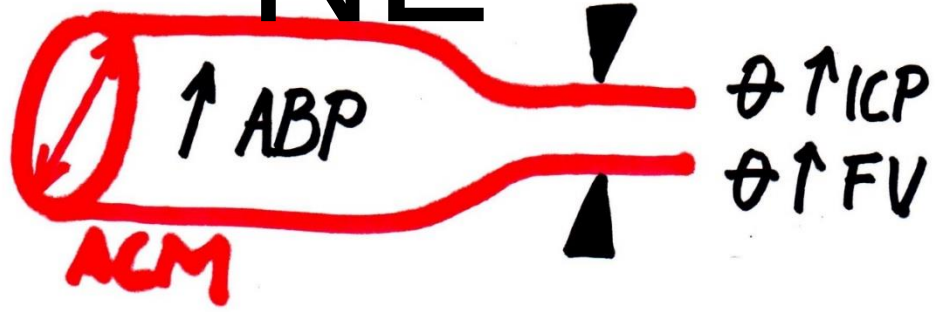
NE

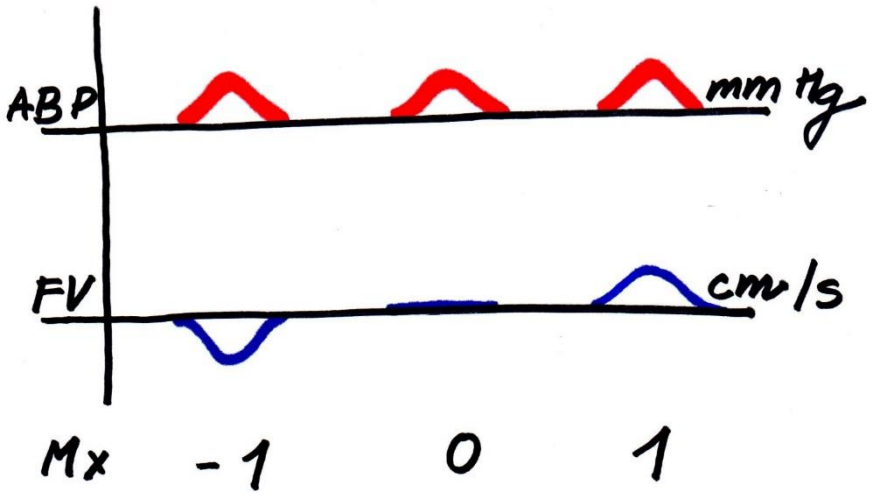


AUTOREGULACE – MONITOROVÁNÍ

INDEXY

PRx = pressure reactivity index:
korelace CPP a ICP

Mx = mean velocity index:
korelace ABP a FV (rychlost toku krve TCD)



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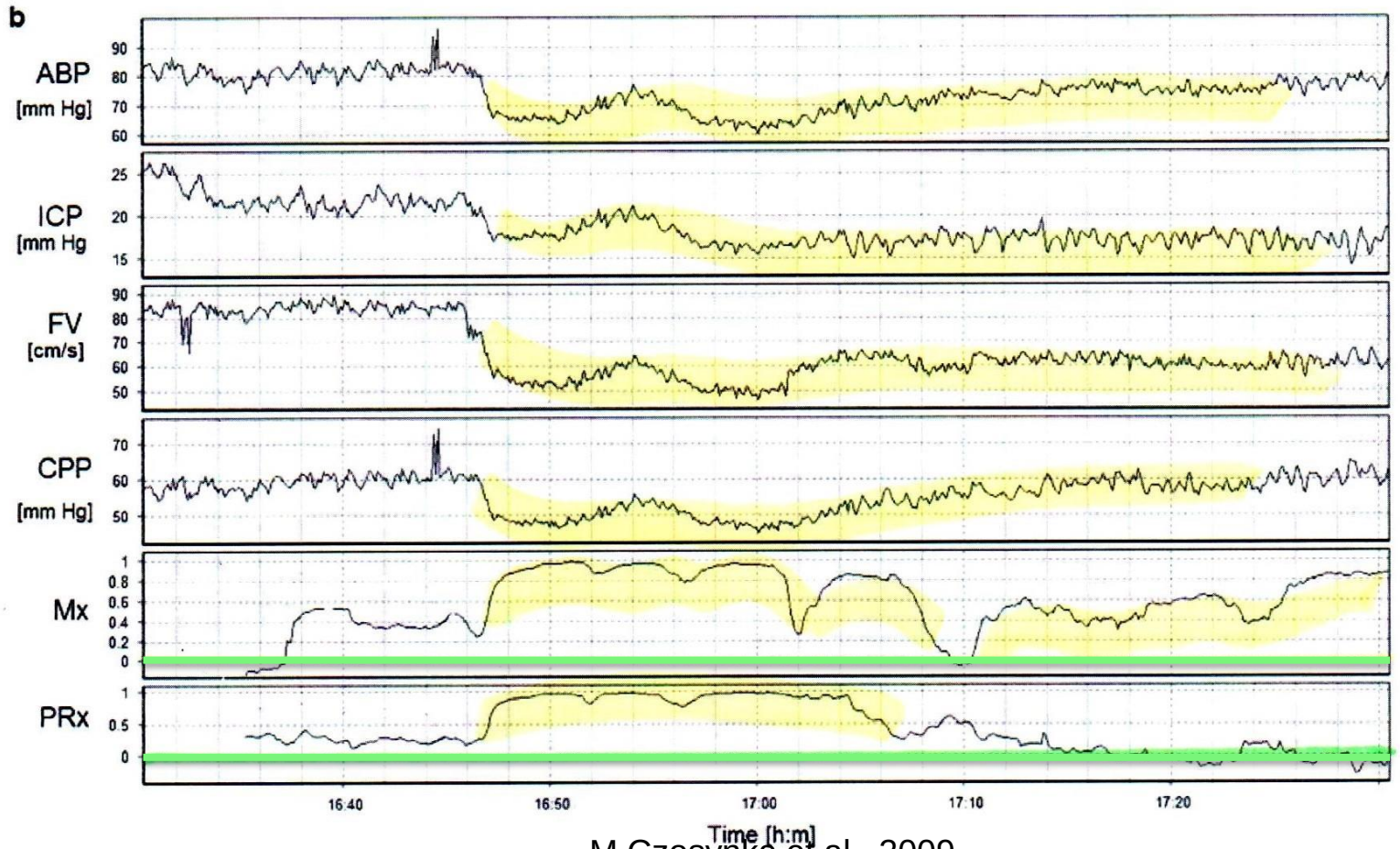


M.Czosynka et al., 2009

PRAKTICKÝ ZÁVĚR 1

Primární postižení mozku→ globální nebo regionální zhoršení tkáňového metabolismu

Schopnost autoregulace krevního průtoku je zhoršená

Pečlivá kontrola ABP je pro prognózu pacienta důležitá

Funkční monitorování adekvátnosti průtoku

PRŮTOK KRVE
INDEXY

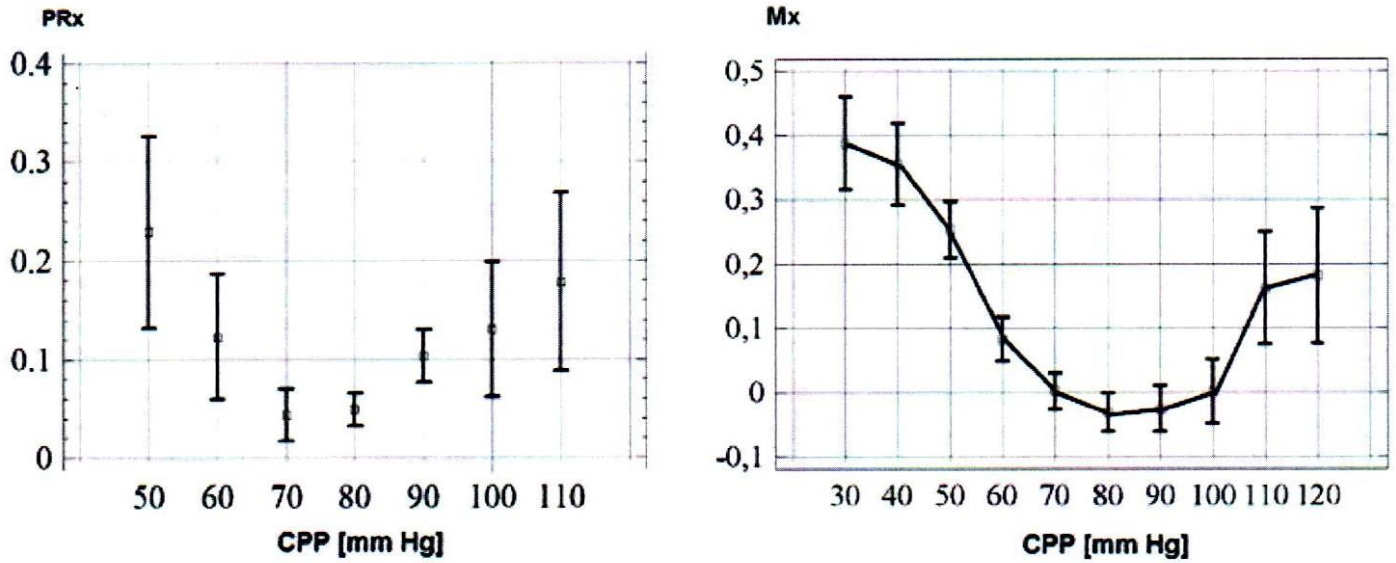


Fig. 6 Relationship between the autoregulation index Mx and the pressure-reactivity index PRx versus CPP in large-group statistics (right panel–270 TBI patients monitored intermittently daily, left panel–397 TBI patients monitored continuously). Both indices show that vascular responses are disturbed at low values of CPP (ischemia) and high values of CPP (hyperemia). There is a range of CPP (70–90 mmHg) 'optimizing' cerebrovascular control. From this finding, the Hypothesis was proposed and since positively verified, that in individual cases, by time-averaging the relationship between PRx and CPP, the delineation of 'optimal CPP' is possible [52]

M. Czosynka et al., 2009

PRAKTICKÝ ZÁVĚR 2

Nemáte-li funkční monitorování k dispozici,

udržujte u pacientů s primárním poškozením mozku perfusní tlak (CPP)

mezi 70 a 80 mmHg

HEMODYNAMIKA

MOZKOVÝ KREVNÍ PRŮTOK (CBF)

&

CEREBRÁLNÍ PERFÚZNÍ TLAK (CPP)

Vypočítaná hodnota ze středního arteriálního tlaku a ICP

MONROOVA – KELLIEHO DOKTRÍNA

V mozku + V krve + V likvoru = konstatní

HEMODYNAMIKA

EUVOLEMIE

Stejný cíl pro všechny typy primárního onemocnění mozku

Hypovolemie
Hypervolemie

MONROOVA – KELLIEHO DOKTRÍNA

V mozku + V krve + V likvoru = konstatní

Stroke

JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association | American Stroke Association

Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage

Stroke 2012;43:00-00

EUVOLEMIE

Maintenance of euvolemia and normal circulating blood volume is recommended to prevent DCI
Revised recommendation from previous guidelines

I. třída, úroveň B

Prophylactic hypervolemia or balloon angioplasty before the development of angiographic spasm is not recommended **New recommendation**

III. třída, úroveň B

3H Hemodiluce, Hypervolemie, Hypertenze → 1H Hypertenze

HEMODYNAMIKA

EUVOLEMIE

BILANCE TEKUTIN

24h, 6h, i na 1h

Příjem tekutin perorální, sonda, intravenózní

Výdej tekutin diuréza (polyurie), zvracení, průjem, pocení 500-1000 ml, krevní ztráty, drény, sonda, ileus, zvýšená tělesná teplota (200 ml na 1° C nad 37° C)

MONROOVA – KELLIEHO DOKTRÍNA

V mozku + V krve + V likvoru = konstatní

HEMODYNAMIKA

EUVOLEMIE

PŘÍJEM TEKUTIN

Základní denní příjem tekutin u dospělých

40ml/kg/den podle BMI u dospělých
do 25 = podle hmotnosti
nad 25 = podle ABW (adjusted body weight)

Substituční příjem tekutin - substituce za ztráty:

polyurie, zvracení, průjem, pocení 500-1000 ml, krevní ztráty, ileus, pankreatitis, teplotou, apod.

HEMODYNAMIKA

EUVOLEMIE

PŘÍJEM TEKUTIN

Fyziologický roztok

0,9% NaCl = 308 mmol/kg

HYPEROSMOLÁLNÍ

Osmolalita plasmy 275 – 295 mmol/kg

MONROOVA – KELLIEHO DOKTRÍNA

V mozku + V krve + V likvoru = konstatní

KREVNÍ TLAK

HYPOTENZE

CPP = MAP – ICP

60 80 20

Zajištění euvolemie s relativně časnou podporou vazopresorů

CÍL

STEJNÝ & RŮZNÝ

KREVNÍ TLAK

HYPOTENZE

NEPŘÍZNIVÉ

- ✓ kolísání TK
- ✓ náhlý pokles TK

→ hypoperfúze ve vulnerabilní oblasti (penambra)

CÍL

STEJNÝ & RŮZNÝ

KREVNÍ TLAK

HYPERTENZE

- ✓ Zvyšuje ICP
- ✓ Zvyšuje riziko krvácení

MONROOVA – KELLIEHO DOKTRÍNA

V mozku + V krve + V likvoru = konstatní

Stroke

JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association | American Stroke Association

Guidelines for the Early Management of Patients With Acute Ischemic Stroke

Stroke. 2013;44:870-947;

INTRAVENÓZNÍ TROMBOLÝZA

S	rtPA	< 185/110 mmHg	I. třída, úroveň B
Bez	rtPA	220/120 mmHg	I. třída, úroveň C

Stroke

JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association | American Stroke Association

Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage

Stroke 2012;43:00-00

HYPERTENZE

Riziko reruptury cíl STK <160 mmHg — IIa. třída, úroveň C

Vasospasmy 1 H — I. třída, úroveň B

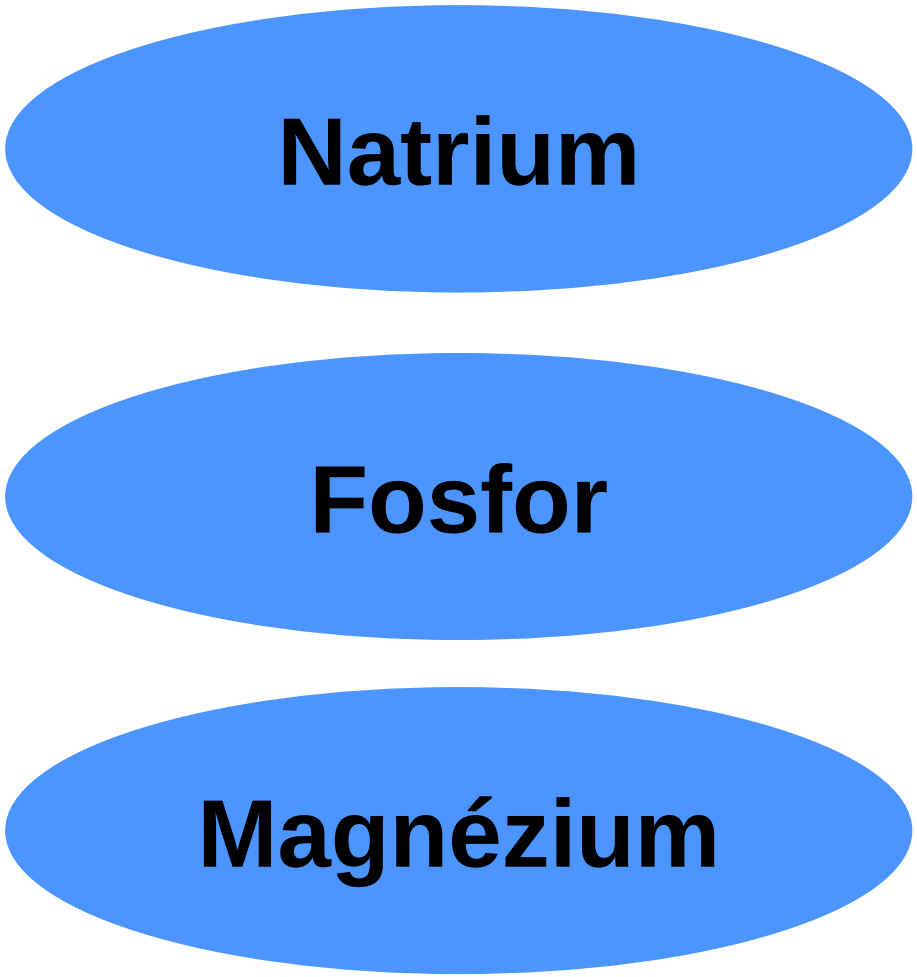
Stroke
JOURNAL OF THE AMERICAN HEART ASSOCIATION

New AHA/ASA Guideline on Spontaneous ICH

HYPERTENZE

For patients with ICH presenting with systolic blood pressure (SBP) between 150 and 220 mm Hg and without contraindication to acute BP treatment, acute lowering of SBP to 140 mm Hg is safe (Class I; level of evidence A) and can be effective for improving functional outcome (Class IIa; level of evidence B; revised from the previous guideline).

MINERÁLY V NEUROINTENZIVNÍ PÉČI



MINERÁLY V NEUROINTENZIVNÍ PÉČI

Fosfor

Zabránit hypofosfatemii

MINERÁLY V NEUROINTENZIVNÍ PÉČI

Magnézium

Zabránit hypomagnezenemii

MINERÁLY V NEUROINTENZIVNÍ PÉČI

Natrium

Hypo / Hypernatremie

Poruchy efektivní osmolality

DYSNATREMIA

Diagnostic management

organism‘s compensatory response

ADH – kidney axis

EFEKTIVNÍ OSMOLALITA

OTÁZKA

20letý pacient přijat v bezvědomí (GCS 5) v ebrietě pro kraniocerebrální trauma: subdurální hematom a kontuze mozku s přesunem středových struktur. Pacient je indikován k akutní operaci.

A. Osmoterapie manitolem.

B. Osmoterapie hypertonickým roztokem natria.

C. Osmoterapie ne.

OTÁZKA

20letý pacient přijat v bezvědomí (GCS 5) v ebrietě pro kraniocerebrální trauma: subdurální hematom a kontuze mozku s přesunem středových struktur. Pacient je indikován k akutní operaci.

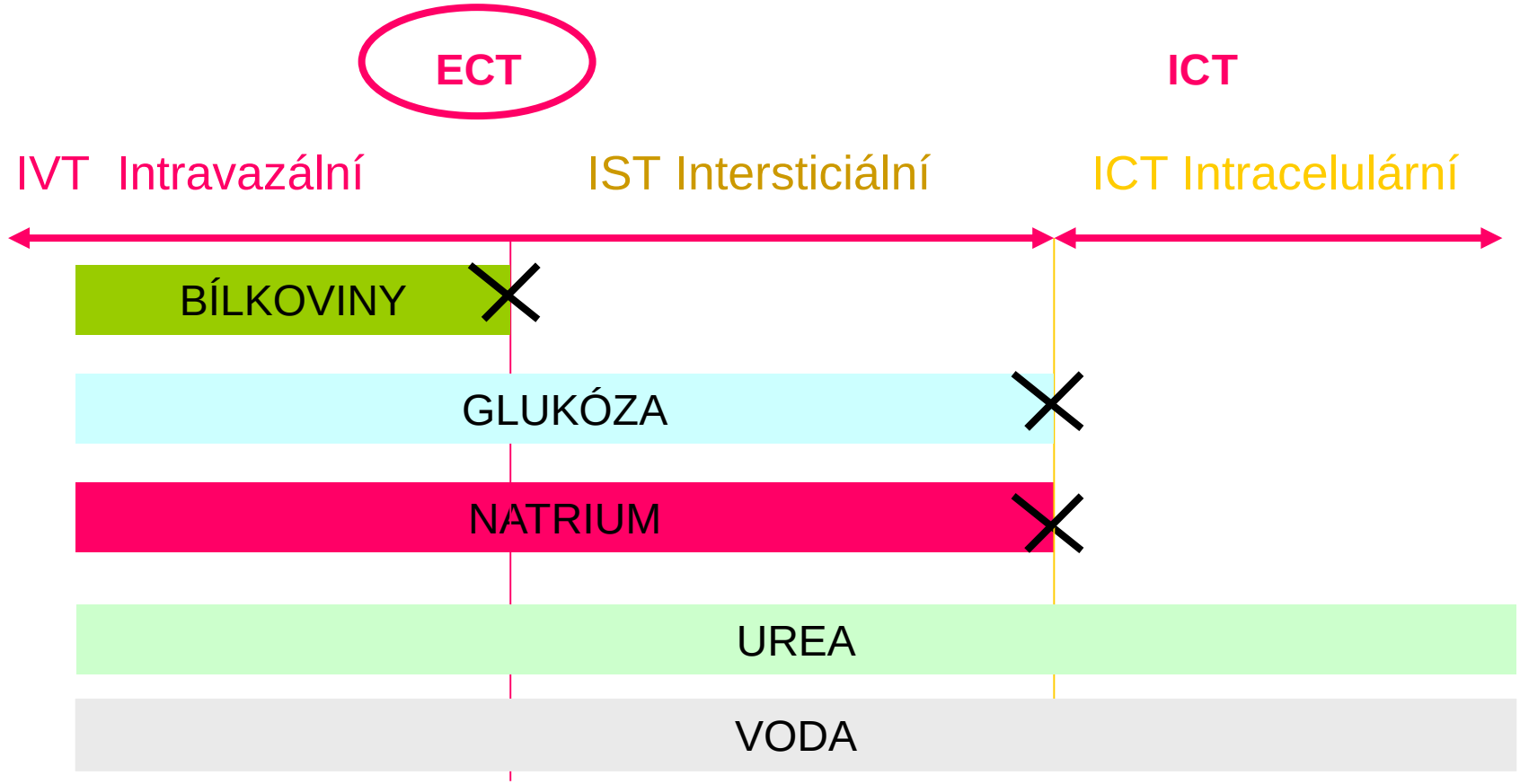
A. Osmoterapie manitolem.

B. Osmoterapie hypertonickým roztokem natria.

C. Osmoterapie ne.

EFEKTIVNÍ OSMOLALITA

ovlivňují jen látky, které se distribuují pouze
v některém prostoru, volně neprochází



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

OSMOLALITA

Měřená serová osmolality

Osmometr

OSMOLALITA

Osmolalita séra měřená SOsm

Osmolalita séra vypočtená SOsmV

2 x SNa + SGLU + SUREA

Osmolalita séra efektivní SOsmE

2 x SNa + SGLU

OSMOLALITA

OSMOLAL GAP > 10 mmol

OG = SOsm – SOsmC

2 x SNa^+ + SGLU + SUREA + Manitol, Alkohol
Metanol

Hlavní extracelulární kationt

Největší podíl na efektivní osmolalitě ECT

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

EFEKTIVNÍ OSMOTICKÝ TLAK

- změna koncentrace v ECT
- vzniká osmotický gradient mezi ECT a ICT
- vyrovnávání

- přesun vody

- edém nebo dehydratace buněk

NATRIUM

HYPO / HYPERNATREME

1. **Akutní poškození mozku**
2. **Následek terapeutických postupů**
3. **Iatrogenní příčiny**

MONROOVA – KELLIEHO DOKTRÍNA

V mozku + V krve + V likvoru = konstatní

NATRIUM

HYPO / HYPERNATREME

NEUROLOGICKÉ PŘÍZNAKY

Edém mozku
Nitrolební hypertenze

Dehydratace mozku
Nitrolební hypertenze

EFEKTIVNĚ OSMOLÁLNÍ DYSNATREMIE

HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

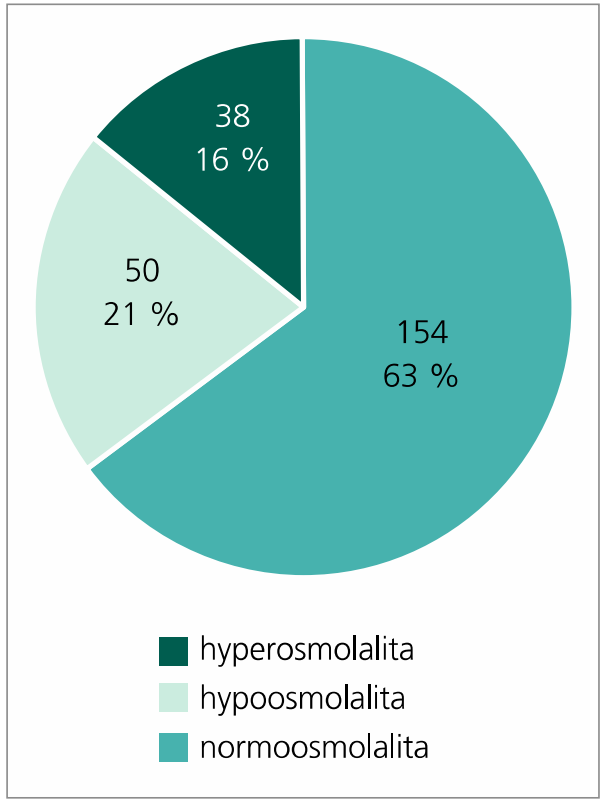
1. krok v diagnostice hyponatrémií

Hodnota měřené sérové osmolality

Diagnosis of Hyponatraemia in Neurointensive Care: the Role of Renal Function Parameters

V. Špatenková[1], A. Kazda[2], P. Barsa[1], V. Beneš[1], P. Škrabálek[3], D. Králová[4], P. Suchomel[1]

[1] Neurocentrum, Krajská nemocnice Liberec, a.s.
[2] Katedra klinické biochemie IPVZ, Praha
[3] Oddělení klinické biochemie, Krajská nemocnice Liberec, a.s.
[4] Institut biostatistiky a analýz, LF a PřF MU, Brno



Graf 1. Počet pacientů s hyponatremií ve vztahu k měřené sérové osmolalitě.

Cesk Slov Neurol N 2008; 71/104(2): 156–162

Acta Neurol Belg (2013) 113:139–145
DOI 10.1007/s13760-012-0137-7

ORIGINAL ARTICLE

Outcome and frequency of sodium disturbances in neurocritically ill patients

Vera Spatenkova · Ondrej Bradac · Pavel Skrabalek

Received: 22 August 2012 / Accepted: 13 September 2012 / Published online: 2 October 2012

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 ($SNa^?$) \ 135 mmol/l (hyponatremia) or $SNa^?$ [150 mmol/l (hypernatremia). Hypoosmolality was defined as measured serum osmolality (SOsm) \ 275 mmol/kg, hyperosmolality as SOsm [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 $SNa^?$ 131.78 ± 2.89 mmol/l, SOsm 279.46 ± 11.84 mmol/kg) and 75 (5 %) pts with hypernatremia (mean $SNa^?$ 154.38 ± 3.76 mmol/l, SOsm 326.07 ± 15.93 mmol/kg). Hypoosmolal hyponatremia occurred in 50 (20 % of hyponatremic patients) pts (mean $SNa^?$ 129.62 ± 4.15 mmol/l; mean SOsm 267.35 ± 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[1] Ondrej Bradac[2] Pavel Skrabalek[3]

[1] Neurocenter, Regional Hospital, Liberec, Czech Republic
[2] Department of Neurosurgery, Military University Hospital and First Medical School, Charles University, Prague, Czech Republic
[3] Department of Clinical Biochemistry, Regional Hospital, Liberec, 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 (SNa^+) < 135 mmol/L and hypernatremia $SNa^+ > 150$ mmol/L. The sodium protocol involved measuring 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; $p = 0.002$).

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.

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

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November 1, 2013
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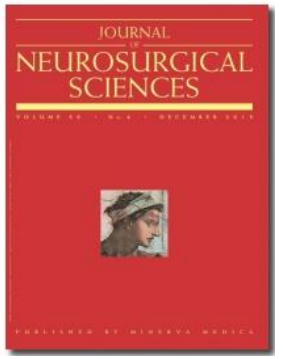
ISSN 2193-6315.

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



Spatenkova V. [1], Bradac O. [2], de Lacy P. [3], Skrabalek P. [4], Suchomel P. [5]

[1] Neurocenter, Neurointensive Care Unit, Regional Hospital, Liberec, Czech Republic; [2] Department of Neurosurgery, Central Military Hospital, Charles University, Prague, Czech Republic; [3] Department of Neurosurgery, Royal Hallamshire Hospital, Sheffield, United Kingdom; [4] Department of Clinical Biochemistry, Regional Hospital, Liberec, Czech Republic; [5] 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±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±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.

NATRIUM

HYPO / HYPERNATREME

1. **Akutní poškození mozku**
2. **Následek terapeutických postupů**
3. **Iatrogenní příčiny**

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DYSNATREMIA

Diagnostic management

organism‘s compensatory response

ADH – kidney axis

RENÁLNÍ FUNKČNÍ PARAMETRY

Posouzení osy ADH-ledviny s využitím parametru EWC

Hypoosmolalita, hypotonicita – sérová osmolalita < 280 mmol/kg

EWC > 0,116 ml/s (10 l/den)……..normální odpověď ADH-ledviny

EWC 0,006 – 0,116 ml/s…….zhoršená odpověď osy ADH-ledviny

EWC < 0,006 ml/s (0,5 l/den)……..abnormální odpověď ADH-ledviny

Hyperosmolalita, hypertonicita – sérová osmolalita > 295 mmol/kg

EWC < 0,005 ml/s (0,4 l/den)…….normální odpověď ADH-ledviny

EWC ≥ 0,005 ml/s…….abnormální odpověď ADH-ledviny

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

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

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cDI

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HYPONATREMIE V NEUROINTENZIVNÍ PÉČI

1. **Akutní poškození mozku**
   CSWS, SIADH

2. **Následek terapeutických postupů**
   Thiazidy

3. **Iatrogenní příčiny**
   Hypotonický roztok
   Iatrogenní SIADH (normonatremie a desmopressin)

HYPERNATREMIE V NEUROINTENZIVNÍ PÉČI

1. **Akutní poškození mozku**
   Centrální diabetes insipidus (CDI)

2. **Následek terapeutických postupů**
   Osmoterapie – Manitol, NaCl, Furosemid

3. **Iatrogenní příčiny**
   Zvýšený příjem soli, profuzní pocení

CO JE PODSTATNÉ

Postoperative care of neurosurgical patients: general principles

Gahan Bose, Astri M.V. Luoma

Anaesthesia and Intensive Care Medicine
June 2017Volume 18, Issue 6, Pages 296–303

The primary aim of care immediately after neurosurgery is to detect and prevent neurological deterioration while supporting systemic and neurological homoeostasis. Surgical-, anaesthetic- or disease- related factors may contribute to a slow return or failure to regain a patient's preoperative status. A period of specific monitoring and observation by nursing and medical staff accustomed to neurosurgical and neurocritical care procedures should be planned for the immediate postoperative period. In many neurosurgical centres the period of postoperative observation may be relatively short (e.g. limited uneventful craniotomies); however, if complicating factors such as cerebral oedema, intracranial haemorrhage, seizures or significant premorbid conditions are present, a period of higher dependency care over several days may be anticipated. In common with all postoperative care safe management of the airway, weaning of ventilatory support, control of circulation and fluid balance, feeding, sedation and analgesia are the mainstays of care. Meticulous attention to each of these is essential in the post neurosurgical patient as poor management can profoundly affect neurological outcome. Thus a robust perioperative plan is mandatory for management of the airway, control of blood pressure, and to ensure continuation of preoperative medication. Furthermore, the plan may entail elective creation of tracheostomy and percutaneous endoscopic gastrostomy. The early postoperative neurosurgical patient continues to require a high degree of clinical vigilance.

NEUROMONITORACE

GLASGOW COMA SCALE

NEUROLOGICKÉ VYŠETŘENÍ

NEUROMONITORACE

GLASGOW COMA SCALE

Správně hodnotit

NEUROLOGICKÉ VYŠETŘENÍ

GLASGOW COMA SCALE (GCS)

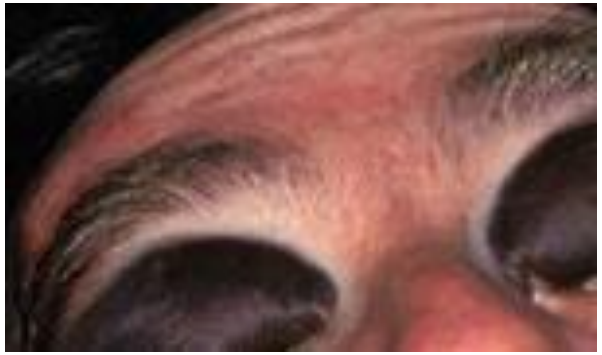
Škála hodnotící stupeň poruchy vědomí

3	4	5	6	7	8	9	10	11	12	13	14	15
Těžké						Střední				Lehké		

SPRÁVNĚ HODNOTIT

- ~~GCS 3, analgosedace~~

- GCS nelze hodnotit, analgosedace, popsat, co dělá při analgosedaci



NEUROMONITORACE

PACIENT V BEZVĚDOMÍ

PACIENT V ANALGOSEDACI

~~NEUROLOGICKÉ VYŠETŘENÍ~~

Intensive Care Med (2014) 40:1189–1209
DOI 10.1007/s00134-014-3369-6

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Gretchen M. Brophy
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Consensus summary statement of the International Multidisciplinary Consensus Conference on Multimodality Monitoring in Neurocritical Care

A statement for healthcare professionals from the Neurocritical Care Society and the European Society of Intensive Care Medicine

We recommend against performing sedation interruption or wake-up tests among brain-injured patients with intracranial hypertension, unless benefit outweighs the risk. (Strong recommendation, low quality of evidence.)

SPECIÁLNÍ NEUROMONITOROVACÍ TECHNIKY

~~BLACK BOX~~

Prof. Stephan A. Mayer, MD,
Neurointenzivista, USA

XXVI. kongres ČSARIM, Brno, 2019

Intensive Care Med (2014) 40:1189–1209
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NOVÉ METODY NEUROMONITORACE

NOVÉ METODY

STANDARDNÍ METODY

MULTIMODÁLNÍ NEUROMONITORACE

INVAZIVNÍ, NEINVAZIVNÍ

KONTINUÁLNÍ, NEKONTINUÁLNÍ

GLOBÁLNÍ, REGIONÁLNÍ

MULTIMODÁLNÍ NEUROMONITORACE

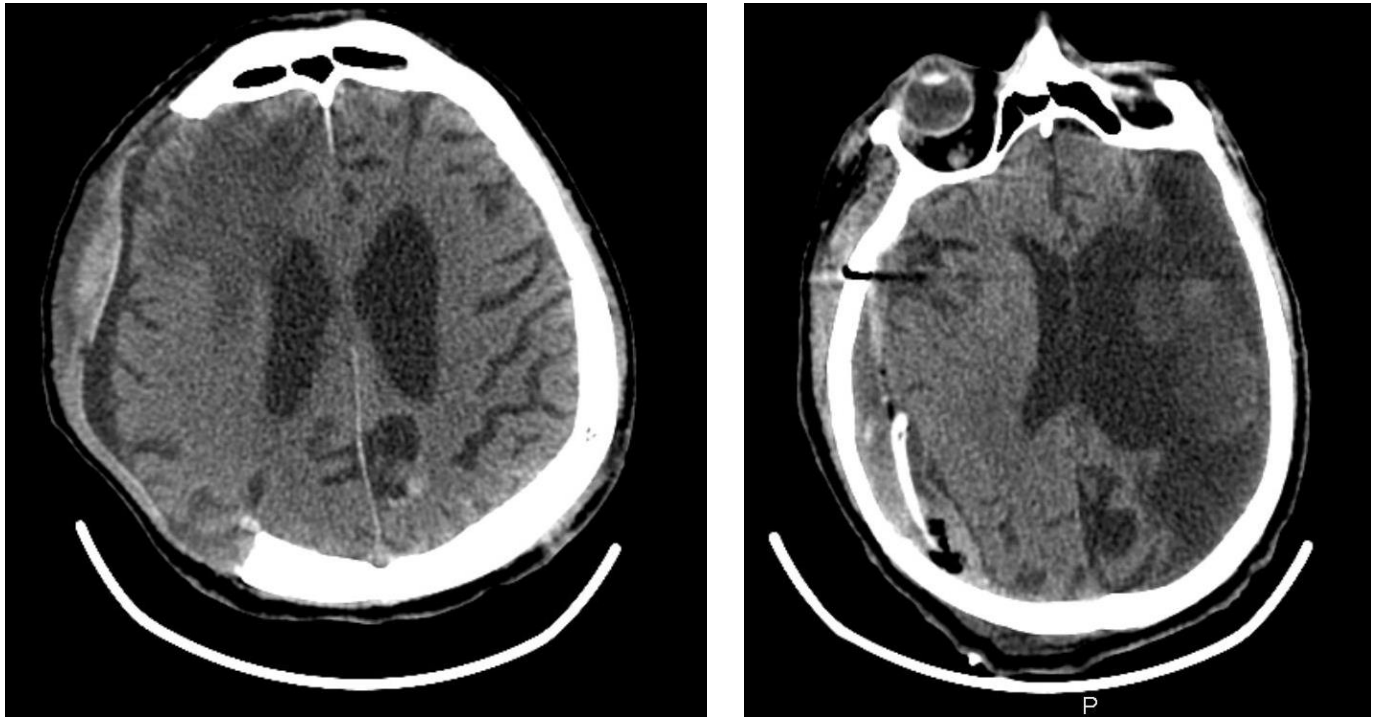
MULTIMODÁLNÍ MONITORACE

MOBILNÍ CT MOZKU

RTG oddělení - 24 hodin / 7 dní

MULTIMODÁLNÍ MONITORACE

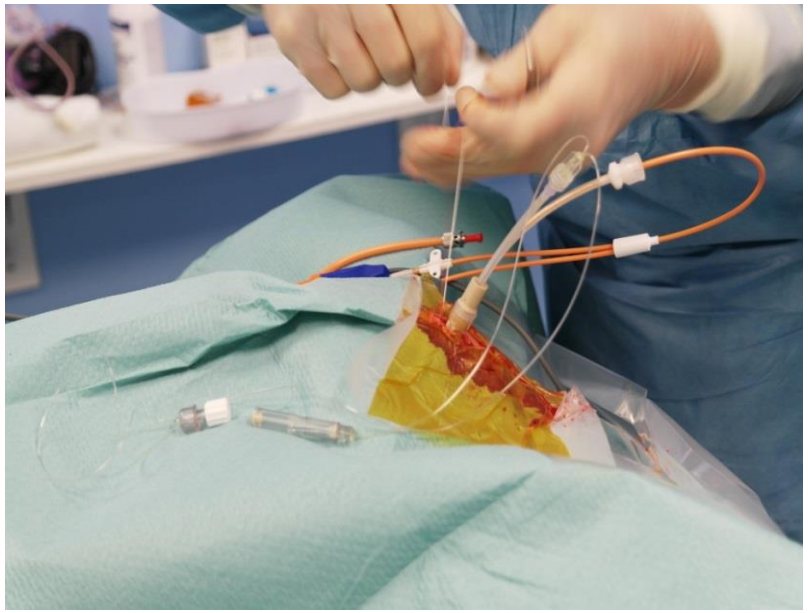
MOBILNÍ CT MOZKU



RTG oddělení - 24 hodin / 7 dní

MULTIMODÁLNÍ MONITORACE

MOZKOVÁ OXYMETRIE



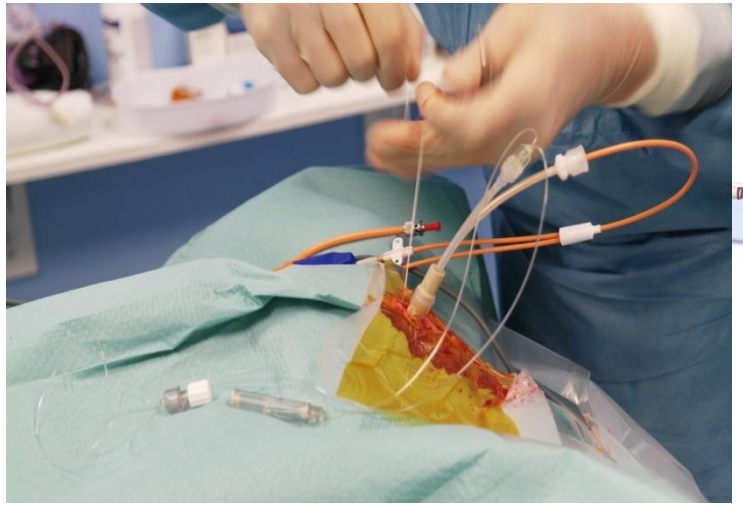
MULTIMODÁLNÍ MONITORACE

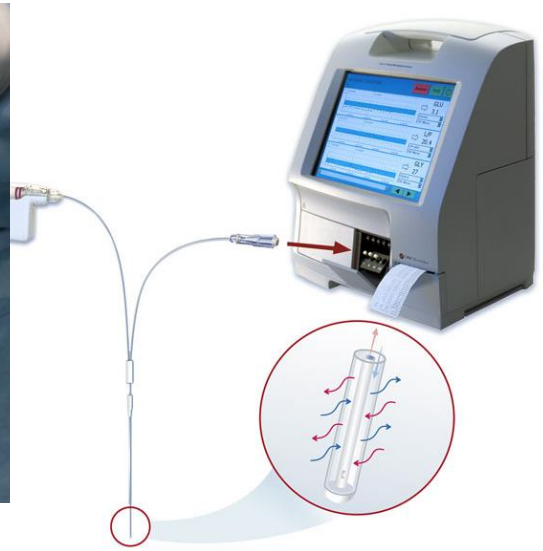
MOZKOVÁ MIKRODIALÝZA

Invazivní, semikontinuální, regionální bedside metoda

Metabolity z extracelulárního prostoru mozkové tkáně:

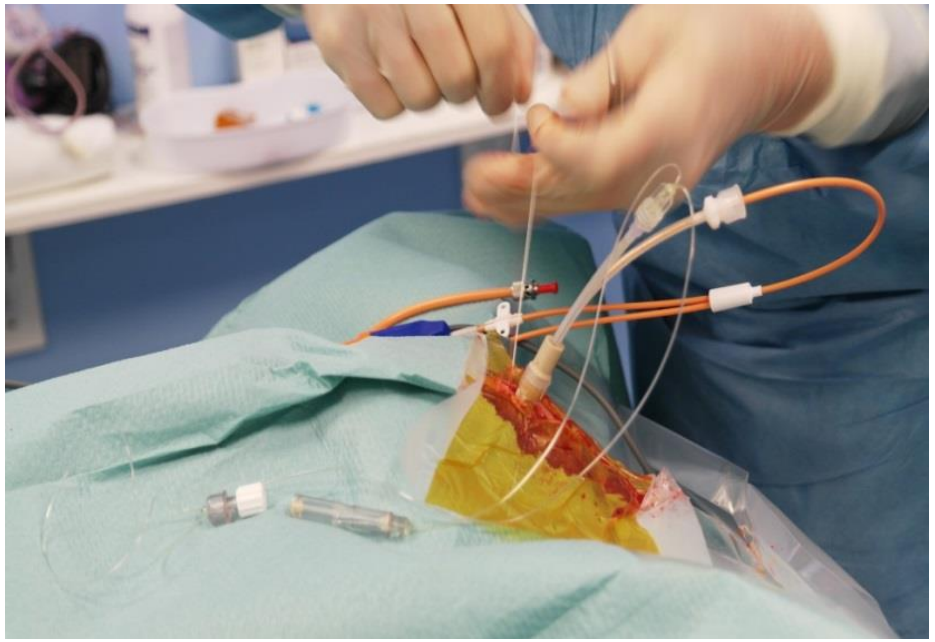
Glukóza
Laktát
Pyruvát
Glutamát
Glycerol





MULTIMODÁLNÍ MONITORACE

MOZKOVÁ MIKRODIALÝZA
MOZKOVÁ OXYMETRIE



MULTIMODÁLNÍ MONITORACE

MOZKOVÁ MIKRODIALÝZA

Intensive Care Med (2015) 41:1517–1528
DOI 10.1007/s00134-015-3930-y

CONFERENCE REPORTS AND EXPERT PANEL

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Consensus statement from the 2014 International Microdialysis Forum

used. There is now evidence from large numbers of patients on how abnormal brain chemistry relates to clinical outcome. The measurement of glucose, lactate and the LP ratio are now considered more useful than glutamate and glycerol. The LP ratio, interpreted in the light of absolute pyruvate concentrations and $PbtO_2$, can be used to differentiate ischemic from non-ischemic causes of energy dysfunction. Importantly, there is increasing evidence of how different therapeutic manoeuvres influence brain chemistry. Microdialysis is well placed to help guide the management of patients in an individualized and targeted fashion. For its effective use, microdialysis should be integrated into brain multi-modal monitoring systems and interpreted with knowledge of catheter location and clinical context. Future clinical research should focus on assessing

ZÁVĚR

NEUROINTENZIVNÍ PÉČE

Pacient po neurochirurgickém výkonu

Klinický výsledek

Určen nejen velikostí, lokalizací a typem primárního onemocnění mozku,
ale i rozsahem
sekundárního poškození mozku.

TIME IS BRAIN

ČASOVÁ NALÉHAVOST

Diagnostických a terapeutických postupů

primárního
sekundárního

poškození mozku

PREVENCE
SEKUNDÁRNÍ POŠKOZENÍ MOZKU
IATROGENNÍ PŘÍČINY

PREVENCE
POMOCÍ APLIKOVANÉ FYZIOLOGIE

NČSIM | NEUROINTENZIVNÍ SEKCE ČESKÉ SPOLEČNOSTI INTENZIVNÍ MEDICÍNY

AKADEMIE NEUROINTENZIVNÍ MEDICÍNY

Vzdělávání v neurointenzivní péči

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XXVI. kongres ČSARIM, Brno, 2019

Děkujeme za pozornost

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