

Kóma, intoxikácie



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II. klinika anestéziológie a intenzívnej medicíny
Fakultná nemocnica s poliklinikou F. D. Roosevelta
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MINISTERSTVO
PÔDOHOSPODÁRSTVA
A ROZVOJA VIDIEKA
SLOVENSKEJ REPUBLIKY



Vedomie – „fyziológia“

- Plné uvedomovanie
 - seba
 - svojho vzťahu k okoliu
- Schopnosť reagovať na vonkajšie podnety a vnútorné potreby
 - uvedomele
 - adekvátne

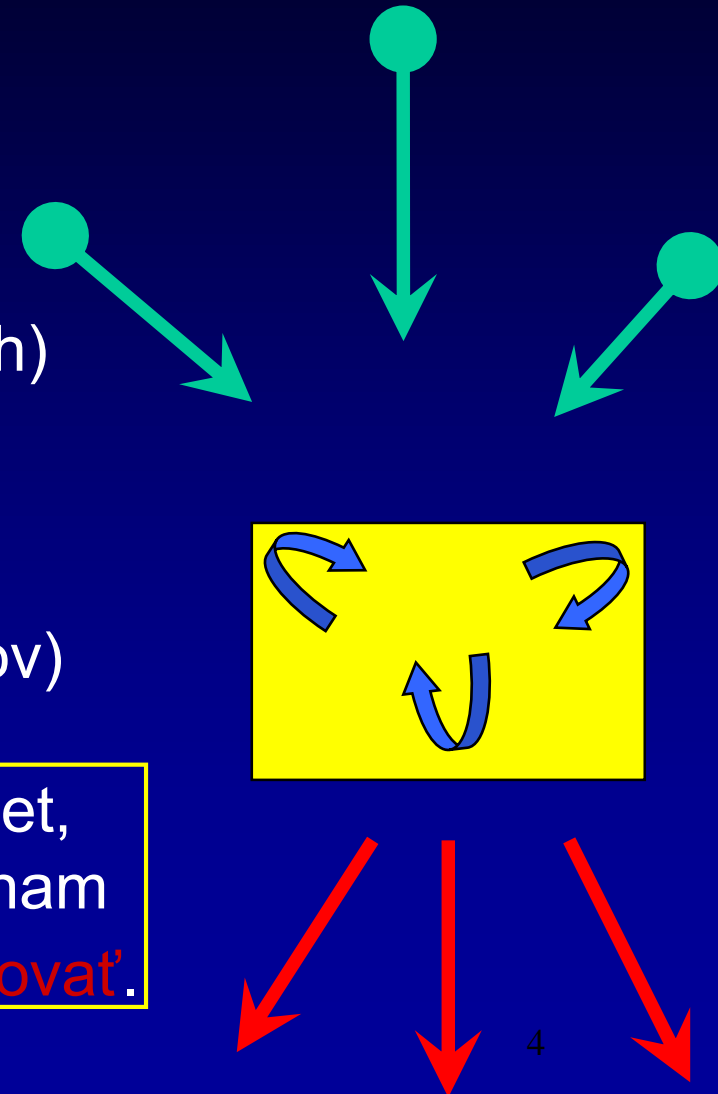


Vedomie – štruktúra procesu

Proces uvedomovania a reakcie:

- **Podnet** (stav snímačov a dostredivých dráh)
- **Spracovanie** v centre (integrita centier)
- **Reakcia** (stav odstredivých dráh a efektorov)

Podmienky: subjekt dokáže registrovať podnet, pochopiť jeho význam a na základe toho **vie, môže a chce** reagovať.



Zmeny vedomia

- **Kvalitatívne**

kognitívne
behaviorálne
afektívne



poruchy

- **Kvantitatívne**

plné vedomie

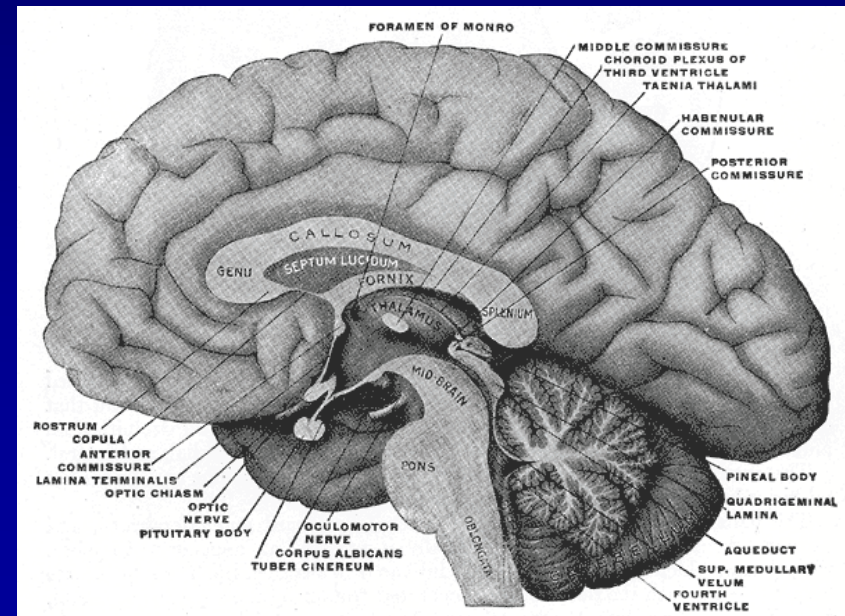
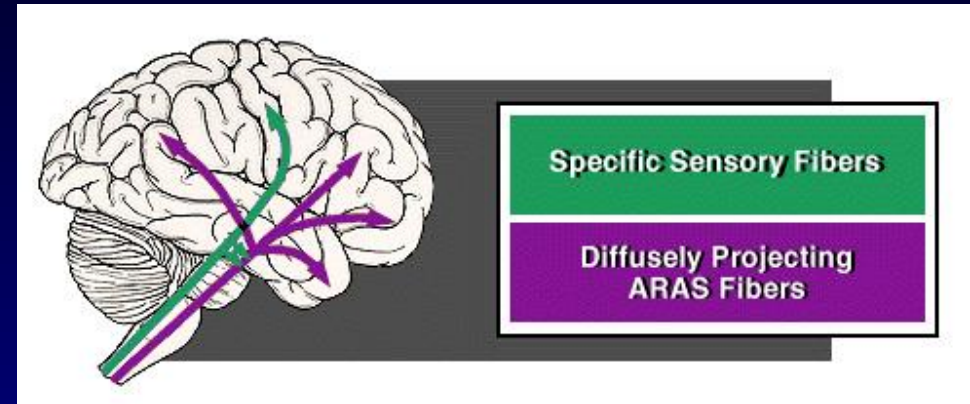


areflexná kóma
(mozgová smrť)

Vedomie – „anatómia“

Komponenty vedomia:

- **Bdelosť (alertness, arousal)**
ascendentný retikulárny
aktivačný systém (ARAS)
(mozgový kmeň, mezencefalon,
diencefalón)
- **Uvedomovanie (awareness)**
suma kôrových funkcií
(kognitívnych, afektívnych)



Stavy vedomia

Obsah vedomia (uvedomovanie)

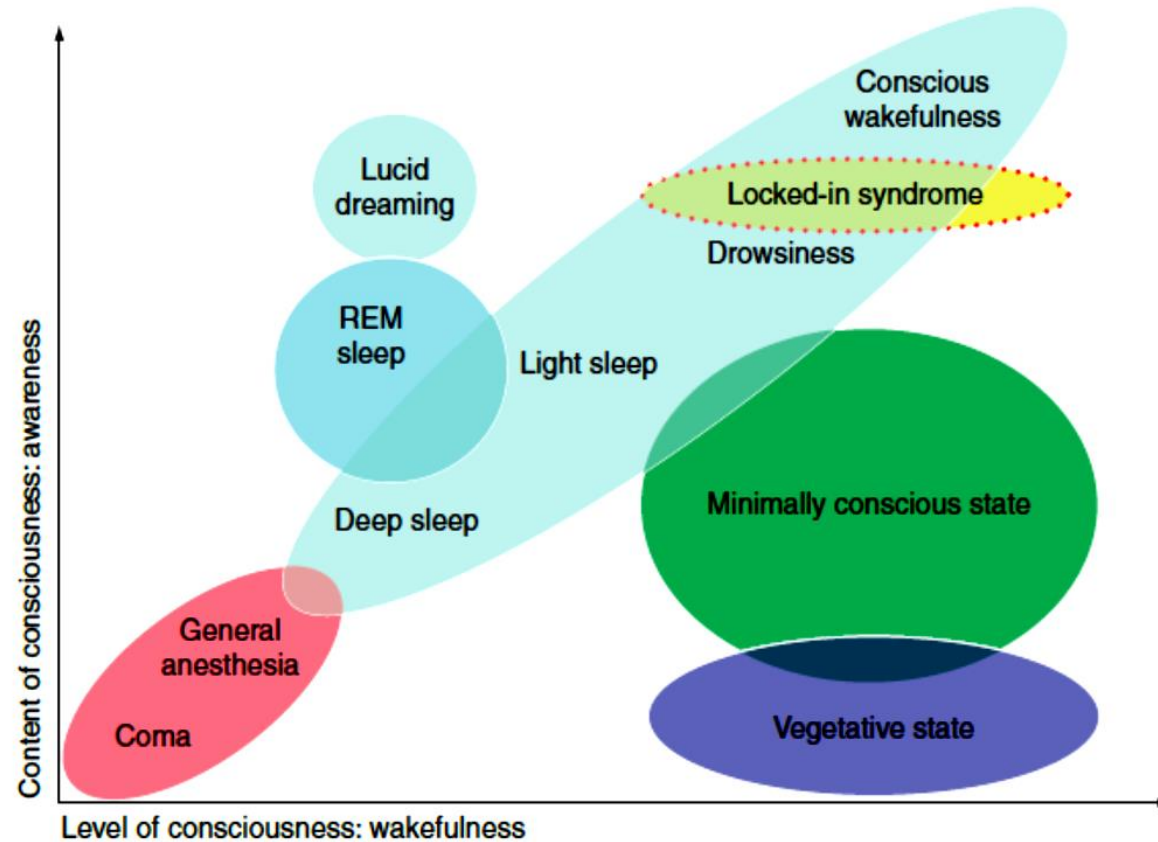


Figure 1 Oversimplified illustration of the two major dimensions of consciousness: the level of consciousness (i.e., arousal or wakefulness) and the content of consciousness (i.e., awareness or experience). Adapted from Laureys S (2005) The neural correlate of (un)awareness: Lessons from the vegetative state. *Trends in Cognitive Sciences* 9: 556–559.

Úroveň vedomia (bdelosť)

Stavy vedomia

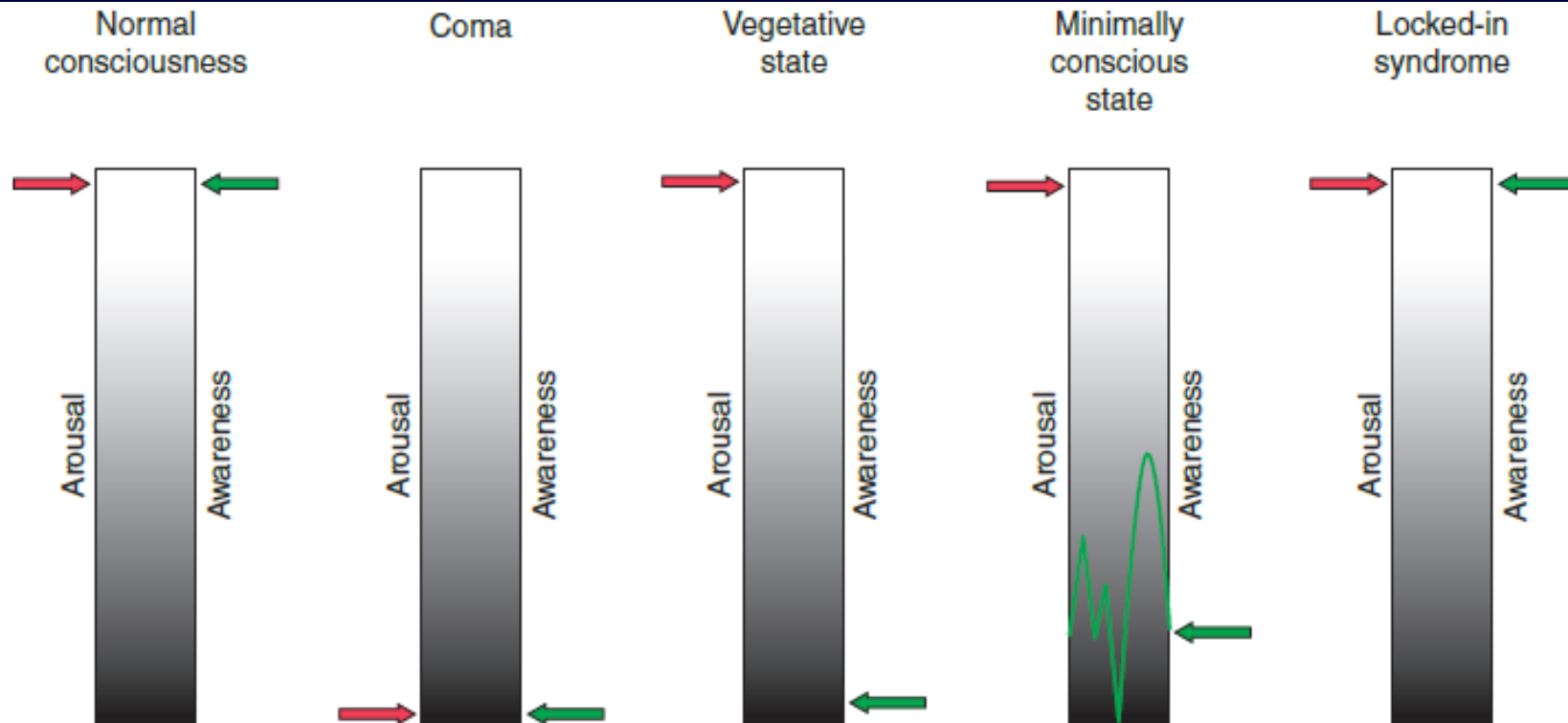


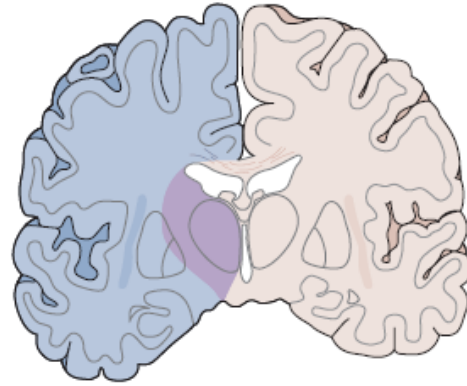
Figure 2 Graphical representation of the two dimensions of consciousness: arousal (red arrow) and awareness (green arrow) and their alterations in coma, the vegetative state, the minimally conscious state, and the locked-in syndrome. Adapted from Laureys S, Owen AM, and Schiff ND (2004) Brain function in coma, vegetative state, and related disorders. *Lancet Neurology* 3: 537–546.

Bezprostredná príčina kómy

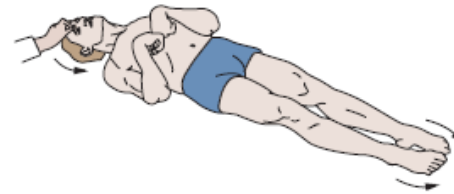
Poškodenie

- **kôrových štruktúr (bilaterálne)** a/alebo
- **retikulárneho aktivačného systému**
- **Primárne** poškodenie
etiologické faktory (dej, ktorý sa už stal: úraz, otrava, hypoxický infarkt atď.)
- **Sekundárne** poškodenie
(potenciálne) ohrozenie ďalším patofyziologickým vývojom

A Metabolic encephalopathy



B Upper midbrain damage



C Upper pontine damage

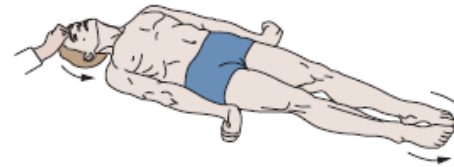
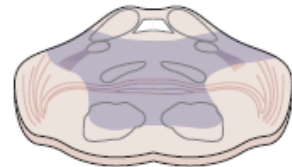


Figure 2–10. Motor responses to noxious stimulation in patients with acute cerebral dysfunction. Levels of associated brain dysfunction are roughly indicated at left. Patients with forebrain or diencephalic lesions often have a hemiparesis (note lack of motor response with left arm, externally rotated left foot, and left extensor plantar response), but can generally make purposeful movements with the opposite side. Lesions involving the junction of the diencephalon and the midbrain may show decorticate posturing, including flexion of the upper extremities and extension of the lower extremities. As the lesion progresses into the midbrain, there is generally a shift to decerebrate posturing (C), in which there is extensor posturing of both upper and lower extremities. (From Saper, C. Brain stem modulation of sensation, movement, and consciousness. Chapter 45 in: Kandel, ER, Schwartz, JH, Jessel, TM. *Principles of Neural Science*. 4th ed. McGraw-Hill, New York, 2000, pp. 871–909. By permission of McGraw-Hill.)

Posúdenie kvantitatívnych zmien vedomia I

- **Subjektívne**

(z hľadiska osoby posudzujúceho)

- **Objektívne**

(skórovacie systémy)

Posúdenie kvantitatívnych zmien vedomia II

- Verbálny opis

Akútne zmeny

Obnubilácia

Delírium

Sopor

Stupor

Kóma

Locked-in sy.

Smrť mozgu (mozgového kmeňa)

Subakútne alebo chronické zmeny

Demencia

Hypersomnia

Abúlia

Akinetický mutizmus

Minimálne vedomie

Vegetatívne stavy

Posúdenie kvantitatívnych zmien vedomia III

- Objektivizácia:
skórovacie systémy

Glasgow Coma Scale
(GCS)

Full Outline UnResponsiveness System
(FOUR)

Glasgow Coma Scale

Glasgow Coma Scale

(3 – 15 b.)

| Bodové ohodnotenie | Očná reakcia | Slovná reakcia | Motorická reakcia |
|--------------------|------------------|----------------|---|
| 6 | - | - | vykoná príkaz |
| 5 | - | orientovaná | lokalizuje bolesť |
| 4 | otvorí spontánne | zmätená | normálna flexia na bolesť |
| 3 | otvorí na výzvu | neadekvátne | abnormálna flexia na bolesť („dekortikačné“ postavenie) |
| 2 | otvorí na bolesť | nezrozumiteľná | extenzia na bolesť („decerebračné“ postavenie) |
| 1 | žiadna | žiadna | žiadna |

Holdgate A, Ching N, Angonese L (2006)
 Variability in agreement between physicians and nurses
 when measuring the Glasgow Coma Scale
 in the emergency department limits its clinical usefulness
 Emergency Medicine Australasia 18 (4), 379–384.

FOUR System

Full Outline Un-Responsiveness

(0 – 16 b.)



Eye response

- 4 Eyelids open or opened, tracking, or blinking to command
- 3 Eyelids open but not tracking
- 2 Eyelids closed but open to loud voice
- 1 Eyelids closed but open to pain
- 0 Eyelids remain closed with pain

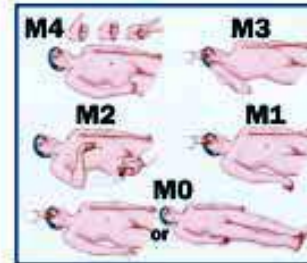


Eye response (E)

Grade the best possible response after at least 3 trials in an attempt to elicit the best level of alertness. A score of **E4** indicates at least 3 voluntary excursions. If eyes are closed, the examiner should open them and examine tracking of a finger or object. Tracking with the opening of 1 eyelid will suffice in cases of eyelid edema or facial trauma. If tracking is absent horizontally, examine vertical tracking. Alternatively, 2 blinks on command should be documented. This will recognize a locked-in syndrome (patient is fully aware). A score of **E3** indicates the absence of voluntary tracking with open eyes. A score of **E2** indicates eyelids opening to loud voice. A score of **E1** indicates eyelids open to pain stimulus. A score of **E0** indicates no eyelids opening to pain.

Motor response

- 4 Thumbs-up, fist, or peace sign to command
- 3 Localizing to pain
- 2 Flexion response to pain
- 1 Extensor posturing
- 0 No response to pain or generalized myoclonus status epilepticus



Motor response (M)

Grade the best possible response of the arms. A score of **M4** indicates that the patient demonstrated at least 1 of 3 hand positions (thumbs-up, fist, or peace sign) with either hand. A score of **M3** indicates that the patient touched the examiner's hand after a painful stimulus compressing the temporomandibular joint or supraorbital nerve (localization). A score of **M2** indicates any flexion movement of the upper limbs. A score of **M1** indicates extensor posturing. A score of **M0** indicates no motor response or myoclonus status epilepticus.

Brainstem reflexes

- 4 Pupil and corneal reflexes present
- 3 One pupil wide and fixed
- 2 Pupil or corneal reflexes absent
- 1 Pupil and corneal reflexes absent
- 0 Absent pupil, corneal, and cough reflex

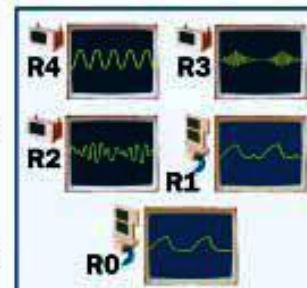


Brainstem reflexes (B)

Grade the best possible response. Examine pupillary and corneal reflexes. Preferably, corneal reflexes are tested by instilling 2-3 drops of sterile saline on the cornea from a distance of 4-6 inches (this minimizes corneal trauma from repeated examinations). Cotton swabs can also be used. The cough reflex to tracheal suctioning is tested only when both of these reflexes are absent. A score of **B4** indicates pupil and cornea reflexes are present. A score of **B3** indicates one pupil wide and fixed. A score of **B2** indicates either pupil or cornea reflexes are absent. **B1** indicates both pupil and cornea reflexes are absent, and a score of **B0** indicates pupil, cornea, and cough reflex (using tracheal suctioning) are absent.

Respiration

- 4 Not intubated, regular breathing pattern
- 3 Not intubated, Cheyne-Stokes breathing pattern
- 2 Not intubated, irregular breathing pattern
- 1 Breathes above ventilator rate
- 0 Breathes at ventilator rate or apnea



Respiration (R)

Determine spontaneous breathing pattern in a nonintubated patient and grade simply as regular **R4**, irregular **R2**, or Cheyne-Stokes **R3** breathing. In mechanically ventilated patients, assess the pressure waveform of spontaneous respiratory pattern or the patient triggering of the ventilator **R1**. The ventilator monitor displaying respiratory patterns is used to identify the patient-generated breaths on the ventilator. No adjustments are made to the ventilator while the patient is graded, but grading is done preferably with P_{aCO_2} within normal limits. A standard apnea (oxygen-diffusion) test may be needed when patient breathes at ventilator rate **R0**.

Validation of a new coma scale: The FOUR score
Eelco F. M. et al.,
Division of Critical Care Neurology, Mayo Clinic College of
Medicine, Rochester, MN

Full Outline of UnResponsiveness (FOUR skóre)

| Bodové ohodnotenie | Očná reakcia | Motorická reakcia | Kmeňové reflexy | Dýchanie |
|--------------------|--|-----------------------|--|-------------------------------|
| 4 | Sleduje pohyb prsta, zažmurká na výzvu | Stlačí prsty na výzvu | Fotoreakcia a korneálny reflex bilat. prítomné | Spontánne, pravidelné |
| 3 | Otvára spontánne | Lokalizuje bolesť | Fixovaná mydriáza na jednom oku | Spontánne, Cheyne - Stokesovo |
| 2 | Otvára na hlasnú výzvu | Flekčná odpoveď | Chýba FR alebo korneálne reflexy | Spontánne, Biotovo |
| 1 | Otvára na bolesť | Extenčná odpoveď | Chýba FR a korneálne reflexy | Pac. triggeruje ventilátor |
| 0 | Neotvára | Nič alebo myoklonus | Chýba FR, korneálne reflexy a kašľací reflex | Pac. netriggeruje ventilátor |

| Condition | Coma |
|-----------------------------|------------------------------|
| Self Awareness | No |
| Cyclical Eye Opening | No |
| Motor function | Reflex & Postural |
| Glasgow Coma Scale | E1-2 M1-4 V1-2 |
| EEG activity | Typically slow wave activity |
| Experience pain | No |
| Respiratory Function | Depressed or varied |
| Auditory Function | None |
| Visual Function | None |
| Communication | None |
| Emotion | None |

| Condition | Coma | Vegetative State |
|-----------------------------|------------------------------|--|
| Self Awareness | No | No |
| Cyclical Eye Opening | No | Yes |
| Motor function | Reflex & Postural | Postures or withdraws. Occasional non-purpose occasionally stereotypic movements |
| Glasgow Coma Scale | E1-2 M1-4 V1-2 | E4 M1-4 V1-2 |
| EEG activity | Typically slow wave activity | Typically slow wave activity |
| Experience pain | No | No |
| Respiratory Function | Depressed or varied | Normal |
| Auditory Function | None | Startle. Brief orientation |
| Visual Function | None | Startle. Brief orientation |
| Communication | None | None |
| Emotion | None | None or reflex smiling crying |

| Condition | Coma | Vegetative State | Minimal Conscious State |
|-----------------------------|------------------------------|--|--|
| Self Awareness | No | No | Partial |
| Cyclical Eye Opening | No | Yes | Yes |
| Motor function | Reflex & Postural | Postures or withdraws. Occasional non-purpose occasionally stereotypic movements | May localise, reach for objects but inconsistently |
| Glasgow Coma Scale | E1-2 M1-4 V1-2 | E4 M1-4 V1-2 | E4 M1-5 v1-4 |
| EEG activity | Typically slow wave activity | Typically slow wave activity | Insufficient data |
| Experience pain | No | No | Not known |
| Respiratory Function | Depressed or varied | Normal | Normal |
| Auditory Function | None | Startle. Brief orientation | Localise. Inconsistent commands-following |
| Visual Function | None | Startle. Brief orientation | Sustained visual fix. Sustained pursuit. |
| Communication | None | None | Contingent vocalisation Inconsistent verbalisation or gesture |
| Emotion | None | None or reflex smiling crying | Contingent smiling or crying |

| Condition | Coma | Vegetative State | Minimal Conscious State | Locked-in Syndrome |
|-----------------------------|------------------------------|--|---|--|
| Self Awareness | No | No | Partial | Full |
| Cyclical Eye Opening | No | Yes | Yes | Yes |
| Motor function | Reflex & Postural | Postures or withdraws. Occasional non-purpose occasionally stereotypic movements | May localise, reach for objects but inconsistently | Quadriplegic Vertical eye movement |
| Glasgow Coma Scale | E1-2 M1-4 V1-2 | E4 M1-4 V1-2 | E4 M1-5 v1-4 | E4 M1 V1 |
| EEG activity | Typically slow wave activity | Typically slow wave activity | Insufficient data | Typically normal |
| Experience pain | No | No | Not known | Yes |
| Respiratory Function | Depressed or varied | Normal | Normal | Normal |
| Auditory Function | None | Startle. Brief orientation | Localise. Inconsistent commands-following | Preserved |
| Visual Function | None | Startle. Brief orientation | Sustained visual fix. Sustained pursuit. | Preserved. |
| Communication | None | None | Contingent vocalisation Inconsistent verbalisation or gesture | Aphonic/ anarthric. Vertical eye movement and blinking |
| Emotion | None | None or reflex smiling crying | Contingent smiling or crying | Preserved |

Iná klasifikácia príčin kómy

Z hľadiska potenciálnych liečebných modalít

- Ložiskové („chirurgické“) poškodenie
lokalizované, fokálne
- Difúzne (metabolické, multifokálne) poškodenie
vrátane intoxikácií

Príčiny kómy v %

| Ložiskové | 40 | Difúzne | 60 |
|--------------------------------|------|------------------------|------|
| Intraparenchýmové krvácanie | 17.4 | Otrava (lieky, drogy) | 22.6 |
| Subarachnoidálne krvácanie | 7.8 | Alkohol | 7.8 |
| NCPM ischemická - infarkt | 4.3 | Hypoglykémia | 4.3 |
| Subdurálny hematóm | 3.5 | Sepsa | 4.3 |
| Epidurálny hematóm | 2.6 | Pneumónia | 4.3 |
| Kontúzia mozgu | 1.7 | Kardiálne zlyhanie | 2.6 |
| Tumor mozgu | 0.9 | Hypoxia | 1.7 |
| Trombóza intrakraniálnych žíl | 0.9 | Kapnonarkóza | 1.7 |
| Centrálne pontínna myelinolýza | 0.9 | Hepatálna kóma | 1.7 |
| | | Krčový stav | 1.7 |
| | | Urémia | 1.7 |
| | | Hyponatrémia | 0.9 |
| | | Diabetická ketoacidóza | 0.9 |
| | | Astma | 0.9 |
| | | Úpal | 0.9 |
| | | Neznáma etiológia | 1.7 |

*Jan-Dec 1999, 446 pacientov
Okinawa Chubu Hospital, Japan*

*Y Tokuda, N Nakazato, G H Stein
Postgrad Med J 2003;79:49–51*

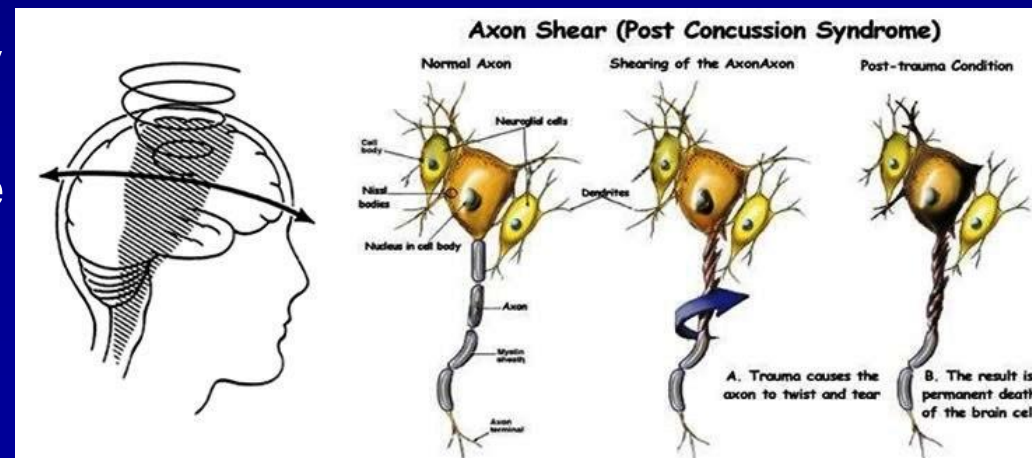
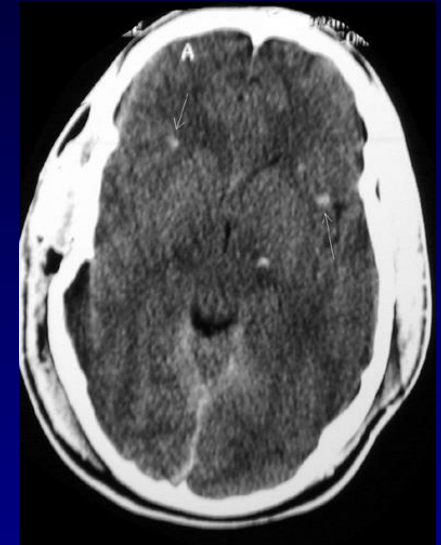
Ložiskové („chirurgické“) inzulty

- **Kompresívne (supra- a infratentoriálne)** hematómy, tumory, abscesy, herniácie mozgu
- **Deštruktívne** kontúzie

lokalizovaná akútna a subakútna anoxia (hypoxia) kôry a podkôrovej bielej hmoty

infarkt v thalame, v mezencefale a ponte

difúzne axonálne poškodenie (DAI)



Multifokálne, difúzne a metabolické inzulty

- Deficit substrátov a kofaktorov energetického metabolizmu neurónov
hypoxia, ischemia, hypoglykémia, hypovitaminózy
(thiamín, pyridoxín, foláty, ...)
- Orgánové insuficiencie (cerebrotoxická metabolitov)
napr. hepatálna kóma, uremická kóma, kapnonarkóza
- Endokrinné dysfunkcie
napr. hypofýza, štítna žľaza, prištítna telieska,
m. Addison, m. Cushing, diabetes mellitus

Multifokálne, difúzne a metabolické inzulty

Poruchy metabolizmu vody, minerálov a acidobázickej rovnováhy

aj iatrogénne (pri liečbe iných príčin kómy)

Primárne ochorenie neurónov a glie

Kreuzfeld-Jacobova choroba, gliomatóza

Zápalové postihnutie CNS (s infekciou alebo bez nej)

encefalitída, meningitída, „toxická“ encefalopatia, subarachnoidálne krvácanie

Poruchy tepelnej homeostázy

podchladenie, úpal

Multifokálne, difúzne a metabolické inzulty

Exogénne toxické látky (lieky, drogy, jedy)

- **sedatívne** pôsobiace (hypnotiká, trankvilizéry, etanol, opioidy)
- **psychotropné** (tricyklické antidepresíva, anticholinergiká, amfetamíny, fenotiazíny, inhibítory MAO, lítium, fencyklidín, LSD, meskalín)
- **kyseliny** a ich prekurzory (paraldehyd, metanol, etylénglykol, chlorid amónny)
- **iné** (antikonvulzíva, steroidy, kardioglykozidy, penicilín, salicyláty, kyanidy, ťažké kovy, organofosfáty)

Patofyziológia sekundárneho poškodenia mozgu I

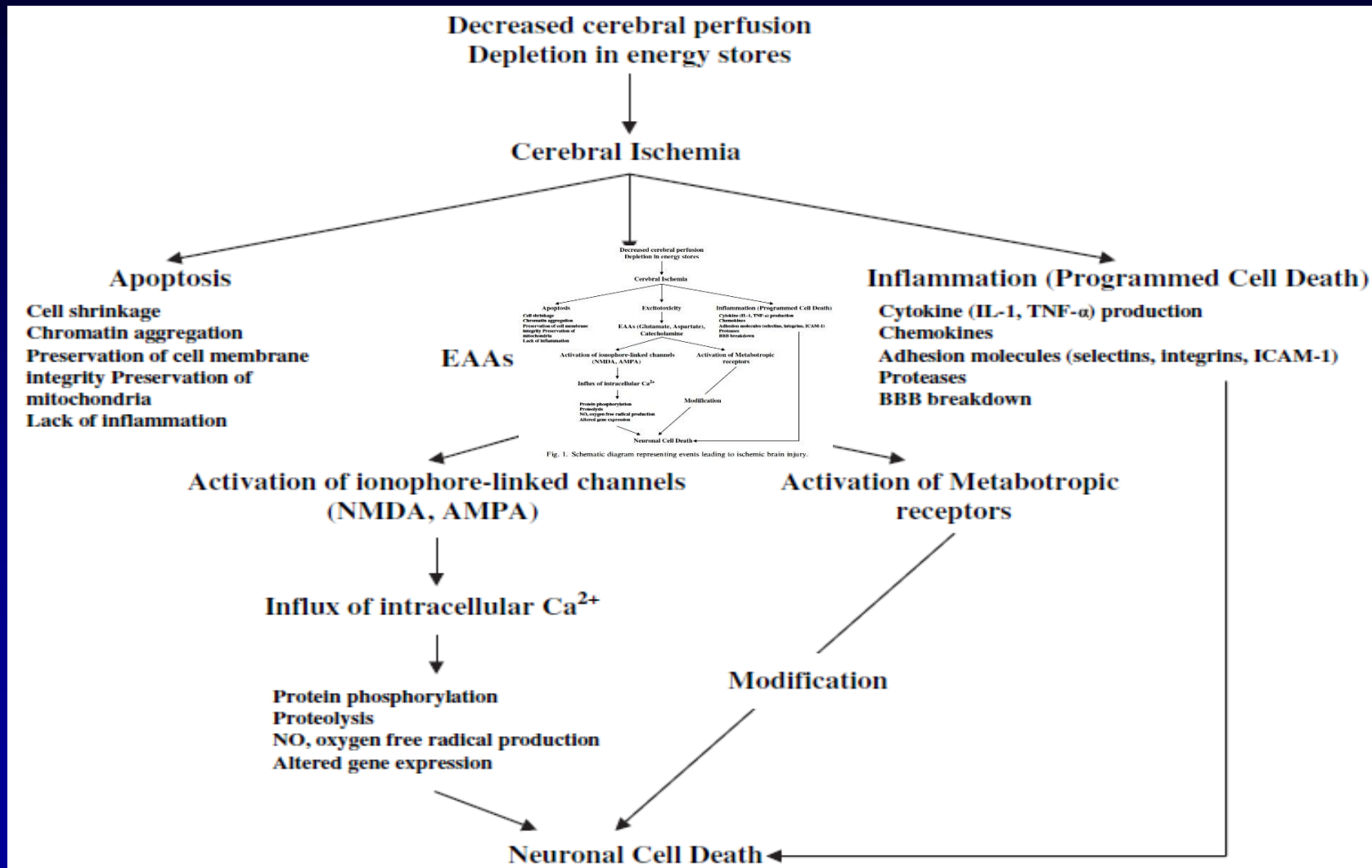
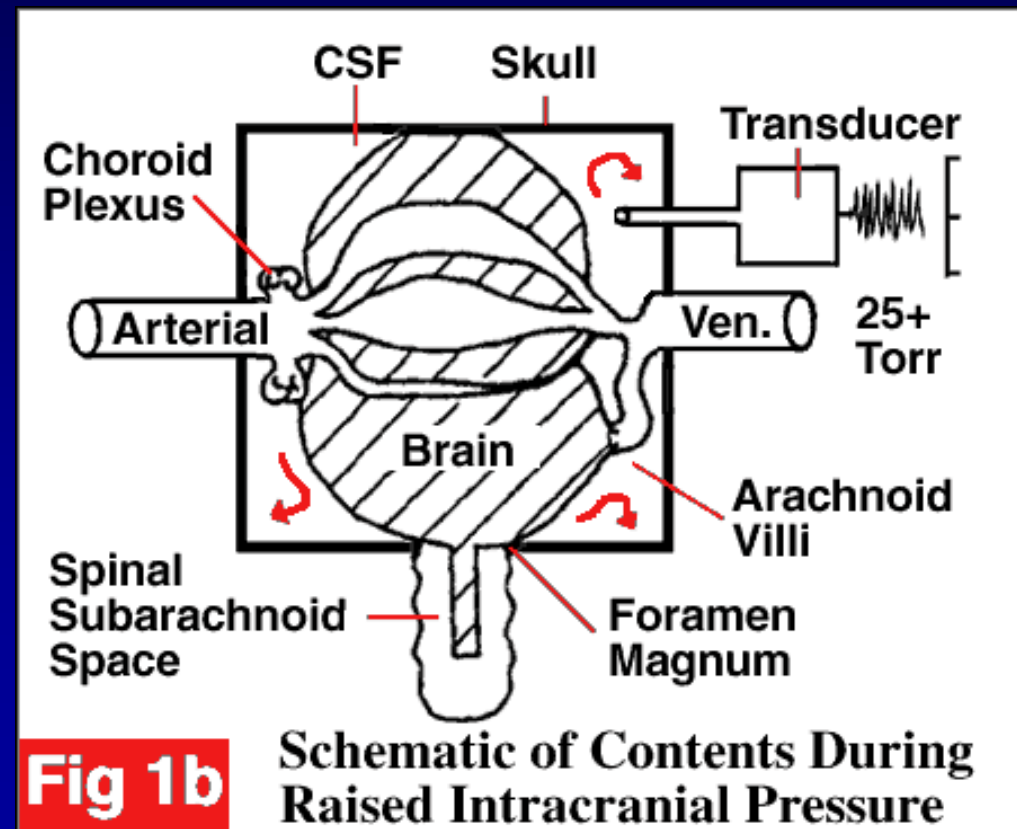
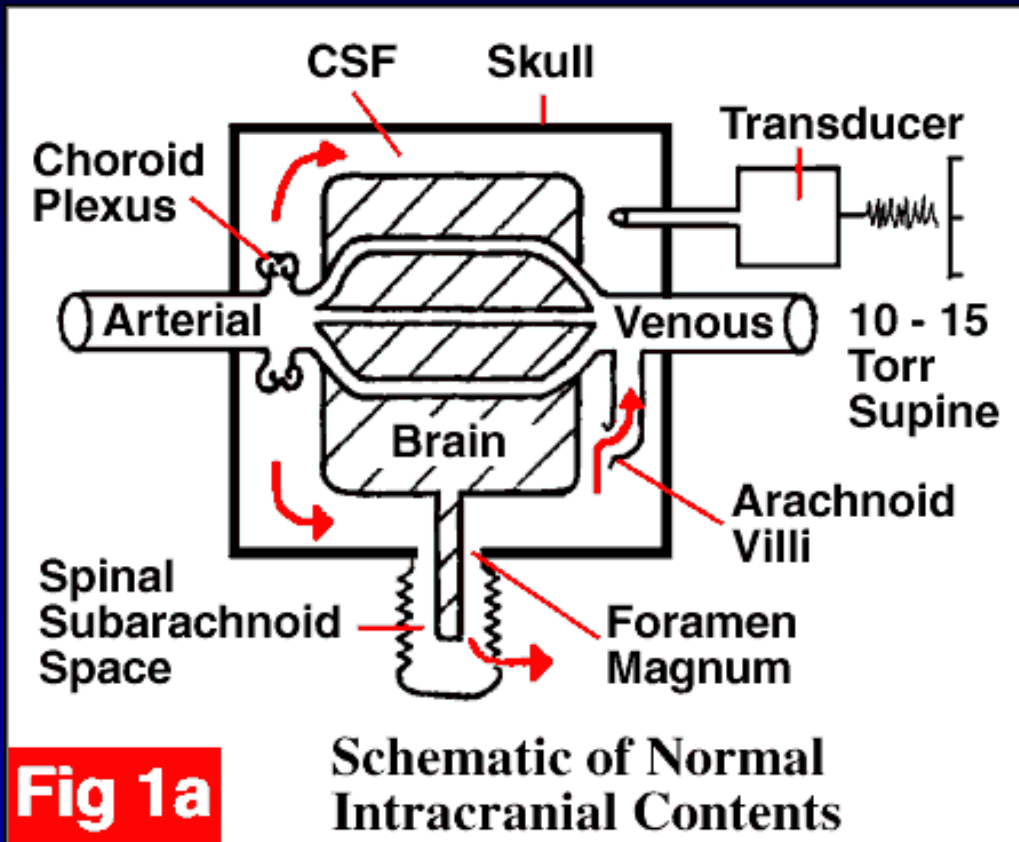
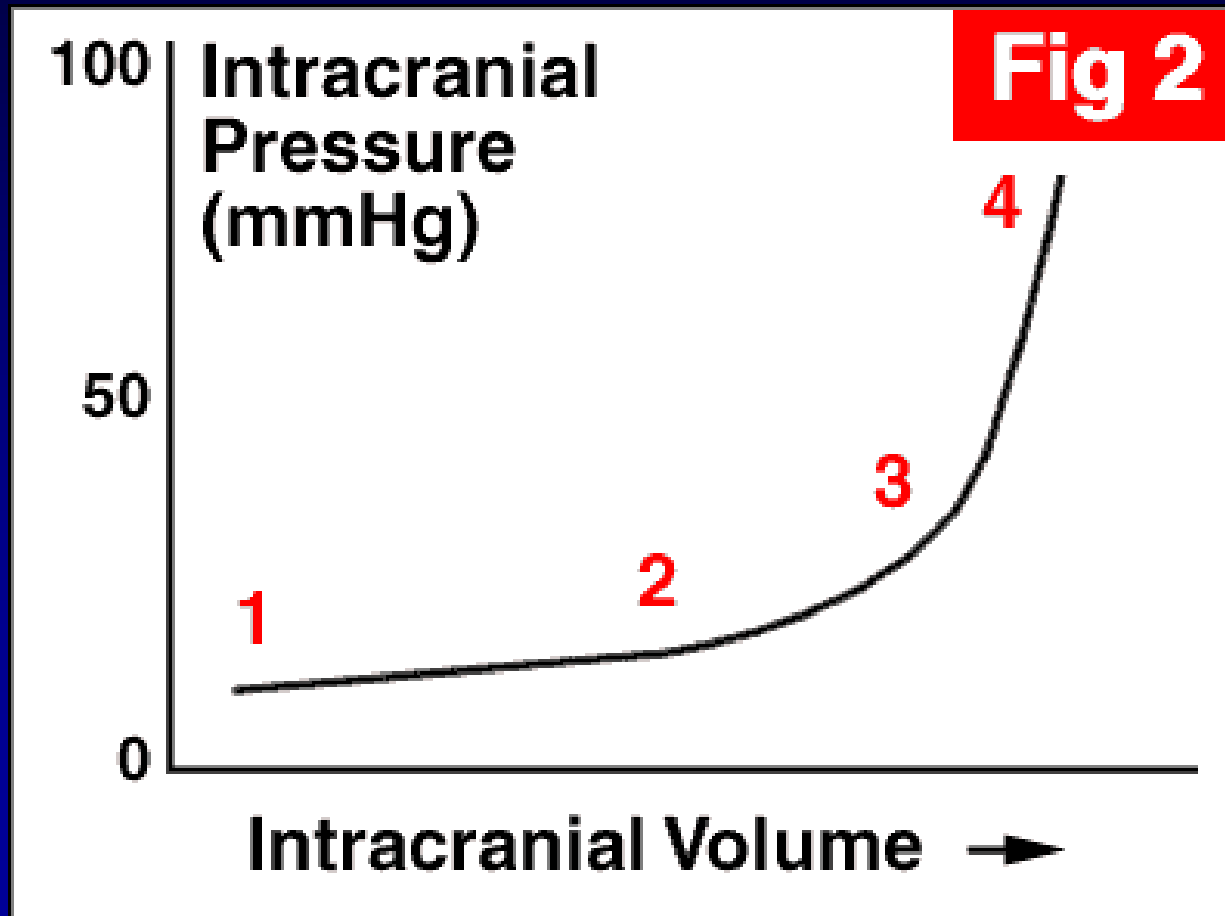


Fig. 1. Schematic diagram representing events leading to ischemic brain injury.

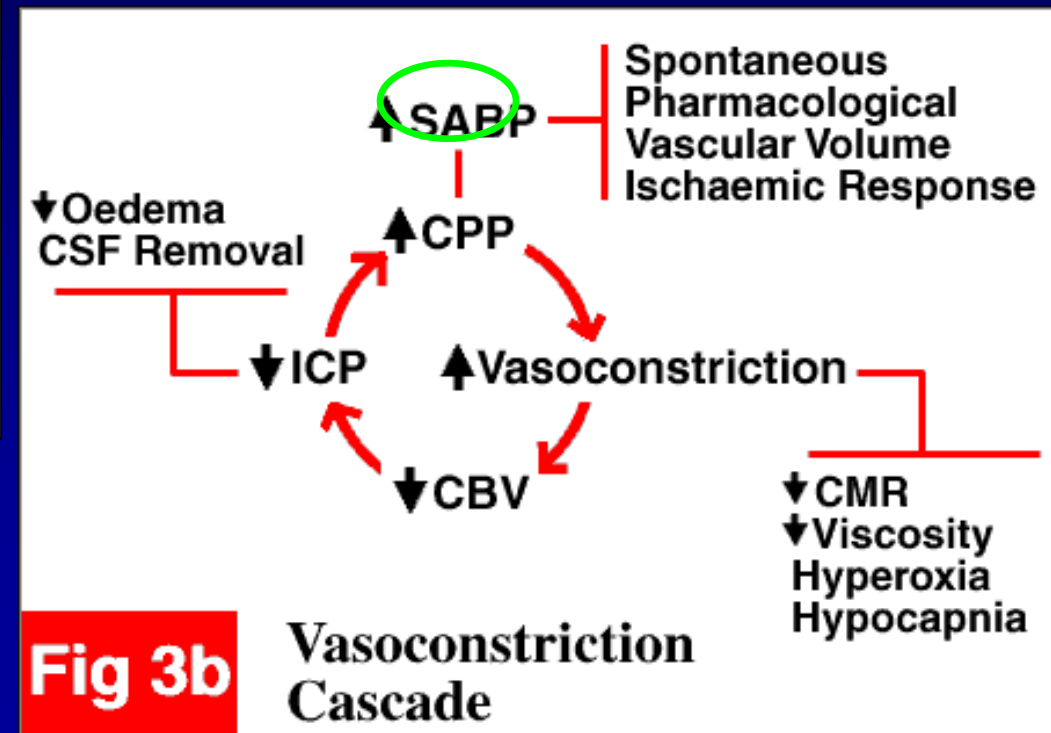
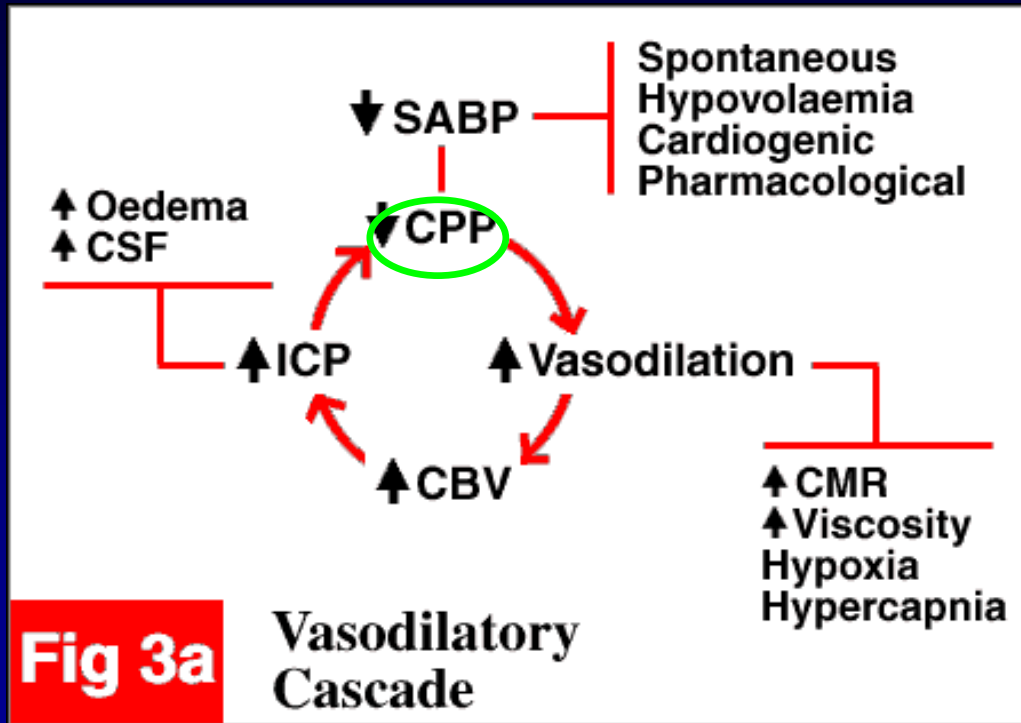
Patofyziológia sekundárneho poškodenia mozgu II



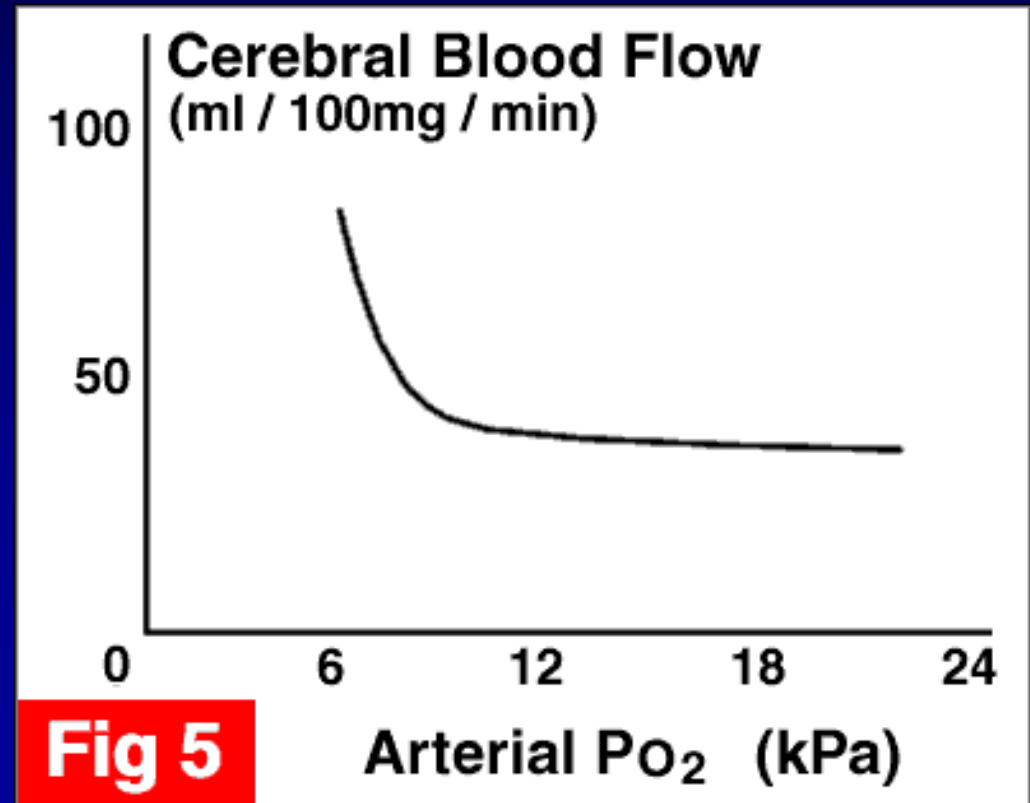
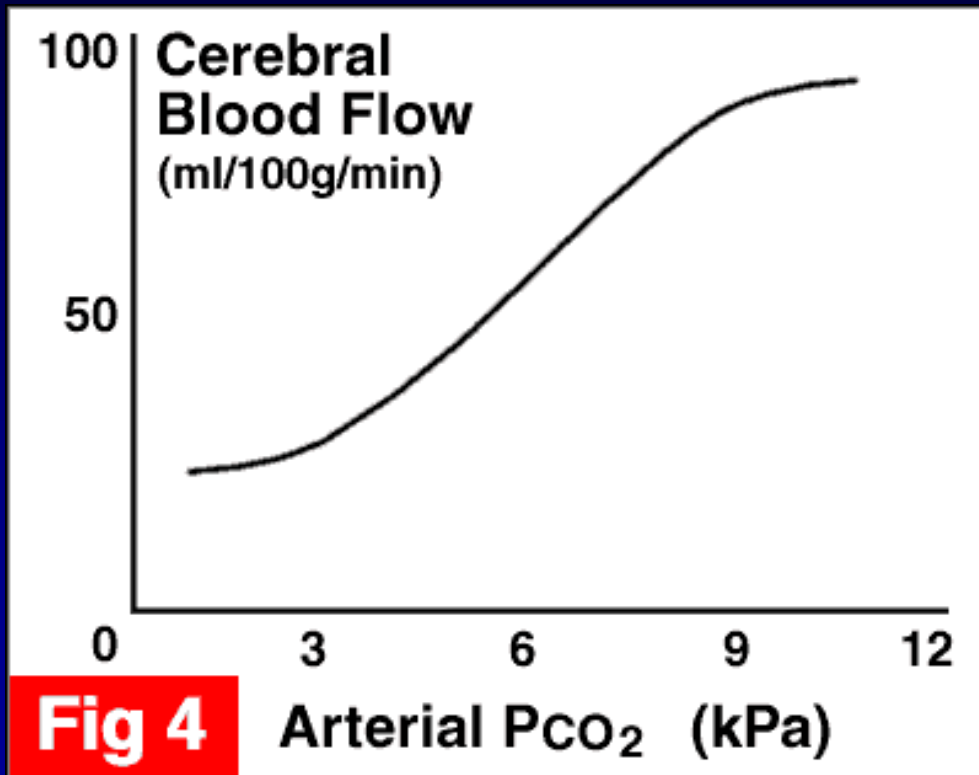
Patofyziológia sekundárneho poškodenia mozgu III



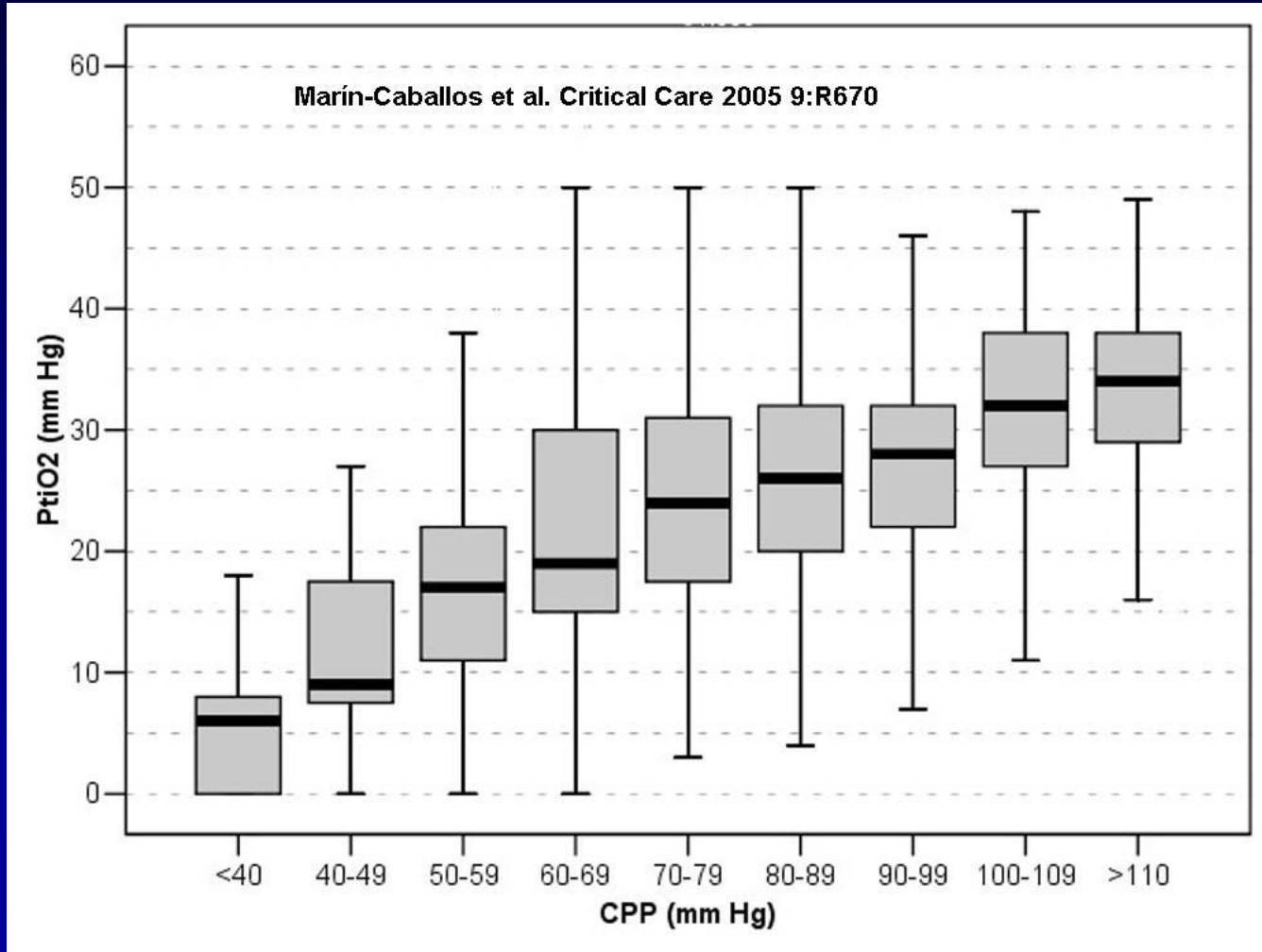
Vazodilatačná a vazokonstrikčná kaskáda



Výmena plynov v pľúcach a perfúzia mozgu



Perfúzia mozgu a výmena plynov v tkanivách



Vyšetrenie komatózneho pacienta

- Kóma je **urgentná** medicínska situácia

= vyšetrenia a liečba musia prebiehať súbežne

- zhodnotenie úrovne vedomia (neformálne i formálne)
- anamnéza (od svedkov, príbuzných, RZP)
- klinické (fyz.) vyšetrenie (všeobecné i neurologické)
- pomocné vyšetrenia (lab., zobrazovacie, funkčné testy)

- zabezpečenie adekvátnej perfúzie a oxygenácie mozgu

Vyšetrenie komatózneho pacienta

Anamnéza (od príbuzných, sprievodcu, záchranárov)

Rýchlosť nástupu kómy (náhle, postupne)

Subjektívne ťažkosti (bolesti hlavy, depresia, oslabnutia končatín, závrat)

Nedávne úrazy

Systemové ochorenia (diabetes, ochorenia srdca, obličiek)

Psychiatrická anamnéza

Prístup k liekom a drogám (alkohol, sedatíva, psychotropné látky)

Vyšetrenie komatózneho pacienta

Ale čo keď dovezú v bezvedomí

- *febrilného depresívneho epileptika,*
- *ktorý sa lieči na hypertenziu a diabetes,*
- *posledné dni ho bolela hlava a mal závraty*
- *a pri jednom závrate dnes spadol zo schodov ?*



Vyšetrenie komatózneho pacienta

Ale čo keď dovezú v bezvedomí

- ***febrilného depresívneho epileptika,***
- ***ktorý sa lieči na hypertenziu a diabetes,***
- ***posledné dni ho bolela hlava a mal závraty***
- ***a pri jednom závrate dnes spadol zo schodov ?***



Vyšetrenie komatózneho pacienta

Všeobecné fyz. vyšetrenie

TK, P, TT, frekvencia dýchania

Známky úrazu

Známky akútnych a chronických systémových ochorení

Známky aplikácie liekov a drog (vpichy, alkoholový foetor)

Rigidita šije (ak sa už vylúčil úraz krčnej chrbtice)

Vyšetrenie komatózneho pacienta II

Neurologické vyšetrenie

- Slovné reakcie
- Dychový typ a rytmus
- Tonus kostrového svalstva
- Motorická odozva
obranné pohyby a reflexy
šľachovo-okosticové reflexy
(prítomnosť patologických reflexov)
- Spontánne pohyby očí
- „Očné“ reflexy
viečkový reflex, korneálny reflex
veľkosť zreníc, fotoreakcia
okulocefalický reflex
ak sa vylúčil úraz C – chrbtice,
okulovestibulárny reflex
- Očné pozadie

Vyšetrenie komatózneho pacienta

Neurologické vyšetrenie

Ložiskové príznaky (lateralizácia)

= ložisko

normálny nález ho nevylučuje !

*(bilat. subdurálny hematóm
subarachnoidálne krvácanie
frontálne lokalizované ložisko, . . .)*

Vyšetrenie komatózneho pacienta

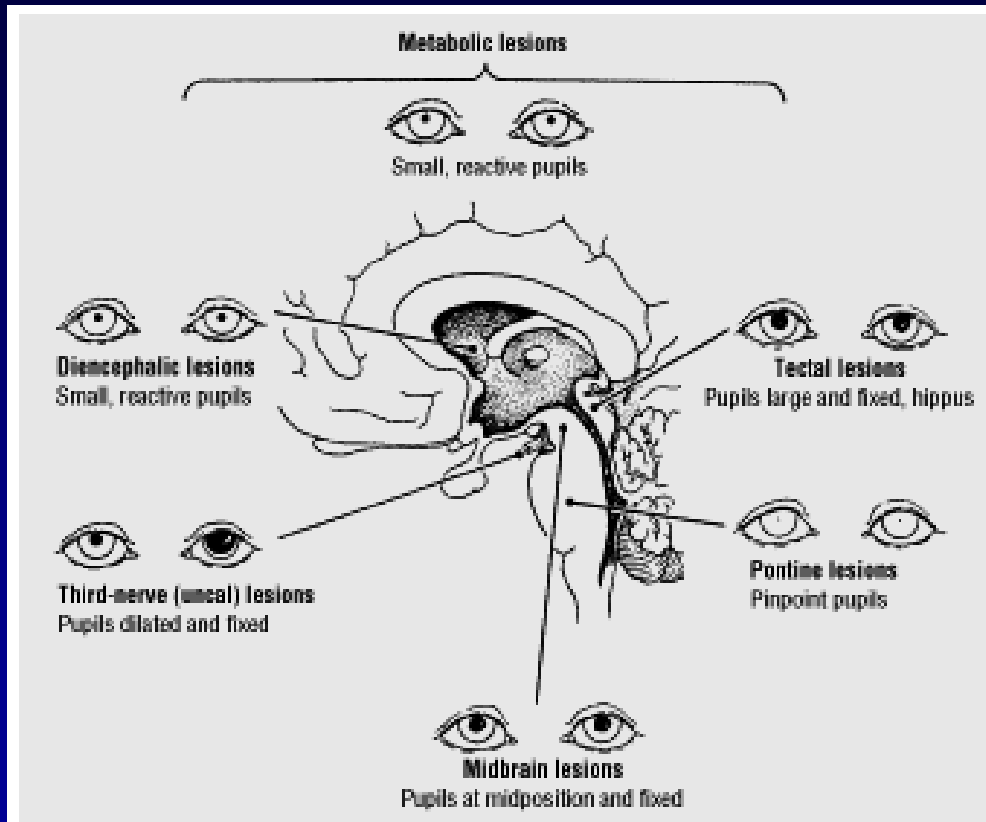


Figure 1. Pupils in comatose patients provide valuable clues to cause of coma.

Adapted, with permission, from Plum and Posner.¹

Veľkosť zreníc, fotoreakcia

Vyšetrenie komatózneho pacienta

| | Oculocephalic responses | | | | Caloric responses | | | |
|--|-------------------------|-----------|-----------|--------------|-------------------|-----------|-----------|------------|
| | Turn right | Turn left | Tilt back | Tilt forward | Cool water | | | Warm water |
| | | | | | Right side | Left side | Bilateral | Bilateral |
| A Brainstem intact (metabolic encephalopathy) | | | | | | | | |
| B Right lateral pontine lesion (gaze paralysis) | | | | | | | | |
| C MLF lesion (bilateral internuclear ophthalmoplegia) | | | | | | | | |
| D Right paramedian pontine lesion (1 1/2 syndrome) | | | | | | | | |
| E Midbrain lesion (bilateral) | | | | | | | | |

Okulocefalický reflex
(ak sa vylúčil úraz C-chrbtice)

Okulovestibulárny reflex

Vyšetrenie komatózneho pacienta

Niektoré diagnostické tipy a triky

| | |
|--|--|
| exantém (purpura) | = meningokoková sepsa |
| okuliarový hematóm | = fraktúra lebečnej bázy |
| bilaterálna mydriáza bez fotoreakcie | = otrava atropínom |
| jednostranná mydriáza, spomalená fotoreakcia | = lézia n. III, temporálny kónus |
| stredne široké zrenice bez fotoreakcie | = lézia v mezencefale |
| bilaterálna bodkovitá mióza s fotoreakciou | = otrava opioidmi |
| Hornerov syndróm | = lézia v oblongáte |
| oponujúca šija | = meningitída |
| gynekomastia, pavúčkové névy, flapping | = hepatálna encefalopatia |
| myoklonus | = hypoxická encefalopatia |
| hypoventilácia | = mozgový kmeň |
| Cheyne - Stokesovo dýchanie | = hlboké bilaterálne subkortikálne lézie |
| apneustické dýchanie | = lézia v ponte |

Vyšetrenie komatózneho pacienta

- Pomocné vyšetrenia

glykémia *(orientačne glukomerom)*

biochémia: *glykémia, aABB, Na, K, Cl, Ca, kreatinín, urea, hepatálne testy*

krvný obraz + dif., koagulačný skríning

toxikologický skríning *(prvý moč, krv)*

alkohol *(biochem. a súdnolek.)*

CT, MRI, sonografia (transkraniálny Doppler)

EEG, evokované potenciály

Likvor - lumbálna punkcia ? *(kontraindikovaná pri susp. ↑ICP)*

Liečba komatózneho pacienta

Všeobecné postupy (resuscitačné)

1. Rýchla korekcia hypoxie a hypotenzie (objem, mimetiká)
2. ETI (od GCS < 9) (ochrana pred aspiráciou a zatečením do dýchacích ciest, profylaxia sekundárnej hypoxie, uľahčenie vyšetrení)
3. Ventilačná podpora (na dosiahnutie normálneho pO_2 a pCO_2)
4. Korekcia hypoglykémie (20-25 g glukózy i.v., 50-60 ml G40%)
5. U alkoholickéj anamnézy thiamín 100mg i.v. (ev. aj pyridoxín a B_{12})
6. Korekcia hydratácie, minerálov, osmo- a onkotických parametrov
(pozor na prudkú úpravu hyponatrémie !)

Liečba komatózneho pacienta

Všeobecné postupy (resuscitačné) – iný algoritmus:

(pokiaľ nie je zjavný úraz hlavy)

glukóza i.v.

O₂, objem, mimetiká

thiamin i.v.

ETI, ...

naloxon i.v.

flumazenil i.v. – zvážiť riziká (poruchy rytmu, vracanie, aspirácia, ...)

Liečba komatózneho pacienta

Všeobecné postupy (profylaktického charakteru)

7. Profylaxia ventilátorovej pneumónie a VILI
8. Profylaxia stresových ulcerácií GITu, včasná enterálna výživa
9. Profylaxia tromboembolických komplikácií
10. Profylaxia dekubitov, svalovej atrofie a kontraktúr
11. Antikonvulzívna profylaxia
12. Nootropná liečba, pasívna a aktívna rehabilitácia, bazálna stimulácia

Liečba komatózneho pacienta

Korekcia a prevencia hypoxie mozgu (z hypoperfúzie)

1. Monitorovanie

- vnútrolebečného tlaku – ICP
- cerebrálneho perfúzneho tlaku – CPP
- spotreby kyslíka v mozgu – S_{ij}vO₂ (NIRS ?, ...)

2. Optimalizácia dodávky O₂ do mozgu

- mozgová perfúzia
- transportná kapacita krvi
- korekcia vazospazmov
- korekcia porúch cievnej permeability a mikrocirkulácie

3. Zníženie CMRO₂

- hypotermia
- barbituráty, propofol, etomidát
- opioidy, svalová relaxácia

Kauzálna liečba a prevencia sekundárneho poškodenia mozgu

Korekcia a prevencia hypoxie mozgu (z hypoperfúzie)

- Liečba edému mozgu (vazoaktívny, cytotoxický)
- Chirurgická korekcia (evakuácia ložiska, dekompresia, drenáž likvoru)
- Obnovenie lokálnej perfúzie
(nimodipín, trombolýza, transluminálna angioplastika)

Protektívny efekt hypotermie ?

Is keeping cool still hot? An update on hypothermia in brain injury.

Neuroscience

Current Opinion in Critical Care. 10(2):116-119, April 2004.

Clifton, Guy L

Abstract:

Purpose of review: The purpose of this review is to examine recent research results for hypothermia as a treatment for brain injury.

Recent findings: One potential application for hypothermia is as a means of control of elevated intracranial pressure in which hypothermia is induced when intracranial pressure becomes uncontrollable by conventional means. A second application is as a neuroprotectant in which hypothermia is induced very early and maintained for a specified period as a means of diminishing the biochemical cascade that produces secondary brain injury. The clinical data indicate that hypothermia reduces elevated intracranial pressure, but no conclusion can be drawn as to whether this improves outcome over existing techniques (eg, mannitol and barbiturates). There is little evidence that hypothermia acts as a neuroprotectant in trials, all of which used treatment windows of over 4 hours.

Summary: Hypothermia is a **useful adjunct to barbiturates and mannitol** to control elevated intracranial pressure. The results of trials that have tested systemic **hypothermia as a neuroprotectant have been negative or equivocal**, and cooling may have been induced outside the treatment window.

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Protektívny efekt hypotermie ?

Is keeping **Pro - con debate** still hot? An update on hypothermia in brain injury.

Hypothermia Does Not Improve Outcome From Traumatic Brain Injury

I. SEPPOLT
Department of Intensive Care Medicine, University of Sydney, Nepean Hospital, Sydney, NEW SOUTH WALES

ABSTRACT

Therapeutic hypothermia is a potentially dangerous treatment with a very narrow therapeutic index. It is of no benefit and may cause harm in other contexts, such as intermediate severity neonatal asphyxia. In traumatic brain injury there has been much provocative early evidence. While it is clear that hypothermia decreases intracranial pressure, a major phase III trial demonstrated no improvement in neurological outcomes with hypothermia, in an unselected group of patient with severe head injury. More focused phase III trials are underway but until the results are known this treatment should not be offered to patients outside the context of a clinical trial. (Critical Care and Resuscitation 2005; 7: 233-237)

and mannitol to control
e tested systemic
equivocal, and cooling

Protektívny efekt hypotermie ?

Pro - con debate

Hypothermia Does Not Improve Outcome From Traumatic Brain Injury

I. SEPPELT

Department of Intensive Care Medicine, University of Sydney, Nepean Hospital, Sydney, NEW SOUTH WALES

ABSTRACT

Therapeutic hypothermia is a potentially dangerous treatment with a very narrow therapeutic index. It is of proven benefit in certain conditions, including post ventricular fibrillation cardiac arrest and intermediate severity neonatal asphyxia. It is of no benefit and may cause harm in other contexts, such as elective neurovascular surgery. In traumatic brain injury there has been much provocative early evidence. While it is clear that hypothermia decreases intracranial pressure, a major phase III trial demonstrated no improvement in neurological outcomes with hypothermia, in an unselected group of patient with severe head injury. More focused phase III trials are underway but until the results are known this treatment should not be offered to patients outside the context of a clinical trial. (Critical Care and Resuscitation 2005; 7: 233-237)

Protektívny efekt hypotermie ?

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Hypothermia Therapy after Traumatic Brain Injury in Children

CONCLUSIONS

In children with severe traumatic brain injury, hypothermia therapy that is initiated within 8 hours after injury and continued for 24 hours does not improve the neurologic outcome and may increase mortality. (Current Controlled Trials number, ISRCTN77393684.)

N ENGL J MED 358;23 WWW.NEJM.ORG JUNE 5, 2008

Protektívny efekt hypotermie ?

Association between therapeutic hypothermia and long-term quality of life in survivors of cardiac arrest: A systematic review

Jignesh K. Patel^{a,*}, Puja B. Parikh^b

RESUSCITATION

OFFICIAL JOURNAL OF THE
EUROPEAN RESUSCITATION COUNCIL



Abstract

June 2016 Volume 103, Pages 54–59

Objectives

Therapeutic hypothermia (TH) has increasingly become a part of the current standard of care for treating patients with cardiac arrest (CA). However, little is known regarding the association between TH and long-term quality of life (QoL) in adult survivors of CA. We conducted a systematic review to investigate the association between TH implementation and long-term QoL outcomes in adult survivors of CA following hospital discharge.

Methods

We systematically searched MEDLINE and Cochrane databases to identify randomized and observational studies from January 2005 to January 2016 investigating the relationship between TH implementation immediately post-CA and long-term QoL in CA survivors post-hospital discharge.

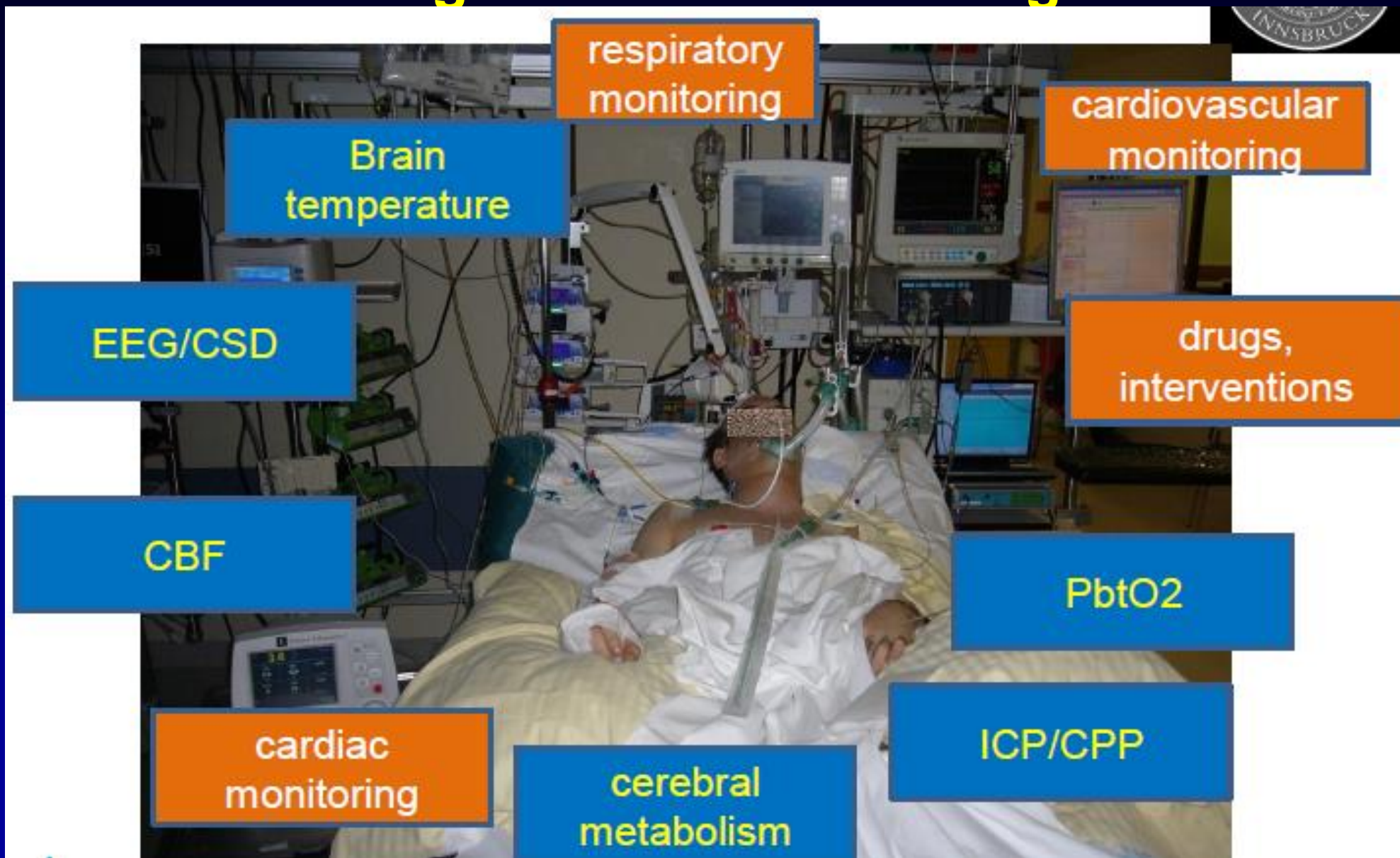
Results

We included 9 studies with a total of 801 patients. Six of these were prospective cohort studies, 2 were substudies of randomized controlled trials, and 1 was a retrospective cohort study. Six studies included patients only with out-of-hospital CA while 3 included patients with both in-hospital and out-of-hospital CA. There was marked between-study heterogeneity with respect to study population, TH implementation, and QoL assessment tool. TH was not associated with long-term QoL in this population.

Conclusions

In this systematic review, the included studies do not suggest any association between TH implementation in CA with long-term QoL in CA survivors. Further larger scale studies are needed to investigate the sustainability of TH effects long term in this patient population.

Monitorovanie faktorov ovplyvňujúcich energetickú bilanciu mozgu



Lund concept : **„*novinka*“ stará 25 rokov ?**

Asgeirsson B, Grände P O, Nordström C H:

A new therapy of post-trauma brain oedema based on
haemodynamic principles for brain volume regulation.

Intensive Care Med 20:260–267, 1994

Nordström C H:

Physiological and biochemical principles underlying volume-
targeted therapy--the "Lund concept".

Neurocrit Care 2(1):83-95, 2005

Department of Neurosurgery, **Lund University Hospital, Sweden**
carl-henrik.nordstrom@neurokir.lu.se

Lund concept

Podstata:

Fyziologická regulácia objemu vnútrolebečných kompartmentov

$$V_{\text{intrakran}} = V_{\text{krv}} + V_{\text{mozog}} + V_{\text{likvor}} + V_{\text{lézia}}$$

Cieľ:

Optimalizácia dodávky a spotreby kyslíka v energetickom metabolizme neurónu

Princíp:

1. Potlačenie stresovej odpovede organizmu, **zníženie energetického metabolizmu mozgu**
2. Zníženie **hydrostatického tlaku** v kapilárach mozgovej cirkulácie
3. Stabilizácia **koloidne-osmotického tlaku** a tekutinovej bilancie
4. Zníženie **objemu krvi** v mozgu

Lund concept

1. Potlačenie stresovej odpovede organizmu, zníženie energetického metabolizmu

napr.: thiopental 0,5 – 3 mg/kg/hod + fentanyl 2 – 5 mcg/kg/hod

2. Zníženie hydrostatického tlaku v kapilárach mozgovej cirkulácie

metoprolol (beta1 lytikum) 0,2 – 0,3 mg/kg/24 hod i.v.

klonidín (alfa2 mimetikum) 0,4 – 0,8 mcg/kg 4-6 x i.v.

Po evakuácii ložiska, dosiahnutí normovolémie, norm. CVT, albumínu a Hb

Cieľový perfúzny tlak (CPP)

60 – 70 torr

Lund concept

3. Stabilizácia koloidne-osmotického tlaku a tekutinovej bilancie

Transfúzia : cieľový Hb 125 – 140 g/l

Infúzia plazmy alebo albumínu: cieľový albumín nad 40 g/l

Bilancia tekutín vyrovnaná alebo mierne negatívna (furosemid)

Nutričný prísun: 15 – 20 kcal/kg/24 hod

4. Zníženie objemu krvi v mozgu

Arteriálna časť riečiska reaguje na **THP**

Venózna časť riečiska reaguje na **DH-ergotamín**

1.deň 0,8 mcg/kg/hod

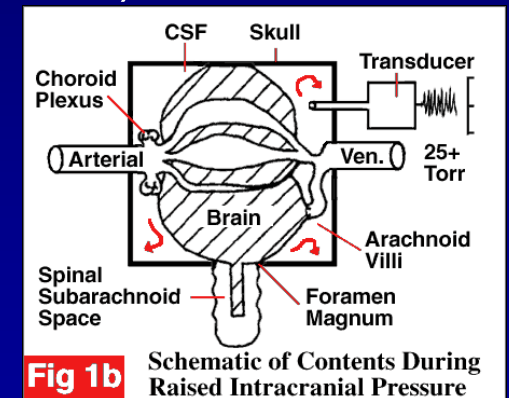
4.deň 0,2

2.deň 0,6

5.deň 0,1 mcg/kg/hod

3.deň 0,4

(aplikovať max. 5 dní, riziko periférnej ischémie) 59



Lund concept

Výsledky autorov

| | pred zavedením konceptu | po zavedení konceptu |
|-------------------------------------|-------------------------|----------------------|
| Glasgow Outcome Scale | 1982 – 1986 | 1989 - 1994 |
| zotavený alebo mierna dysfunkcia | 42 % | 79 % |
| ťažká disabilita alebo smrť | 58 % | 21 % |

Potenciálne perspektívy vo farmakologickej prevencii a liečbe

- **MgCl** – v experimente redukuje rozsah poškodenia mozgovej kôry
- **Melatonín** – scavenger voľných radikálov, bráni dezintegrácii bunkových membrán
- **Cannabinoidy** – protekcia pred excitotoxickými transmitermi
- **Karnitín** – stabilizuje mitochondrie (podávaný 4 týždne pred experimentálnym inzultom ???)
- **Cyklosporín** – dtto, zatiaľ sa nedostal za fázu II testov

Potenciálne perspektívy vo farmakologickej prevencii a liečbe

Editorial

**Pharmacotherapy in neurointensive care
an update on new options and developments**

Oddo, Mauro

Current Opinion in Critical Care: [April 2019 - Volume 25 - Issue 2 - p 95–96](#)

Critical care outcomes in resource-limited settings

Vukoja, Marija; Riviello, Elisabeth D. Schultz, Marcus J

Current Opinion in Critical Care: [October 2018 - Volume 24 - Issue 5 - p 421–427](#)

Prognóza komatózneho pacienta

- Pri prácach s funkčnou (metabolickou) MRI – fMRI sa ukázalo, že miera zachovania (aspoň parciálneho) úrovne medzineurónovej komunikácie koreluje s vyššou pravdepodobnosťou zotavenia z kómy (2015).

fMRI ako prognostický nástroj ?

- Sledovanie účinkov systematickej sluchovej stimulácie komatóznych pacientov hlasmi rodinných príslušníkov a blízkych osôb (FAST - familiar auditory sensory training) naznačujú zreteľné zlepšovanie mozgových funkcií (2015).

príspevok k rehabilitácii mozgových funkcií ?

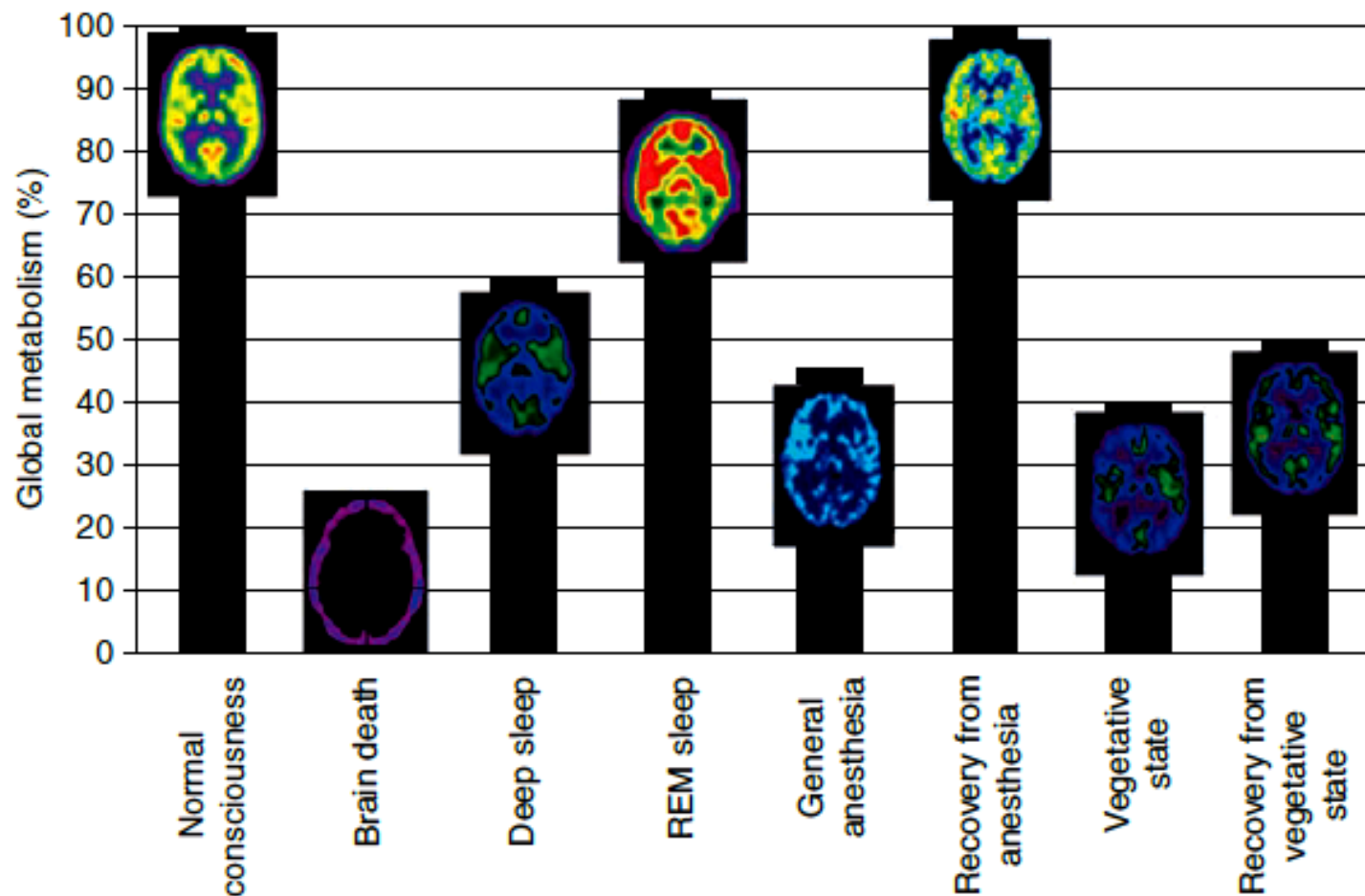


Figure 3 Cerebral metabolism in conscious wakefulness, in brain death (hollow skull sign confirming the absence of neuronal function in the whole brain in irreversible coma), physiological (slow wave sleep) and pharmacological (general anesthesia) modulation of arousal reflecting massive global decreases in cortical metabolism (in REM sleep, metabolic activity is paradoxically prominent); and in wakefulness without awareness (i.e., the vegetative state). Note that recovery from the vegetative state may occur without substantial increase in overall cortical metabolism, emphasizing that some areas in the brain are more important than others for the emergence of awareness. Images of halothane-induced loss of consciousness from Alkire M, Pomfrett CJ, Haier RJ, et al. (1999) Functional brain imaging during anesthesia in humans: Effects of halothane on global and regional cerebral glucose metabolism. *Anesthesiology* 90: 701–709.

Prognóza komatózneho pacienta

Assessing brain injury after cardiac arrest, towards a quantitative approach

Cronberg, Tobias

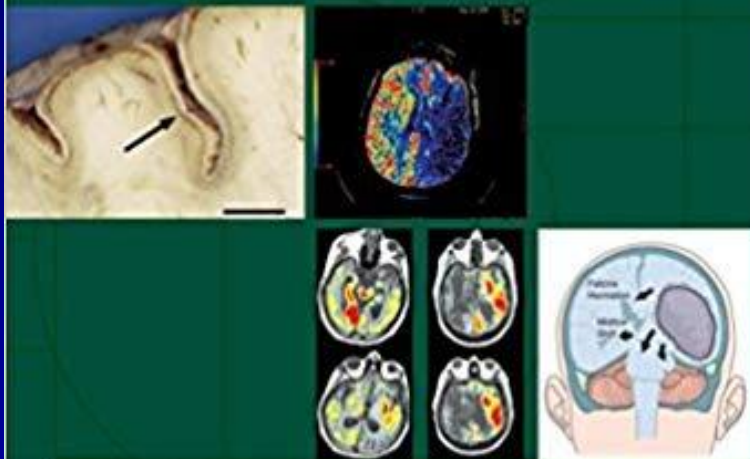
Current Opinion in Critical Care: [June 2019 - Volume 25 - Issue 3 - p 211–217](#)

- Currently, the only quantitative method for assessment of postarrest brain injury in regular clinical use is the measurement of **neuron-specific enolase in serum**.
- Since 2015 several promising methods to standardize assessment have been introduced including **pupillometry, standardized electroencephalography interpretation and the quantification of somatosensory evoked potentials, computed tomography and MRI-signals**. In addition, **novel and superior blood biomarkers** are on the verge of clinical introduction.

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FOURTH EDITION

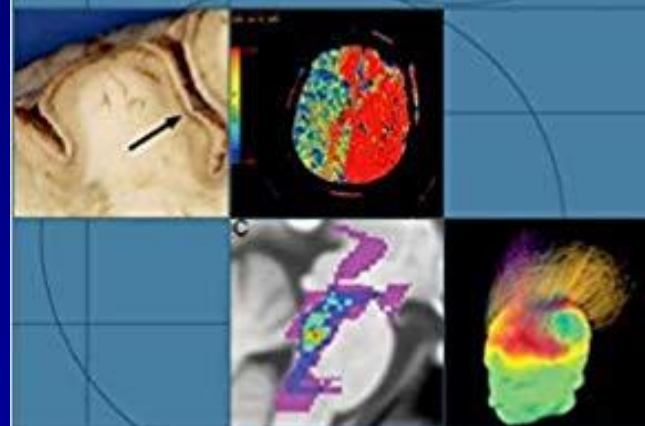


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CONTEMPORARY NEUROLOGY SERIES

PLUM AND POSNER'S DIAGNOSIS AND TREATMENT OF STUPOR AND COMA

FIFTH EDITION



JEROME B. POSNER
CLIFFORD B. SAPER
NICHOLAS D. SCHIFF
JAN CLAASSEN

OXFORD

Intoxikácie ako príčina kómy

- priamo – neurotoxicita
- nepriamo – prostredníctvom zlyhania obehu, dýchania, metabolickým rozvratom atď.

liekové

(nežiadúce účinky / predávkovanie)

anestetiká, benzodiazepíny, opioidy

antidepresíva, salicyláty, ...

alkohol, drogy

jedy

rastlinné

živočíšne

„priemyselné“

náhodné

úmyselné

Jaroslav Prokeš et al.

ZÁKLADY TOXIKOLOGIE

Obecná toxikologie a ekotoxikologie

základy

Galén
Univerzita Karlova v Praze,
Nakladatelství Karolinum



SPRINGER
REFERENCE

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Editors

Critical Care Toxicology

Diagnosis and Management of the
Critically Poisoned Patient

Second Edition



Systematický postup u intoxikácií

- Resuscitácia
- Identifikácia látky
- Eliminácia látky
- Špecifické postupy (napr. antidota živočíšnych jedov)
- Podporná a symptomatická liečba

Resuscitácia

- základná + rozšírená KPCR
- hypotermia *(vekové extrémny, alkohol)*
- hypertermia *(salicyláty, anticholinergiká, malígna hypertermia, sepsa)*
- rhabdomyolýza *(pri hypotermii, kóme, traume, prolongovaných kŕčoch)*

Identifikácia látky

- čo, koľko, kedy, ako
- okolnosti (*úmysel, svedkovia, prázdne obaly, trauma*)
- anamnéza (*predošlé pokusy, pridružené ochorenia, alergia*)
- príznaky (*prejavy, podaná prvá pomoc, kompletne fyzické vyšetrenie*)
- pomocné vyšetrenia
(*toxikológia, biochémia, ABR + krvné plyny, EKG, rtg hrudníka*)

Eliminácia látky

- externá dekontaminácia (*organofosfáty, hydrokarbóny*)
- ~~indukované vracanie~~ (*obsolentné, u kómy NIKDY! – aspirácia, sy. Mallory-Weiss*)
- laváž žalúdka (*do 1 hod., teofylín a salicyláty i neskôr*
závery metaanalýz Cochrane Library – neovplyvňuje dlhodobú prognózu)
- aktívne uhlie (*veľmi účinné, ALE:*
NIE u elementárnych kovov – Fe, Li,
NIE u pesticídov – DDT, malathion,
NIE u alkoholov, kyanidov)

Cochrane Library – neovplyvňuje dlhodobú prognózu



Eliminácia látky

- chirurgickou cestou (*„bodypackers“ – pašeráci drog*)
- laxanciá (*(riziko dehydratácie a iónového rozvratu, parafínový olej u lipofilných substancií)*)
- forsírovaná diuréza (*(riziko prevodnenia, edém pľúc, mozgu alkalizácia moču – barbituráty, salicyláty)*)
- hemodialýza (*(malá mol. hmot., malá väzba na proteíny, malá lipofília, malý spontánny klírens)*)
- hemoperfúzia (*(s aktívnym uhlím, so živicom lipofilné látky – teofylín, barbituráty)*)

Špecifické postupy - antidota

Látka / stav

acetaminofen (paracetamol)

anticholinergiká

anticholinesterázy

benzodiazepíny

etylénglykol

hypoglykemizujúce látky

kyanidy

metanol

methemoglobinémia

opioidy

organofosfáty

oxid uhoľnatý

Antidotum

N-acetylcysteín

fyzostigmín

atropín

flumazenil

etanol / fomepizol, tiamín

a pyridoxín

glukóza, glukagón, oktreotid

amylnitrit, Na-thiosulfát,

hydroxocobalamín

etanol / fomepizol, ac. folicum

metylénová modrá

naloxon

atropín, pralidoxamín

kyslík, HBO

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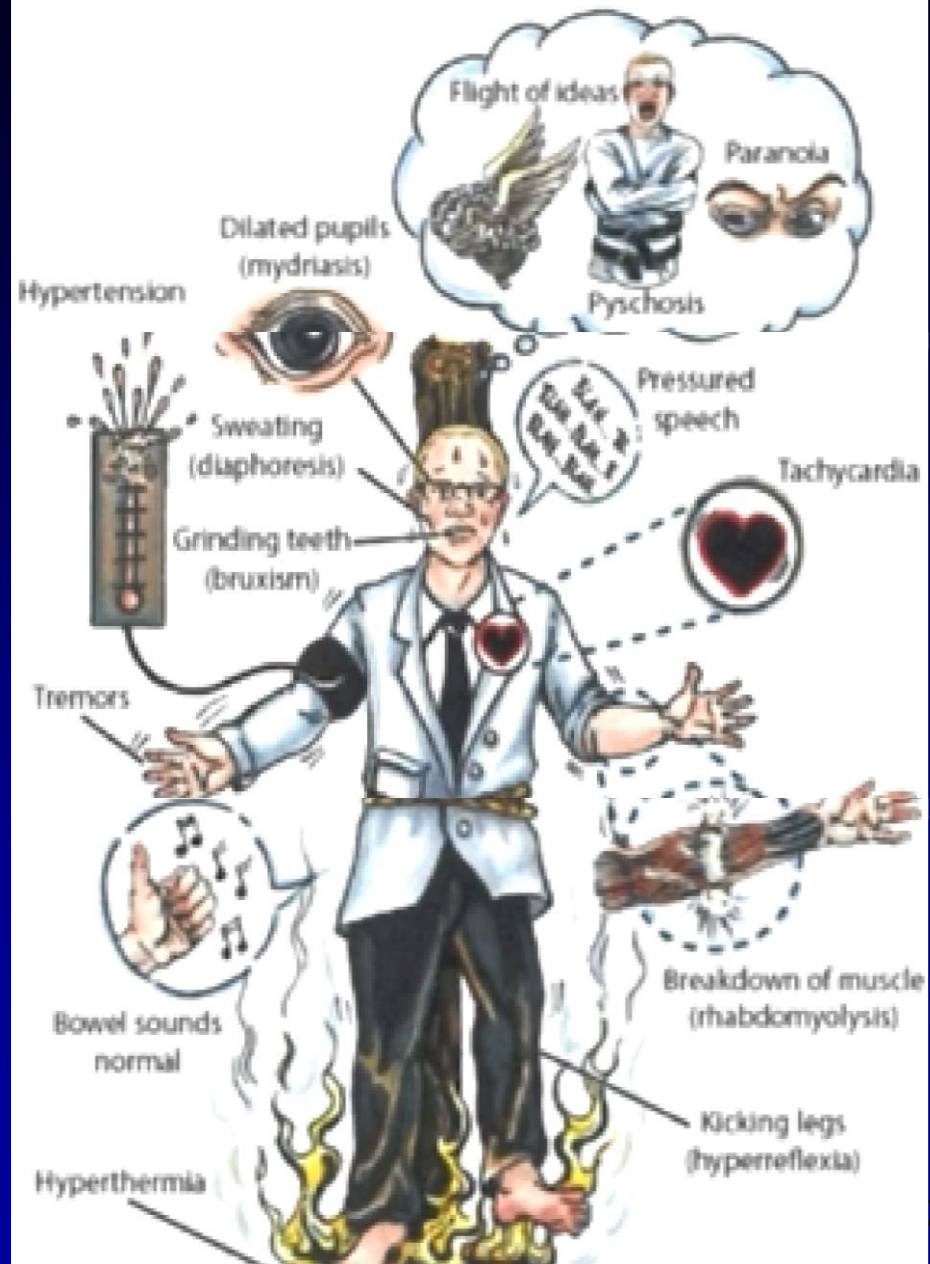
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Kokaín

(príznaky)

Sympathomimetic Toxidrome





Ďakujem za pozornosť

