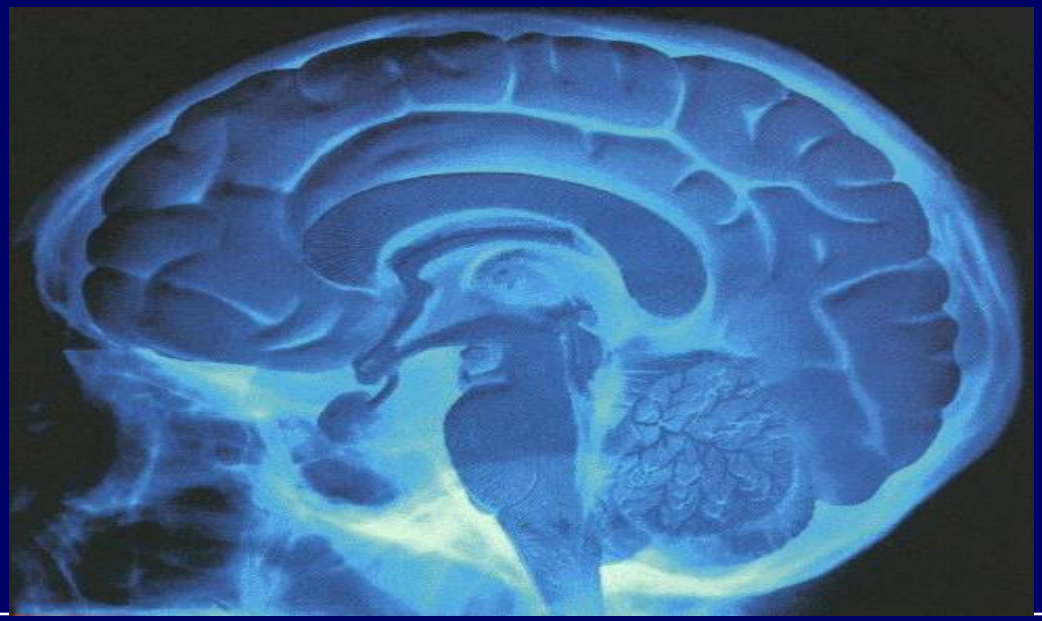
HEAD INJURY in CHILDREN

ד"ר שי מנשקו
היחידה לנוירולוגית ילדים
בה"ח ספרא לילדים



Head Injury

Management of:

- skull fractures
- epidural hematomas
- subdural hematomas

Intensive care management of severely head injured children.

- The most accurate way to define a mild closed head injury is from a ***functional*** **rather than a** ***neurological perspective***

- Children who sustain mild head injury may appear unimpaired until they attempt to return to school…at that point, memory difficulties, impaired concentration abilities and a general decrease in their ability to learn efficiently are detected

- Other concerns include increased irritability, hyper-activity or stubbornness

MINIMAL HEAD INJURY

- GCS 15 and…
  - No or only mild headache and nausea
  - No L.O.C.
  - No antegrade amnesia
  - No seizure
  - No vomiting
  - 2< AGE< 65
  - Likelihood of CT abnormality essentially 0%

MILD HEAD INJURY

- GCS 14 or 15 and….

- Any L.O.C., seizure or vomiting
- Coagulopathy
- Clinical skull fracture, facial injury or large scalp haematoma
- Focal neurological abnormality
- Abnormal pupillary reactions

- Likelihood of abnormal CT @ 10%
- Neurosurgical intervention <1%

Signs and Symptoms of Mild Brain Injury

COGNITIVE SYMPTOMS	PHYSICAL SYMPTOMS	BEHAVIORAL CHANGES

pattern

Late Signs and Symptoms of Mild Head Injury

- Light-headedness
- Persistent headache
- Memory problems
- Poor concentration
- Easy fatigability
- Irritability
- Visual disturbances
- Noise intolerance
- Sleep disturbances
- Deterioration of school performance
- Changes in behavior

APOE Σ - 4 (apolipoprotein-E) **allele** has been recognized as having a correlation with increased incidence of Alzheimer disease as does head injury (plaque formation)

- short term recovery from mild to moderate injury over a 6 week period - consistently lower scores for patients positive for APOE Σ - 4

- conflicting studies – if control for variables only a slower recovery rate (J. Neurotrauma 2007)

WHY?

- Amyloid precusor protein and APOE upregulate immediately following brain injury (produces secondary inflammation in the injured area)

- Increased levels of amyloid pathology caused by APOE Σ- 4 may induce immune-mediated delays in recovery

- Σ - 4 allele inhibits neurite outgrowth in injured areas
- Σ - 4 subtype offers the least protection against oxidative stress
- APOE may degrade neuronal or synaptic function in complex circuits for working memory

Is there a marker for brain injury?

Required characteristics

- High specificity for brain injury
- High sensitivity for brain injury
- Rapid, immediate analysis available/Point-of-Care test

Candidate markers

- Neuron-specific enolase (**NSE**)
- **S100B**
- Myelin-basic protein (**MBP**)

Neuron-specific enolase (NSE)

- An enzyme found in the cytoplasm of neurons
- Measurable in the CSF and serum
- ELISA, takes 1 hour to complete
- Serum half-life: 24 hours
- May be more specific for outcome if measured with GFAP
- Difficulties in correlating with GOS (J. Neurotrauma Jan 2007)
- *Limitation*:

  -- Sensitive to hemolysis

S100B

- Calcium binding protein with alpha and beta subunits
- Found in the cytosol of astroglial cells
- Measurable in the CSF and serum
- ELISA, takes 1 hour to complete
- Not affected by hemolysis
- If normal – no brain injury
- Serial measurements-if sustained increase may predict outcome and extent of injury
- *Limitations*:
  - Contribution of extracerebral sources?

Myelin-basic protein (MBP)

- Abundant protein in CNS myelin/marker of axonal injury
- Measurable in the CSF and serum
- ELISA, takes 1 hr to complete
- Not affected by hemolysis
- Serum half-life: 12 hours
- May be involved in delayed degeneration of initially spared neurons by T-cell dependent neuroprotective response

Is there a marker for brain injury?

Current Status

- No markers predictive of PCS
- No clear correlation or predictive value for S100B or NSE – not valid markers for outcome

COGNITIVE AND PSYCHOLOGICAL SEQUELAE

- Mild closed head can produce various deficiencies, including a ***decrease*** *in intellectual functioning, language skills, attention and memory, executive function, academic achievement and even different forms of behavioral adjustment*

- *Long term difficulties following head injuries are reported in* ***language skills***

Outcome and Recovery

- The range of neuropsychological stress, suggests that even though they may be considered unaffected by their trauma, a number may have *one or more difficulties*

- Ewing-Cobbs et al. showed that the neuropsychological problems seen in children involved in a traumatic brain injury indicates the need for assessment of the children, *no matter what their level of severity seems to be* during the immediate period after their injury
  (Ewing-Cobbs et al. Psychology Press 2000)

- **Attention** problems are another complaint in children after mild to severe head injury

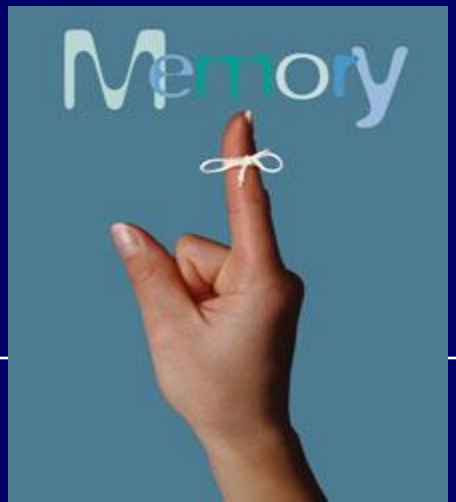
- Issue of emerging injury features in adolescence

- When compared to a matched group of children, these difficulties are more impressive among *young children* with head trauma than the somewhat older group of children with the same type of injury
  (Jeffrey et al. Journal of the International Neuropsychological Society 1999)

- **Memory** is another area in which changes are noted in children with closed head injury

- The severity of the memory problems correlates well with the severity of the injury

- Difficulties have been reported on a wide variety of verbal tests, including tests for recognition of memory for words and words listing learning (Hanten at al. Neuropsychology. 2004)



BIOMECHANICS OF BRAIN PATHOLOGY

- The pathophysiology of head trauma begins at the time of impact, then continues for a prolonged period (weeks to months)
- Observable injuries can be classified as:

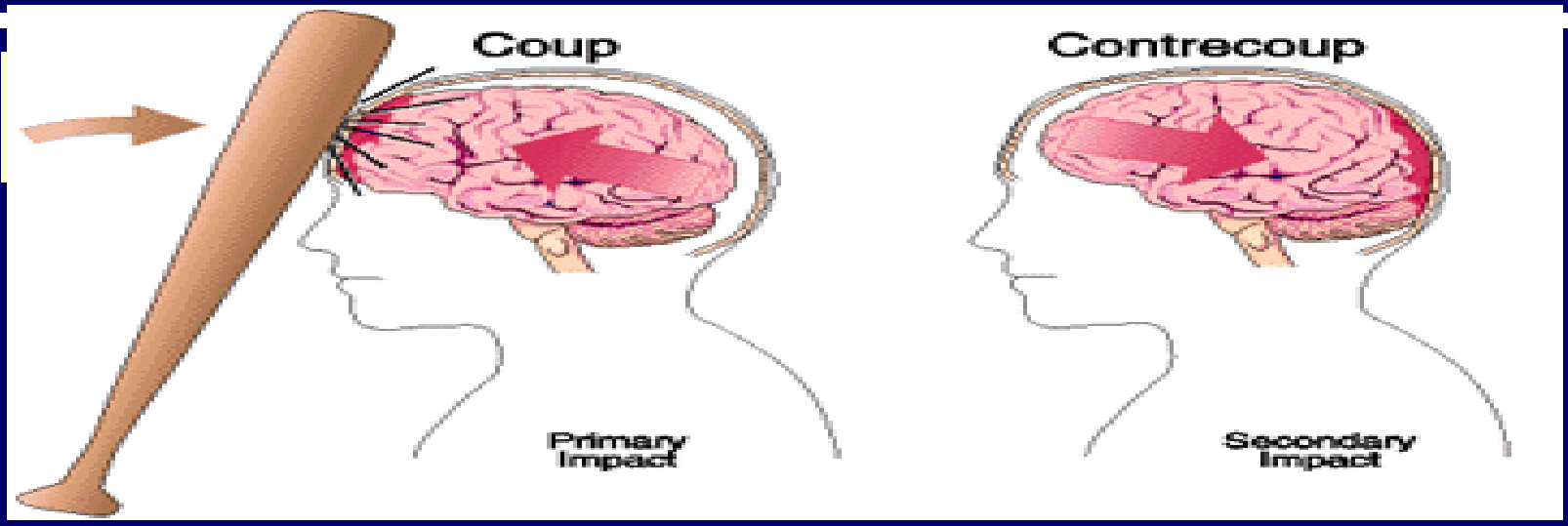
* *Primary* injuries result directly from the trauma itself and may include skull fractures, contusions, lacerations and mechanical injuries to nerve fibers and blood vessels

* *Secondary or Second* injuries arise indirectly from the trauma including edema, hypoxia, fever/infection and increased intracranial pressure, hematomas with shift (McLean et al. Head Injury 1997)

Pathophysiology

- It has been demonstrated that brain function can be severely impaired, with no observable pathophysiological evidence on primary diagnostic testing or follow-up MRI (although present on co-incidental autopsy)

- The primary injuries that arise after head trauma reflect the different biomechanical forces, and it can involve either **impact/impression** or **acceleration-deceleration and rotation**

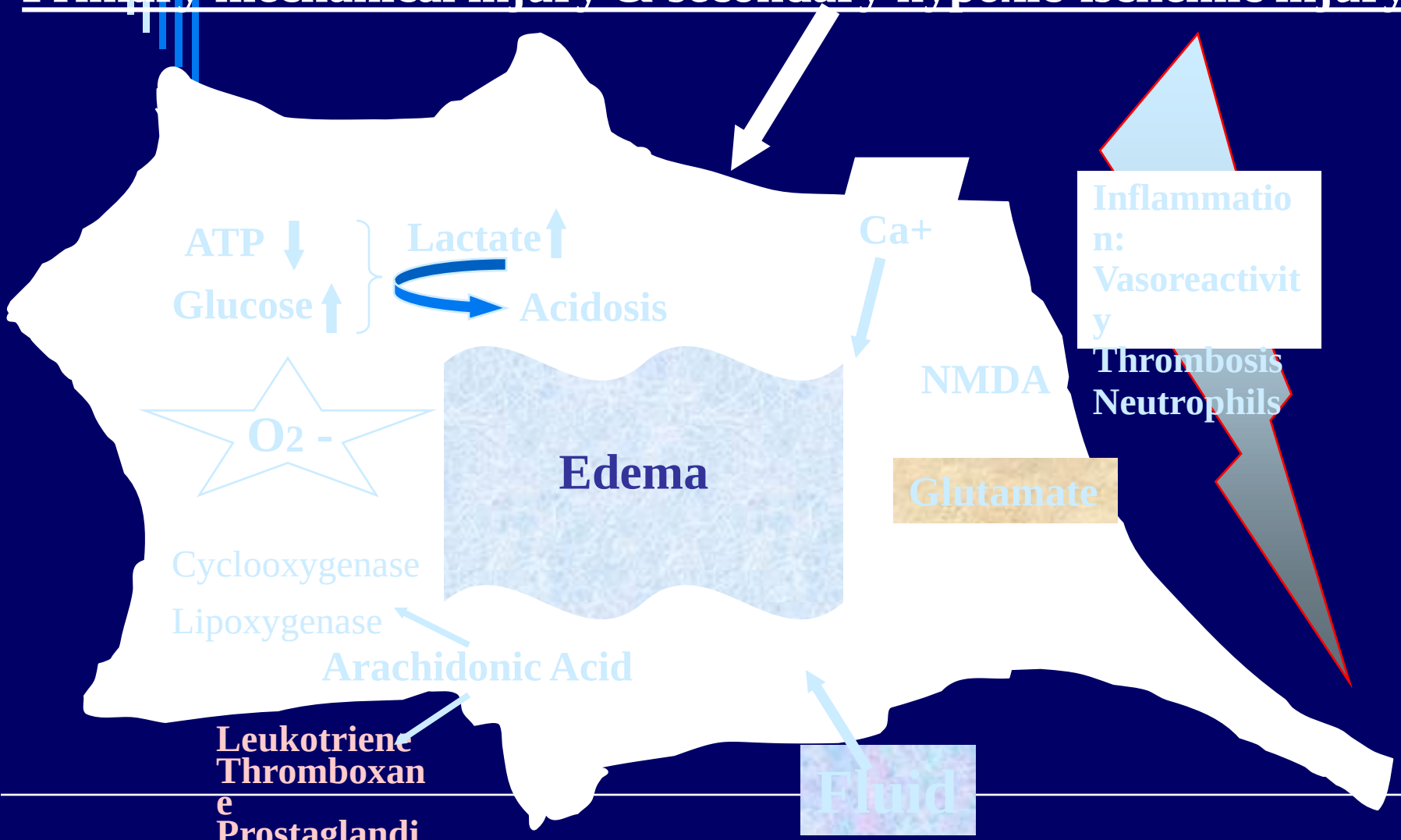


- Contrecoup - is a term used to describe an injury to the brain which is on the **opposite side** of the original impact to the head

- One hypothesis is that it is a consequences of rapid and localized pressure changes originating near the surface of the brain tissues with cavitation effects arising from the brain moving relatively to the bony skull cavity

Neuronal Response to Injury

Primary mechanical injury & secondary hypoxic-ischemic injury



At Present…

- We still know very little about the real implication of mild closed head injury and their outcome
- Protection, protection and protection!
- Very little about the overall **quality of life**, the **need for health care utilization**, **influence of the family** in these circumstances, the **effect of school** and **school performance** on children after **MBI**
- Prospective longitudinal studies, to follow children over a **period of years** utilizing advances in **neuroimaging** and **neuro-psychological outcome** may help in better understanding the predictors and the possible outcomes of children after **MBI.**