



Quiz 43

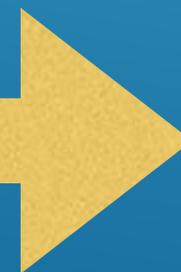
TRAUMATIC BRAIN INJURY 101

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Start



1. A 25 Y/O MALE PATIENT PRESENTS WITH A GUN SHOT WOUND TO THE HEAD. WHICH OF THE FOLLOWING IS **TRUE** REGARDING MANAGEMENT OF TRAUMATIC BRAIN INJURY (TBI):

- A. Severity of initial insult determines ultimate clinical outcome in TBI.
- B. Intracranial pressure monitor can accurately determine progression and guidance to treatment for TBI.
- C. Individually tailored approaches are best guided by monitoring physiologic variables .
- D. Glasgow coma scoring can accurately determine outcome in all TBI patients.

Go to Q2

A. SEVERITY OF INITIAL INSULT DETERMINES ULTIMATE CLINICAL OUTCOME.

This is not True. Clinical outcomes are determined not only by the severity of the initial injury but also by biochemical, excitotoxic, and inflammatory responses that lead to further (secondary) brain injury. Hence, prevention of secondary injury leads to improved outcomes.

Bramlett HM, Dietrich WD: Long-term consequences of traumatic brain injury: Current status of potential mechanisms of injury and neurological outcomes. J Neurotrauma 2015; 32:1834–48

Incorrect
Try again

B. INTRACRANIAL PRESSURE MONITOR CAN ACCURATELY DETERMINE PROGRESSION AND GUIDANCE TO TREATMENT FOR TBI.

This is not true. Multimodal monitoring measure several variables like ICP, CPP, cerebral autoregulation, cerebral oxygenation, simultaneously and provides a more comprehensive picture of the (patho)physiology of the injured brain and its response to treatment.

Bouzat P, Marques-Vidal P, Zerlauth JB, Sala N, Suys T, Schoettker P, Bloch J, Daniel RT, Levivier M, Meuli R, Oddo M: Accuracy of brain multimodal monitoring to detect cerebral hypoperfusion after traumatic brain injury. Crit Care Med 2015; 43:445–52

Incorrect
Try again



C. INDIVIDUALLY TAILORED APPROACHES ARE BEST GUIDED BY MONITORING PHYSIOLOGIC VARIABLES .

This is True. Multimodal neuromonitoring helps in assessment of cerebral hemodynamics, oxygenation, and metabolic status and allows for individually tailored approach to management and prevention of secondary ischemic brain injury. After injury: in this patient treatment decisions can be guided by monitoring changes in physiologic variables rather than by predefined, generic thresholds.

Makarenko S, Griesdale DE, Gooderham P, Sekhon MS: Multimodal neuromonitoring for traumatic brain injury: A shift towards individualized therapy. J Clin Neurosci 2016; 26:8-13

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D. GLASGOW COMA SCORING CAN ACCURATELY DETERMINE OUTCOME IN ALL TBI PATIENTS

This is not true. Although Glasgow coma scoring is still the most widely used scoring system to assess severity of TBI and prognosis, it has limitations as in an intubated patient where verbal response cannot be detected and it does not provide information on brain stem function. Full Outline of UnResponsiveness (FOUR) score that assesses four components of neurologic function— eye, motor, brainstem, and respiratory functions—has been developed to overcome some of these limitations. It also identifies a locked-in state, and detects the presence of a vegetative state.

Wijdicks EF, Bamlet WR, Maramattom BV, Manno EM, McClelland RL: Validation of a new coma scale: The FOUR score. *Ann Neurol* 2005; 58:585–93

Incorrect
Try again

2. ALL ARE TRUE ABOUT THE MECHANISM OF PROGRESSION IN TBI , **EXCEPT:**

- A. Primary brain injury causes shearing of brain tissue and cell death.
- B. Secondary brain injury occurs with efflux of Calcium and influx of potassium.
- C. Hypotension and hypoxemia are often avoidable secondary injury factors for TBI.
- D. Secondary injury continues for months even years post TBI.

Go to Q3

A. PRIMARY BRAIN INJURY CAUSES SHEARING OF BRAIN TISSUE AND CELL DEATH.

This is true. The primary insult/injury to the brain leads to shearing and tearing of the brain parenchyma and blood vessels causing breakdown of the cell membranes and release of cellular content and cell death. This leads to extensive brain tissue deformity.

McIntosh TK. Neurochemical sequelae of traumatic brain injury: therapeutic implications
Cerebrovasc Brain Metab Rev. 1994 Summer; 6(2):109-62.

Incorrect
Try again



B. SECONDARY BRAIN INJURY OCCURS WITH EFFLUX OF CALCIUM AND INFLUX OF POTASSIUM.

This is not true. It is the influx of calcium and efflux of potassium that is responsible for secondary brain injury. Immediately following the primary injury, there is excessive release of the excitatory amino acid neurotransmitters glutamate and aspartate with the subsequent activation of glutamate receptors, a process named excitotoxicity. The release of glutamate results in cellular influx of Na^+ and Ca^{2+} and efflux of K^+ . The influx of calcium ions is regarded to be a key event early post-TBI leading to mitochondrial damage, an increase in free radical production, changes in gene expression and activation of calcium-dependent proteases including caspases, calpains and phospholipases resulting in extensive cytoskeletal damage

Israelsson C, Bengtsson H, Kylberg A, Kullander K, Lewén A, Hillered L, Ebendal T
Distinct cellular patterns of upregulated chemokine expression supporting a prominent inflammatory role in traumatic brain injury.
J Neurotrauma. 2008 Aug; 25(8):959-74.

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C. HYPOTENSION AND HYPOXEMIA ARE OFTEN AVOIDABLE SECONDARY INJURY FACTORS FOR TBI.

This is true. Hypoxemia $\text{PaO}_2 < 60$ mmHg, hypotension < 90 mmHg, fever, seizures, hypo/hyper glycemia are all avoidable and treatable factors. Clinically increased ICP and decreased perfusion pressure should be urgently detected and treated to prevent secondary brain injury. Hence should be monitored continuously.

Marklund N, Hillered L. Animal modelling of traumatic brain injury in preclinical drug development: where do we go from here? *British Journal of Pharmacology*. 2011;164(4):1207-1229

Incorrect
Try again

D. SECONDARY INJURY CONTINUES FOR MONTHS EVEN YEARS POST TBI

This is true. Neuronal and glial damage that occurs at the time of primary injury is markedly exacerbated by a complex cascade of pathophysiological and neurochemical events during the course of the initial hours and days. Progressive encephalomalacia (softening or loss of brain tissue) continues for many years post-injury secondary to diffuse axonal injury.

Büki A, Povlishock JT
All roads lead to disconnection?--Traumatic axonal injury revisited.
Acta Neurochir (Wien). 2006 Feb; 148(2):181-93; discussion 193-4.

Incorrect
Try again

3. ALL OF THE FOLLOWING STATEMENTS REGARDING INTRACRANIAL PRESSURE MONITORING IN TRAUMATIC BRAIN INJURY ARE CORRECT *EXCEPT* :

- A. Salvageable patients with severe TBI with abnormal head CT should have ICP monitoring.
- B. Microtransducer device measures global ICP.
- C. Salvageable TBI patient with normal scan in a patient >40 yr and SBP <90 mmHg, should have ICP monitoring.
- D. Salvageable patients after evacuation of an acute supratentorial hematoma should have ICP monitoring.

Go to Q4

A. SALVAGEABLE PATIENTS WITH SEVERE TBI WITH ABNORMAL HEAD CT SHOULD HAVE ICP MONITORING.

This is true. According to the brain trauma foundation guidelines an intracranial monitor should be placed in a salvageable patient with severe TBI with an abnormal CT scan of the head- intracranial hematoma, contusions, brain swelling, herniation, compressed basal cisterns.

Incorrect
Try again



B. MICROTRANSDUCER DEVICE MEASURES GLOBAL ICP.

This is false. Two types of devices are used to measure ICP in clinical practice. Microtransducer device is seated on the brain parenchyma and measures localized ICP, and Ventricular catheter measures global ICP and has the advantage of draining CSF.

Smith M: Monitoring intracranial pressure in traumatic brain injury. *Anesth Analg* 2008; 106:240-8

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C. SALVAGEABLE TBI PATIENT WITH NORMAL SCAN IN A PATIENT >40 YR AND SBP <90 MMHG, SHOULD HAVE ICP MONITORING:

This is True. An ICP monitor is placed in a salvageable patient with severe TBI with normal scan with 2 or more of the following risk factors- >40 yr, SBP < 90 mmHg, and/or posturing (uni/bilateral)

Incorrect
Try again

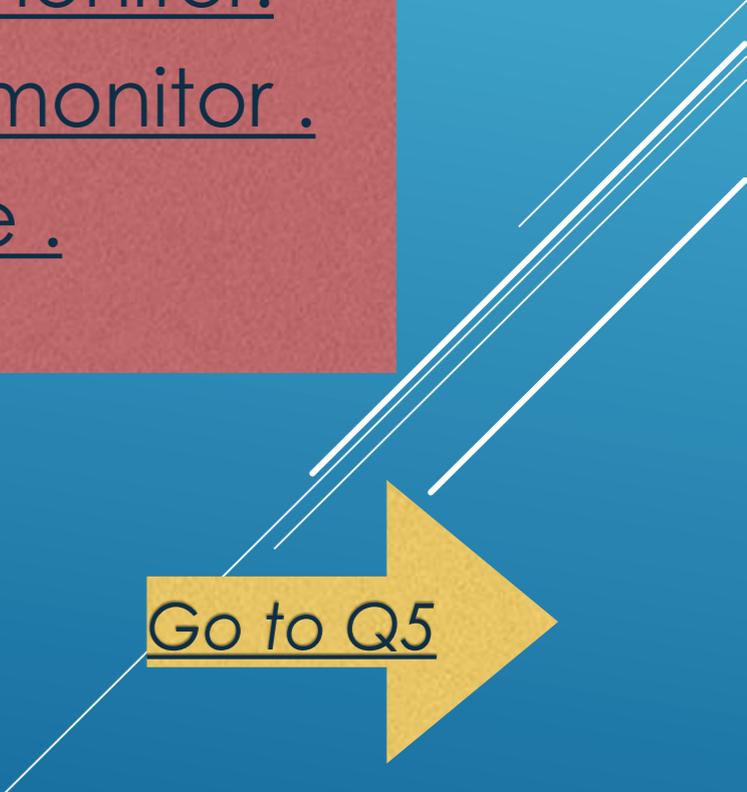
D. SALVAGEABLE PATIENTS AFTER EVACUATION OF AN ACUTE SUPRATENTORIAL HEMATOMA SHOULD HAVE ICP MONITORING

This is true. ICP should also be monitored in patients with Glasgow coma scale motor score ≤ 5 , pupillary abnormalities, prolonged hypoxia and/or hypotension, midline shift > 5 mm. and after secondary decompressive craniectomy to monitor effectiveness of ICP control and guide ongoing management In polytrauma patients requiring multiple procedures under general anesthesia or prolonged sedation.

Incorrect
Try again

4. ALL ARE TRUE ABOUT INTRACRANIAL MONITORING TECHNIQUES FOR CEREBRAL OXYGENATION, **EXCEPT**:

- A. Jugular venous oximetry is a global trend monitor.
- B. Brain tissue oxygen monitor is a focal monitor.
- C. Near infrared spectroscopy is a focal monitor .
- D. Brain tissue oxygen monitor is real time .



Go to Q5

A. JUGULAR VENOUS OXIMETRY IS A GLOBAL TREND MONITOR

This is true. Jugular venous oximetry is a global, flow weighted measure that may miss critical regional ischemia. It is real time and continuous and simple to interpret. It is an invasive procedure, hence can have complications- infection, carotid puncture, thrombosis. Intervention is required when JVO2 \leq 50-55%

Schell RM, Cole DJ: Cerebral monitoring: Jugular venous oximetry. Anesth Analg 2000; 90:559-66)

Incorrect
Try again

B. BRAIN TISSUE OXYGEN MONITOR IS A FOCAL MONITOR

This is true. Brain tissue oxygen monitor is a focal monitor and directly measures brain tissue partial pressure of oxygen in patients with focal lesions such as traumatic contusions, intracerebral hemorrhage. Recent guidelines from the Neurocritical Care Society recommend that brain tissue O₂ monitoring can be used to titrate individual targets for CPP, arterial partial pressure of carbon dioxide (PaCO₂), arterial partial pressure of oxygen (PaO₂), and hemoglobin concentration, and to manage intracranial hypertension in combination with ICP monitoring. It is a gold standard for bedside tissue oxygen monitoring.

Kirkman MA, Smith M: Brain oxygenation monitoring. *Anesthesiol Clin* 2016; 34:537–56

Incorrect
Try again

C. NEAR INFRARED SPECTROSCOPY IS A FOCAL MONITOR .



This is false. NIRS is a noninvasive regional cerebral tissue oxygenation monitoring technique based on the transmission and absorption of near infrared light (700 to 950nm) as it passes through tissue. Has a high spatial and temporal resolution. Not routinely used. Ischemic threshold for intervention not defined.

Smith M: Shedding light on the adult brain: A review of the clinical applications of near-infrared spectroscopy. Philos Trans A Math Phys Eng Sci 2011; 369:44

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D. BRAIN TISSUE OXYGEN MONITOR IS REAL TIME

This is true. Brain tissue oxygen monitor is real time and continuous. Is invasive but has a low complication rate. Therapeutic intervention is warranted when $PO_2 \leq 15-20$ mmHg

Kirkman MA, Smith M: Brain oxygenation monitoring. Anesthesiol Clin 2016; 34:537-56

Incorrect
Try again

5. DERANGEMENT OF BRAIN METABOLISM AND ENERGY DYSFUNCTION INFLUENCE TBI OUTCOMES. ALL ARE TRUE ABOUT METABOLIC MARKERS AND INTERVENTION, EXCEPT:

- A. Cerebral dialysis catheter allows collection of small molecules by diffusion.
- B. Cerebral microdialysis measured lactate:pyruvate ratio is a marker for ischemia.
- C. Glycerol is a marker for cell membrane breakdown.
- D. Periods of high brain glucose with high lactate:pyruvate ratio correlates with poor outcome.

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A. CEREBRAL DIALYSIS CATHETER ALLOWS COLLECTION OF SMALL MOLECULES BY DIFFUSION

This is true. Cerebral dialysis catheter has a semipermeable membrane at the tip which allows diffusion of small molecules with molecular weight less than 20kDa, like glucose, lactate, pyruvate, glycerol, glutamate along their concentration gradient.

Belli A, Sen J, Petzold A, Russo S, Kitchen N, Smith M:
Metabolic failure precedes intracranial pressure rises in
traumatic brain injury: A microdialysis study. Acta Neurochir
(Wien) 2008; 150:461–70

Incorrect
Try again

B. CEREBRAL MICRODIALYSIS MEASURED LACTATE:PYRUVATE RATIO IS A MARKER FOR ISCHEMIA.

This is true. LP ratio is a sensitive marker of tissue ischemia. After TBI, a high LP ratio has been found to correlate with the severity of clinical symptoms and fatal outcome. Increased lactate:pyruvate ratio in the presence of low pyruvate indicates a profound reduction in energy substrate supply and classic ischemia, whereas elevated lactate:pyruvate ratio in the presence of normal or high pyruvate indicates a non-ischemic cause related to mitochondrial dysfunction with or without increased metabolic demand.

Belli A, Sen J, Petzold A, Russo S, Kitchen N, Smith M:
Metabolic failure precedes intracranial pressure rises in
traumatic brain injury: A microdialysis study. Acta Neurochir
(Wien) 2008; 150:461–70

Incorrect
Try again

C. GLYCEROL IS A MARKER FOR CELL MEMBRANE BREAKDOWN

This is true. Glycerol is considered to be a marker of cell membrane degradation and thus cellular lysis.

Microdialysate glycerol is a marker of severe tissue damage, as seen immediately after brain injury or during profound tissue hypoxia.

Clausen T, Alves OL, Reinert M, Doppenberg E, Zauner A, Bullock R: Association between elevated brain tissue glycerol levels and poor outcome following severe traumatic brain injury. J Neurosurg 2005; 103:233–8

Incorrect
Try again

D. PERIODS OF HIGH BRAIN GLUCOSE WITH HIGH LACTATE:PYRUVATE RATIO CORRELATES WITH POOR OUTCOME.



This is false. Periods of low brain glucose concentration (less than 0.7 to 1mM) combined with elevated lactate:pyruvate ratio (more than 40) suggest severe hypoxia/ischemia and correlate with poor outcome. After TBI, the normal relationship between serum glucose concentration, glycemic control, and brain glucose may be lost, and brain glucose may fall to levels that are insufficient to meet metabolic demand even when serum glucose concentration is within a normal range.

Timofeev I, Carpenter KL, Nortje J, Al-Rawi PG, O'Connell MT, Czosnyka M, Smielewski P, Pickard JD, Menon DK, Kirkpatrick PJ, Gupta AK, Hutchinson PJ: Cerebral extracellular chemistry and outcome following traumatic brain injury: A microdialysis study of 223 patients. Brain 2011; 134:484-94

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End of set

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