Severe traumatic brain injury complications

- Distinct clinical phenotypes and their neuroanatomic correlates in chronic traumatic brain injury
- Research progress in the use of botulinum toxin type a for post-stroke spasticity rehabilitation: a narrative review
- Anesthetic and perioperative management of pregnant patients undergoing neurosurgery: a case series from a single center in Morocco (2017-2024)
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- Risk factors for the development of hydrocephalus in traumatic brain injury: a systematic review and meta-analysis
- Disorders of Electrophysiology Following Severe Traumatic Brain Injury
- Challenges to Neurocritical Care Management in Polytrauma

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see Traumatic Intracranial Hypertension

56–60% of patients with GCS score ≤ 8 have 1 or more other organ system injured [1].

25% have “surgical” lesions. There is a 4–5% incidence of associated spine fractures with significant head injury (mostly C1 to C3).

Traumatic brain injury (TBI) is independently associated with Deep-Vein Thrombosis (Deep-vein thrombosis) and pulmonary embolism (PE). However, early prevention with heparinoids is often withheld for its anticoagulant effect.

Evidence suggests low molecular weight heparin reduces cerebral edema and improves neurological recovery following stroke and TBI, through blunting of cerebral leukocyte (LEU) recruitment. It remains unknown if unfractionated heparin (UFH) similarly affects brain inflammation and neurological recovery post TBI.

Prophylaxis was associated with decreased risk of pulmonary embolism and Deep-Vein Thrombosis, but no increase in risk of late neurosurgical intervention or death. Early prophylaxis may be safe and should be the goal for each patient in the context of appropriate risk stratification [2].

Unfractionated heparin (UFH) after TBI reduces LEU recruitment, microvascular permeability and brain edema to injured brain. Lower UFH doses concurrently improve neurological recovery while higher UFH may worsen functional recovery. Further study is needed to determine if this is due to increased bleeding from injured brain with higher UFH doses [3].

Severe traumatic brain injury in childhood can lead to permanent pituitary dysfunction; Growth hormone deficiency and Central precocious puberty may appear after many years. Dassa et al., recommended systematic hormonal assessment in children one-year after severe traumatic brain injury and a prolonged monitoring of growth and pubertal maturation. Recommendations should be elaborated for the families and treating physicians [4].

Patients with severe traumatic brain injury (sTBI) are at risk of adverse events (AEs) during hospitalization, and providing nursing interventions can help reduce the negative impact of AEs. This study primarily discusses the influence of early cluster nursing intervention on nursing efficacy and AEs in patients with sTBI. We enrolled 109 sTBI patients treated in the First Affiliated Hospital of Shanghai Jiao Tong University School of Medicine between October 2022 to June 2023. We grouped them as follows based on different nursing approaches: regular group (n=52) with routine nursing intervention and research group (n=57) with early cluster nursing intervention. Parameters such as nursing satisfaction, incidence of AEs (bed falls, agitation, indwelling needle withdrawal, and skin loss), and scores of Fugl-Meyer Assessment (FMA), Functional Independence Measure (FIM), Glasgow Coma Scale (GCS), National Institutes of Health Stroke Scale (NIHSS), and quality of life assessment instrument (QOL-100) were comparatively analyzed. The analysis showed a higher nursing satisfaction degree and a lower incidence of AEs in the research group compared with the regular group; in addition, FMA, FIM, GCS, and QOL-100 scores were higher in the research group versus the control group after nursing, while the NIHSS score was lower; all of these differences were statistically significant ($P < .05$). Therefore, early cluster nursing intervention is highly effective in the care of sTBI patients. It can effectively improve patients' nursing satisfaction and prevent AEs while enhancing their motor function, functional independence, consciousness, neurological function, and quality of life [5].

Sleep disturbance

Following moderate to severe traumatic brain injury (TBI), sleep disturbance commonly emerges during the confused post-traumatic amnesia (PTA) recovery stage. However, the evaluation of early sleep disturbance during PTA, its recovery trajectory, and influencing factors is limited. This study aimed to evaluate sleep outcomes in patients experiencing PTA using ambulatory gold-standard polysomnography (PSG) overnight and salivary endogenous melatonin assessment at two timepoints (a hormone which influences the sleep-wake cycle). The relationships between PSG-derived sleep-wake parameters and PTA symptoms (i.e., agitation and cognitive disturbance) were also evaluated. In a patient subset, PSG was repeated after PTA had resolved to assess the trajectory of sleep disturbance. Participants in PTA were recruited from Epworth HealthCare's inpatient TBI Rehabilitation Unit. Trained nurses administered overnight PSG at the patient bedside using the Compumedics Somté portable PSG device (Compumedics Ltd, Australia). Two weeks after PTA had resolved, PSG was repeated. On a separate evening, two saliva specimens were collected (at 24:00 and 06:00 hours) for melatonin testing. Routine daily hospital measures (i.e., Agitated Behavior Scale and Westmead PTA Scale) were also collected. Twenty-nine patients were monitored with PSG (mean: 41.6 days post-TBI; standard deviation [SD]: 28.3). Patients mean sleep duration was reduced (5.6 hours, SD: 1.2), and was fragmented with frequent awakenings (mean: 27.7, SD: 15.0). Deep, slow-

wave restorative sleep was reduced, or completely absent (37.9% of patients). The use of PSG did not appear to exacerbate patient agitation or cognitive disturbance. Mean melatonin levels at both timepoints were commonly outside of normal reference ranges. After PTA resolved, patients (n=11) displayed significantly longer mean sleep time (5.3 hours [PTA]; 6.5 [out PTA], difference between means: 1.2, p=.005). However, disturbances to other sleep-wake parameters (e.g., increased awakenings, wake time and sleep latency) persisted after PTA resolved. This is the first study to evaluate sleep disturbance in a patient cohort as they progressed through the early TBI recovery phases. There is a clear need for tailored assessment of sleep disturbance during PTA, which currently does not form part of routine hospital assessment, to suggest new treatment paradigms, enhance patient recovery and reduce its long-term impacts [6].

1)
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2)
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3)
Nagata K, Kumasaka K, Browne KD, Li S, St-Pierre J, Cognetti J, Marks J, Johnson VE, Smith DH, Pascual JL. Unfractionated heparin after TBI reduces in vivo cerebrovascular inflammation, brain edema and accelerates cognitive recovery. J Trauma Acute Care Surg. 2016 Aug 16. [Epub ahead of print] PubMed PMID: 27533909.
4)
Dassa Y, Crosnier H, Chevignard M, Viaud M, Personnier C, Fletchner I, Meyer P, Puget S, Boddaert N, Breton S, Polak M. Pituitary deficiency and precocious puberty after childhood severe traumatic brain injury: a long-term follow-up prospective study. Eur J Endocrinol. 2019 Mar 1. pii: EJE-19-0034.R1. doi: 10.1530/EJE-19-0034. [Epub ahead of print] PubMed PMID: 30884465.
5)
Jiang Y, Jiang C, Hu H, Zhuang Y, Fei K. Influence of Early Cluster Nursing Intervention on Nursing Efficacy and Adverse Events in Patients with Severe Traumatic Brain Injury. Altern Ther Health Med. 2024 Mar 29:AT9633. Epub ahead of print. PMID: 38551416.
6)
Following moderate to severe traumatic brain injury (TBI), sleep disturbance commonly emerges during the confused post-traumatic amnesia (PTA) recovery stage. However, the evaluation of early sleep disturbance during PTA, its recovery trajectory, and influencing factors is limited. This study aimed to evaluate sleep outcomes in patients experiencing PTA using ambulatory gold-standard polysomnography (PSG) overnight and salivary endogenous melatonin assessment at two timepoints (a hormone which influences the sleep-wake cycle). The relationships between PSG-derived sleep-wake parameters and PTA symptoms (i.e., agitation and cognitive disturbance) were also evaluated. In a patient subset, PSG was repeated after PTA had resolved to assess the trajectory of sleep disturbance. Participants in PTA were recruited from Epworth HealthCare's inpatient TBI Rehabilitation Unit. Trained nurses administered overnight PSG at the patient bedside using the Compumedics Somté portable PSG device (Compumedics Ltd, Australia). Two weeks after PTA had resolved, PSG was repeated. On a separate evening, two saliva specimens were collected (at 24:00 and 06:00 hours) for melatonin testing. Routine daily hospital measures (i.e., Agitated Behavior Scale and Westmead PTA Scale) were also collected. Twenty-nine patients were monitored with PSG (mean: 41.6 days post-TBI; standard deviation [SD]: 28.3). Patients mean sleep duration was reduced (5.6 hours, SD: 1.2), and was fragmented with frequent awakenings (mean: 27.7, SD: 15.0). Deep, slow-wave restorative sleep was reduced, or completely absent (37.9% of patients). The use of PSG did not appear to exacerbate patient agitation or cognitive disturbance. Mean melatonin levels at both

timepoints were commonly outside of normal reference ranges. After PTA resolved, patients (n=11) displayed significantly longer mean sleep time (5.3 hours [PTA]; 6.5 [out PTA], difference between means: 1.2, p=.005). However, disturbances to other sleep-wake parameters (e.g., increased awakenings, wake time and sleep latency) persisted after PTA resolved. This is the first study to evaluate sleep disturbance in a patient cohort as they progressed through the early TBI recovery phases. There is a clear need for tailored assessment of sleep disturbance during PTA, which currently does not form part of routine hospital assessment, to suggest new treatment paradigms, enhance patient recovery and reduce its long-term impacts