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REVIEW
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Neuromonitoring in neurocritical care for traumatic brain injury in the Thai context

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ABSTRACT:

The purposes of this review were to identify the incidence and types of traumatic brain injury (TBI) in Thailand, and recommend neuro-monitoring for limited resources in Thai patients. The monitoring methods focus on the targeted and personalized management in severe TBI such as intracranial pressure (ICP), pressure reactivity index (PRx), regional cerebral saturation (rSO_2), cerebral autoregulation (CA), as well as noninvasive methods for example near-infrared spectroscopy (NIRS) and optic nerve sheath diameter (ONSD). These monitors are aimed to optimize brain oxygen delivery and prevent further neurologic deterioration in terms of increased ICP and decreased cerebral perfusion pressure (CPP). Some of these were implemented in neurological monitoring protocol and clinical practice guidelines for severe TBI. However, cost-effectiveness is concerned. Even though considering CA and the advance monitoring methods in continuous assessment are widely used, current therapeutic interventions which appear entirely to bedside approach for correct dysregulated CA are limited. In addition, understanding of basic molecular and cellular pathways involved in cerebral homeostasis (brain oxygen delivery, cerebral blood flow, CA and cerebral vascular reactivity to oxygen and carbon dioxide) as well as secondary brain injury prevention are still necessary for improving TBI outcomes. In summary, further study in Thailand is required to determine optimal cerebral physiologic-based technology, monitoring parameters, and individualized thresholds to optimize CA and potentially improve neurologic outcomes across a spectrum of TBI patients, which focus in Thai rural areas where invasive monitoring is not routinely performed due to resources limitation. Encourage and training of non invasive methods might solve these issues.

Keywords: Traumatic brain injury; Neuro-monitoring; Cerebral autoregulation; Cerebral homeostasis; Secondary brain injury

INTRODUCTION

Traumatic brain injury in Thailand

Traumatic brain injury (TBI) is defined as a lesion that disrupts the normal functioning of the brain [1]. Incidence in Thailand was about 1 million patients per year [2,5] of which 80% were mild TBI, 10% were moderate TBI, and 10% were severe TBI. Causes of TBI in Thailand were motor vehicle-traffic accidents (66%), and 32.8% fell injuries, both at the same level and at a different level (Tables 1-3). The characteristics of TBI patients when performing a computer tomography (CT) scan were a 30% abnormal CT scan and 3-5% surgical indication that surgery was needed [2]. After following these patients for 6 months, we found that 61% of patients with moderate to severe TBI experienced a disability, a high rank in the volume of patients, mortality, and a combination of both death and disability [3].

Neuro-trauma in Thailand was a major cause of disability and death. The severity and impact on relationships vary greatly, from physical to cognitive, psycho-emotional, and behavioral, are significant medical, healthcare, and social issues due to heterogenic and complex pathology, considering the vast array of causes, mechanisms, clinical picture, management, and post-injury outcomes that effect the burden on society because of high mortality rates and bring about the need for surgical treatment and, rehabilitation [4].

In Thailand, factors that affect neurosurgeon, neurologist, and neuro-intensivist management have been guided by neurologic examination and neuroimaging, and while both provide invaluable clinical insights, in isolation, these approaches do not support an understanding of the ongoing dynamic pathobiological processes, which neuro-monitored can guide medical and surgical intervention in real time continuously [5]. There has been an increased focus on the development and utilization of neuromonitoring techniques and neurophysiologic measurement, which allow for enhanced surveillance and recovery of brain physiologic parameters in order to detect secondary brain injury and allow for goal-directed interventions to overcome irreversible brain damage. One of the advantages of the expanded use of multimodality monitoring (MMM) was the capability to characterize cerebral autoregulation (CA) capacity, which has been validated as an important prognostic indicator and can be useful to guide neuro-hemodynamics management with the goal of optimizing brain oxygen delivery and individualized cerebral perfusion approaches. Autoregulation status has impacted recognition as a crucial preventive homeostatic mechanism and an essential determinant of mortality and functional outcome in patients with severe TBI [6].

KEY MESSAGES:

- Traumatic brain injury (TBI) is a heterogeneous range of sequelae pathologic processes that can often lead to irreversible neurologic insult.
- Primary brain injury is a cascade of maladaptive and deleterious neurophysiologic processes that can ensue in the form of neuro-inflammation as well as impaired cerebral autoregulation (CA), leading to ischemic brain injury and subsequent neuronal death.
- Prevention of secondary brain injury is very important in neurocritical care management with the goal of optimizing brain oxygen delivery and attempting to maximize the potential for recovery.

Table 1. Injury characteristics of TBI in Thailand in dominant type of injury [2].

Dominant type of injury	Incidence (%)
Blunt	95.2
Penetrating	2
Combined Blunt and Penetrating	0.9
Unknown	1.9

Table 2. Injury characteristics of TBI in Thailand in intention of injury [2].

Intention of injury	Incidence (%)
Accidental	90
Self-inflicted	4.5
Assault	1.8
Unknown	3.6
Other	0.1

Table 3. Injury characteristics of TBI in Thailand in location of injury [2].

Location of injury	Incidence (%)
Road	61.1
Workplace	5.2
Home	23.3
Sport facility	1.8
Other	4.7
Unknown	3.9

MAIN BODY

Neuro-trauma chain of care and Neuro critical care management in Thailand evolved over time with important landmarks including The integration of brain-specific treatments into Thai guideline for pre-hospital life support for the initial resuscitation of the severe head Injury patient in 1996, Thai guideline for intracranial pressure critical pathway for severe head injury patient in 2000, Setting intracranial pressure threshold and Thai guideline adapted from guideline for the management of severe traumatic brain injury in 2007, Thai guideline adapted from Guideline for the management of severe traumatic brain injury in 2016, collaborated with the Thai society of critical care medicine for set program for critical care course for neurosurgeon in 2018-2019 and Thai guideline adapted from Seattle international severe traumatic brain injury consensus conference in 2019 (Figure 1).

The main objectives of performing systematic neuro-critical care in Thailand, together with neuro-monitoring, were to optimize brain oxygen delivery, improve practical applications, and provide education to encourage humanization in neurotrauma and neurocritical care, education, and changing the approach–promoting teamwork and multidisciplinary techniques, verifying patient care, caregiver education, and strengthening and long-term follow-up on the impact of neurocritical care patients on the quality of life of the patients, their families, and their social circle [3].

Neuromonitoring in traumatic brain injury

For the setting and organization of the Neuro-critical Care Unit (NCCU), intensivists should be concerned about the policy for organ donation and "do not resuscitation", which is composed of guidelines for palliative care and end of life care with clinical flow and guidelines for potential organ donation and the brain death declaration committee. NCCU should develop guidelines or protocol such as stroke guideline, brain tumor surgery flow or Enhanced Recovery After Surgery (ERAS) protocol for brain tumor surgery, severe TBI care guideline, status epilepticus guideline, Massive blood transfusion protocol, Heavy sedation protocol and miscellaneous for example admission criteria and discharge policy especially in patients with devastating brain injuries, NCCU should exactly prepare a tool that had the most accuracy in prognostication aimed to prevent premature withdrawal of organ support, treatment strategies bias and used a 72-hour observation period to determine clinical response and delaying decisions regarding withdrawal of therapy co-operated with family support for allowing family presence at the bedside and made a policy and guideline for end of life care. Technology in NCCU: intensivists should prepare special equipment such as intracranial pressure (ICP) monitoring sets, continuous electroencephalography (EEG), near-infrared spectrometry (NIRS), regional cerebral oximetry for brain oxygenation monitors, brain tissue oxygen tension, and cerebral microdialysis (CMD)-biomarker analysis (glucose, lactate, and pyruvate) (CMD was a future aspect in Thailand) [6, 24].

The differences between the NCCU and the medical or surgical intensive care unit (ICU) for neurosurgical and neurological patients were longer lengths of stay, more invasive intracranial monitoring and hemodynamic monitoring, more tracheostomies, more nutritional support, and less intravenous sedation.

Intensive care management in severe TBI in the Thai context was based on the objective of decreasing ICP and optimizing cerebral perfusion pressure (CPP). Basic physiology in brain oxygen delivery is equal to the product of cerebral blood flow (CBF) and arterial oxygen contents (CaO_2). The important knowledge is the identification of factors that affect CBF, such as CPP, autoregulation status, and arterial partial pressure of oxygen and carbon dioxide. To prevent sequelae of elevated ICP (herniation syndrome such as uncal ,central transtentorial, subfalcine and tonsillar herniation and cerebral ischemia and hypoxia comprised decreased in CPP and brain oxygen delivery) is to perform neuro-monitoring that classified into monitor in driving pressure (ICP and CPP), CBF (Transcranial Doppler (TCD) ultrasound for detect local and regional blood flow), brain oxygen delivery (Jugular venous oxygen satura-

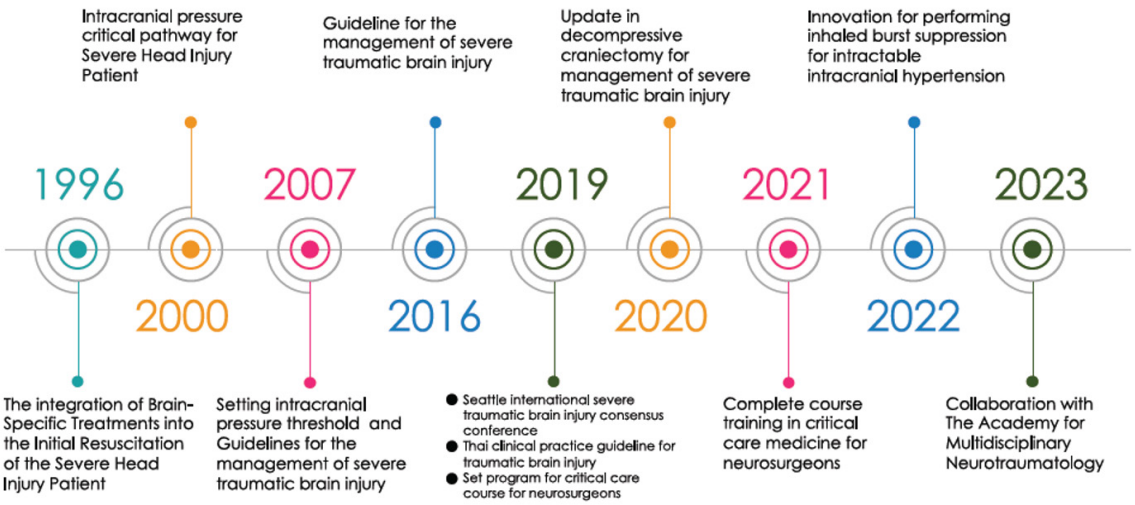

Figure 1. Milestone of neuro-trauma and neuro-critical care in Thailand.

tion ($Sjvo_2$), Near-infrared spectroscopy (NIRS) and Brain tissue oxygenation ($PbtO_2$), cerebral metabolism (CMD to measure cerebral lactate-pyruvate ratio, cerebral lactate and glucose) and central nervous system function (EEG and electrophysiology study) that recommend threshold level in Table 4 [6-11,24] and the diagram of the neuro-monitoring strategy in traumatic brain injury in Figure 2.

We recommend neuro-monitoring in the Thai context to begin with ICP monitoring with analysis of PRx combination with non-invasive neuro-monitoring such as NIRS and ONSD. We start with ICP monitoring in all severe TBI patients because when the Glasgow Coma Scale (GCS) was below 8, they had a low-quality to closed-monitoring level of consciousness. In non-traumatic brain injury, we recommend performing ICP monitoring in patients with high risk for elevated ICP, such as patients with hydrocephalus or at high risk for developing hydrocephalus, patients with SAH, ICH, and other non-TBI conditions. In patients who are at risk of elevated ICP based on clinical and/or imaging features, all poor-grade SAH patients should be monitored and considered for MMM, as should patients who undergo hemicraniectomy in the setting of cerebral edema [13,15]. Contraindications for invasive ICP monitoring were concurrent use of anticoagulant drugs, bleeding disorders, scalp infections, and brain abscesses [13,24]. The gold standard for ICP monitoring was an intraventricular catheter that had the advantages of being most accurate, able to recalibrate, having a lower cost, and allowing therapeutic CSF drainage. Some disadvantages were found, for example, being difficult to insert into compressed or displaced ventricles, easy obstruction of the fluid column in the catheter, e.g., blood clots, the transducer must be consistently maintained at a fixed reference point relative to the patient's head, and the risk of infection and intracerebral hemorrhage. Arterial and intracranial pressure waveforms (Figure 3) consist P1 (percussion wave), which refers to arterial pulsation from the left ventricle contracting to send cardiac output to the brain; P2 (tidal wave), which refers to intracranial compliance and terminates in the dicrotic notch; and P3 (dicrotic wave), which refers to the reflection of aortic valve closure. The important wave was P2, due to the change in the morphology of P2, which can predict the failure of the cerebral autoregulation systems and therefore serves as an early indicator of increased ICP [13]. Autoregulation monitoring can be performed by the PRx, which is a moving Pearson's correlation that expresses the correlation coefficient between ICP and mean arterial pressure (MAP) (Figure 4). If MAP increases, ICP increases together, resulting in a positive correlation coefficient that means disruption of autoregulation. On the other hand, if MAP increases but ICP remains constant but decreases, a zero or negative correlation coefficient results in normal or intact autoregulation. The aim of PRx in MAP and CPP planning is to keep the lowest PRx or PRx threshold < 0.3 [11]. The lowest level of PRx refers to the optimum CPP and MAP for stabilized dysfunctional autoregulation in the lowest ICP and maximum CPP for an individualized target in each severe TBI patient. Another method to assess autoregulation status is the MAP challenge by using ICP monitoring, a titrated vasopressor, and an increase in MAP. When the increased dose of vasopressor effect increased in MAP, if ICP remained constant or decreased, it meant that it was intact in autoregulation status because this mechanism created cerebral vasoconstriction that resulted in cerebral blood flow decreasing, then ICP decreased. If ICP increases, it means autoregulation status dysfunction because this mechanism leads to cerebral vasodilatation, producing CBF that increases and increases in ICP [10].

Another non-invasive neuro-monitoring method, such as NIRS and ONSD, can be useful for guided intervention and assisted neuro-monitoring when ICP monitoring cannot be performed or during adjusted ventilation, especially in patients with severe TBI with lung injury or

Table 4. Characteristics of neuromonitoring classified into invasive and non-invasive methods and recommend threshold levels [7-11, 14-16, 24].

Invasive neuro-monitoring	Non-invasive neuro-monitoring
ICP monitoring threshold level ICP < 22 mmHg CPP 60-70 mmHg Monitor P2 waveform	Near-infrared spectroscopy (NIRS) rSO_2 > 75 % and change from baseline and between hemisphere ≤ 10% Optimal mean arterial pressure (MAP) or CPP based on autoregulation monitor in cerebral oximetry index
Pressure reactivity index (PRx) threshold level Optimum CPP based on autoregulation monitoring Keep Lowest PRx or PRx threshold < 0.3	Transcranial Doppler Ultrasound Threshold for Cerebral vasospasm Mean Flow Velocity (MFV) of Middle Cerebral Artery (MCA) > 120 cm/sec and Lindegaard ratio (LR) > 3 MFV of MCA > 200 cm/sec and LR > 6 is strongly suggestive Cerebral Vasospasm
Brain tissue oxygenation ($PbtO_2$) > 20 mmHg	Optic nerve sheath ultrasound Diameter > 0.5 cm correlates with ICP > 20 mmHg
Jugular venous oxygen saturation ($Sjvo_2$) 55-75 %	EEG Detection of seizure and assess cerebral function
Cerebral microdialysis threshold level Lactate-Pyruvate ratio < 25 Lactate < 4 mmol/L Glucose > 0.8 mmol/L	

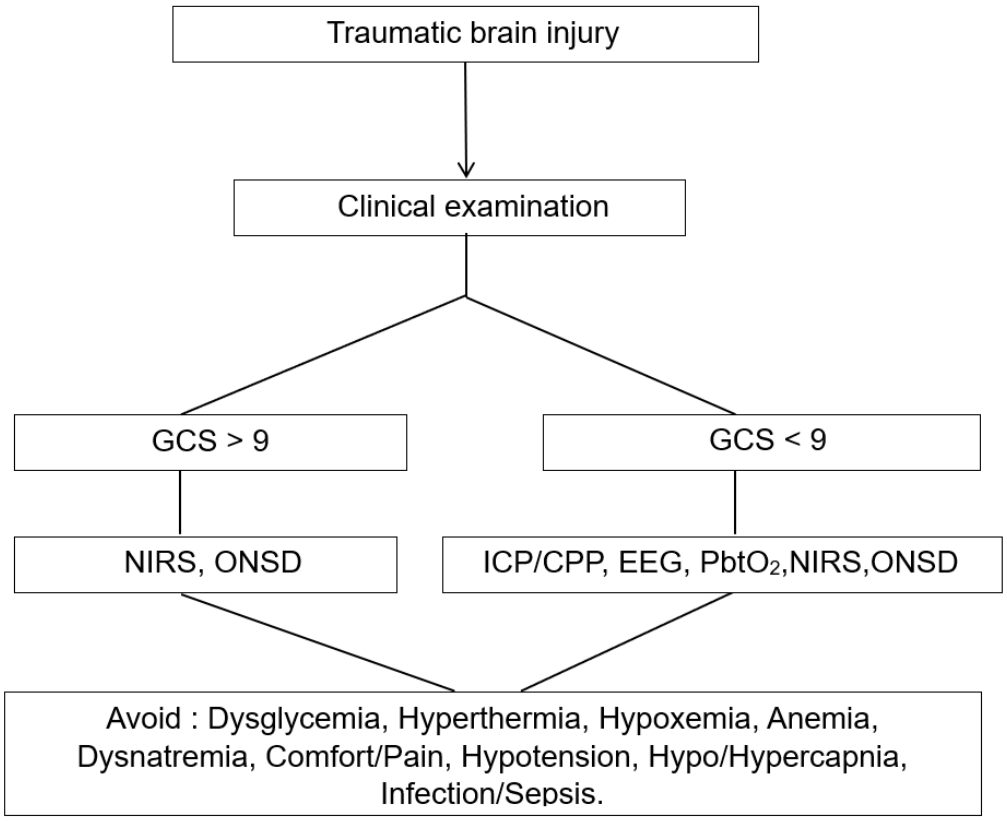

Figure 2. The diagram of the neuromonitoring strategy in traumatic brain injury.

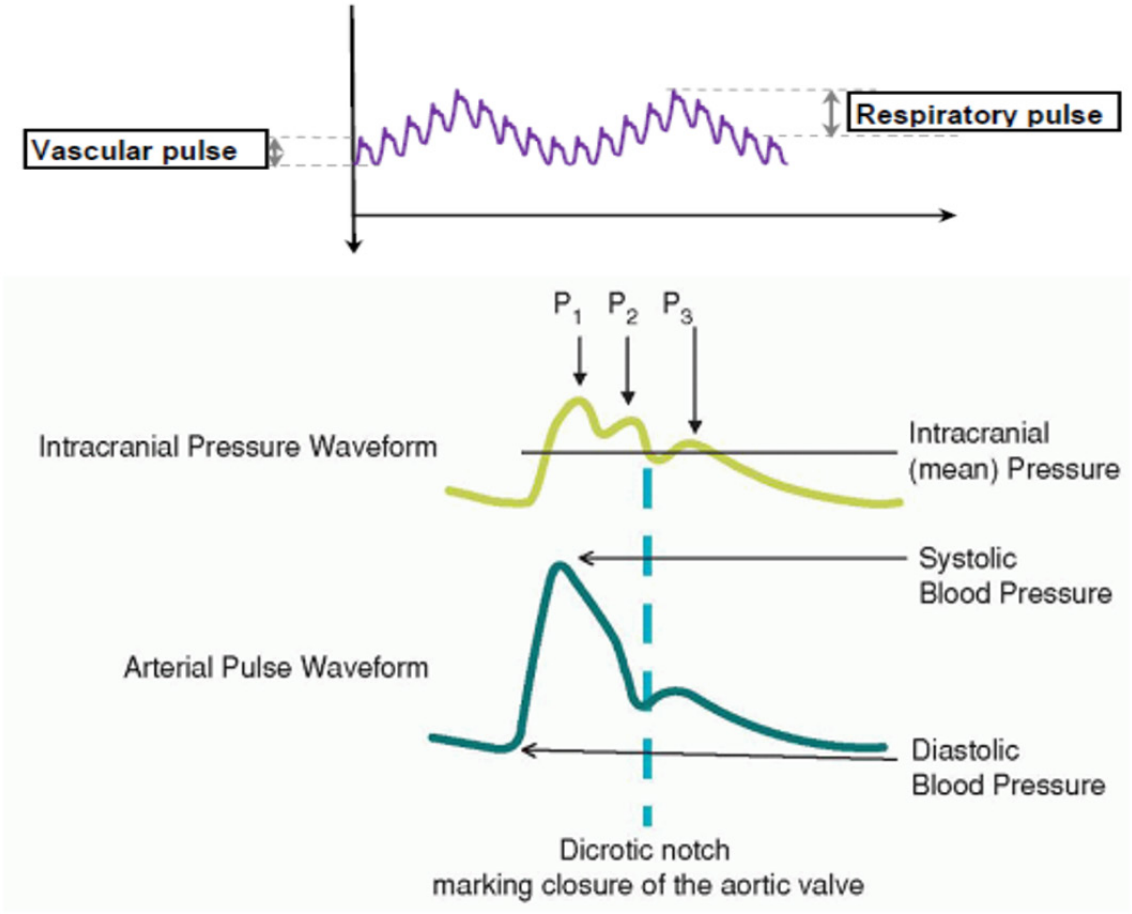

Figure 3. Arterial and Intracranial pressure waveforms.

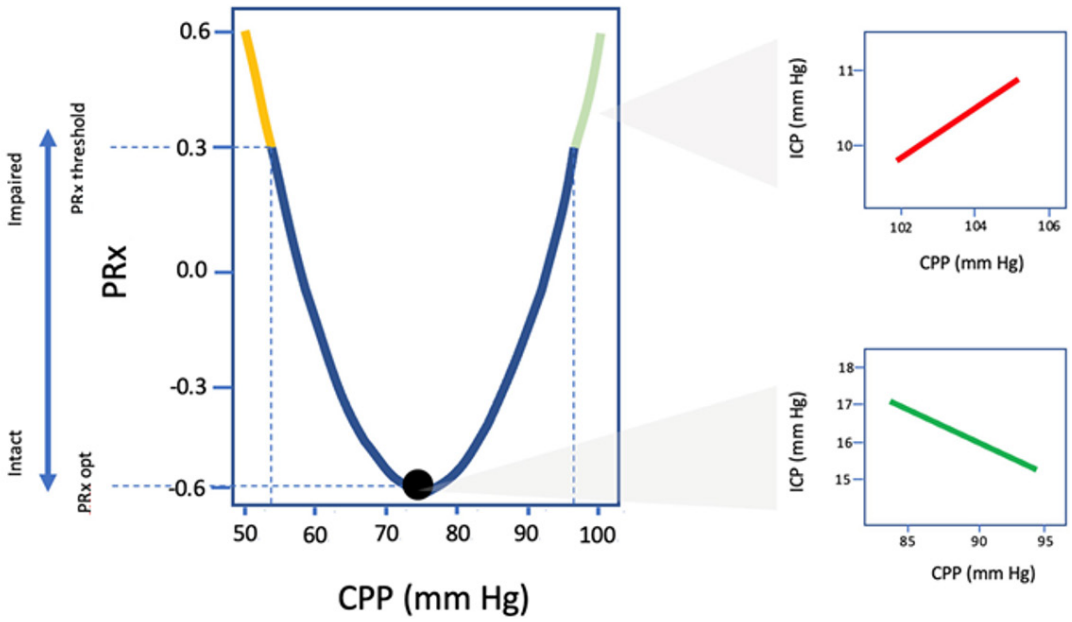

Figure 4. U-shaped curve of PRx value as a function of CPP, CPPOPT identified as the CPP value with the lowest associated PRX and most optimal autoregulatory state and correlation coefficient of both positive and negative correlation between CPP and ICP.

acute respiratory distress syndrome (ARDS), or for guided intervention in comatose patients to avoid cerebral desaturation. Not only can NIRS be performed in a neurosurgical intensive care unit, but it can also be useful in the medical ICU when used for extracorporeal membrane oxygenation (ECMO) and ventilation management, vascular surgery such as carotid endarterectomy (CEA), thoracic aortic aneurysm (TAA) surgery, and cardiac surgery to monitor perioperative stroke. This technique provides estimates of total hemoglobin concentration as well as the ratio of cerebral oxyhemoglobin to total cerebral hemoglobin concentration, called regional cerebral oxygen saturation (rSO_2). It reflects the balance between oxygen supply and demand within the distal arterial, venous, and capillary territories [24]. The majority of NIRS applications involve placing optodes over the forehead and measuring signal over the frontal lobe gray matter and watershed area of anterior and middle cerebral arteries (MCA) territory with the normal range of rSO_2 between 55–80% [15-16]. Intraoperative management guided by the use of cerebral oximetry (CeOx) was associated with a reduction in postoperative complications such as stroke, ICU and hospital length of stay, and the incidence of postoperative delirium. Severe TBI with a decrease in cerebral oximetry is associated with multiple impaired systemic microcirculations, more morbidities, and mortality rate. When using the brain as an index organ, interventions to improve brain oxygen delivery may have systemic benefits for these patients. NIRS can assess the bifrontal regional cortex, and regional cerebral oxygen saturation (rSO_2) has shown a correlation between severe TBI patients with low rSO_2 values and impaired cognitive function. CeOx has been cleared by the United States Food and Drug Administration for monitoring regional cerebral oxygen saturation [4], and cerebral deoxygenation (rSO_2 below 55%) has been shown to correlate with a variety of adverse systemic complications and multi-organ failure, for example, renal failure, overall well-being, prolonged ventilation, cortical dysfunction, cerebral hypoxia, and low cardiac output syndromes. Many studies [1,7,12,16] showed that CeOx with NIRS correlated with CPP estimation, the Glasgow Outcome Score Scale, and mortality in patients with severe TBI, and NIRS has the capability to provide an early warning of cerebral ischemia and infarction. These impacts of continuous monitoring showed the above correlations between regional cerebral saturation and systemic outcomes, and most of the intraoperative measures taken to optimize cerebral rSO_2 and oxygen delivery potentially affect systemic perfusion (e.g., alterations in $PaCO_2$, cardiac output, arterial blood pressure, etc.). We suggested that using the brain as an index organ and attempting to make interventions to optimize rSO_2 would have a systemic benefit to improve global tissue perfusion and clinical outcomes. A review of CeOx [15-16] showed that avoiding decline of rSO_2 prevented prolonged desaturations and was associated with a shorter ICU length of stay [24]. The intervention protocol undertaken to increase rSO_2 to baseline resulted in a rapid improvement in rSO_2 in most cases and did not add any risks to the patients. While none of the interventions undertaken are outside the range of standard clinical practice for TBI, it is clear that in the absence of feedback from a specific indicator of end-organ compromise (e.g., cerebral desaturations), the ability of neuro-intensivists to detect and optimize otherwise silent but potentially adverse perturbations in clinical variables remains limited and indicates a clinical benefit to monitoring and managing cerebral oxygen saturation in the ICU [16]. The data on the benefit of near-infrared spectroscopy to monitor regional cerebral oxygen saturation in patients with traumatic brain injury is limited. The variability in baseline saturation readings is even below the

normal limit because of the loss of normal CA, and the subsequent changes from this baseline are more difficult to interpret, as is the problem with intracranial hypertension, the important secondary brain injury, and ischemic insults (Figure 5-6) (Informed consent was obtained from either the patient himself or his family). NIRS has the advantage of being completely non-invasive, portable, and capable of providing regional assessments of cerebral physiology and oxygenation [12]. In contrast to TCD ultrasound, however, NIRS has the added superior benefit of being non-operator dependent, easily used in the operative, prehospital, emergency department, and ICU environments without complexity in setting and getting data continuously. There are some limitations that must be addressed. NIRS requires a close spatial relationship between the cortex and cranium and is prone to inaccurate readings in the setting of post-neurosurgical procedures, such as pneumocephalus, skin pigmentation, scalp edema, extracranial hemodynamics, frontal contusions, and hemorrhage, which are all common in neuro-ICU patients. It is sensitive to changes in the superficial cortical anatomy, often restricted to the frontal lobe region, and does not have the capability to detect distant ischemic events [15-16]. The other important limitation of NIRS is rSO_2 value is not well-correlated with $PbtO_2$ [26].

The other non-invasive neuro-monitoring was optic nerve sheath ultrasound to measure ONSD for estimating ICP, which has been widely reported in several pieces of literature [17-25]. This can be useful for assistive neuro-monitoring when neurosurgeons or neurologists cannot perform ICP monitoring and can be easily applied at the bedside together with repeats for follow-up. Recently, ONSD has come into the field for the diagnosis of increased ICP [23]. It has been preferred for neuromonitoring due to its ease of application, particularly in patients with TBI in pre-hospital care and emergency units [19]. Increased ICP, or cerebrospinal fluid (CSF) pressure, is directly transmitted to the subarachnoid space around the perineurium, resulting in enlargement of the perioptic dural sheath. The measurement of ONSD was performed by applying a linear probe placed transversally over the closed eyelid to the patient, then measuring at 3 millimeters from the globe when the diameter was more than 0.5 centimeter and the ICP was more than 20 mmHg [18] (Figure 7-8) (Informed consent was obtained from her family). The optic nerve sheath surrounds the optic nerve, which continues with subarachnoid spaces and CSF collected inside [21]. As a result [22-24], it had a susceptible variant in diameter that responded to ICP fluctuations. The optic nerve is characterized by a mean diameter of 3 mm, while ONSD has an average thickness of 4 mm. The subarachnoid space between them measures about 1 mm [22]. Based on those findings [21], it can be estimated that the ONSD measures approximately 4 mm under physiological conditions. A direct connection exists between the subarachnoid space of the optic nerve and the chiasmatic cistern compartment, thus allowing for the communication of the CSF between these compartments. The threshold for ONSD was found to be 5.0 mm. Higher values may indicate an increased ICP. In a meta-analysis [22-25], ONSD was found to have a high probability of detecting increased ICP (>20 mmHg). ONSD has recently come into use to diagnose and follow-up on increased ICP in emergency departments, ICUs, and NCCUs because it is less expensive, does not have radiation effects, can be easily used at the bedside, and can be repeated for follow-up in severe TBI patients [19].

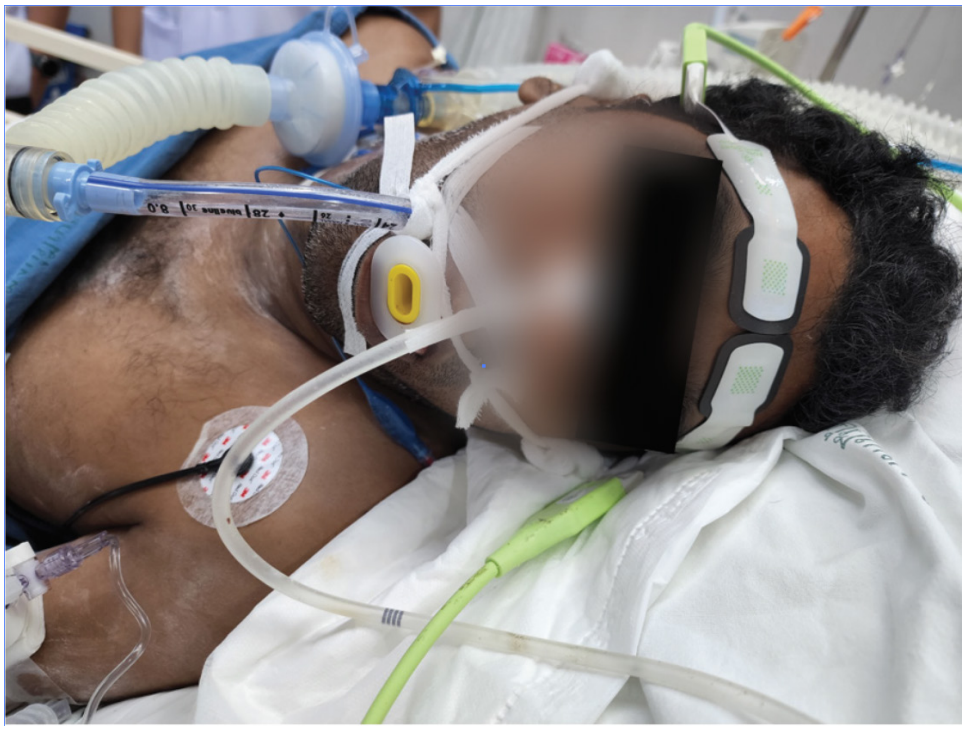

Figure 5. NIRS applies guided intervention and assisted neuro-monitoring when ICP monitoring is not performed in severe TBI patients.

Figure 6. The use of NIRS in combination with hemodynamic monitoring guided intervention in severe TBI patients.

Figure 7. Optic nerve sheath ultrasound applied guided intervention and assisted neuro-monitoring when cannot perform ICP monitoring in severe TBI patients.

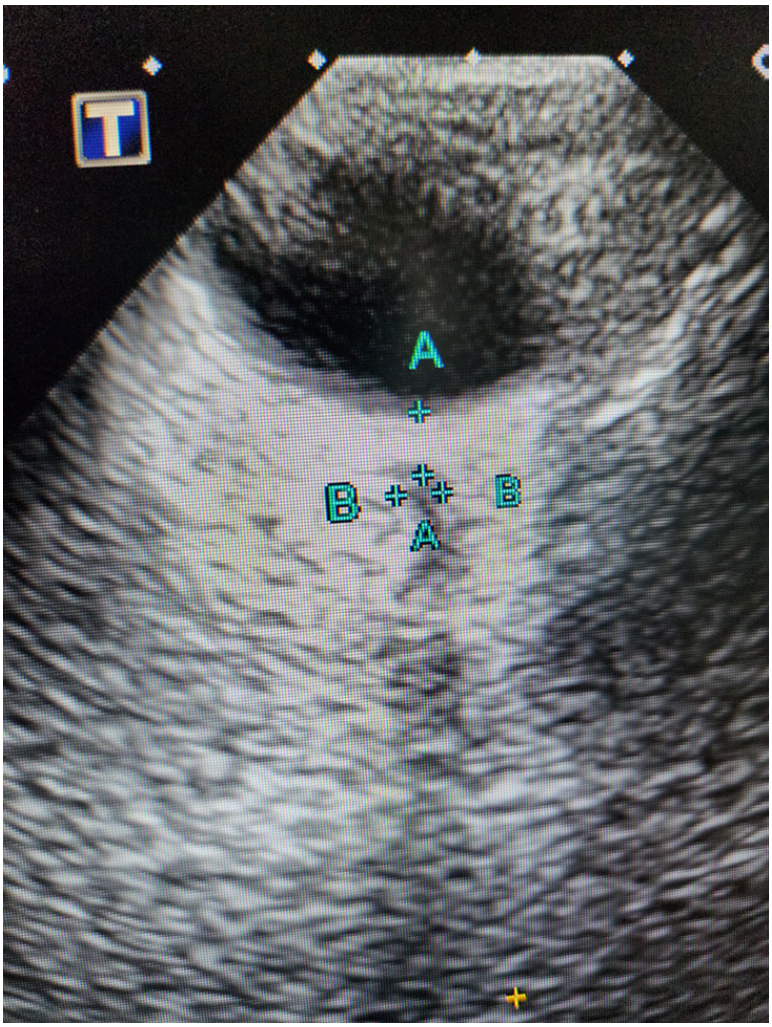

Figure 8. Optic nerve sheath ultrasound measurement by application of a linear probe placed transversally over the closed eyelid of the patient then measuring at 3 millimeters from the globe when the diameter is greater than 0.5 centimeter, it correlates with ICP greater than 20 mmHg (A = Eye globe and B = Optic nerve sheath).

CONCLUSION

The management of critically ill neurological and neurosurgical patients was complex and different from that of general patients. The therapeutic targets are different from those of general critically ill patients. Good knowledge and clinical assessment skills on the NCCU team are the keys to success in neurological and neurosurgical patient care.

Although non-invasive ICP measurement currently exists, many physical properties of the head remain to be evaluated for these pressure measurements. The best solution, according to current viewpoints, will probably involve combining several methods and calibrating them at intervals (e.g., performing ONSD every 6-8 hours or using near-infrared spectroscopy to apply guided intervention and assisted neuromonitoring when ICP cannot be performed in continuous data). The continuing development of non-invasive methods with suitable feedback mechanisms is necessary to strengthen the confidence of physicians and further improve the methods of caring for severe TBI patients in the Thai context. Noninvasive neuromonitoring, such as optic nerve sheath ultrasound and near-infrared spectroscopy, may be used to guide treatment in traumatic brain injury patients, but further research is needed to determine the clinical benefit of those monitoring techniques.

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ABBREVIATIONS

CA: Cerebral autoregulation; CaO_2: Arterial oxygen contents; CBF: Cerebral blood flow; CEA: Carotid endarterectomy; CeOx: Cerebral oximetry; CMD: Cerebral microdialysis; CPP: Cerebral perfusion pressure; CSF: Cerebrospinal fluid; CT: Computer Tomography; ECMO: Extracorporeal Membrane Oxygenation; EEG: Electroencephalography; ERAS: Enhanced Recovery After Surgery; GCS: Glasgow Coma Scale; ICH: Intracranial hemorrhage; ICP: Intracranial pressure; ICU: Intensive care unit; LR: Lindegaard ratio; ONSD: Optic nerve sheath diameter; MAP: Mean arterial pressure; MCA: Middle cerebral artery; MFV: Mean flow velocity; MMM: Multimodality monitoring; mmHg: Millimeters of mercury; NCCU: Neuro-critical Care Unit; NIRS: Near-infrared spectroscopy; $PaCO_2$: Partial pressure of carbondioxide; $PbtO_2$: Brain tissue oxygenation; PRx: Pressure Reactivity Index; rSO_2: Regional cerebral saturation; SAH: Subarachnoid hemorrhage; $SjvO_2$: Jugular venous oxygen saturation; SpO_2: Peripheral O_2 saturation; TAA: Thoracic aortic aneurysm; TBI: Traumatic Brain Injury; TCD: Transcranial Doppler

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