Review Article

Critical Care in Neurosurgery in Developing Countries

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Abstract

Neurological patients managed in specialized intensive care units have better outcomes than those managed in general intensive care units. However, such management strategies cannot be applied to Nepalese healthcare entirely. Effective management is affected by its difficult geographical location (difficulty in transporting sick patients due to the lack of motorable roads and almost nonexistent effective air transport systems) and virtual nonexistence of neurointesivists in the country. While some 'supplies' for the treatment of serious neurosurgical patients are readily available (e.g. central venous pressure lines, arterial lines, ventricular drainage catheter etc.) in major neurosurgical centers in Nepal, others are scarce (e.g. adequate operating room capacity, intracranial pressure monitors, Swan Ganz catheters etc.). In this review an attempt has been made to distill the pertinent literature in the field and recommend what is applicable in the third world countries like Nepal where a profound physical and human resource constrains exist. In addition, realistic proposals for improvement of the current situation are made.

Key Words: Intensive care unit, head injury, neurocritical care, neurointensivists

Introduction

Neurocritical care is a relatively new subspecialty in the field of neuroscience that intersects with many neuroscience and allied specialties. The main aim of this field is to advance the care of the critically ill neurological patients. The group of health care practitioners that are specially trained to take care of such patients are called neurointensivists. Neurological patients cared for in specialty neuro-intensive care units (NICUs) often undergo more invasive intracranial and hemodynamic monitoring, tracheostomy, and receive less sedation than patients in general intensive care units (ICUs). These differences in care probably explain the lower mortality and better outcome in patients treated in specialized NICUs than in general ICUs. The main argument for the necessity of neurointensivist training is that the care of often critical neurological patients mandates special training in clinical physiology of intracranial pressure, cerebral blood flow and metabolism, brain and neuromuscular electrophysiology, and ventilator management. Neurocritical care has made a tremendous difference in the care of patients mainly with acute ischemic stroke, intracerebral and subarachnoid hemorrhages, intracranial neoplasms, traumatic brain and spinal cord injury, status epilepticus, and neuromuscular respiratory failure. The goal is to minimize deaths, and to convert many "deaths" and "vegetative states" into "good outcomes". Today, many advances in imaging, therapeutics, and neuromonitoring have made it possible to affect outcome of critically ill patients. [1] One thorny issue, however, is the cost incurred in setting up the ICUs and training of the skilled manpower which is often the rate-limiting factor in establishing the good neurocritical care service in the less developed countries. However, as neurologically disabled and vegetative patients consume vast economic resources, it will be cheaper in the long run.

Though a relatively new field, Neurocritical Care as a specialty has undergone rapid developments over last several decades. Though developing countries like ours are struggling to have a modest neurocritical care, let alone a state-of-the-art one, it is imperative for all of us to be familiar with what the essential components of a modern Neurocritical Care are.

Early Developments

The appropriate starting point for a history of neurocritical care is a matter of debate, as there is no agreement as to what constitutes neurocritical care.

The concept of modern neurosurgical ICUs was first developed by Dr. Walter Dandy in 1932 at Johns Hopkins Hospital when two adjoining semiprivate rooms of the

Neurosurgical unit, called Halsted 7, were dedicated to manage neurosurgical patients postoperatively.[1,2,3] All patients were taken to these rooms after surgery and, when conscious and alert, they were moved to the ward. More seriously ill patients with craniotomies were observed here one or more days until they became stable. There was a nurse in each of these two adjacent rooms continuously and interns and residents made more frequent rounds. Many consider his creation of this special unit to be the first real ICU. The first formal neurocritical care training in the United States were set up in the late 1970s to early 1980s offering fellowships. The Neurocritical Care Society was founded in 2003 with the support of the Society of Critical Care Medicine. There has been simultaneous growth of this specialty from the Europe and has subsequently spread to the rest of the world. Neurocritical Care is now a viable and respectable subspecialty on its own.

Neurocritical care involves a) Clinical assessment, b) Monitoring, and c) Treatment of specific abnormalities.

Initial Clinical Assessment

During the last decade intracranial pressure monitoring, calculation of cerebral perfusion pressures, evolving microsensor technology, etc. have changed the perception of clinicians involved with sick neurological patients. Previously, clinical neurological examination was the only means of monitoring. In patients, for example, with mild or moderate head injury (HI) clinical assessment is still the most important information. By using the Glasgow coma scale (GCS) the level of consciousness in trauma patients can be followed efficiently in a standardized fashion. In severe HI patients, however, clinical assessment and the value of GCS scoring is limited, for patients are usually sedated and intubated. Still, the best motor response can be an important indicator for neurological deterioration. Whenever possible sedation should be withhold for neurological assessments in a scheduled fashion, the only exception to this being patients with critically increased intracranial pressure (ICP). Any decrease in the GCS by two or more points is clinically significant and additional diagnostic tests, e.g. computed tomography (CT) scanning, should be undertaken to rule out developing intracranial pathology. Additionally, careful examination of the pupillary light response, width of the pupils and caudal cranial nerve functioning is substantial and may be used to confirm ICP measurements or question artificial readings. Focal neurological deficits, like hemiparesis mandate diagnostic work-up including CT.

Monitoring

In monitoring critically ill patients, basic monitoring has to be differentiated from extended neuromonitoring. A combination derived from basic and extended neuromonitoring is the so called multimodal (or multiparametric) cerebral monitoring.

Basic Monitoring

Basic monitoring of common physiological parameters is mandatory for all patients with a GCS of eight or less, especially those with increased ICP. 4 Basic monitoring is not different from the monitoring applied in other critical care patients. Discontinuous blood pressure monitoring may be sufficient in mild or moderate HI, whereas severe HI and other neurologically critical patients require continuous, automatic blood pressure measurements to ensure stability in the mean arterial pressure (MAP) (and thus cerebral perfusion pressure (CPP)). For this pressure transducers need to be leveled with the ICP measurement. Conventionally, the external acoustic meatus is chosen as a reference. Arterial lines, nowadays standard in many ICUs, have low procedural and infectious risks and are furthermore helpful for routine blood gas analyses. Additionally, continuous monitoring of the arterial oxygen saturation curve is fairly simple and cheap. Both blood gas analyses and saturation monitoring complement each other and have become conditio sine qua non in modern intensive care medicine. According to the recommendations of the European Brain Injury Consortiums (EBIC) the following arterial blood gas parameters should be maintained: arterial saturation of oxygen >95%, arterial partial pressure of oxygen (pa O2) >100 mmHg, arterial partial pressure of carbon dioxide (pC O2) >35 mmHg.4 The other parameters that need to be monitored are temperature, sodium levels, hemoglobin levels, glucose levels, coagulation status (discussed below).[5,6,7,8]

Cerebral Monitoring (ICP/CPP)

Monitoring and treatment of ICP is meant to reduce the number and severity of secondary ischemic injuries to the brain. Intracranial hypertension can only be treated adequately with consequent measurement of the ICP. In severe HI, intracranial hemorrhages, contusions, pericontusional edema or global brain edema can be the cause for increased ICP and reduced CPP. ICP should be monitored whenever these lesions are present, especially in conjunction with compressed basal cisterns. In unconscious patients with severe HI and a normal CT, the risk for intracranial hypertension is increased if at least two of the following items apply: [9]

- Age 40 and above;
- Flexor or extensor posturing;
- Systolic blood pressure less than 90 mmHg.

ICP monitoring is not indicated in patients with mild or moderate HI where the neurological exam can be followed (i.e. GCS ≥9). In the presence of massive cerebral contusions there is always a risk of sudden deterioration, so that in patients who are "on the edge," the indication for placement of an ICP monitor needs to be assessed individually. The hydrostatic pressure derived from the frontal horn of the lateral ventricle via an external ventricular drainage (EVD) is the "gold standard" for ICP measurement. By definition ICP is referenced to the level of the Foramen of Monro. This is in line with the external acoustic meatus, which serves as a skin landmark when leveling pressure transducers. An argument in favor of a ventriculostomy is the therapeutic value of cerebrospinal fluid (CSF) drainage, while the risks of infection and hemorrhage have to be contemplated. Direct measurement by tip transducers placed in the brain parenchyma has a significantly lower morbidity, but the system cannot be recalibrated after implantation and is prone to drift and technical error. Epidural measurements of the ICP depend on dural tension and are technically inferior to the aforementioned systems. Especially overestimations of ICP have been found; hence epidural ICP monitors are used less nowadays.[10] The threshold for intervention is 20 mmHg since the neurological outcome of HI patients seems to be affected the more the longer the ICP stays above this level.[11] It has to be underscored, however, that an ICP of 15–20 mmHg is already pathologically elevated.

It has been known for long that with CPP decreasing below the threshold for autoregulation, cerebral perfusion is diminished and finally ceases. The crucial question is, where this pressure threshold exactly is in individual patients. It is recommended to maintain the CPP above 60 mmHg for adequate cerebral perfusion and oxygenation.[12]

Many arguments have been raised based on clinical and experimental findings regarding the critical question of whether ICP or CPP should be followed. Current opinion is that both are important. Both therapy regimens are not diametric to each other since they have in common that ICP is treated whenever >20 mmHg and CPP is maintained >60 mmHg. In conclusion, the European Brain Injury Consortium (EBIC) and the American Association of Neurological Surgeons (AANS) have suggested the following thresholds for intervention: ICP 20 mmHg, and CPP 60 mmHg.4,11,12 To maintain a CPP of 60 mmHg the ICP should be kept lower than 20 mmHg by conservative treatment measures. If this fails the use of catecholamines should be considered. In the AANS guidelines, the prophylactic elevation of the CPP beyond these targets, however, has been discouraged. [11,12]

Extended Cerebral Monitoring (Oxygenation, Metabolism, Cerebral Blood Flow)

As mentioned earlier, ICP/CPP monitoring yields surrogate parameters to avoid deterioration of cerebral perfusion in comatose patients. Normal or moderately increased ICPs, however, are only indirect indicators of sufficient perfusion, whereas a high ICP is not necessarily a herald of pending ischemia. In the early 90's high expectations had been raised by the emerging field of (direct) invasive cerebral oximetry. Here a sensor is placed in a representative region of the brain (e.g. tissue oximetry, pti O2), in the jugular-venous return from the cranium (jugular bulb oximetry, Sjv O2), or (non-invasively) on the skin and adjacent to the region of interest (near-infrared spectroscopy, NIRS). A clear distinction has to be made between global and local measurements. Both Sjv O2 and pti O2 are sensitive parameters for the risk of an unfavorable outcome, if desaturations (<50%) or critical decreases in the partial pressure of oxygen occur frequently (<10 mmHg). Both measurements allow for a more individualized (targeted) ICP/CPP therapy.

In the last two decades the so-called bedside- or onlinemicrodialysis has been introduced in neurocritical care. This method is based on a double-lumen microcatheter with a semipermeable membrane in the cerebral parenchyma. Neurochemical analyses of the cerebral extracellular space can be performed using an automated photometer. Within minutes information about concentrations of glucose, lactate, pyruvate, glutamate and glycerol in the brain becomes available to the clinical team and can be used for therapeutic decisions.[13]

It has become obvious that monitoring involves more than one parameter for clinical decision making. Multiple parameters have to be interpreted in context and their interrelations have to be understood. This is the basis for the so called multiparametric or multimodal cerebral monitoring approach, where all parameters are processed and recorded. Clinical decisions are driven by direct and/or post-hoc interpretations of a given subset of parameters chosen by the neurointensivist.

Maintenance of Normal Physiological Balance

Critical care of neurosurgical patients aims at maintaining normal physiological balance, i.e. normovolemia, normotension, normothermia and normoglycemia.

Normovolemia

Normovolemia is guided by the clinical assessment (peripheral edema, pulmonary congestion and urine output).

Both hypo- and hypervolemia should be avoided. Substitutes for blood volume are packed red blood cells, colloid solutions like albumin as well as isotonic crystalloids. The use of hypertonic saline has been propagated, especially for fluid resuscitation of patients in shock. Currently experimental and clinical data do not suggest any harm to the injured cerebrum if administered acutely. Hypertonic saline as a second-tier therapy for refractory intracranial hypertension has been recommended as an option by the American Brain Foundation.[12]

Temperature Control

With the renaissance of hypothermic treatment, monitoring of core temperature has been intensified in NICUs. It is well known that hyperthermia aggravates brain injury by increasing energy metabolism and demand.[5] Therefore hyperthermia ought to be treated aggressively in all patients with cerebral lesions. While transient controlled hypothermia decreases cerebral metabolism and ICP, prophylactic and longer lasting use of hypothermia is discussed controversially. In the US American National acute brain injury study: hypothermia, severe HI patients were randomized to moderate hypothermia (33o C) for 48 hours or normothermia. No effect on mortality and neurologic outcome was observed. Systemic complications (coagulopathy, pneumonia, sepsis) even led to a worse outcome in the subgroup of patients aged 45 years or older.[14,15] Fever caused by infections has to be treated promptly (physical, pharmacological, and causal treatment, i.e. antibiotics). The most common nosocomial infection in neurosurgical critical care patients is pneumonia. Its incidence has been progressively lowered during the last years, probably since corticosteroid treatment for HI is no longer favored and because general intensive care is qualitatively better today. Traditional methods of fever treatment such as cooling blankets are not that effective. Newer cooling techniques such as surface cooling and catheter-based heat-exchange systems have been found to be more beneficial than traditional cooling methods. Empiric, calculated or targeted antibiotic treatment is indicated based on the degree of suspicion or proof of infection.

Normoglycemia and Nutritional Balance

Several studies have shown that hyperglycemia is associated with significantly worse clinical outcomes.[6,7] Consequently, normoglycemia is the goal in all neurologically critical patients. Hyperglycemia is observed in neuro-critical patients especially in those receiving steroids. If necessary, insulin is administered to maintain serum glucose at 100–200 mg/dl. Nutritional balance has to be kept in order to respond to the altered requirements of post-injury metabolism. Energy requirements and requirements of other nutritional supplies should be measured and monitored or calculated if possible.

Sodium and Hemoglobin Balance

Since both hypo- and hypernatremia increase the risk of edematous brain swelling, prevention or cautious normalization needs to be undertaken. Similarly, hemoglobin concentrations should be maintained above 10 g/dl in all severe HI patients to ensure adequate tissue oxygenation.

Coagulation Status

Bleeding and hypocoagulable states are not infrequent seen in HI and may contribute to enlarging hemorrhagic contusions as well as traumatic intracerebral hematomas.[8] Hence, monitoring and stabilization of coagulation parameters are of paramount objectives.

Sedation/Analgesia

Adequate analgosedation is necessary to avoid stress, pain and fear in patients who are often intubated and ventilated for several days. Sedation also efficiently reduces cerebral metabolism, cerebral blood volume, and therefore supports ICP treatment. On the other hand, the need for neurological assessments requires to minimize sedation as much as possible. Commonly, a combination of a benzodiazepine (e.g. midazolam 0,09 mg/ kg/h) and an opioid (e.g. fentanyl 0,0012 mg/kg/h) is used. Individual variations exist and increased ICP eventually makes a higher sedation level desirable.

Treatment of Intracranial Hypertension

In the following discussion specific treatment of critical patients with head injuries, often with an increased ICP will be discussed. But the principle applies well in other neurosurgical conditions e.g. subarachnoid hemorrhage (SAH) and spontaneous parenchymal hematoma too. In Table 1 principal treatment options, mechanisms and pros and cons of this treatment principle are summarized.

Table 1: Table showing treatment options for intracranial hypertension; cerebral blood flow (CBF), cerebral blood volume (CBV), cerebral perfusion pressure (CPP), cerebrospinal fluid (CSF), electroencephalography (EEG), mean arterial pressure (MAP).

Therapy	Mechanism	Pros	Cons
Head elevation	venous drainage↑, CBF↑ intracranial volume↓	simple, efficient	CPP↓
CSF drainage	vasoconstriciton	simple, efficient	invasive, risk of infection↑
Hyperventilation *Moderate (pCO2 =30mmHg* *Forced (pCO2 <30mmHg)*	CBV↓, CBF↓ osmotic gradient, "dehydration", improved rheology	simple, efficient simple	risk of cerebral ischemia↑ nephrotoxicity (keep plasma osmolarity <320 mosmol)
Osmodiuretics (mannitol)	metabolism ↓, CBF↓, CBV↓ intracranial space↓		EEG monitoring necessary, risk of pulmonary infection, MAP↓ risk of operation
Barbiturates	metabolism ↓, CBF↓, CBV↓		technically difficult, systemic side effects
Surgical decompression			
Hypothermia			

Positioning

Both ICP and CPP are significantly affected by positioning of the head. Venous drainage can be reduced if the head is turned sideways, the neck is extended or flexed, and jugular veins get compressed. Tight dressings, cervical collars, cervical hematoma, subcutaneous emphysema, and high positive end expiratory pressures are to be avoided. Elevation of the head and upper torso has been discussed controversially in the past. The dogma that severe head injury patients have to be positioned in elevation has been disputed by various investigations showing a increased ICP, but constant CPP, CBF and oxygenation in patients with resting flat.[14] Mild elevation by 15-30o results in an improvement of cerebrovenous return and ICP while CPP and cerebral oxygenation remain constant.[17]

Ventricular Drainage

As previously discussed ventricular drainage systems can be of use for ICP monitoring and therapeutic CSF drainage. Reducing the partial volume of the intracranial CSF spaces compensates for vasocongestion and brain edema. An EVD is a very simple, yet efficient, measure in the treatment of intracranial hypertension though the risk of bacterial contamination and meningitis has to be taken into consideration. In principle there are two regimens of CSF drainage: continuous drainage into an external container leveled at a fixed height above the external acoustic meatus, or intermittent drainage at fixed time intervals or whenever a threshold ICP has been reached. Several series have shown that external CSF drainage enables reduction in the intensity of other ICP treatment options (i.e. mannitol, hyperventilation).[18]

Hyperventilation

Cerebral vasoconstriction is the physiological mechanism of hyperventilation on ICP. Moderate and forced hyperventilation have to be differentiated (pCO2 30–35 mmHg and pCO2 <30 mmHg, respectively). Hyperventilation diminishes ICP rapidly. On the other hand undue vasoconstriction increases the risk of secondary ischemic injuries. In a controlled clinical trial, the clinical outcome of patients with severe HI was adversely affected by prolonged forced hyperventilation (pCO2 ˜ 25 mmHg). [19] In clinical practice, prophylactic hyperventilation has

therefore been abandoned. Transient hyperventilation, although efficiently reducing the ICP and improving CPP, is associated with a decrease in cerebral oxygenation. Critically impaired oxygenation, however, occurs with forced hyperventilation only.

Currently it is recommended to use hyperventilation transiently for instances of acute neurological deterioration whenever intracranial hypertension is refractory to sedation, CSF drainage and osmodiuretic treatment.[11,12] Forced hyperventilation should only be used if continuous surveillance of cerebral oxygenation is feasible.

Osmodiuretics

ICP can be efficiently lowered with osmodiuretics. Typically mannitol (20%) is administered intravenously. In adults approximately 300 ml (i.e. 1.0 g/kg) are infused as a bolus over 10–20 minutes. If a more profound reduction of ICP is necessary, e.g. intraoperatively, up to 250 ml can be given. Higher doses have been suggested for pre-operative use whenever patients have clinical signs of impending herniation and some time is required for Operation Room transfer.[20] Future investigations will show if hypertonic saline could be a potential substitute if osmodiuretics are contraindicated because of hypovolemia and/or hypotension. Routine use in patients with increased ICP is either intermittently scheduled (e.g. every eight hours) or whenever ICP reaches a defined threshold. Most clinicians prefer the former mode, where boluses are scheduled every 8, 6, 4 or even 2 hours. With repeated boluses, mannitol can cumulate and therefore plasma osmolality has to be checked before continuing osmotherapy. Plasma osmolalities beyond 320 mosmol/l carry the risk of nephrotoxicity and acute tubular necrosis.

There are at least two mechanisms which are complementary in the use of mannitol: increasing the osmolar gradient of plasma and cerebral interstitium transiently leads to an unspecific "dehydration" of well-perfused tissue. Additionally hemorheology is improved acutely leading to enhanced cerebral perfusion.[21] Rebound phenomena have been postulated to occur in patients with breakdown of the blood brain barrier (BBB). Mannitol is thought to transit into the cerebral parenchyma and bind water locally. Therefore accumulation over time could theoretically lead to increased ICP. Both clinical and experimental experience, however, demonstrates that patients with contusions (and proven BBB breakdown) can be treated with mannitol over several days without adverse effects on ICP.[22] With prolonged osmodiuretic treatment strict charting of fluid intake and output is necessary.

Barbiturates

Barbiturate coma is known to effectively lower cerebral metabolism which is coupled with cerebral perfusion and consecutively with decreased cerebral blood volume. Therefore ICP is affected beneficially. Further (wanted) properties are its anticonvulsive activity, reduced liberation of free oxygen radicals, inhibition of lysosomal enzymes, and moderate reduction of the core temperature.[23] Unwanted side effects are due to systemic vasodilation (hypotension), reduction of leukocyte counts (infections. Hepatotoxicity can complicate treatment and, in the worst case, lead to acute liver failure. To avoid overdosage barbiturate coma should be monitored by electroencephalography (EEG) where a "burst suppression pattern" is contemplated.

To predict the effect of barbiturate therapy individually, a test dose of 5 mg/kg thiopental should be infused over 30 minutes. Continued barbiturate coma is not reasonable if CPP decreases. A maintenance dose of 5 mg/kg/h is recommended, but therapeutic serum levels vary individually. Therefore continuous EEG monitoring is necessary to titrate exactly to the therapeutic goal where the effect of thiopental on cerebral metabolism is maximal (i.e. burst suppression). According to the AANS guidelines barbiturate coma is a "second-tier" therapy that should be used after CSF drainage, moderate hyperventilation and osmotherapy.[11,12]

Catecholamines

Catecholamines are used in the core concept of CPP management.[24] The treatment of intracranial hypertension should aim at reducing ICP through measures like moderate hyperventilation, CSF drainage and osmodiuretics. If ICP remains high despite these measures, catecholamines can be introduced under continuous monitoring of ICP, MAP and CPP. Normovolemia should be maintained and kidney function monitored thoroughly when patients are receiving catecholamines.

Decompressive Craniectomy

Decompressive craniectomy with duraplasty has seen a renaissance in neurosurgery over the last two decades. In earlier times epidural and subdural hematomas where removed without reimplanting bone flaps acutely. This procedure (even with extended size and with duraplasty) has not yielded better clinical results compared to acute reimplantation.

Decompressive craniectomy nowadays means a secondary intervention if conservative treatment of posttraumatic brain swelling fails and intracranial hypertension necessitates "second-tier" treatment.[11,12] Surgical decompression can

be carried out uni- or bilaterally, fronto-temporo-parietally or bifrontally. Especially young patients without primary papillary dysfunction and brainstem injuries seem to benefit most from this treatment. A prospective study has shown a favorable outcome in 58% of all patients in this subgroup.[25] In patients above the age of 50, decompressive hemicraniectomy does not improve outcome significantly.[25] Recent randomized trial (DECRA trial) and a meta-analysis, however, failed to show any long term benefit though the procedure could effectively decrease ICP and increase CPP.[26,27]

Based on aforementioned measures, treatment pathways for intracranial hypertension have been developed. These are oriented on treatment thresholds for ICP/CPP of 20 and 60 mmHg respectively. First line measures (sedation, positioning, ventriculostomy, osmotherapy, moderate hyperventilation) have to be differentiated from so-called "second-tier" therapies (e.g. barbiturate coma, decompressive craniectomy). Cerebral oxygenation measurements (Sjv O2 or pti O2) have not become clinical standard yet, but are recommended for use with forced hyperventilation (a second-tier therapy).

Pharmacotherapy:

Glucocorticosteroids

In the 60s of the last century glucocorticosteroids were introduced in the treatment of peritumoral brain edema. The mechanisms are manifold: vascular permeability is reduced in and surrounding brain tumors, CSF production is down-regulated, generation of free radicals and calcium influx into neurons and glia are reduced. All these mechanisms could potentially be neuroprotective in traumatic lesions as well. In a number of initial experimental studies beneficial effects of steroids following trauma were demonstrated. Trials to reproduce these effects clinically have failed, however.[28] The most recent international study (CRASH, "Corticosteroid Randomization After Significant Head injury") was stopped after more than 10 thousand patient enrollments. High dose methylprednisolone treatment over 48 hours had led to a significantly higher mortality at two weeks following trauma in the treatment arm.[29] In summary, the use of steroids is not recommended for the treatment of patients following TBI.[11,12]

Calcium Antagonists

The mechanism of nimodipine, which has been the focus of calcium antagonist studies, is a reduction of calcium influx in neurons injured by hypoxia or ischemia. Relaxation of smooth muscle cells and inhibition of vasospasm has been hypothesized as a mechanism of action in SAH, but neither does it prevent or ameliorate vasospastic vessel narrowing in itself nor does it improve CBF. Though data for supporting its use in spontaneous SAH is positive, the same in head injury is more complex, however.[30] Studies have failed to prove an effect of nimodipine in patients with SHI. Accordingly the general use of calcium antagonist in severe HI patients is not recommended.[31] Yet, subgroup analyses have indicated a positive effect in patients with traumatic SAH. [31] In summary, calcium antagonists like nimodipine might have an indication in the treatment of patients with TBI and traumatic SAH. More comprehensive trials will be necessary for a clear recommendation, however.

Anticonvulsants

Seizures affect a high percentage of critically ill neurosurgical patients especially those with focal lesions (i.e. contusions, tumors). The incidence for trauma has been reported between 5 and 40%, reflecting uncertainties in the diagnosis. Early posttraumatic seizures (within 7 days after trauma) have to be differentiated from late posttraumatic seizures. Anticonvulsants like phenytoin and carbamazepine do reduce the incidence of early posttraumatic seizures significantly when given prophylactically. The occurrence of late posttraumatic seizures and the clinical outcome are unaffected, however.[12,32] Therefore prophylactic treatment is generally not recommended (standard), but, according to the US-American guidelines, can be used optionally to prevent early post-traumatic seizures in patients at high risk (e.g. with cortical lesions). It has to be underscored that these recommendations apply to prophylactic use, while anticonvulsant treatment should certainly be initiated with proven occurrence of posttraumatic seizures.[32]

Conclusions

Critical care is a rapidly emerging field in neurology and neurosurgery. However, it is difficult for Nepalese healthcare to adopt these contemporary management strategies immediately. Though the number of clinicians interested in neurology and neurosurgery is growing yearly, there is not a person to be qualified as a neurointensivist in the country yet. So the clinicians should update themselves in neurocritical care to make any meaningful and recognizable improvement in the overall neurological care in the national perspective.

The following recommendations can be made to improve the situation:

- Establishment of regional trauma centers with basic 3-6 months condensed and intensive training of neurosurgeons and neurologists in critical care. Simple, yet efficient, treatment options like ventricular drainage

should be made possible at every trauma hospital.

- Prioritizing the neurocritical care from the existing health institutions from the country. Timely intervention of people with neurotrauma is frequently delayed even in the tertiary care facility due to the lack of adequate operating room (OR) time. Epidural hematomas occasionally have to compete with, for example, long bone fractures or obstructed hernias as only one emergency OR is available
- ICP/ CPP-directed treatment for increased ICP should be instituted. As far as possible protocols should be followed so that there is uniformity in managing and reporting the outcome among various centers in the country.

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