The role of neurosciences intensive care in neurological conditions

The neurosciences intensive care unit provides specialized medical and nursing care to both the neurosurgical and neurological patient. This second of two articles describes the role it plays in the management of patients with neurological conditions.

Neurosciences intensive care is a rapidly evolving specialty. Historically the majority of patients cared for on neurosciences intensive care units have been postoperative neurosurgical cases. Increasingly there is the perception that patients with primary and secondary neurosurgical or neurological conditions will benefit from the care of a dedicated specialized intensivist team. An audit has demonstrated that over one-third of patients on neurosciences intensive care units have a neurological diagnosis that has resulted in their admission, while the remaining two-thirds are neurosurgical in nature (M Smith, personal communication, 2006).

This article will discuss the commonest neurological conditions presenting to neurosciences intensive care unit (*Table 1*). They can be divided into those involving the central or peripheral nervous system and those related to a neurological complication secondary to an alternative primary diagnosis, i.e. intensive care unit-acquired weakness and critical illness neuropathy. Space dictates that disorders in which the major management decisions take place outside the intensive care unit (e.g. thrombolysis for acute stroke) are excluded, and conditions familiar to other specialities will not be discussed even though they can require intensive care unit treatment in severe cases (e.g. infectious diseases of the CNS).

Table 1. Neurological conditions managed in the neurological intensive care unit

Central nervous system	Cerebrovascular disease	Malignant middle cerebral artery stroke
		Posterior fossa stroke
	Cerebral venous sinus thrombosis	
	Recurrent seizures or status epilepticus	
Peripheral nervous system	Guillain–Barré syndrome	
	Myaesthenia gravis	
	Intensive care acquired weakness	
	Critical illness neuropathy	

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Central nervous system admissions

Cerebrovascular disease

Patients with neurovascular conditions have limited potential for recovery if there has been extensive neuronal loss. However, specialist care is justified in certain cases as a number of specific treatment regimens have evolved which improve outcome (Kiphuth et al, 2010; Stroke Unit Trialists' Collaboration, 2013).

Malignant middle cerebral artery stroke

Up to 10% of infarcts in the anterior part of the circle of Willis, particularly those caused by proximal occlusion of the middle cerebral artery, can develop life-threatening focal swelling, hence the term malignant (Hacke et al, 1996). In most cases, this occurs within 24 hours of onset of the stroke, but some cases, possibly as a result of conversion of ischaemic penumbra to infarction, may take up to 72 hours. Malignant middle cerebral artery occlusion is fatal in over 80% of cases as a result of uncal herniation and brainstem compression (Berrouschot et al, 1998). Patients with a diminished level of consciousness, pronounced motor deficit and impairment of language function (i.e. high National Institutes of Health Stroke Scale score; ≥20 involving the left hemisphere or ≥15 in the right hemisphere out of a total of 40 points) are particularly at risk (Glymour et al, 2007). Early nausea and vomiting, obtundation or other clinical signs of raised intracranial pressure and early computed tomography hypodensity of over 50% within the middle cerebral artery territory can be predictive of the development of malignant middle cerebral artery stroke (Krieger et al, 1999).

Patients at risk of developing malignant middle cerebral artery infarction require aggressive medical treatment and should be referred to a neurosciences centre for monitoring and consideration for neurosurgical intervention. Management includes close neuro-observations and prevention of secondary insults. Medical therapies alone have not proven effective in its management (Hofmeijer

et al, 2003). Therapeutic hypothermia reduces mortality (Schwab et al, 2001) and remains the subject of clinical trials (www.eurohyp1.eu). Decompressive hemicraniectomy in three randomized controlled trials (DECIMAL, DESTINY and HAMLET) has been recognized as an effective treatment (Vahedi et al, 2007). Taken together these three studies demonstrate that early decompressive craniectomy in young patients (<50 years) reduces mortality with a number needed to treat of four in the prevention of severe neurological deficit (Rankin scale of 3 or less) (Vahedi et al, 2007). Although the survival rate is also improved in older patients, the benefit regarding functional outcome is not clear. What is clear is that decompression within 48 hours is critical in the management of these patients, and early admission to neurosciences intensive care unit facilitates the 'work-up' and management of these cases before surgery (Bershad et al, 2008).

Posterior fossa stroke

Cerebellar strokes represent 20% of all strokes, with patients presenting with dizziness, diplopia, dysarthria, dysphagia, dystaxia and prominent headache. Rapid deterioration is common and caused by compression of the brainstem as a result of mass effect, worsening hydrocephalus and/or brainstem infarction. The clinical course of these patients is unpredictable and it is recognized that their management in the first week is best carried out in a neurosciences intensive care unit where continuous monitoring will rapidly identify neurological deterioration (Jensen and St Louis, 2005).

Cerebral venous sinus thrombosis

Cerebral venous sinus thromboses constitute approximately 1% of strokes. The diagnosis of cerebral venous sinus thromboses has been greatly simplified by the use of magnetic resonance imaging and computed tomography venography. The vast majority of cases are caucasian females who present with headache and in whom imaging demonstrates thrombosis within the superior sagittal sinus. The majority will make an uneventful recovery following prompt anticoagulation (Ferro et al, 2004). Nevertheless, fulminant venous thrombosis may cause life-threatening intracranial hypertension, infarction and secondary haemorrhage. Up to 19% of cases may present with stupor or coma on admission. The mortality rate of these cases may be up to 50% (Mehraein et al, 2003; Ferro et al, 2004). A statement by the American Stroke Association has highlighted the need for organized high quality care given by a multidisciplinary team using protocols in the management of cerebral venous sinus thrombosis (Saposnik et al, 2011).

The European Federation of Neurological Sciences task force (Einhaupl et al, 2006) has developed a therapeutic management plan. Their recommendations are that patients should be anticoagulated irrespective of concomitant intracranial haemorrhage, as intracerebral haemorrhage is a likely secondary consequence of venous outflow obstruction. This obviously carries risk and warrants expert guidance. In cases where surgery or repeated lumbar punctures for raised intracranial pressure might be required intravenous heparin is preferred. When no immediate surgical intervention is indicated low molecular weight heparin is recommended. In complex cases the development of raised intracranial pressure should in the first instance be managed on a neurosciences intensive care unit while assessment is made regarding surgical or radiological intervention. In those patients with visual deterioration secondary to intracranial hypertension serial lumbar punctures are an option and anticoagulation should be withheld for 24 hours after the last lumbar puncture. Clinicians need to be wary when managing these patients as complications can develop rapidly. Prompt referral of complex cases to a specialist unit can improve outcomes and reduce morbidity and mortality (Saposnik et al, 2009).

Recurrent seizures and status epilepticus

Status epilepticus is continuous seizure activity lasting longer than 5 minutes (Lowenstein et al, 1999). Between 30 and 50% of these cases are resistant to first-line anticonvulsants and are termed refractory (Treiman et al, 1998). Status epilepticus is a common neurological emergency which has significant morbidity and mortality. Of those patients admitted to the neurosciences intensive care unit for management of status epilepticus over one-third are refractory to first and second-line anticonvulsant medication (Treiman et al, 1998; Mayer et al, 2002; Holtkamp et al, 2005).

Encephalitis is the commonest reason for refractory status epilepticus. Less frequently refractory seizures result from inadequate levels of anticonvulsant medication (Holtkamp et al, 2005). Of those cases that fail to respond to two anticonvulsant agents, 42% will fail to respond to a third agent. The identification of underlying medical conditions, adequate anticonvulsant therapy and management in a neurosciences intensive care unit can reduce the likelihood of status epilepticus becoming refractory status epilepticus.

Clear guidelines for the management of status epilepticus have been formulated (Shorvon, 2011) (*Figure 1*). The initial treatment for status epilepticus in the prehospital stage is benzodiazepines (typically 10 mg buccal midazolam). Lorazepam 0.1 mg/kg over <5 minutes is a common first choice in early status epilepticus in hospital. Lack of response after 5 minutes is defined as failure, and higher doses do not help. The established phase of status epilepticus requires intravenous anticonvulsants, traditionally phenytoin or fosphenytoin, but levetiracetam at doses of 1–3 g or high doses of sodium valproate can be effective. If seizure control is not achieved within 60–90 minutes, continuous intravenous treatment, intubation and intensive care unit admission is critical.

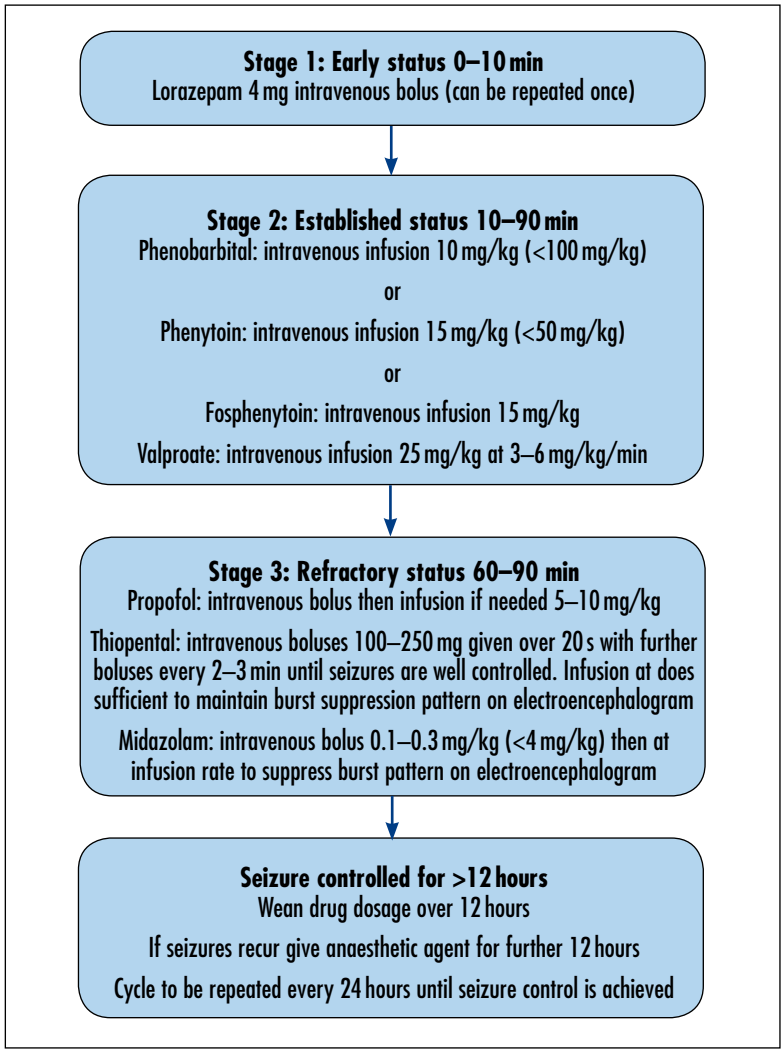

Figure 1. Management of status epilepticus (modified from www.neuroicu.org.uk).

Treatment options include high dose benzodiazepines or a barbiturate, with a loading dose followed by a continuous infusion until clinical and electroencephalographic seizure activity ceases. Electroencephalographic monitoring is needed to establish whether seizures are truly suppressed; cessation of clinical seizure activity is not reliable. This normally means a flat electroencephalograph, because the 'burst-suppression' pattern often advised in the literature is not easy to maintain, and bursts are frequently difficult to differentiate from residual seizure activity. Intravenous medication must be continued for 24 hours after seizure control, then tapered gradually under long-term anticonvulsant cover to avoid recurrent status epilepticus.

The duration of status epilepticus should not be a limitation to continued care, with good outcomes documented in cases persisting for more than a month (Shorvon, 2011; Brophy et al, 2012). The complications associated with status epilepticus and refractory status epilepticus are the primary determinants of mortality. Hypotension is common in refractory status epilepticus and complicates management with inability to tolerate high doses of anticonvulsants. Active fluid management and the use of vasoactive agents in neuroscience intensive care units can improve anticonvulsant effectiveness. Patients with prolonged refractory status epilepticus may also suffer rhabdomyolysis and hyperthermia which are best managed in the intensive care setting.

More recently the issue of electroencephalographic monitoring in both non-convulsive status epilepticus and refractory status epilepticus has been addressed in a consensus statement from the neurointensive care section of the European Society of Intensive Care Medicine. This recommends continuous over intermittent electroencephalographic monitoring for refractory status epilepticus and also for comatose patients with unexplained and permanently altered consciousness (Riviello et al, 2013).

Peripheral nervous system admissions

Guillain–Barré syndrome

Guillain–Barré syndrome has an incidence of 10–20 per million per year. Over 50% of cases follow a prodromal respiratory (e.g. *Mycoplasma pneumoniae*) or gastrointestinal (e.g. *Campylobacter jejuni*) infection. Distal paraesthesiae, back pain, ascending weakness and loss of reflexes are often observed. The average nadir of weakness is around day 10. About 30% of all cases become bed-bound and, of these, one-third will need intubation as a result of a combination of bulbar and respiratory muscle weakness. Of those requiring intubation, up to 50% will be ventilator-dependent for at least 3 weeks. Additionally, autonomic dysfunction may occur in up to 60% and can cause orthostatic hypotension, diabetes insipidus, ileus or cardiac dysrhythmmias. Overall mortality is 5–10%, predominantly as a result of infection or cardiac arrhythmias.

Critical care management in patients with Guillain–Barré syndrome starts outside the intensive care setting. Close surveillance of vital capacity and inspiratory and expiratory mouth pressures is essential. The treating team should apply the 20/30/40 rule (Hughes, 1998; Lawn et al, 2001) to assess the need for intubation (vital capacity below 20 ml/kg, peak inspiratory pressure below 30 cmH_2O, and peak expiratory pressure below 40 cmH_2O) with a defined protocol in situ as to when to involve the neurosciences intensive care unit (*Figure 2*) (Wijdicks and Roy, 2006). It must be noted that these are respiratory threshold values that indicate that progression to respiratory failure is likely and as such indicate the need for pre-emptive measures such as admission to the neurosciences intensive care unit (Hughes, 1998). Additionally, electrocardiographic monitoring is needed in bedbound patients, with cardiac pacing readily available in case of symptomatic bradycardia. The threshold for elective intubation of patients with respiratory decline should be low. Patients should be warned of the likelihood of tracheostomy before ventilation is started. The evidence base for intravenous immunoglobulin therapy and plasma exchange in the management of Guillain–

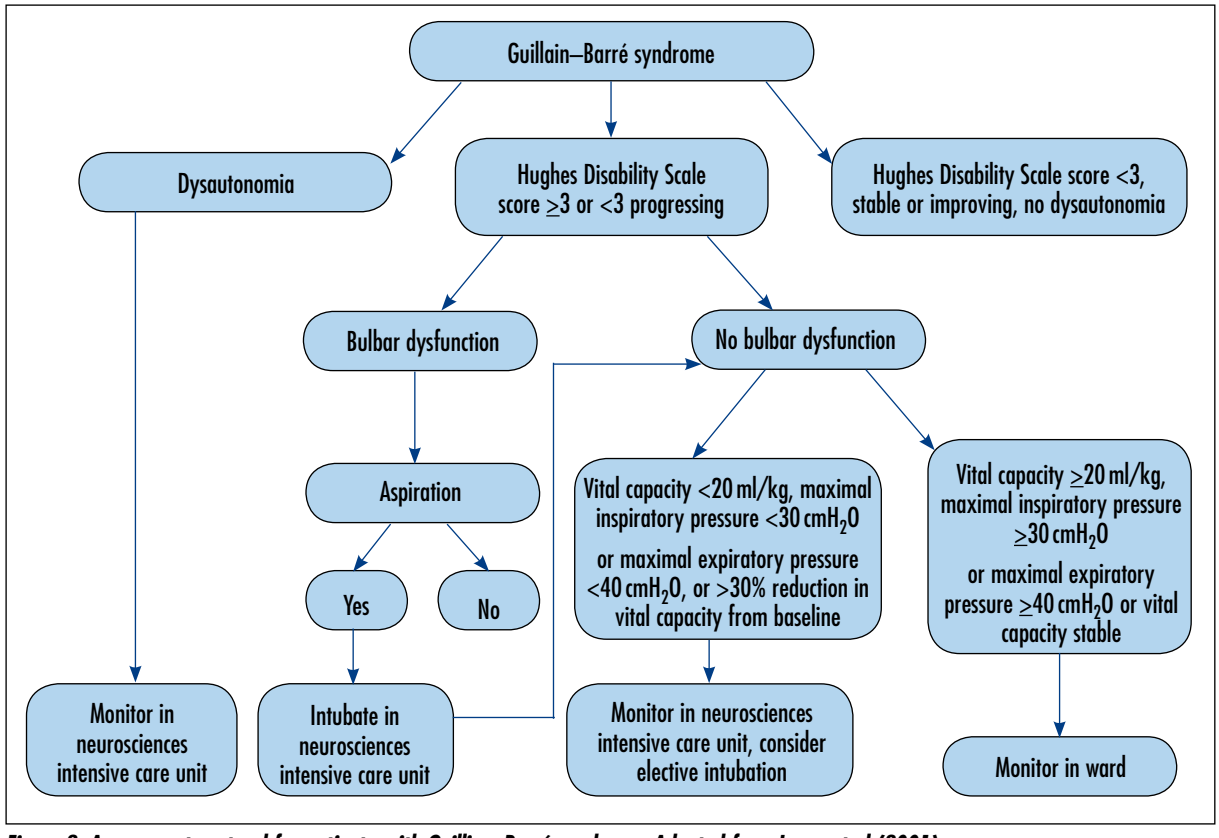

Figure 2. Assessment protocol for patients with Guillian–Barré syndrome. Adapted from Lawn et al (2001).

Barré syndrome is well established with delivery best within the neurosciences intensive care unit (van der Meché and Schmitz, 1992).

Myasthenia gravis

Myasthenia gravis is an autoimmune condition of the neuromuscular junction; most cases are caused by T-cell derived antibodies to the nicotinic acetylcholine receptor at the postsynaptic motor endplate. Seronegative patients (without acetylcholine receptor antibodies) suffer from myasthenia caused by an antibody against a muscle-specific kinase. The cardinal symptom of myasthenia is fatiguability following sustained exertion. It often initially affects extraocular muscles, but becomes generalized in 85% of cases. Treatment consists of the cholinesterase inhibitor pyridostigmine, which provides symptomatic relief. In most cases, immunosuppressive treatment (initially incremental corticosteroids, later a steroid-sparing agent such as azathioprine) is required for sustained benefit.

Neurological intensive care teams become involved when bulbar or respiratory weakness affects respiratory function, the 'myasthenic crisis', which is commonly precipitated by infections or inappropriate medications. More rarely a second type of crisis occurs as a result of excessive medication. This typically presents with cholinergic features such as miosis, bradycardia, increased bronchial secretions, cramps, fasciculations and diarrhoea. The initial challenge is to recognize the impending myasthenic crisis early. Close monitoring of fatiguability, respiratory function, cough, swallowing, peripheral oxygen saturations and alveolar–arterial gradient is required. Treatment principles on the neurosciences intensive care unit include identification of potential sources of sepsis (a common precipitant of crises), rehydration, and cessation of inappropriate medication (e.g. timolol, verapamil and quinine). Initially, pyridostigmine is often discontinued, then gradually reintroduced. Immunomodulatory treatment with intravenous immunoglobulin or plasma exchange are used. There is limited evidence for the use of high-dose steroids (Schneider-Gold et al, 2005). In refractory cases, more profound immunosuppression (e.g. rituximab) may be necessary. Intensive respiratory therapy and initial non-invasive ventilation may reduce ventilator days (Seneviratne et al, 2008). Often only a short period of ventilation is needed, meaning that tracheostomy can be avoided. However, there is a relatively high risk of reintubation, especially in patients with atelectasis, and standard predictors of extubation success may not apply.

Intensive care unit-acquired weakness and critical illness neuromyopathy

Critical illness neuromyopathy and polyneuropathy are frequent complications in critically ill patients receiving long-term ventilation, presenting as neuromuscular weakness and sensory loss (Bolton et al, 1986; Faragher et al, 1996; Hermans et al, 2008). Symmetric flaccid

paralysis of the limbs, often sparing facial muscles, is noted in patients who have a combination of critical illness polyneuropathy and critical illness neuromyopathy. Primarily motor and sensory axons are affected, with abnormal sensory nerve action potentials typical for critical illness polyneuropathy and reduced response to direct muscle stimulation in critical illness neuromyopathy (Bolton et al, 1986; Faragher et al, 1996). The two entities are often difficult to separate by routine electrophysiological examination and there is considerable overlap (Latronico et al, 1996; Bednarik et al, 2003; Hermans et al, 2008). A series of 33 Spanish patients (Fernandez-Lorente et al, 2010) demonstrated myopathic findings in 26/33, evidence of neuropathy in 11/33, and a combination of both in 7/33. Muscle biopsies revealed significant muscle cell necrosis in 19 with loss of myosin in and mitochondrial abnormalities in 17. The study suggests that there are three major subtypes of critical illness neuromyopathy: a diffuse non-necrotizing myopathy affecting type 2 fibres predominantly, acute myosin-loss myopathy, and acute necrotizing myopathy (Hermans et al, 2008). Myopathic abnormalities are now recognized to generally predominate, but the importance of separating critical illness polyneuropathy and critical illness neuromyopathy is unclear (Hermans et al, 2008).

The development of these conditions typically delays weaning from the ventilator and the start of rehabilitation, and in some cases results in long-term disability. A careful search to exclude metabolic, acute cerebral and spinal disorders is often necessary as the mode of onset is frequently unclear. Risk factors appear to be the same for both; about one third of patients who are ventilated for over 7 days have some degree of neuromuscular disturbance, rising to 60–70% in those with sepsis, systemic inflammatory response syndrome or acute respiratory distress syndrome, and up to 100% in those with multiple organ failure (Hermans et al, 2008). Myosin-loss myopathy has been described more frequently in patients receiving steroids and neuromuscular-blocking agents. Treatment remains unclear, although there have been reports that strict glycaemic control may reduce the incidence of critical illness polyneuropathy or critical illness neuromyopathy (Hermans et al, 2008), but the overall benefit of intensive insulin therapy on survival is currently controversial. Approximately one half of patients with critical illness polyneuropathy or critical illness neuromyopathy recover fully, but 25% retain significant long-term disability and long-term follow-up studies are needed (Hermans et al, 2008).

KEY POINTS

- Patients with middle cerebral artery stroke or posterior fossa stroke can deteriorate rapidly may benefit from transfer to a specialist neurosciences unit for monitoring and possible neurosurgical intervention.
- Recent recommendations are for anticoagulation in all cases of cerebral venous sinus thrombosis irrespective of the presence of intracerebral haemorrhage. Given the risks of further bleeding, these patients should be managed in specialist units where rapid access to neurosurgery is available.
- Many patients with status epilepticus can be managed successfully in general intensive care units. Advice should be sought regarding the necessary investigations and consideration made for referral of refractory status epilepticus to specialised centres.
- At present there is a lack of evidence to support the cohorting of patients with severe Guillain–Barré syndrome and myasthenia gravis in neurosciences intensive care units.

Conclusions

Critical care patients with neurological conditions are commonly cared for in general intensive care units. The role of the neurosciences intensive care unit is often limited to unusual or difficult cases. Improving clinical communication between units is perhaps the first step but further research into the value of cohorting such patients is warranted. **BJHM**

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