Managing post-acute brain injury patients in general medicine

While most patients with an acquired brain injury (ABI) receive appropriate post-acute care and rehabilitation via established pathways from critical care units and tertiary neurosciences centres, a small number of these patients do fall through the cracks and end up in the general medical ward. Classic examples include the patient with hypoxic brain injury secondary to an out-of-hospital cardiac arrest and the neurosurgical patient who has been repatriated. It can be particularly challenging to work out what to do with these patients. This is especially so in Scotland, where the rehabilitation prescription is not implemented.[1] We

Table 1. Management options for spasticity

Intervention		Maximum daily dose	Common side effects	Comments
Non-pharmacological	Trigger factor management	n/a	n/a	Addressing trigger factors reduces the sensory drive for worsening spasticity and spasms. Common trigger factors include infections, skin breaks, ingrown toe nails, urinary retention and constipation.
	Passive stretching	n/a	n/a	Refer to physiotherapy. Patients and family members can also be involved in performing passive stretching.
	Splinting	n/a	n/a	Refer to physiotherapy/orthotics. Common orthotic devices include resting arm splints and AFOs.
Pharmacological – oral	Baclofen	100 mg in three to four divided doses	Sedation, confusion and reduced seizure threshold	$GABA_B$ receptor agonist which acts centrally. A starting dose of 5 mg three times a day is reasonable. Avoid sudden withdrawal as it can lead to baclofen-withdrawal syndrome.
	Tizanidine	32 mg in three to four divided doses	Sedation, dry mouth, hypotension and liver function derangement	Alpha-2 noradrenergic receptor agonist which acts centrally. Starting dose of 2 mg once daily. LFTs need to be monitored monthly for the first 4 months.
	Dantrolene	400 mg in four divided doses	Sedation, diarrhoea and potential fatal hepatotoxicity	Ryanodine receptor antagonist (inhibits Ca^{2+} ions release from sarcoplasmic reticulum) which acts peripherally. Starting dose of 25 mg once daily. Usual maintenance dose of 75 mg three times daily. LFTs need to be monitored at regular intervals indefinitely.
	Benzodiazepines	Variable	Sedation, confusion and potential dependence	$GABA_A$ receptor positive allosteric modulator which acts centrally. Diazepam is useful for acute spasms. Clonazepam has a longer duration of action and is particularly useful for overnight spasms. A starting dose of 0.5 mg at bedtime is reasonable.
	Gabapentin	3,600 mg in three to four divided doses	Sedation, constipation and weight gain	Gabapentinoid (inhibits $\alpha 2\delta$ subunit-containing VDCC) which acts centrally. Starting dose of 300 mg daily either as a single dose or in three divided doses. Particularly useful for patients with concurrent neuropathic pain.
Pharmacological – intramuscular	Botulinum toxin A	Variable	Muscle weakness and risks involved with local invasive procedures	Refer to local specialist service. Effectively causes chemical denervation of NMJ which lasts for 3–4 months. Usually used for focal spasticity and in conjunction with physical intervention programmes.

AFOs = ankle-foot orthoses; GABA = gamma-aminobutyric acid; LFTs = live function tests; NMJ = neuromuscular junction; VDCC = voltage-dependent calcium channel

provide a brief guide on key medical management issues in ABI patients.

Agitation

Disturbed behaviour can be challenging to manage in a ward not used to it. A reasonable initial approach is to address any reversible causes (eg infection, alcohol withdrawal, secondary brain injury) and identify triggers for agitation (eg pain, incontinence, substance withdrawal). Prior to resorting to chemical sedation, environmental and behavioural measures can be effective and these include using a quiet side room, re-orientation strategies and staff consistency. A registered mental health nurse can be an invaluable resource if available. If pharmacological intervention is required, propranolol (up to 80 mg three times daily as tolerated) is effective without being sedating or disorienting but early liaison psychiatry involvement to establish an antipsychotic or antidepressant regime is advised.[2]

Spasticity

Spasticity is a disorder of sensory-motor control resulting from an upper motor lesion, resulting in hyper-excitability of the tonic stretch reflex and persistent muscle over-activity.[3,4] It can present in various patterns (focal, regional or generalised) and establishing treatment goals are important as spasticity can be helpful in some patients to maintain function. Adverse effects of spasticity include functional restriction, abnormal posturing leading to skin vulnerability, pain and contracture development. Early referral is paramount as contracture can develop within as short as 2 weeks of initial presentation.[5] Table 1 highlights the management options for spasticity.

Onward management

It is important to refer to the ward physiotherapist and occupational therapist for an initial assessment. An early referral to the local neuropsychologist, brain injury liaison nurse or neuro-rehabilitation unit will also be helpful. This will allow a suitable rehabilitation plan to be formulated, expectations to be managed or advice about signposting and discharge planning given.

We have only skimmed the surface of the issues that may be encountered in ABI patients on the general medical ward. There is a brain injury e-learning resource commissioned by the Scottish Acquired Brain Injury Network, available at www.acquiredbraininjury-education.scot.nhs.uk, which general physicians may find useful.

It is important that ABI patients are managed appropriately and offered the opportunity to undergo timely rehabilitation that can potentially reduce their long-term care needs. This requires basic knowledge on dealing with potential clinical issues that may arise and an awareness of local rehabilitation pathways. A prompt management plan for these patients will also reduce delays for onward referral and discharge planning, ultimately improving bed turnover in the general medical ward. ■

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References

1 British Society of Rehabilitation Medicine. *Rehabilitation for patients in the acute care pathway following severe disabling illness or injury: BSRM core standards for specialist rehabilitation*. London: BSRM, 2014.
2 Scottish Intercollegiate Guidelines Network (SIGN). *SIGN 130: Brain injury rehabilitation for adults*. Edinburgh: SIGN, 2013.
3 Lance JW. What is spasticity? *Lancet* 1990;335:606.
4 Burridge JH, Wood DE, Hermens HJ *et al.* Theoretical and methodological considerations in the measurement of spasticity. *Disabil Rehabil* 2005;27:69–80.
5 Clavet H, Hébert PC, Fergusson D *et al.* Joint contracture following prolonged stay in the intensive care unit. *CMAJ* 2008;178:691–697.

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