Meaningful Assessment in Patients with Acquired Brain Injuries

Thomas Watanabe, MD[a,b,*]

KEYWORDS

• Upper motor neuron syndrome • Acquired brain injury • Spasticity • Goal setting
• Physical examination

KEY POINTS

- Knowledge of the implications of the type and severity of the acquired brain injury is important to developing a treatment strategy for patients with upper motor neuron syndrome (UMNS).
- Spasticity is often not the main factor limiting joint function or range of motion after acquired brain injuries; assessment of other components of the UMNS or other neuromedical complications is essential to optimize outcomes.
- Ongoing access to care must be considered when choosing specific interventions for patients with UMNS.
- Determination of a treatment plan should be based on the identification of relevant and attainable goals, with input from, as appropriate, the rehabilitation team, patients, and families.

Acquired brain injuries (ABIs) take many different clinical forms. Not surprisingly, spasticity, or perhaps more appropriately termed the *upper motor neuron syndrome* (UMNS), also presents in quite a varied fashion in different patients with ABI. Additionally, the clinician must consider other factors when assessing patients with ABI with respect to management of spasticity. This article primarily considers the similarities and differences between traumatic brain injuries (TBIs) and hypoxic-ischemic brain injury (HIBI) when addressing the cause. Relevant objective measures of spasticity and other elements of the UMNS are discussed. The impact of severity, prognosis, and chronicity of the ABI on assessment and treatment planning also needs to be considered. Variables related more specifically to the presentation of spasticity,

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[a] MossRehab at Elkins Park, Einstein Healthcare Network, Elkins Park, PA, USA; [b] Department of Physical Medicine and Rehabilitation, Temple University School of Medicine, Philadelphia, PA, USA
* MossRehab at Elkins Park, 60 Township Line Road, Elkins Park, PA 19027.
E-mail address: Watanabt@einstein.edu

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such as extent, severity, complications that limit function, resources available, and appropriate goals, are also important to consider in the assessment and to formulate a management plan.

TYPE OF BRAIN INJURY

TBIs may be relatively focal or more diffuse. The extent and location of cerebral injury will affect the presentation of spasticity and its functional implications. HIBI is always diffuse, although certain areas of the brain are more susceptible to hypoxic injury than others, such as the hippocampus, basal ganglia cortex, and cerebellum.[1] HIBI also tends to lead to other movement disorders that might be confused with spasticity but may require significantly different management strategies, such as dystonia, rigidity, ataxia, and myoclonus, although these problems may also be seen after TBIs. Some of these disorders may have a delayed presentation.[2] Of course, patients who sustain TBIs may also have elements of HIBI depending on the complications associated with their traumatic injury. Careful examination including an evaluation of functional activities may help the clinician to differentiate among various movement disorders. When assessing passive range of motion (ROM), rigidity or cogwheeling will present with resistance to ROM that is not velocity dependent. Dystonic movements will be present even in the absence of ranging a joint. Another movement disorder that sometimes is seen after ABIs, especially HIBI, is myoclonus. Myoclonus may worsen with activity or be present at rest. Positive myoclonus results from sudden muscle contractions and may be brought on by stimuli, including movement.[3]

The difference in causes may also affect clinical decision-making because of the effects on long-term prognosis. This concern is especially relevant for patients with more severe injuries. It is known that the likelihood for significant functional recovery is worse for patients with HIBI relative to those with TBIs.[4] The choice of interventions should be based in part on the goals identified. Patients with, for example, disorders of consciousness, will likely benefit more from goals such as ease of passive care rather than more cognitively and motorically complex functional activities. Treatment options based on goals of active function might be favored if the anticipation is that further neurologic recovery is to be expected.

The contrasts in the likelihood of meaningful functional recovery for these two types of ABIs is exemplified by older literature that used the terms *persistent* versus *permanent* vegetative state.[5] Permanence was said to be determined if there was no recovery of consciousness after 3 months in a nontraumatic injury and 12 months after a TBI. One does not, of course, need to wait for these specific time points to guide decisions regarding the types of goals and interventions for spasticity. These definitions help in the description and prognostication of more severe ABIs. Other functional goals will demand different degrees of preservation of cognitive function, and management of the UMNS should be guided to a large degree by the likelihood that patients will have the cognition necessary to achieve such goals. This point is just one of several examples of why spasticity management must be individualized.

MEASURES OF SPASTICITY AND OTHER ELEMENTS OF UPPER MOTOR NEURON SYNDROME

The UMNS is often described as having positive and negative signs. Positive signs include heightened muscle stretch reflexes, spastic cocontraction, dystonia, and muscle spasms. Negative signs include weakness and loss of selective muscle control.[6] As part of the assessment, it is useful to incorporate measures that quantify some of these elements as a way to characterize the specific patient's condition and to