The Neuro-orthopaedic Approach

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KEYWORDS

• Spasticity • Upper motor neuron disease • Neuro-orthopaedics • Contracture • Deformity

KEY POINTS

- Upper motor neuron injury or disease leads to muscle overactivity or nonfunction, leading to development of functional deficiencies, contractures, pain, and poor hygiene.
- A variety of nonsurgical and surgical management tools are used to improve function, alleviate pain, and improve hygiene and cosmesis.
- Preoperative assessment should determine contributing muscle forces to each deformity because spasticity may mask any underlying volitional muscle control.
- Surgical techniques include tendon lengthenings, releases, transfers, osteotomies, and bony fusions.

GENERAL PRINCIPLES OF NEURO-ORTHOPAEDICS

Upper motor neuron disease or injury leads to a variety of both positive and negative signs, notably including muscle spasticity and weakness throughout the body. Patients with spasticity are at risk for development of a variety of deformities caused by these imbalances in muscle forces, leading to functional impairments, contractures, pain, and poor hygiene. The approach to neuro-orthopaedic patients is by necessity multidisciplinary, because a variety of nonsurgical and surgical options are available. In evaluating each patient, surgeons must consider the severity and direction of any deformity, potential for improvement in function, the ability to alleviate pain, and potential for improvement in hygiene and cosmesis. Similar to the physiatry analog of botulinum toxin versus oral spasticity agents, surgery can

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be local in the form of muscle releases, fusions, joint arthroplasty, and other procedures, or it can be more global and far reaching, such as nerve ablation or dorsal rhizotomy.

In the setting of acute injury, a nonsurgical approach may be best because patients with traumatic brain injury may continue to regain functionality up to 6 months after injury. Patients with stroke may continue to recover for up to 1 year. Intervention in the acute setting is aimed at interventions that provide symptomatic relief and reduce the severity of downstream deformities. Nonsurgical approaches in the neuro-orthopaedist's toolbox are varied and include antispasmodic medications, Botox administration, chemodenervation, serial casting, splinting, orthoses, and physical and occupational therapy.

Operative interventions may be considered thereafter when the extent of recovery has been maximized, or when significant obstruction to rehabilitation has been refractory to nonoperative means. Keeping in mind the natural history of neurologic recovery, it is important to keep the following overarching management principles in mind: (1) operate early, before deformities become fixed and rigid; (2) distinguish between the function of the limb and the function of the patient; (3) spastic muscles are weak muscles, and surgery necessarily weakens them further; (4) be sure to get the diagnosis right and optimize length-tension relationships.

Preoperative assessment should determine contributing muscle forces to each deformity. Often, the spasticity of antagonizing musculature masks any underlying volitional control of agonists across a joint. Dynamic electromyography (EMG) studies can determine the primary causes of deformity,[1–4] whereas selective anesthetic blockade of antagonistic musculature can determine the extent of any volitional control. In general, patients with volitional control benefit from selective lengthening of the deforming musculature or tendon transfers to optimize function. Those without voluntary control can undergo selective releases or bony fusion procedures to optimize hygiene and passive function.

CONSIDERATIONS IN SPINAL CORD INJURY

In the United States, there are approximately 400,000 patients living with spinal cord injury (SCI), and about 11,000 new cases occur per year. Common causes of SCI vary from motor vehicle accidents and gunshot trauma to sports injuries and falls. In general, patients follow a bimodal distribution: younger patients sustain injuries from higher-energy trauma, and older patients with spinal stenosis have lower-energy trauma.

SCI is classified by the spinal cord level and number of affected extremities (paraplegic vs tetraplegic) and completeness of the injury (complete vs incomplete). The American Spinal Injury Association (ASIA) classification further characterizes the extent of injury. Several well-known patterns of SCI, such as Brown-Séquard, central cord, and anterior cord syndromes, occur depending on the location of injury within the spinal cord and can be elucidated by their characteristic physical examination findings.

In the setting of acute SCI, patients may show signs of spinal shock, a temporary loss of spinal cord function and reflex activity below the level of injury. Diagnosis of a complete SCI cannot be made until after the resolution of spinal shock, which is assessed by the return of the bulbocavernosus reflex, in which the contraction of the anal sphincter is felt while squeezing the glans penis or clitoris. Complete SCI results in a permanent disruption in the reflex arc and the bulbocavernosus reflex does not return. Resolution of spinal shock is variable but generally returns within 48 hours.