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Spasticity and hyperselective neurectomy in the upper limb

Spasticité et neurectomie hypersélective au membre supérieur

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ABSTRACT

Spasticity is a complex pathology, both in terms of assessment and treatment. This article focuses on the clinical examination (objective, capacity, performance and function), which is key for choosing a treatment and can be helped by botulinum toxin injections. The treatment involves physical therapy, occupational therapy, medications and surgery. Neurectomy has been used in the upper limb since 1912 and is one of the therapeutic options for spasticity. This treatment is usually reserved for nonfunctional hands. Cadaver studies have helped us better understand nerve anatomy and improve the hyperselective neurectomy (HSN) technique. This article describes the history of neurectomy, how anatomical dissections apply to surgery, the HSN technique in the musculocutaneous nerve, median nerve and ulnar nerve and results of preliminary prospective studies. Spasticity, mobility, performance and function were evaluated a few months after HSN and about 12 months later to assess the permanence of the results in children and adult spastic patients. No matter the nerve or function targeted (elbow extension, wrist extension, or supination), spasticity was reduced with improvements in the functional House score and appeared stable at the last follow-up. HSN seems to be a good, reliable therapeutic option for spasticity, including functional hands.

Mots clés :
Spasticité
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RÉSUMÉ

La spasticité est une pathologie complexe, aussi bien pour l'évaluation que pour le traitement. Cet article fait le point sur l'examen clinique (analytique, capacités, performances et fonction) qui est un élément clé dans le choix du traitement et peut être aidé par les injections de toxine botulique. Le traitement comprend la kinésithérapie, l'ergothérapie, les traitements médicaux et la chirurgie. La neurectomie a été proposée au membre supérieur depuis 1912, c'est est l'une des options thérapeutiques contre la spasticité elle-même. Ce traitement était habituellement réservé à la main non fonctionnelle. Des études cadavériques ont permis de mieux comprendre l'anatomie des nerfs et d'améliorer la technique de neurectomie hypersélective (NHS). Cet article décrit l'histoire de la neurectomie, l'application des dissections anatomiques à la chirurgie, la technique de NHS pour le nerf musculocutané, le nerf médian et le nerf ulnaire et les résultats des études prospectives préliminaires. La spasticité, la mobilité, la performance et la fonction ont été évaluées après NHS à un temps précoce et au dernier recul afin d'évaluer la pérennité des résultats chez les patients spastiques enfants et adultes. Quel que soit le nerf concerné et la fonction ciblée (extension du coude, extension du poignet ou supination), la spasticité diminuait avec une amélioration du score fonctionnel de House et semblait stable au dernier recul. La NHS semble être une option thérapeutique satisfaisante et fiable pour la spasticité, y compris pour les mains fonctionnelles.

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1. Introduction

Spasticity is the result of hyperexcitability of the stretch reflex [1] resulting from damage to neurons of the corticospinal tracts and loss of inhibitory supraspinal influences [2]. It is characterized by a velocity-dependent increase in tonic stretch reflexes (“muscle tone”) with exaggerated deep tendon reflexes.

In a spastic patient, reflex responses are faster, larger and spread to other muscle groups [3]. Moreover, the passive resistance to stretch increases with a “spastic catch”: velocity-dependent triggering of muscle contraction, which then releases slowly. Other characteristics of upper motor neuron syndrome are flexor and extensor spasms, clasp-knife phenomenon, exaggerated cutaneous reflexes and contractures. The degree of spastic hypertonia varies during the day, depending on the patient’s tiredness, cold, stress and many other factors.

Assessment is not easy and the goals of treatment have to be well defined to evaluate the potential benefits and risks [3]. The goals are usually comprehensive: better independence, better strength and better coordination. Others goals are now more feasible such as control of painful spasms and decreased resistance to passive movement. The difficulty resides in determining whether the patient’s complaint is due to hypertonia or other features such as weakness, decreased dexterity, increased flexion withdrawal reflexes, clonus or orthopedic deformity.

Treatment usually consists of physical therapy, occupational therapy, medical treatment with oral drugs and surgery that is often performed for sequelae: contracture, bone deformity, joint dislocation and scoliosis. In the upper limb, wrist fusion, muscle lengthening and tendon transfers can improve appearance and function [4]. This article reviews the etiology of spasticity, its examination and outcome scales and its treatment.

Hyperselective neurectomy (HSN) is another approach for treating the spasticity itself, reducing the nerve’s stimulation and therefore the spasticity. Neurectomy has been performed in the upper limbs since 1912. The technique has good outcomes but is typically proposed for nonfunctional hands; its history is summarized in this article. Cadaver studies have improved our understanding of nerve anatomy and the HSN technique. This article describes the HSN technique for the musculocutaneous, median and ulnar nerves, and the results of preliminary prospective studies.

2. Pathophysiology

Spasticity is the sequelae of damage to neurons of the corticospinal tracts, with hyperexcitability of the myotatic reflex responsible for exaggerated, velocity-dependent, stretch reflexes and tendon reflexes, resulting from loss of cortical inhibitory influence. Cutaneous reflex contractures appear to be due to hyperactivity of polysynaptic reflexes responsible for triple flexion or extension. In intramedullary lesions with interruption of the afferent motor neurons from the posterior roots, the hyperexcitability is intrinsic to the motor neuron. The appearance of spasticity is usually delayed due to neuronal plasticity responsible for perturbation of down control, and reorganization of spinal circuits with hypersensitivity of receptors without any other afferents, sprouting from neighbor fibers.

Neurectomy involves not only the motor neuron axon, but also the sensory proprioceptive neuron dendrite (Fig. 1). Neurectomy suppresses the stretch reflex by acting on both kinds of fibers; 6 months later, motor recovery occurs because of sprouting from the remaining axons (1/4 to 1/5) after the neurectomy and sometimes because of regrowth of the cut axons, but the spasticity does not reappear because the transected dendrites are unable to regrow and the sensory part of the stretch reflex remains interrupted.

3. Etiology

3.1. Children

During childhood, cerebral palsy is the most common cause of spasticity, but the prevalence and incidence are not well known and likely underestimated. It is increased especially in infant survivors of normal birth weight, according to a prevalence study in the United States [5], likely related to improvements in neonatal care and better documentation. The overall reported prevalence in children aged 3–10 years in 1991 was 2.4 per 1000 children [3]. Cerebral palsy is the result of central nervous system damage during *in utero* development, during delivery or during the first 2 years of life [6]. Very low birth weight infant survivors and neonatal intensive care increase the risk of cerebral palsy. Eighty percent of the risks factor for cerebral palsy are prenatal [7]: prematurity, low birth weight, intrauterine growth restriction, multiple births, intracranial hemorrhage, white mater injury, cerebral malformations, maternal age more than 35 years, severe maternal iodine deficiency, chorioamnionitis, maternal infection, antepartum vaginal bleeding, untreated hyperbilirubinemia. Ten percent are perinatal (peripartum asphyxia, maternal infection) and 10% are postnatal (head trauma and hypoxia within the first two years of life, meningitis, intentional injury). The severity of the lesions differs based on the patient. Associated disorders are very

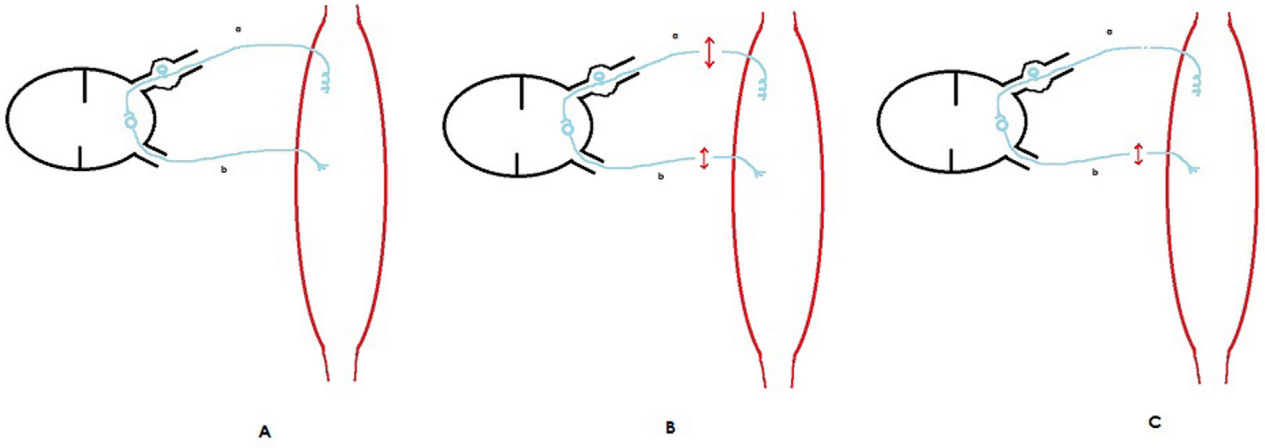

Fig. 1. Myotatic reflex arch with motor neuron (a) and sensory proprioceptive neuron dendrite (b) (A). Neurectomy of the motor neuron axon and sensory proprioceptive neuron dendrite (B). Regrowth of the cut axon; sensory proprioceptive neuron dendrite remains interrupted (C).