The Evaluation of "Spasticity"

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ABSTRACT: Lesions of the upper motor neuron cause: 1. *Alterations in segmental reflex activity.* For example increased tendon jerks and velocity dependent stretch reflexes ("spasticity"), clonus, the clasp knife response, release of flexion reflexes and extensor plantar reflexes. 2. *Impaired ability to activate motoneurons* rapidly and selectively. Voluntary movements may also be *restrained* by co-contraction of antagonists muscles, by segmental reflexes (enhanced during voluntary effort) or by contractures. A combination of these factors may impair *overall functional ability.* Segmental reflexes, voluntary power and overall functional abilities can be assessed using *clinical scoring systems.* Recordings of muscle *length, tension and EMG* offer more objective measures of reflex and voluntary activity and of overall functions such as locomotion, and can separate weakness from co-contraction, spasticity from contracture. Methods are now available for exploring *individual* (transmitter specific) segmental *reflex pathways* and *descending pathways* in man. Lesions of the upper motor neuron are complicated by *secondary changes* in segmental neurons. Segmental reflex activity and muscle mechanics depend on the *immediate past history* of events. These factors must be taken into account.

RÉSUMÉ: Evaluation de la spasticité Les lésions du neurone moteur supérieur causent: 1. *Des altérations dans l'activité réflexe segmentaire:* par exemple, l'augmentation des réflexes ostéotendineux et des réflexes myotatiques dépendants de la vitesse ("la spasticité"), le clonus, la réponse en lame de canif, la diffusion des réflexes de flexion et des réflexes cutanés plantaires. 2. *Des altérations de l'abilité à activer les motoneurones rapidement et sélectivement.* Les mouvements volontaires peuvent également être restreints par la contraction concomitante de muscles antagonistes, par des réflexes segmentaires (accrus pendant un effort volontaire) ou par des contractures. Une combinaison de ces facteurs peut altérer la capacité fonctionnelle globale. Les réflexes segmentaires, la puissance volontaire, et la capacité fonctionnelle globale peuvent être évalués par des échelles d'évaluation clinique. L'enregistrement de la longueur et de la tension des muscles et l'EMG fournissent d'autres mesures objectives de l'activiteś réflexe et volontaire et des fonctions globales telle la locomotion, et peuvent séparer la faiblesse de la contraction simultanée, la spasticité de la contracture. Des méthodes pour explorer les voies réflexes segmentaires individuelles (spécifiques aux différents transmetteurs) et les voies descendantes sont maintenant disponibles chez l'homme. Les lésions du neurone moteur supérieur sont compliquées par des changements secondaires dans les neurones segmentaires. L'activité réflexe segmentaire et la mécanique musculaire dépendent du cours des évènements les plus récents. On doit tenir compte de ces facteurs dans l'évaluation de la spasticité.

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Lesions of the upper motor neurons give rise to:

1. *Exaggerated segmental reflexes* including a velocity dependent increase in tonic stretch reflexes and exaggerated tendon jerks, ("spasticity"),[1] clonus, the clasp knife phenomenon, release of the flexion reflex and the Babinski response.

2. *Reduced ability to activate motoneurons* quickly or selectively and/or disordered patterns of innervation of motor neurons.

Voluntary movements may also be restrained by co-contraction, contracture, or the inappropriate elicitation of stretch reflexes. Together these deficits can lead to an impairment of *overall functional ability.*

As functional impairment is not necessarily caused by "spasticity" as defined above[2,3] the evaluation of a patient with the upper motor neuron syndrome is of little value unless all of these factors are taken into account. An evaluation can be based on *clinical assessments,* can be supplemented by *objective measures of limb position and EMG* activity or can be focused on the *examination of individual (? transmitter specific) reflex pathways.*

Clinical Evaluation

Clinical evaluation requires no special equipment except a reliable observer and the tools for a neurological examination. The activity of *segmental reflexes* can be recorded using a scoring system for tendon jerks, clonus, stretch reflexes (both in response to the velocity of a movement and in response to static stretch) and the flexion reflex.[4] Abnormalities of these reflexes do not, necessarily, occur together. Dohrman and Nowack,[5] for example, have confirmed the lack of correlation between exaggerated tendon jerks and tonic stretch reflexes which is a common clinical observation.

Maximum *voluntary power* can be scored using the MRC scale.[6] Some measure of skilled movement may also be useful.

The patient's *overall functional ability* can be assessed using one of the many scoring systems for neurological function or degree of disability. Some of these require detailed neurological examination while others can be based on a patient

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questionnaire. Each system differs in the number of grades assigned to slightly, moderately, or severely disabled patients. A scoring system with many grades would be best suited to detect subtle differences produced by drugs. This topic has been well reviewed by Grynderup[7] and Feldman et al.[8]

Objective Measurements

Recordings of the EMG and of the length and tension of skeletal muscles can provide objective measurements of neurological dysfunction and help to elucidate the underlying pathophysiology. Such recordings can be used to distinguish weakness resulting from impaired recruitment of motoneurons from that resulting from the restraints of co-contraction or contracture and can be used to dissect the various components of the *stretch reflex*.

The force detected by an examiner attempting to move a limb arises from several sources. When there is no muscle contraction the focuses are those related to the length dependent, stiffness (elasticity), and the velocity dependent, resistance (viscosity) of the muscle and connective tissues as well as acceleration dependent inertia of the limb. If the movement induces a reflex muscle contraction the net force produced by the agonists and antagonists is added.[9] These major components can be separated in the following way. The passive, mechanical, properties of the muscle and connective tissue can be examined in isolation by recording muscle force and length when EMG activity either did not occur or was blocked by local anesthesia of nerves or ischemia of the limb. The inertial component can be calculated from the acceleration and an estimate of the mass of the limb. The stiffness can be estimated from the tension during static stretch or stretch at constant velocity[10] and the viscous and frictional resistance can be determined from the area of the hysteresis curve of the force-length plot.[11] The contribution of each of these factors to the force opposing movement of the limb can thus be separated.[10] Several authors have studied the passive properties of muscle in patients with upper motor neuron lesions using ischemia or nerve block. For example Herman[12] demonstrated an increase in muscle stiffness (and an increase in velocity dependent resistance in the patients with contracture), Hufschmidt and Mauritz[11] showed an increase in muscle stiffness and in the plastic or frictional resistance.

The contribution of the central nervous system to stretch reflexes can be assessed by recording the EMG in response to limb movements of various lengths and velocities.[13] The behaviour of the stretch reflex can then be classified as length or velocity dependent, phasic or tonic. The averaged responses to sinusoidal stretch can provide the same information.[13] In the upper motor neuron syndrome the reflex EMG generally increases with increasing velocity of stretch and may be greater with increasing length of the muscle (except for the quadriceps which show the reverse).[14] Sustained responses to static stretch may be seen in certain muscles for example the biceps.[13] In patients with spasticity the regression between integrated EMG and the velocity of stretch provides a repeatable measure of spasticity that can be used in drug trials.[4,15] This is perhaps the simplest objective measure of spasticity.

More precisely timed and controlled muscle stretches can be generated with a torque motor. The EMG within the first hundred milliseconds is segmented into two or more peaks. The first occurs at "monosynaptic" latency, the second about 30 ms later. The amplitude depends on the background EMG and the velocity of stretch. In the upper motor neuron syndrome the first response is exaggerated, the second is variable often being decreased or delayed.[16,17]

The evaluation of *voluntary movements* can also be rendered more objective by recording EMG and limb position. Ballistic movements, for example, are generated by precisely timed bursts of EMG activity in the agonist, the antagonist and then the agonist again. In the upper motor neuron syndrome the first agonist burst is prolonged[18,19] and there is a longer delay between the onset of EMG and the onset of movement.[20] The prolongation of the agonist burst may represent an attempt by the nervous system to compensate for the reduced motor neuron output as it has been shown that the firing rate of motor neurons is lower and cannot be adjusted so rapidly in the upper motor neuron syndrome.[21] The EMG records can also be used to decide whether restricted voluntary movement is due to contracture, elicitation of the stretch reflex, or inappropriate central programming. Sahrmann and Norton,[2] for example, found that the primary impairment of rapidly alternating movement was not due to restriction by stretch reflexes but to prolongation of agonist activity. McLellan et al[3] observed inappropriate activity in the biceps during extension of the forearm in subjects with the upper motor neuron syndrome attempting sinusoidal movements. As this was also present when the subject made sinusoidal changes in muscle tension with the limb in a fixed position the activity was clearly not due to a stretch reflex but due to an abnormality of central programming.

The evaluation of certain *overall functions* such as locomotion can be improved by recording multiple channels of EMG and the position of each limb segment during walking. In the upper motor neuron syndrome Conrad et al,[22] for example, have shown prolongation of agonist contractions resulting in co-contraction of agonist and antagonist muscles and reduced EMG prior to heel strike all suggestive of a disorder of central programming. There was an increase in the stretch reflexes on heel strike and during extension of the calf muscles and Knutsson[23] suggested that this may contribute to the hyperextension of the knee in such patients. Such studies may separate patients who are likely to benefit from a reduction in the stretch reflex from those who would not.

Evaluating the Function of Individual (? Transmitter Specific) Pathways

Many individual *spinal reflex pathways* can now be examined in man. Microneurography has allowed deductions about muscle receptor activity and, indirectly, about the fusimotor system.[24] More or less selective cutaneous or large muscle afferent volleys can be generated by electrical stimulation of peripheral nerves and more complex afferent volleys produced by tendon taps or muscle stretch. The reflex effects from these afferents can be shown by conditioning monosynaptic H-reflexes. The responses represent the net effects of the input on a pool of motor neurons and pre and postsynaptic effects cannot be easily distinguished. By recording changes in the firing probability of voluntarily activated motor neurons in man in response to an afferent volley it is possible to derive subthreshold events (EPSPs and IPSPs) in single motor neurons in man. In this way it is possible to obtain information similar to that obtained by intracellular recordings. The projection frequency of afferents and the amplitude or ("effectiveness") and rise time of postsynaptic potentials can all be documented. There are now estab-

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lished techniques using H reflexes to examine the Ia monosynaptic,[25] Ia polysynaptic,[26] Ia inhibitory,[27] Ib,[27] group II,[28] and cutaneous pathways.[29] Renshaw cell activity[30] and presynaptic inhibition[31] can be assessed. Post stimulus time histograms of the firing of single motor units have been used to demonstrate homonomyous and heteronomyous Ia facilitation,[32] Ia polysynaptic effects,[26] Ia inhibition,[32] Ib,[32] and cutaneous[33] actions on individual motor neurons. Group II effects may also be demonstrable.[34] Presynaptic inhibition can be shown.[35]

These techniques are now being applied to the study of patients with the upper motor neuron syndrome. So far there has been no evidence for increased spindle excitability in spasticity.[36] The Ia EPSP appears little altered.[37] Presynaptic inhibition of Ia facilitatory effects is less,[38] the flexion reflex is released[39] and the normal facilitation of antagonist Renshaw cell activity during a voluntary contraction is lacking.[40]

The use of electrical or magnetic stimulation of the brain may allow *descending pathways* to motor neurons and interneurons to be assessed in a similar manner.

Problems and Cautions

The upper motor neuron syndrome may be complicated by *secondary changes at segmental level* including degeneration of motor neurons, changes in motor unit fiber type, contracture of muscles and tendons, and compression of peripheral nerves.[42] These will all have a profound effect on clinical and neurophysiological measurements. For example the elastic stiffness of a muscle depends on its cross sectional area[11] the EMG/tension relationship is altered in the upper motor neuron syndrome.[4] Thus the neurophysiological evaluation of a patient with the upper motor neuron syndrome must be preceded by an appropriate assessment of sensory and motor peripheral nerves and of muscle bulk.

Recordings can be affected by the *immediate past history* of events for the limb. The thixotropic properties of muscle can affect not only the recorded tension[11] but the afferent activity and reflex consequences.[44]

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