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Update article

Coefficients of impairment in deforming spastic paresis

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ABSTRACT

This position paper introduces an assessment method using staged calculation of coefficients of impairment in spastic paresis, with its rationale and proposed use. The syndrome of deforming spastic paresis superimposes two disorders around each joint: a neural disorder comprising stretch-sensitive paresis in agonists and antagonist muscle overactivity, and a muscle disorder ("spastic myopathy") combining shortening and loss of extensibility in antagonists. Antagonist muscle overactivity includes spastic cocontraction (misdirected descending command), spastic dystonia (tonic involuntary muscle activation, at rest) and spasticity (increased velocity-dependent reflexes to phasic stretch, at rest). This understanding of various types of antagonist resistance as the key limiting factors in paretic movements prompts a stepwise, quantified, clinical assessment of antagonist resistances, elaborating on the previously developed Tardieu Scale. Step 1 quantifies limb function (e.g. ambulation speed in lower limb, Modified Frenchay Scale in upper limb). The following four steps evaluate various angles X of antagonist resistance, in degrees all measured from 0°, position of minimal stretch of the tested antagonist. Step 2 rates the functional muscle length, termed X_{V1} (V1, slowest stretch velocity possible), evaluated as the angle of arrest upon slow and strong passive muscle stretch. X_{V1} is appreciated with respect to the expected normal passive amplitude, X_N, and reflects combined muscle contracture and residual spastic dystonia. Step 3 determines the angle of catch upon fast stretch, termed X_{V3} (V3, fastest stretch velocity possible), reflecting spasticity. Step 4 measures the maximal active range of motion against the antagonist, termed X_A, reflecting agonist capacity to overcome passive (stiffness) and active (spastic cocontraction) antagonist resistances over a single movement. Finally, Step 5 rates the residual active amplitude after 15 seconds of maximal amplitude rapid alternating movements, X_{A15}. Amplitude decrement from X_A to X_{A15} reflects fatigability. Coefficients of shortening $(X_N - X_{V1})/X_N$, spasticity $(X_{V1} - X_{V3})/X_{V1}$, weakness $(X_{V1} - X_A)/X_{V1}$ and fatigability $(X_A - X_{A15})/X_A$ are derived. A high (e.g., >10%) coefficient of shortening prompts aggressive treatment of the muscle disorder – e.g. by stretch programs, such as prolonged stretch postures –, while high coefficients of weakness or fatigability prompt addressing the neural motor command disorder, e.g. using training programs such as repeated alternating movements of maximal amplitude.

Six decades ago, Tardieu defined spasticity as an increase in stretch reflexes that could be characterized and measured by the speed required to elicit the reflexes [1]. After these initial briskness measurements, Tardieu moved to a measurement of the angle of the muscle reaction to fast stretch [2]. During that period, Ashworth published an ordinal scale to rate resistance to passive movement in patients with spastic paresis [3]. In the era of botulinum toxin in particular, the Ashworth scale would become considered as a tool rating spasticity and would see its use markedly increase [4]. Fifteen years after the publication of that scale, a complex consensus definition of the word spasticity was proposed, largely following Tardieu's inspiration but not always understood or followed since [5].

As will be seen below, such a strict definition of spasticity has been useful as a way to characterize patients affected with a common syndrome; however, it failed to adequately represent the key issues that hamper function and quality of life in paretic patients. The author has given the full name deforming spastic paresis to the clinical syndrome caused by lesions involving the corticospinal pathways. This syndrome combines a neural disorder made of agonist paresis and antagonist overactivity (see below the definitions of each of these items) and a soft tissue disorder combining shortening and loss of extensibility, in muscle in

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particular. Sufficient data exist today to individualize this specific muscle disorder, which may be termed spastic myopathy [6]. In the full name deforming spastic paresis, the adjective deforming finds double justification: first, patients see their bodies deformed in this syndrome, a cosmetic aggression that causes a significant component of their loss of quality life [7]; second, if no appropriate therapeutic program is implemented, the syndrome continues to dynamically deform bodies over time. It seems important to remind of such a fundamental component of a syndrome in its name.

After revisiting the definitions and characterizations of the key phenomena in deforming spastic paresis, this position paper revises a stepwise method to quantify their assessment, by proposing the staged calculation of four coefficients of impairment.

1. Definitions – pathophysiology – taxonomy in deforming spastic paresis

Deforming spastic paresis is thus a syndrome combining a neural disorder of motor command and a muscle disorder of extensibility loss [8]. The neural disorder entails two components, superimposed around each joint and acting synergistically to challenge active movements: stretch-sensitive paresis in agonists and muscle overactivity in antagonists [8,9]. The muscle disorder, which can be termed spastic myopathy, can be clinically subdivided into two constitutive elements, physical shortening and visco-elastic loss of extensibility, as a part of soft tissue contracture, challenging both active and passive movements [6,8]. The neural and muscular disorders of deforming spastic paresis are unevenly distributed around joints, which creates force imbalances and thus deformities and asymmetrical impairments of active movement, with both muscular and neural resistances greater in attempts at moving against the more shortened muscles [8,9]. These symptoms of deforming spastic paresis appear in the following order after a lesion to the central pathways of motor command execution:

- stretch-sensitive paresis is defined as a quantitative reduction of the voluntary recruitment of agonist motor units, further diminished by antagonist stretch [8–10]. Paresis is chronologically the first manifestation of a central lesion to the motor pathways. In addition, paresis of central origin will act as the trigger of a cascade of nervous system and soft tissue adaptations, in particular muscle adaptations that will lead to stretch-sensitivity of virtually all subsequent symptoms [10,11]. One example is the sensitivity of agonist paresis to antagonist stretch, which represents one of the consequences of soft tissue adaptations [8–10];
- soft tissue contracture manifests itself as a two-fold clinical issue: physical shortening and – at equal length – loss of extensibility (stiffness) by increased muscle viscosity and elasticity, particularly obvious when applying high tensions [8,12–20]. Soft tissue contracture originates in the muscle aggression represented by the immobilization (complete or partial) in short position of some muscles, which begins with the onset of paresis [12,19]. Such immobilization is often insufficiently compensated, or sometimes promoted, by healthcare teams. Muscle contracture then occurs through acute modifications of gene transcription in muscle fibers immobilized in short position, with deleterious quantitative and qualitative changes [21,22]. Overall reduced rates of protein synthesis and induced expression of genes for disuse atrophy promoters (REDD1, REDD2, MAFbx, MuRF1) represent changes not seen when the muscle fiber is immobilized in long position [23]. This phenomenon follows an acute time course, as most of the contracture has actually developed by the end of the acute/subacute period, within the days and weeks following immobilization onset [24]. Muscle contracture, which can be termed spastic myopathy, as a form of myopathy characterized by both increased muscle tension and stretch-sensitive evolution, thus represents a true muscle disorder, essentially avoidable, that comes to superimpose on the neurological disorder. Contracture becomes both the first factor of body deformity in patients with deforming spastic paresis [25] and, through increased spindle sensitivity in the contractured muscle [19], a factor greatly limiting passive and active movement (see below) [26];
- spastic muscle overactivity comprises different forms of increased involuntary recruitment of motor units, of which the following three, most often co-existing with one another, are of particular importance:
  - spasticity is simply defined as an increase in the velocity-dependent reflexes to phasic stretch, detected and measured at rest [9]. Using such strict definition, spasticity is a concept that is useful to the clinician for being both a simple marker of this patient population and a clinical parameter quantifiable at bedside (in contrast with functionally more important forms of muscle overactivity, see below), provided a valid and precise measure is used [1,2,27–31]. In addition, spasticity is somewhat correlated with other forms of spastic muscle overactivity as it may partially reflect both motoneuronal hyperexcitability and spindle responsiveness [9,19,32–37]. However, spasticity *per se* is not the main factor limiting active movement in patients with deforming spastic paresis, with the likely exception of attempts at fast or ballistic movements [9],
  - spastic dystonia is an excessive, chronic, tonic muscle activation of supraspinal origin, detected and measured at rest, potentially reduced after maintained stretch of the dystonic muscle [35–39]. Spastic dystonia is likely related to increased involvement of brainstem descending pathways (rubro-, vestibulo-, tecto-, and ipsilateral reticulo-spinal pathways) undergoing abnormal branching onto deafferented hyperexcitable motor neurons following higher lesions [40–44]. Most of these pathways are excitatory and have reduced capacities of neuronal rest, compared to the corticospinal pathway [45–47]. Within the most shortened muscles, spastic dystonia comes to superimpose on soft tissue contracture to represent a second major factor of deformity in patients with spastic paresis [35–39],
  - spastic cocontraction is an excessive degree of antagonistic activation elicited by voluntary agonist command [8,9,11,48–51]. This type of overactivity is thus revealed and measured only during voluntary command directed to the agonist; it has supraspinal origin and is aggravated by stretch of the cocontracting muscle [9,11,50]. Antagonist coactivation actually was the first identified form of muscle overactivity in patients with deforming spastic paresis, before the term spasticity was coined [48]. Spastic cocontraction is a critical factor of limitation – sometimes reversal – of active movement in subjects with deforming spastic paresis [9–11,48–51].

Stretch-sensitive paresis, soft tissue contracture and spastic muscle overactivity make up the syndrome of deforming spastic paresis and represent the three main factors hindering movement in this syndrome. Reciprocal potentiation ends up developing between the muscle disorder, soft tissue contracture in the shorter of the two muscles around a joint, and the neural disorder, antagonist muscle overactivity in that muscle and stretch-sensitive paresis in the opposing muscle [8,9,17,52].

A behavioral modification typically comes to further aggravate this picture, as a self-imposed behavior of general hypo-activity, reduced social participation and therefore sensorimotor restriction