

Synergistic muscle activation during maximum voluntary activation in children with or without spastic CP

The traditional model of motor weakness derived from the works of Hughlings Jackson is that of a negative phenomenon following a cortical injury such as stroke, in contrast to the positive phenomena of brisk reflexes and the release of primitive, complex reflexes. The physiology of orderly motor neuron recruitment and de-recruitment elaborated by Henneman,¹ in which the smallest, weakest, and slowest motor neurons are recruited first followed by increasingly large, powerful, and fast motor neurons, provided a physiological explanation for weakness following a cortical insult. This was especially so with the later work of Evarts² who demonstrated that the frequency of firing of Betz cells (upper motor neurons) in the cerebral cortex specified the strength of isometric contractions. Maintaining a contraction is underpinned by pools of upper and lower motor neurons being recruited in rotation by relay as it were. Reduced populations and sizes of such upper motor neurons would result in poorly sustained firing of neurons and hence weakness. However, in cerebral palsy (CP), the injury is seldom a focal lesion in the motor cortex but more usually bilateral lesions affecting subcortical white matter.³ Moreover, the brains of children with CP are immature and still developing. The work of Sutherland⁴ demonstrating persistence of the wrap-around electromyogram discharges throughout the gait cycle, and subsequently of Leonard,⁵ support a model of 'developmental co-contraction' as the default muscle activation pattern out of which, in the uninjured state, refined, selective motor strategies emerge to provide fluency and elegance of movement. As Leonard and colleagues demonstrated, children with CP never shake off this co-contracting pattern which results in clumsy, poorly executed, and energy-intensive movements. According to the landmark study by Tedroff et al. (p 789) children who have a hemiplegia or diplegia frequently select the wrong muscles when attempting to perform a maximum voluntary contraction. This miss-selection often involves an antagonist or remote muscle from the intended joint of action. A consequence of these findings necessitates a revision of the essence of the motor disorders traditionally referred to as 'spastic'.

Twenty-three years ago, Nashner, in an elegant series of studies of children with CP standing on tilt platforms, demonstrated that they exhibited a reverse wave of muscle activation compared with controls, a phenomenon unrelated to spasticity: a movement disorder.⁶

More useful to carers and parents is the concept of CP as a movement disorder with important developmental consequences. According to this, abnormal synergies, co-contractions, and miss-selections can be viewed as developmental, task-dependent (as with unfamiliar tasks such as walking fast),⁷ or pathological.⁸

After more than 60 years of viewing CP as a series of irreversible muscle contractures waiting to happen, and in an age of functional imaging coupled with a growing interest of cerebral plasticity in recovery from injury, the findings of Tedroff et al. should spawn further fruitful insights into developing motor function.

Their study indicates that muscle strength testing encompasses more than meets the eye and should reawaken an interest in the motor physiology of early brain disorders.

Jean-Pierre Lin

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