Upper Limb Motor Impairment After Stroke



Preeti Raghavan, мо

KEYWORDS

• Stroke • Arm • Weakness • Hemiparesis • Motor control

KEY POINTS

- Weakness or paresis is the key impairment early on that leads to learned nonuse. Sensory impairment, immobility, and chronic pain may further contribute to learned nonuse.
- Spasticity, spastic cocontraction, and abnormal motor synergies occur as recovery proceeds and may lead to abnormal compensatory movements, which if repeated and reinforced lead to learned bad use.
- Impairment in sensorimotor adaptation can lead to transient retention of new skills despite extensive practice; this is referred to as forgetting.

THE NATURE OF UPPER LIMB MOTOR IMPAIRMENT

According to the International Classification of Functioning, Disability and Health (ICF) model,¹ impairments may be described as (1) impairments of body function such as a significant deviation or loss in neuromusculoskeletal and movement-related function related to joint mobility, muscle power, muscle tone, and/or involuntary movements or (2) impairment of body structures such as a significant deviation in structure of the nervous system or structures related to movement, for example, the arm and/or hand. A stroke may lead to both types of impairments. Upper limb impairments after stroke cause the functional limitations in using the affected upper limb after stroke, therefore a clear understanding of the underlying impairments is necessary for restoration of upper limb function. However, understanding upper limb impairments in any given patient is complex for two reasons. (1) The impairments may change; therefore the treatment needs to evolve to target the impairment(s) contributing to dysfunction at a given point in time. (2) Multiple impairments may be present simultaneously, that is, a patient may present with weakness of the arm and hand immediately after a

Funding: NIH R01HD071978.

E-mail address: Preeti.Raghavan@nyumc.org

Phys Med Rehabil Clin N Am 26 (2015) 599–610 http://dx.doi.org/10.1016/j.pmr.2015.06.008 1047-9651/15/\$ – see front matter © 2015 Elsevier Inc. All rights reserved.

pmr.theclinics.com

Motor Recovery Research Laboratory, Department of Rehabilitation Medicine, Rusk Rehabilitation, New York University School of Medicine, 240 East 38th Street, 17th Floor, New York, NY 10016, USA

stroke, which may not have resolved when spasticity sets in a few weeks or months later; hence there may be a layering of impairments over time making it difficult to decide what to treat first. It is useful to review the progression of motor recovery as described by Twitchell² and Brunnstrom³ to understand how impairments may be layered over time (Fig. 1).

UNDERSTANDING MOTOR IMPAIRMENT FROM A FUNCTIONAL PERSPECTIVE

The most useful way to understand how impairments contribute to upper limb dysfunction may be to examine them from the perspective of their functional consequences. There are three main functional consequences of stroke on the upper limb: (1) learned nonuse, (2) learned bad use, and (3) forgetting as determined by behavioral analysis of a task such as reaching for a food pellet and bringing it to the mouth in animal models of stroke.⁴ These consequences are equally valid for human behavior. Each of the functional consequences and the underlying impairments are elaborated in the following sections.

LEARNED NONUSE

Initially after a stroke, individuals may not use their affected upper limb, eventually leading to learned nonuse. Nonuse can result from several impairments. Initially nonuse may occur because of weakness/paralysis or sensory loss. However, as time progresses, nonuse may become habitual and the limb may not be incorporated into functional activities, even though the individual can move it. Now it becomes a learned behavior and is referred to as learned nonuse.

Weakness or paralysis is the predominant impairment that contributes to dysfunction after stroke^{5,6}; it is a direct consequence of the lack of signal transmission from the motor cortex, which generates the movement impulse, to the spinal cord, which executes the movement via signals to muscles. This lack of transmission results in delayed initiation and termination of muscle contraction,⁷ and slowness in developing force,⁸ manifested as an inability to move or move quickly with negative functional consequences. Abnormally increased electromyographic (EMG)-force slopes are



Fig. 1. Sequential progression of motor recovery as described by Twitchell² and Brunnstrom.^{47,48} Note that while recovery is proceeding from one stage to the next, residual impairment from preceding stages may still be present leading to the layering of impairment. Excitatory and inhibitory plasticity are the presumed underlying physiologic processes that could account for progression of recovery from one stage to the next.

Download English Version:

https://daneshyari.com/en/article/4083952

Download Persian Version:

https://daneshyari.com/article/4083952

Daneshyari.com