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Analytical Review: Systematic Search

# Post-stroke Spasticity: Predictors of Early Development and Considerations for Therapeutic Intervention

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**Abstract**

**Objective:** The complexities of post-stroke spasticity (PSS), and the resultant difficulties in treating the disability, present a significant challenge to patients, stroke rehabilitation teams, and caregivers. Reducing the severity of spasticity and its long-term complications may be facilitated by early intervention, making identification of stroke patients at high risk for developing spasticity essential. Factors that predict which patients are at risk for the development of PSS are identified.
**Type:** Systematic search and review
**Literature Survey:** A PubMed search of the following terms was conducted: predictors OR risk factors AND stroke AND spasticity. Studies discussing predictors of early PSS development and factors predictive of motor/functional outcomes and recovery were selected and reviewed in detail.
**Synthesis:** Several predictors of PSS have been proposed, based on studies conducted in patients within 6 months after stroke, including development of increased muscle tone, greater severity of paresis, hemihypesthesia, and low Barthel Index score. Predictors identified in later stages post-stroke (within 12 months) have also proved useful for clinicians, as has the consideration of predictors of motor and functional outcomes and recovery; yet there is a need for additional studies in this area. An understanding of these and other potential predictive factors—such as motor impairment, neurologic and sensory deficit, lesion volume and location, and associated diseases—has not progressed to the same extent and warrants further investigation.
**Conclusion:** The studies discussed in this review support the notion that early identification of factors predictive of PSS should significantly affect the course of intervention, help target individuals who would benefit most from specific types and intensities of therapy, and possibly provide better motor and functional outcomes.

## Introduction

Post-stroke spasticity (PSS) is a complex disorder that can be difficult initially to identify and treat. More than two thirds of stroke survivors develop post-stroke sequelae, including PSS and impaired motor function [1,2]. Early recognition of spasticity, and identification of predictors to assist rehabilitation professionals recognize which stroke patients are at risk for spasticity to develop, could result in earlier treatment and possibly better outcomes. It would be helpful for clinicians and therapists to know which factors help identify patients at high risk for developing severe PSS and for which motor abilities, especially during the initial admission post-stroke. Understanding more about the pathophysiology, evolution of disease, epidemiology, and therapeutic intervention for PSS, along with identifying PSS risk factors, should lead to better care.

### *Definition and Pathophysiology*

Patients with stroke often experience significant impairments that lead to disability. A common motor consequence of stroke is spasticity, defined by Lance as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes ('muscle tone') with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome" [3]. Although Lance's definition is the one most frequently cited, no gold standard definition has been accepted. The difficulty in arriving at a universal definition may

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be due to the enormous variability in the manifestations of spasticity, as evidenced by the many post-stroke patients who exhibit involuntary muscle activity typical of spasticity—and as verified by biomechanical and neurophysiological measures—yet who fail to achieve scores on the Modified Ashworth Scale (MAS) diagnostic for spasticity [4]. Other definitions of spasticity that build on Lance's definition have been proposed, including Young's, which defines the condition as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes that results from abnormal intraspinal processing of primary afferent input, as one component of the upper motor neuron syndrome" [5]. Another definition, from the Task Force on Childhood Motor Disorders, describes spasticity as a form of hypertonia "in which one or both of the following signs are present: (1) resistance to externally imposed movement increases with increasing speed of stretch and varies with the direction of joint movement, and/or (2) resistance to externally imposed movement rises rapidly above a threshold speed or joint angle" [6].

Patients with spasticity comprise a clinically and physiologically recognizable population disabled by two or three factors of motor impairment, including paresis, muscle overactivity, and, with time, soft tissue contracture [7,8] (Figure 1). Paralysis or paresis after the initial neural insult, such as stroke, is defined as having difficulty or being completely unable to voluntarily recruit skeletal motor units to generate torque or movement. Consequently, paresis immediately leaves the affected muscles immobilized. Immobilization of the paretic body part, commonly imposed by the current care environment, causes adaptive shortening of the muscles and joint contracture (soft tissue contracture). Over time, this immobilization and contracture leads to chronic disuse, which further aggravates the consequences of baseline paresis. These gradual changes result in the progressive development of abnormal responses to muscle stretch in the paretic body part, such as increases in velocity-dependent stretch reflexes. Although these abnormal stretch responses have been shown to manifest in multiple ways in patients with central paresis [9,10], the observation of increased velocity-dependent stretch reflexes is a consistent feature [8,11,12]. Hence, patients with spasticity present with a syndrome of muscle overactivity. Muscle overactivity aggravates muscle contracture, which in turn enhances responses to stretch and further aggravates spastic overactivity [7]. Preferential cortical activation of synergistic groups of muscles also interferes with movement and function [13]. Motor impairment in patients with PSS can be described by a cycle of overactivity—contracture—overactivity evolving in parallel with the continuum of paresis—disuse—paresis. To optimize motor recovery and function, both cycles must be disrupted [7,8].

### *Epidemiology, Disability, and Burden of Disease*

The reported prevalence of PSS ranges from 4% to 27% during the first 6 weeks after stroke [14-16]. The rate has been reported to be 19% at 3 months, 21.7% to 42.6% at 4 and 6 months [14-17], and 17% to 38% at 12 months [18-20] post-stroke (Table 1). In addition to the increased muscle tone of PSS, indirect effects, such as

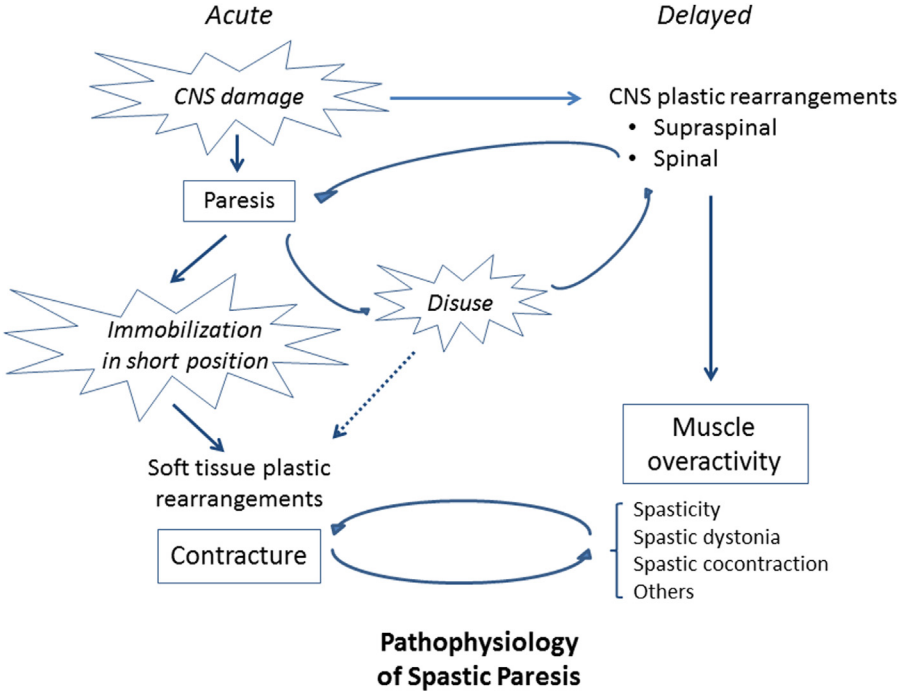


**Figure 1.** Development of spastic paresis [8]. Solid arrows represent established causal relationships; dashed arrow represents a conjectural connection.