



Abnormal Interactions Between Arm and Leg Movements After Cerebral Stroke

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Article Info

Article history:

Received: 06 April 2020

Editor: 08 April 2020

Reviewed: 29 April 2020

Revised: 08 May 2020

Published: 19 May 2020

Keywords:

Stroke; Motor control; Interlimb coordination; Upper limb; Lower limb; Gait; Reaching; Functional connectivity; Rehabilitation; Motor recovery

Abstract

Stroke is a leading cause of long-term disability, frequently resulting in motor impairments affecting both upper and lower limbs. While much research has focused on the independent deficits of the arm and leg, it is increasingly recognized that stroke disrupts the complex interplay and mutual influences between these limbs, critical for coordinated activities like walking, reaching, and maintaining balance. We discuss the neural substrates supporting interlimb coordination and how stroke-induced damage, particularly to descending motor pathways and cortical/subcortical networks, disrupts these mechanisms. The review explores observed abnormalities, including altered gait patterns influenced by upper limb support, changes in upper limb kinematics related to postural stability provided by the lower limbs, and the emergence of pathological coupling or synergies that constrain independent limb movement. Assessment methods for quantifying these interlimb dependencies are discussed, ranging from kinematic analysis during functional tasks to neurophysiological techniques. Finally, we consider rehabilitation strategies that implicitly or explicitly address interlimb coordination deficits, highlighting the importance of task-specific training that integrates upper and lower limb movements. Understanding these abnormal mutual influences is crucial for developing more effective, holistic rehabilitation approaches aimed at restoring functional independence after stroke.

Introduction

Stroke is a devastating neurological event that commonly leads to persistent motor deficits, significantly impacting an individual's ability to perform activities of daily living. The resulting hemiparesis or hemiplegia typically affects one side of the body, impairing voluntary control of both the upper (arm, hand) and lower (leg, foot) limbs [1]. While clinical assessment and rehabilitation often focus on restoring function in each limb individually, it is critical to acknowledge that most functional movements, such as walking, standing up, reaching for objects while standing, or manipulating objects while seated, require coordinated action and interaction between the upper and lower extremities [2]. The nervous system employs intricate neural circuits to ensure smooth and efficient coordination between the limbs. These circuits involve complex interactions between cortical areas, subcortical structures (basal ganglia, cerebellum), brainstem nuclei, and spinal cord networks [3]. Descending pathways, notably the corticospinal tract, play a crucial role in mediating voluntary control and fine motor skills, while other pathways like the reticulospinal tract contribute significantly to posture, balance, and gait [4].

Stroke-induced damage to these neural substrates can disrupt not only the control of individual limbs but also the communica-

tion and mutual influences between them. For instance, impaired trunk control due to stroke can compromise the stable base required for effective upper limb reaching [5]. Conversely, reliance on the less affected upper limb for support can alter weight bearing and gait symmetry in the lower limbs [6]. These abnormal interlimb influences can perpetuate maladaptive movement patterns, limit functional recovery, and increase the risk of secondary complications such as falls [7]. Despite the clear functional importance of interlimb coordination, research and clinical practice have often compartmentalized the assessment and treatment of upper and lower limb impairments after stroke. By examining the neural basis of interlimb coordination, the specific ways stroke disrupts these interactions, methods for their assessment, and relevant rehabilitation approaches, we hope to underscore the need for a more integrated perspective in stroke recovery.

Neural Substrates of Interlimb Coordination

Effective coordination between the upper and lower limbs relies on the seamless integration of sensory and motor information processed by a distributed network of brain and spinal cord regions. Understanding the healthy function of these pathways is essential for appreciating how stroke disrupts them. At the spinal cord level, Central Pattern Generators (CPGs) are thought to contribute to rhythmic activities like walking, coordinating the stepping patterns of the legs [8]. While CPGs are primarily

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Citation: Blake S, Robinson L (2020). Temporal Dynamics of Brain Integration During Sleep and Arousal Shifts. *J Exp Bio Physiol*; 7:039.

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associated with locomotion, there is evidence for interlimb coordination mechanisms within the spinal cord that link activity between upper and lower limbs, particularly during rhythmic movements [9].

Descending motor pathways provide voluntary control and modulate spinal activity. The corticospinal tract, originating primarily from the motor cortex, is crucial for fine motor control and independent limb movements [4]. Fibers from the motor cortex also project to brainstem nuclei, which in turn give rise to pathways like the reticulospinal tract. The reticulospinal tract plays a significant role in postural control, gait, and coordinating gross movements involving multiple body segments, including the trunk and limbs [10]. Damage to these descending pathways due to stroke can directly impair the brain's ability to control and coordinate limb movements. Cortical areas involved in planning and executing movements, such as the primary motor cortex, premotor cortex, and supplementary motor area, interact extensively with subcortical structures like the basal ganglia and cerebellum [11]. The basal ganglia are involved in initiating and scaling movements, as well as selecting appropriate motor programs [12]. The cerebellum is critical for motor learning, coordination, balance, and error correction [13]. Lesions in any of these areas can disrupt the timing, sequencing, and amplitude of movements, leading to discoordination between limbs.

Furthermore, sensory information from the limbs (proprioception, touch) is relayed to the brain via ascending pathways and is crucial for online motor control and adaptation. Integration of this sensory feedback within cortical and subcortical circuits allows for adjustments to ongoing movements and contributes to the smooth interplay between limbs [14]. Stroke can impair sensory processing, further compromising interlimb coordination. The complex interplay between these neural structures ensures that upper and lower limb movements are appropriately timed, scaled, and sequenced to achieve functional goals, whether it's maintaining balance while reaching or using arm swing to enhance gait efficiency.

Normal Interlimb Coordination During Functional Tasks

In healthy individuals, the upper and lower limbs work in concert during a wide range of activities. Examples include:

Locomotion: Walking involves rhythmic, alternating movements of the legs, coupled with coordinated arm swing [15]. Arm swing contributes to balance, reduces metabolic cost, and helps regulate gait speed [16]. The timing and amplitude of arm swing are coupled to the stepping cycle, demonstrating a clear interlimb dependency [15].

Reaching and Grasping While Standing: Reaching for an object while standing requires anticipatory postural adjustments in the lower limbs and trunk to maintain balance as the center of mass shifts [17]. The stability provided by the lower limbs and trunk forms the foundation for accurate and efficient upper limb movement [5].

Sit-to-Stand: This transition involves coordinated extension of the lower limbs and trunk, often accompanied by arm movements (e.g., pushing off from armrests or forward arm swing) that assist in generating vertical momentum [18].

Manipulation While Seated: Even when seated, trunk stability provided by the lower body and core muscles is important for supporting dexterous hand movements [5].

These examples illustrate that upper and lower limb movements

are rarely isolated but are components of larger, integrated motor programs. The nervous system orchestrates these movements by coordinating activity across multiple joints and body segments, relying on the distributed neural networks described previously.

Abnormalities of Interlimb Influences After Stroke

Stroke-induced damage disrupts the neural pathways and networks supporting interlimb coordination, leading to various abnormal patterns of interaction between the upper and lower limbs. The nature and severity of these abnormalities depend on the location and extent of the lesion [19].

Impact on Descending Motor Pathways: Damage to the corticospinal tract is a common consequence of stroke and is strongly associated with impaired voluntary control, weakness, and spasticity in the contralateral limbs [4]. While the corticospinal tract is primarily known for controlling distal limb muscles, it also influences proximal muscles and contributes to the coordination of multi-joint movements. Lesions affecting the corticospinal tract can therefore disrupt the finely tuned timing and scaling of muscle activity required for coordinated interlimb actions [20]. Damage to brainstem pathways, such as the reticulospinal tract, can profoundly affect postural control, balance, and gait [10]. Since these pathways are critical for coordinating trunk and limb movements, their disruption can lead to abnormal coupling between the upper and lower body, affecting activities like walking and reaching while standing [21]. For example, impaired reticulospinal control can result in reduced or absent arm swing during gait [15].

Abnormal Coupling Patterns and Synergies: A hallmark of motor impairment after stroke is the emergence of abnormal muscle synergies – fixed patterns of muscle activation that constrain independent joint movements [22]. While synergies are often described within a single limb (e.g., flexor synergy of the upper limb), there is evidence for abnormal coupling between limbs. For instance, attempts to move the paretic upper limb might involuntarily trigger activity in the paretic lower limb, or vice versa [23]. These pathological interlimb synergies can interfere with functional tasks, making it difficult to perform movements that require dissociation between limb segments. Excessive co-contraction of antagonist muscles is another common abnormality after stroke [24]. While co-contraction can provide stability, excessive levels can impair movement efficiency and flexibility. This can occur not only within a limb but also between limbs, where muscles in one limb co-contract in response to movement demands in the other, hindering smooth coordination [25].

Influence of Upper Limb Impairment on Gait and Balance: Although gait is primarily a lower limb function, upper limb function significantly influences it after stroke. Patients often rely on the less affected upper limb for support, either by holding onto objects or using assistive devices [6]. This reliance can lead to asymmetrical weight-bearing, altered trunk kinematics, and compensatory movements in the less affected leg, contributing to inefficient and unstable gait patterns [26]. Reduced or absent arm swing on the paretic side also contributes to gait asymmetry and reduced balance [15]. Furthermore, impaired upper limb function can limit the ability to use arm movements for reactive balance control, increasing the risk of falls [7].

Influence of Lower Limb Impairment on Upper Limb Function: Conversely, lower limb and trunk impairments after stroke can significantly impact upper limb function. Poor postural stability due to weakness or impaired control in the lower limbs and

trunk provides an unstable base for upper limb movements, particularly during reaching tasks that require shifts in the center of mass [5]. Patients may compensate by reducing reach distance, altering movement trajectories, or relying excessively on the less affected side, all of which limit the functional use of the paretic upper limb [27]. The ability to generate and control ground reaction forces through the lower limbs is also crucial for initiating and executing many upper limb actions, and this is compromised after stroke [28] (Table 1).

Assessment Methods for Interlimb Coordination Abnormalities

Quantifying abnormalities in interlimb coordination after stroke requires methods that can capture the dynamic interplay between the limbs during functional tasks. Traditional clinical scales often assess limbs independently, but more nuanced approaches are needed.

Kinematic Analysis: Motion capture systems can track the trajectories, velocities, and angles of multiple body segments simultaneously during tasks like walking, reaching, or sit-to-stand [29]. Analysis of the relative timing, phasing, and amplitude of movements between upper and lower limbs can reveal discoordination and abnormal coupling patterns [15, 27]. For example, analyzing the coupling between hip flexion/extension and shoulder flexion/extension during gait provides insight into arm-leg coordination [15].

Kinetic Analysis: Force plates and instrumented treadmills can measure ground reaction forces and weight distribution during standing and walking [6]. Analyzing weight-bearing asymmetry or the timing of force application can indicate how upper limb support strategies impact lower limb function [26].

Electromyography (EMG): Surface or intramuscular EMG can record muscle activation patterns in multiple muscles across different limbs [23]. Analyzing the onset, duration, and amplitude of muscle activity during interlimb tasks can reveal abnormal co-contraction or synergistic activation patterns [25].

Functional Connectivity (fMRI, EEG, MEG): Neuroimaging techniques can assess the functional coupling between brain regions involved in motor control during rest or task performance [19]. While challenging to perform during complex movements, these methods can identify altered network interactions that may underlie interlimb discoordination [11]. For instance, re-

duced connectivity between motor areas controlling the upper and lower limbs might be observed.

Clinical Observational Scales: While less quantitative, experienced clinicians can use observational gait analysis or task-specific assessments to identify visible signs of interlimb discoordination, such as reduced arm swing, trunk-limb coupling, or compensatory strategies [1].

Combining these methods provides a more comprehensive picture of the nature and severity of interlimb coordination deficits after stroke.

Rehabilitation Approaches Targeting Interlimb Coordination

Given the functional importance of coordinated interlimb movement, rehabilitation strategies should ideally address these deficits explicitly or implicitly. While many conventional therapies focus on individual limb training, approaches that integrate upper and lower limb activity are gaining recognition.

Task-Specific Training: Engaging patients in functional tasks that naturally require interlimb coordination, such as walking with arm swing, reaching for objects while standing, or practicing sit-to-stand transfers, can promote the relearning of coordinated movement patterns [30]. This approach emphasizes practicing the interaction between limbs in a meaningful context.

Treadmill Training with Body Weight Support: Treadmill training is a common intervention for gait rehabilitation. Incorporating arm swing or using poles can help facilitate more symmetrical and coordinated gait patterns by promoting the natural coupling between upper and lower limbs during locomotion [31].

Dual-Task Training: Requiring patients to perform an upper limb task while simultaneously performing a lower limb task (e.g., manipulating objects while walking) can challenge and potentially improve interlimb coordination and the ability to manage multiple motor demands [32].

Trunk Rehabilitation: Addressing trunk weakness and impaired control is crucial, as trunk stability provides the necessary base for both upper and lower limb function [5]. Exercises targeting core strength and control can indirectly improve interlimb coordination.

Table 1: Examples of Abnormal Interlimb Influences After Stroke.

Impaired Limb/Function	Influenced Limb/Function	Observed Abnormality	Potential Underlying Mechanism
Paretic Upper Limb	Paretic Lower Limb	Involuntary lower limb muscle activation during upper limb movement attempts.	Pathological interlimb synergies, abnormal descending drive
Paretic Lower Limb	Paretic Upper Limb	Impaired reach distance or trajectory due to poor postural stability.	Deficits in anticipatory postural adjustments, trunk control
Trunk Control	Both Upper and Lower Limbs	Reduced ability to dissociate limb movements from trunk, leading to stiff, uncoordinated actions.	Damage to reticulospinal pathways, core muscle weakness/spasticity
Less Affected Upper Limb	Paretic Lower Limb	Increased weight-bearing on less affected side, leading to asymmetrical gait.	Compensatory strategy for balance and propulsion
Reduced Arm Swing	Gait/Balance	Increased gait asymmetry, reduced walking speed, decreased balance confidence.	Disrupted interlimb coupling, loss of counter-rotation assistance

Robotics and Technology: Robotic devices can be used to assist or challenge interlimb coordination during specific tasks, allowing for repetitive, controlled practice [33]. Virtual reality can also create immersive environments for practicing complex interlimb movements in a motivating way [34].

Neurophysiological Interventions: Techniques like transcranial magnetic stimulation (TMS) or transcranial direct current stimulation (tDCS) have been explored to modulate excitability in brain regions involved in interlimb control, potentially facilitating motor learning and coordination [35].

While the evidence bases specifically targeting abnormal mutual influences.

Discussion

Stroke-induced motor deficits extend beyond individual limb impairments to disrupt the complex and crucial mutual influences between the upper and lower extremities. Damage to descending pathways, particularly the corticospinal and reticulospinal tracts, and disruption of cortical-subcortical networks are key factors underlying these abnormalities. The resulting pathological coupling, altered gait patterns influenced by upper limb compensation, and impaired upper limb function due to poor postural support underscore the interconnected nature of post-stroke motor impairments. Understanding these abnormal interlimb influences is not merely an academic exercise; it has significant implications for clinical assessment and rehabilitation. Focusing solely on individual limb function risks overlooking critical barriers to functional recovery. For instance, a patient might regain some isolated strength in their paretic arm, but if trunk instability prevents them from using that arm effectively during standing or walking, the functional gain is limited. Similarly, improving leg strength might not translate into improved gait if abnormal interlimb synergies constrain the movement pattern.

The assessment methods discussed, ranging from kinematic analysis to neurophysiological techniques, provide tools to quantify these complex interactions. Incorporating such assessments into clinical practice could lead to a more precise understanding of a patient's specific interlimb coordination deficits and inform individualized treatment plans. The rehabilitation approaches reviewed suggest that training that integrates upper and lower limb movements within functional tasks is likely to be more effective in restoring coordinated control than isolated limb exercises. Task-specific training, treadmill training with arm swing, and dual-task paradigms all require the nervous system to coordinate activity across different body segments, promoting the relearning of interlimb dependencies. Despite the growing recognition of interlimb coordination issues, there was a need for more standardized and clinically feasible assessment tools specifically designed to quantify interlimb coordination deficits. Furthermore, the optimal parameters and efficacy of different integrated rehabilitation approaches required further investigation through rigorous clinical trials. The precise neural mechanisms underlying the recovery of interlimb coordination after stroke, and how different interventions modulate these mechanisms, were also areas requiring more research.

Future research directions should include longitudinal studies to track the evolution of interlimb coordination deficits over the course of recovery, investigating the relationship between specific lesion locations and types of interlimb abnormalities, and utilizing advanced neuroimaging and stimulation techniques to understand and modulate the underlying neural circuits. De-

veloping and validating clinically practical tools for assessing interlimb coordination would also be a valuable contribution.

Conclusion

Stroke disrupts the intricate mutual influences between the upper and lower limbs, leading to abnormal coordination patterns that significantly impact functional independence. These abnormalities stem from damage to distributed neural networks controlling movement and posture. Recognizing and addressing these interlimb dependencies is crucial for effective stroke rehabilitation. While research prior to 2020 had begun to shed light on the nature of these deficits and potential integrated rehabilitation strategies, continued investigation was needed to refine assessment methods, optimize interventions, and deepen our understanding of the neural mechanisms of recovery. A holistic approach that considers the interplay between the upper and lower limbs within the context of functional tasks holds promise for improving motor outcomes and enhancing the quality of life for stroke survivors.

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