

Journal of Physical Medicine and Rehabilitation Research

Review Article

Prevalence and Risk Factors of post-stroke hemiparesis in stroke survivors

Pedersen M^{1*}, Larsen A² and Karlsson K³

¹Division of Rehabilitation Research, Tajo Center for Physical Medicine, Iceland

²Department of Clinical Rehabilitation Research, Shannon Institute of Rehabilitation Research, Iceland

³Department of Physical Medicine, Mackenzie Center for Physical Medicine, Sweden

*Address for Correspondence: Magnus Pedersen, Division of Rehabilitation Research, Tajo Center for Physical Medicine, Iceland; E-mail: magnus.pedersen@tajo.edu

Received: 29 April 2024; Accepted: 27 July 2024; Published: 27 October 2024.

Citation of this article: Pedersen M, Larsen A, Karlsson K (2024) Prevalence and Risk Factors of post-stroke hemiparesis in stroke survivors. J Phys Med Rehabil Res, 6(1): 01-06. DOI: [10.5281/zenodo.22815091](https://doi.org/10.5281/zenodo.22815091)

Copyright: © 2024 Magnus Pedersen, et al. This is an open access article distributed under the [Creative Commons Attribution License](#), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Background: Hemiparesis affects up to three-quarters of stroke survivors in the acute period and remains the most common persisting motor impairment after stroke, driving much of the long-term disability burden captured in global stroke registries.

Objective: This review synthesizes published evidence on the prevalence of post-stroke hemiparesis across the acute, subacute, and chronic phases of recovery, and on the demographic, clinical, neuroanatomical, and biochemical factors that predict its occurrence and persistence.

Methods: We synthesized population-based incidence studies, hospital and rehabilitation cohort studies, and systematic reviews addressing motor impairment prevalence, upper- and lower-limb weakness, spasticity, and recovery trajectories after ischemic and hemorrhagic stroke.

Findings: Reported prevalence of any motor weakness at onset ranges from about 50% to 80%, with roughly two-thirds of survivors showing residual upper-limb impairment at six months; age, initial stroke severity, lesion volume and location, diabetes, and delayed rehabilitation onset predict poorer motor outcome and persistent hemiparesis, while early intensive therapy and favorable corticospinal tract integrity favor more complete recovery.

Conclusions: Post-stroke hemiparesis is prevalent, multifactorial, and only partly modifiable once established, underscoring early risk stratification, timely rehabilitation, and continued investment in biomarkers identifying survivors most likely to benefit from intensive motor therapy.

Keywords: stroke; hemiparesis; motor impairment; prevalence; risk factors

1. Introduction

Stroke remains one of the leading causes of acquired adult disability worldwide, and among its many consequences, motor weakness of one side of the body, hemiparesis, is the single most common and most functionally consequential impairment survivors face [1,2]. Global burden-of-disease estimates place stroke among the top contributors to disability-adjusted life years lost, and a large share of that burden is attributable not to mortality but to the years survivors spend living with impaired mobility, reduced upper-limb function, and dependence in basic activities of daily living [3]. Hemiparesis arises when ischemic or hemorrhagic injury disrupts the corticospinal and corticobulbar pathways that carry voluntary motor commands from the primary motor cortex, premotor areas, and their descending projections through the internal capsule and brainstem to the spinal cord, and because these pathways are anatomically concentrated in a relatively small volume of tissue supplied largely by the middle cerebral artery, even modest infarcts in strategic locations can produce weakness disproportionate to overall lesion size [4].

Population-based and hospital-based cohort studies converge on a broadly consistent picture despite differences in stroke severity mix, case ascertainment method, and timing of assessment: motor weakness is present in roughly half to four-fifths of patients at stroke onset, and while a substantial proportion recover useful function within the first weeks, a large minority are left with persistent, and often permanent, hemiparesis that shapes their long-term quality of life and caregiving needs [5,6]. This review synthesizes the epidemiological literature on the prevalence of post-stroke hemiparesis across the acute, subacute, and chronic phases of recovery, and examines the demographic, clinical, neuroanatomical, and biological factors that the literature has identified as predicting its occurrence, severity, and persistence, with the goal of clarifying where risk stratification and early intervention are best targeted.

2. Prevalence in the acute phase

Estimates of motor weakness at stroke onset vary considerably depending on how "hemiparesis" is defined and how soon after onset patients are assessed. Lawrence and colleagues, in one of the more comprehensive early population-based surveys of stroke-related impairment, found that approximately 80% of first-ever stroke patients had some degree of limb weakness in the acute period, with roughly half showing weakness severe enough to prevent independent ambulation without assistance [5]. Other population-based incidence studies using less stringent motor thresholds report figures closer to 50-60% for clinically apparent weakness at presentation, reflecting the sensitivity of prevalence estimates to whether mild, subclinical paresis detected only on formal strength testing is counted alongside overt hemiplegia [7].

The Copenhagen Stroke Study, a large prospective cohort that has shaped much of the subsequent literature on stroke motor recovery, documented initial-onset paresis in the great majority of its cohort and used serial Scandinavian Stroke Scale assessments to track the trajectory of that weakness across the following months, establishing benchmark data against which later studies have been compared [8]. Similarly, community-based registries in the Oxfordshire Community Stroke Project found that limb weakness was among the most frequent presenting deficits across all stroke subtypes, though the proportion affected and the severity of weakness differed meaningfully between lacunar, partial anterior circulation, total anterior circulation, and posterior circulation syndromes, with total anterior circulation infarcts carrying the highest prevalence of severe, complete hemiplegia [9]. These classification-based differences matter clinically because they mean prevalence figures pooled across unselected stroke populations obscure how strongly weakness clusters within specific stroke subtypes and vascular territories.

3. Prevalence across the subacute and chronic



phases

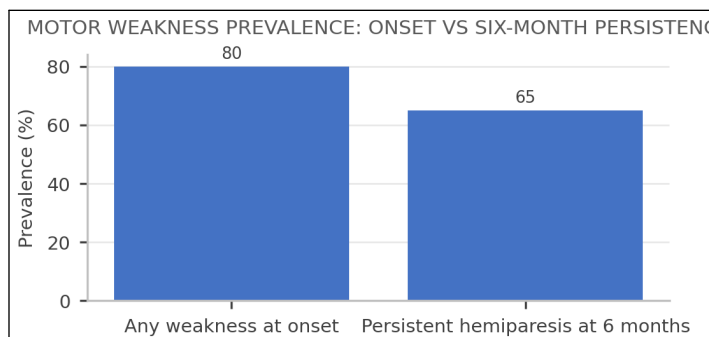


Figure 1: Reported prevalence of any motor weakness at stroke onset compared with the proportion of survivors with persistent residual hemiparesis at six months, drawn from population-based and cohort follow-up data [5,6].

The trajectory of motor recovery after stroke follows a broadly predictable pattern in which the greatest gains occur within the first one to three months, after which the rate of further spontaneous improvement slows substantially, leaving a residual deficit that for many patients persists indefinitely [6,10]. Kwakkel and colleagues, synthesizing longitudinal cohort data on upper-limb recovery, reported that approximately 65% of stroke survivors with initial arm paresis had a persistent upper-limb motor deficit at six months, and that the proportion achieving full, dexterous recovery of the paretic arm was substantially smaller than the proportion regaining basic ambulation, underscoring that lower-limb and upper-limb recovery trajectories diverge considerably [6].

Nakayama and colleagues, following the Copenhagen Stroke Study cohort through one year, found that the plateau in motor recovery for both arm and leg function was typically reached within 11 weeks of stroke onset for most patients, with only a small proportion showing continued meaningful gains beyond that window, a finding that has informed the timing of intensive rehabilitation protocols in subsequent decades [11]. Longer-term follow-up studies extending into the chronic phase, one year and beyond, consistently find that a substantial minority, often cited in the range of 30-40% of

survivors, remain functionally dependent because of persistent hemiparesis and its downstream consequences for gait, balance, and manual dexterity [12,13]. Wade and colleagues, in some of the earliest systematic community-based follow-up work on stroke outcomes, similarly documented that residual motor impairment, more than any single other deficit, predicted whether a survivor returned to independent community living [14]. Hendricks and colleagues, in a systematic review of motor recovery after stroke spanning several decades of cohort data, estimated that roughly 30% of survivors are left with a severe, persistent hemiparetic arm, a figure that has remained relatively stable across studies conducted before and after the widespread adoption of thrombolysis and organized stroke unit care, suggesting that acute reperfusion therapies alone have not eliminated the long-term motor burden of stroke [15]. The Framingham Study's long-term follow-up of community-dwelling stroke survivors, reported by Kelly-Hayes and colleagues, similarly found that hemiparesis persisting beyond six months was strongly associated with reduced likelihood of returning to independent living and with greater need for institutional or family caregiving [16].

4. Demographic and clinical risk factors

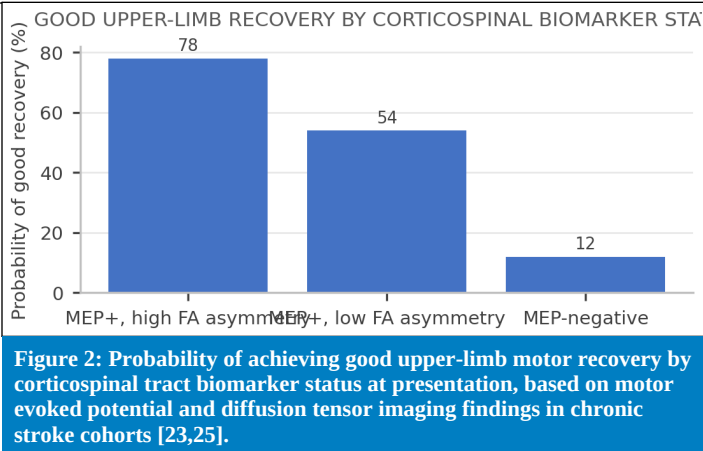
Age is among the most consistently reported predictors of both the occurrence and the severity of post-stroke hemiparesis, with older patients showing not only a higher initial prevalence of weakness but also a slower and less complete recovery trajectory, likely reflecting a combination of reduced neuroplastic capacity, greater burden of pre-existing small-vessel disease, and higher rates of comorbid conditions that independently impair recovery [11,8]. Initial stroke severity, most commonly quantified using the National Institutes of Health Stroke Scale, is the single strongest clinical predictor available at presentation: patients with higher initial NIHSS scores have both a higher probability of presenting with hemiparesis and a substantially lower probability of achieving independent motor function by three to six

months [17].

Table 1: Demographic and clinical factors associated with occurrence and persistence of post-stroke hemiparesis [11,8,18,20].		
Factor	Association with hemiparesis	Proposed mechanism
Older age	Higher initial prevalence, slower recovery	Reduced neuroplastic capacity, comorbid small-vessel disease
Higher initial NIHSS score	Higher prevalence and severity	Larger or more strategically located infarct
Diabetes mellitus	Worse motor outcome	Microvascular disease, impaired collateral flow
Female sex	Inconsistent; largely confounded	Older age at onset, higher atrial fibrillation prevalence

Diabetes mellitus has been repeatedly identified as an independent risk factor for both more severe initial weakness and poorer motor recovery, plausibly through mechanisms involving microvascular disease, impaired collateral circulation, and adverse effects of chronic hyperglycemia on neuronal and axonal repair processes [8,18]. Hypertension, atrial fibrillation, and prior stroke similarly predict worse motor outcomes, both directly through their association with larger or recurrent infarcts and indirectly through their contribution to a less favorable pre-stroke functional and cognitive reserve [19]. Sex differences in hemiparesis prevalence and recovery have been reported inconsistently across studies; some cohorts find women present with more severe initial deficits and slower functional recovery, a pattern that appears to be confounded substantially by women's older age at stroke onset and higher prevalence of atrial fibrillation rather than representing a true biological sex effect [20]. Gresham and colleagues' long-term Framingham community follow-up of stroke survivors similarly identified older age and greater initial neurological deficit as the two clinical variables most strongly and consistently linked to poor long-term functional outcome, a finding echoed by nearly every subsequent large cohort study of stroke recovery [21].

5. Neuroanatomical and lesion-related predictors



Lesion location and volume are central determinants of both the presence and the severity of hemiparesis, reflecting the concentrated anatomy of descending motor pathways. Infarcts or hemorrhages involving the posterior limb of the internal capsule, the corona radiata, or the pyramidal tract in the brainstem tend to produce weakness disproportionate to their volume because these structures carry a densely packed, somatotopically organized bundle of corticospinal fibers with little redundancy [22,4]. Schiemanck and colleagues demonstrated that lesion volume measured on early MRI, together with the degree of corticospinal tract involvement, predicted subsequent motor and functional outcome more accurately than clinical severity scores alone in some cohorts, supporting a role for structural imaging in early prognostication [22].

Diffusion tensor imaging studies have refined this picture further by quantifying corticospinal tract integrity directly rather than inferring it from lesion location. Stinear and colleagues showed that fractional anisotropy asymmetry of the corticospinal tract, combined with the presence or absence of motor evoked potentials elicited by transcranial magnetic stimulation, substantially improved prediction of upper-limb motor outcome relative to clinical assessment alone, forming the basis of the widely used PREP and PREP2 prediction algorithms [23,24]. Patients in whom motor evoked potentials could be elicited early after stroke had a markedly better prognosis

for functional arm recovery than those in whom the corticospinal tract appeared electrophysiologically silent, a distinction with direct implications for how intensively upper-limb therapy should be targeted [23]. Byblow and colleagues extended this biomarker framework prospectively, validating that algorithm-based prediction using clinical scores, transcranial magnetic stimulation, and, where available, imaging measures could stratify patients into recovery categories with clinically useful accuracy soon after stroke onset [25].

6. Spasticity, secondary complications, and functional consequences

Hemiparesis rarely occurs in isolation from other motor abnormalities, and spasticity is among the most clinically significant secondary complications, developing in a substantial minority of patients with more severe initial paresis, typically emerging over the weeks following stroke as altered descending inhibitory control and adaptive changes in spinal reflex circuits take hold [26,27]. Sommerfeld and colleagues found that spasticity, when present, was strongly associated with more severe residual disability and slower functional recovery, though the causal direction, whether spasticity itself impedes recovery or simply co-occurs with more severe underlying corticospinal injury, remains debated in the literature [27].

Shoulder pain, contracture, and falls are common downstream consequences of persistent hemiparesis that compound its direct functional impact. Ada and colleagues catalogued the high prevalence of hemiplegic shoulder pain in patients with more severe arm weakness, linking it to abnormal joint mechanics, disuse, and, in some cases, associated subluxation, all of which further limit rehabilitation participation and quality of life [28]. Fall risk is substantially elevated in stroke survivors with residual hemiparesis because of impaired limb control, reduced proprioceptive feedback, and asymmetric weight-bearing during gait, and falls in this population carry a disproportionately high risk of fracture and further

loss of independence [29].

7. Rehabilitation timing, intensity, and modifiable factors

Among the factors that appear genuinely modifiable, the timing and intensity of rehabilitation stand out as the most actionable. Langhorne and colleagues, reviewing evidence on organized stroke unit care and early mobilization, found that patients treated in dedicated stroke units with structured, early rehabilitation had better motor and functional outcomes than those managed on general medical wards, a finding replicated across multiple health systems and now embedded in international stroke care guidelines [30,12]. Coupar and colleagues, in a systematic review of predictors of upper-limb recovery, similarly found that earlier initiation and greater intensity of task-specific therapy were associated with better motor outcomes, even after accounting for baseline severity, supporting a causal rather than purely prognostic role for rehabilitation dose [31].

Constraint-induced movement therapy and other high-intensity, task-oriented approaches have shown particular promise for patients with residual but usable motor function in the paretic limb, though their benefit appears smaller or absent in patients with the most severe, motor-evoked-potential-negative weakness, reinforcing the importance of biomarker-guided patient selection for intensive interventions [32,24]. Pollock and colleagues, in a Cochrane review of physical rehabilitation approaches for the arm and hand after stroke, found that no single therapeutic approach demonstrated clear superiority over others, but that overall dose of practice, rather than the specific technique used, was the more consistent predictor of improvement, a conclusion with direct implications for how rehabilitation services are resourced [33].

Table 2: Rehabilitation-related predictors of upper-limb motor recovery after stroke, from systematic reviews and randomized trials [30,31,32,33].

Intervention / factor	Evidence source	Association with recovery
Organized stroke unit care, early mobilization	[30]	Better motor and functional outcomes
Earlier, higher-intensity task-specific therapy	[31]	Better upper-limb recovery independent of baseline severity
Constraint-induced movement therapy	[32]	Improved function in patients with usable residual movement
Overall practice dose vs specific technique	[33]	Dose more consistently predicts improvement than technique

8. Discussion

Taken together, the epidemiological literature supports several consistent conclusions about post-stroke hemiparesis. Its prevalence at onset is high, commonly cited in the range of 50-80% depending on ascertainment method and stroke severity mix, and while a meaningful proportion of patients recover substantial motor function within the first three months, a large minority are left with persistent weakness that shapes long-term disability, dependence, and caregiver burden [5,6,12]. Age, initial stroke severity, diabetes, and lesion characteristics, including volume, location, and corticospinal tract integrity, are the risk factors most consistently associated with both the occurrence and the persistence of hemiparesis across studies spanning different populations and decades [11,8,22,23].

The clinical implication of this body of evidence is twofold. First, early and accurate prognostic stratification, increasingly informed by biomarkers such as motor evoked potentials and corticospinal tract fractional anisotropy alongside clinical severity scores, allows rehabilitation resources to be directed toward patients most likely to benefit from intensive, task-specific therapy, while setting realistic expectations for patients unlikely to achieve substantial motor recovery [24,25]. Second, the strength and consistency of the relationship between rehabilitation timing and intensity and eventual motor outcome argues for organized, early, and sufficiently

intensive rehabilitation as one of the few genuinely modifiable levers available once hemiparesis has occurred, a conclusion reinforced by systematic reviews spanning stroke unit organization, task-specific practice, and constraint-induced approaches [30,31,33].

9. Conclusion

Post-stroke hemiparesis remains one of the most prevalent and functionally consequential impairments after stroke, affecting roughly half to four-fifths of survivors at onset and leaving a substantial minority with persistent motor deficits that drive long-term disability [1,5,6]. Its occurrence and severity are shaped by a combination of largely non-modifiable factors, age, lesion location, and corticospinal tract integrity, and modifiable factors centered on the timing and intensity of rehabilitation [4,23,30]. Continued refinement of prognostic biomarkers, alongside sustained investment in early, intensive, and appropriately targeted rehabilitation services, offers the most realistic path to reducing the long-term burden that hemiparesis places on stroke survivors and their caregivers [24,31].

10. References

1. Feigin, V.L., Stark, B.A., Johnson, C.O., Roth, G.A., Bisignano, C., Abady, G.G., et al. (2021) Global, regional, and national burden of stroke and its risk factors, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet Neurology, 20(10): 795-820. DOI: 10.1016/S1474-4422(21)00252-0

2. GBD 2016 Neurology Collaborators (2019) Global, regional, and national burden of neurological disorders, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet Neurology, 18(5): 459-480. DOI: 10.1016/S1474-4422(18)30499-X

3. Johnson, C.O., Nguyen, M., Roth, G.A., Nichols, E., Alam, T., Abate, D., et al. (2019) Global, regional, and national burden of stroke, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet Neurology, 18(5):

- 439-458. DOI: [10.1016/S1474-4422\(19\)30034-1](https://doi.org/10.1016/S1474-4422(19)30034-1)
4. Ward, N.S. (2004) Functional reorganization of the cerebral motor system after stroke. *Current Opinion in Neurology*, 17(6): 725-730. DOI: [10.1097/00019052-200412000-00013](https://doi.org/10.1097/00019052-200412000-00013)
5. Lawrence, E.S., Coshall, C., Dundas, R., Stewart, J., Rudd, A.G., Howard, R., et al. (2001) Estimates of the prevalence of acute stroke impairments and disability in a multiethnic population. *Stroke*, 32(6): 1279-1284. DOI: [10.1161/01.STR.32.6.1279](https://doi.org/10.1161/01.STR.32.6.1279)
6. Kwakkel, G., Kollen, B.J., van der Grond, J., Prevo, A.J. (2003) Probability of regaining dexterity in the flaccid upper limb: impact of severity of paresis and time since onset in acute stroke. *Stroke*, 34(9): 2181-2186. DOI: [10.1161/01.STR.0000087172.16305.CD](https://doi.org/10.1161/01.STR.0000087172.16305.CD)
7. Rathore, S.S., Hinn, A.R., Cooper, L.S., Tyroler, H.A., Rosamond, W.D. (2002) Characterization of incident stroke signs and symptoms: findings from the Atherosclerosis Risk in Communities study. *Stroke*, 33(11): 2718-2721. DOI: [10.1161/01.STR.0000035286.87503.31](https://doi.org/10.1161/01.STR.0000035286.87503.31)
8. Jorgensen, H.S., Reith, J., Nakayama, H., Kammersgaard, L.P., Raaschou, H.O., Olsen, T.S. (1999) What determines good recovery in patients with the most severe strokes? The Copenhagen Stroke Study. *Stroke*, 30(10): 2008-2012. DOI: [10.1161/01.STR.30.10.2008](https://doi.org/10.1161/01.STR.30.10.2008)
9. Bamford, J., Sandercock, P., Dennis, M., Burn, J., Warlow, C. (1991) Classification and natural history of clinically identifiable subtypes of cerebral infarction. *Lancet*, 337(8756): 1521-1526. DOI: [10.1016/0140-6736\(91\)93206-0](https://doi.org/10.1016/0140-6736(91)93206-0)
10. Duncan, P.W., Goldstein, L.B., Matchar, D., Divine, G.W., Feussner, J. (1992) Measurement of motor recovery after stroke. Outcome assessment and sample size requirements. *Stroke*, 23(8): 1084-1089. DOI: [10.1161/01.STR.23.8.1084](https://doi.org/10.1161/01.STR.23.8.1084)
11. Nakayama, H., Jorgensen, H.S., Raaschou, H.O., Olsen, T.S. (1994) Recovery of upper extremity function in stroke patients: the Copenhagen Stroke Study. *Archives of Physical Medicine and Rehabilitation*, 75(4): 394-398. DOI: [10.1016/0003-9993\(94\)90161-6](https://doi.org/10.1016/0003-9993(94)90161-6)
12. Langhorne, P., Bernhardt, J., Kwakkel, G. (2011) Stroke rehabilitation. *Lancet*, 377(9778): 1693-1702. DOI: [10.1016/S0140-6736\(11\)60325-5](https://doi.org/10.1016/S0140-6736(11)60325-5)
13. Lai, S.M., Studenski, S., Duncan, P.W., Perera, S. (2002) Persisting consequences of stroke measured by the Stroke Impact Scale. *Stroke*, 33(7): 1840-1844. DOI: [10.1161/01.STR.0000019289.15440.F2](https://doi.org/10.1161/01.STR.0000019289.15440.F2)
14. Wade, D.T., Wood, V.A., Hewer, R.L. (1985) Recovery after stroke--the first 3 months. *Journal of Neurology, Neurosurgery, and Psychiatry*, 48(1): 7-13. DOI: [10.1136/jnnp.48.1.7](https://doi.org/10.1136/jnnp.48.1.7)
15. Hendricks, H.T., van Limbeek, J., Geurts, A.C., Zwarts, M.J. (2002) Motor recovery after stroke: a systematic review of the literature. *Archives of Physical Medicine and Rehabilitation*, 83(11): 1629-1637. DOI: [10.1053/apmr.2002.35473](https://doi.org/10.1053/apmr.2002.35473)
16. Kelly-Hayes, M., Beiser, A., Kase, C.S., Scaramucci, A., D'Agostino, R.B., Wolf, P.A. (2003) The influence of gender and age on disability following ischemic stroke: the Framingham study. *Journal of Stroke and Cerebrovascular Diseases*, 12(3): 119-126. DOI: [10.1016/S1052-3057\(03\)00042-9](https://doi.org/10.1016/S1052-3057(03)00042-9)
17. Adams, H.P. Jr., Davis, P.H., Leira, E.C., Chang, K.C., Bendixen, B.H., Clarke, W.R., et al. (1999) Baseline NIH Stroke Scale score strongly predicts outcome after stroke: a report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). *Neurology*, 53(1): 126-131. DOI: [10.1212/WNL.53.1.126](https://doi.org/10.1212/WNL.53.1.126)
18. Megherbi, S.E., Milan, C., Minier, D., Couvreur, G., Osseby, G.V., Tilling, K., et al. (2003) Association between diabetes and stroke subtype on survival and functional outcome 3 months after stroke: data from the European BIOMED Stroke Project. *Stroke*, 34(3): 688-694. DOI: [10.1161/01.STR.0000057975.15221.40](https://doi.org/10.1161/01.STR.0000057975.15221.40)

19. Sacco, R.L., Benjamin, E.J., Broderick, J.P., Dyken, M., Easton, J.D., Feinberg, W.M., et al. (1997) American Heart Association Prevention Conference IV: prevention and rehabilitation of stroke. Risk factors. *Stroke*, 28(7): 1507-1517. DOI: [10.1161/01.STR.28.7.1507](https://doi.org/10.1161/01.STR.28.7.1507)
20. Petrea, R.E., Beiser, A.S., Seshadri, S., Kelly-Hayes, M., Kase, C.S., Wolf, P.A. (2009) Gender differences in stroke incidence and poststroke disability in the Framingham Heart Study. *Stroke*, 40(4): 1032-1037. DOI: [10.1161/STROKEAHA.108.542894](https://doi.org/10.1161/STROKEAHA.108.542894)
21. Gresham, G.E., Fitzpatrick, T.E., Wolf, P.A., McNamara, P.M., Kannel, W.B., Dawber, T.R. (1975) Residual disability in survivors of stroke--the Framingham study. *New England Journal of Medicine*, 293(19): 954-956. DOI: [10.1056/NEJM197511062931903](https://doi.org/10.1056/NEJM197511062931903)
22. Schiemanck, S.K., Kwakkel, G., Post, M.W., Kappelle, L.J., Prevo, A.J. (2006) Predictive value of ischemic lesion volume assessed with magnetic resonance imaging for neurological deficits and functional outcome poststroke: a critical review of the literature. *Neurorehabilitation and Neural Repair*, 20(4): 492-502. DOI: [10.1177/1545968306289298](https://doi.org/10.1177/1545968306289298)
23. Stinear, C.M., Barber, P.A., Smale, P.R., Coxon, J.P., Fleming, M.K., Byblow, W.D. (2007) Functional potential in chronic stroke patients depends on corticospinal tract integrity. *Brain*, 130(1): 170-180. DOI: [10.1093/brain/awl333](https://doi.org/10.1093/brain/awl333)
24. Stinear, C.M., Byblow, W.D., Ackerley, S.J., Smith, M.C., Borges, V.M., Barber, P.A. (2017) PREP2: a biomarker-based algorithm for predicting upper limb function after stroke. *Annals of Clinical and Translational Neurology*, 4(11): 811-820. DOI: [10.1002/acn3.488](https://doi.org/10.1002/acn3.488)
25. Byblow, W.D., Stinear, C.M., Barber, P.A., Petoe, M.A., Ackerley, S.J. (2015) Proportional recovery after stroke depends on corticomotor integrity. *Annals of Neurology*, 78(6): 848-859. DOI: [10.1002/ana.24472](https://doi.org/10.1002/ana.24472)
26. Wissel, J., Manack, A., Brainin, M. (2013) Toward an epidemiology of poststroke spasticity. *Neurology*, 80(3 Suppl 2): S13-S19. DOI: [10.1212/WNL.0b013e3182762448](https://doi.org/10.1212/WNL.0b013e3182762448)
27. Sommerfeld, D.K., Eek, E.U., Svensson, A.K., Holmqvist, L.W., von Arbin, M.H. (2004) Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. *Stroke*, 35(1): 134-139. DOI: [10.1161/01.STR.0000105386.05173.5E](https://doi.org/10.1161/01.STR.0000105386.05173.5E)
28. Ada, L., Foongchomcheay, A., Canning, C. (2005) Supportive devices for preventing and treating subluxation of the shoulder after stroke. *Cochrane Database of Systematic Reviews*, (1): CD003863. DOI: [10.1002/14651858.CD003863.pub2](https://doi.org/10.1002/14651858.CD003863.pub2)
29. Weerdesteyn, V., de Niet, M., van Duijnhoven, H.J., Geurts, A.C. (2008) Falls in individuals with stroke. *Journal of Rehabilitation Research and Development*, 45(8): 1195-1213.
30. Langhorne, P., Coupar, F., Pollock, A. (2009) Motor recovery after stroke: a systematic review. *Lancet Neurology*, 8(8): 741-754. DOI: [10.1016/S1474-4422\(09\)70150-4](https://doi.org/10.1016/S1474-4422(09)70150-4)
31. Coupar, F., Pollock, A., Rowe, P., Weir, C., Langhorne, P. (2012) Predictors of upper limb recovery after stroke: a systematic review and meta-analysis. *Clinical Rehabilitation*, 26(4): 291-313. DOI: [10.1177/0269215511420305](https://doi.org/10.1177/0269215511420305)
32. Wolf, S.L., Winstein, C.J., Miller, J.P., Taub, E., Uswatte, G., Morris, D., et al. (2006) Effect of constraint-induced movement therapy on upper extremity function 3 to 9 months after stroke: the EXCITE randomized clinical trial. *JAMA*, 296(17): 2095-2104. DOI: [10.1001/jama.296.17.2095](https://doi.org/10.1001/jama.296.17.2095)
33. Pollock, A., Farmer, S.E., Brady, M.C., Langhorne, P., Mead, G.E., Mehrholz, J., et al. (2014) Interventions for improving upper limb function after stroke. *Cochrane Database of Systematic Reviews*, (11): CD010820. DOI: [10.1002/14651858.CD010820.pub2](https://doi.org/10.1002/14651858.CD010820.pub2)