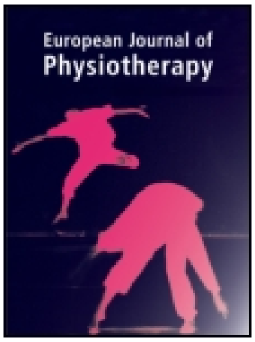

Taylor & Francis
Taylor & Francis Group

European Journal of Physiotherapy

ISSN: 2167-9169 (Print) 2167-9177 (Online) Journal homepage: http://www.tandfonline.com/loi/iejp20

Neuroplasticity in action post-stroke: Challenges for physiotherapists

Gunilla E. Frykberg & Rajul Vasa

To cite this article: Gunilla E. Frykberg & Rajul Vasa (2015) Neuroplasticity in action post-stroke: Challenges for physiotherapists, European Journal of Physiotherapy, 17:2, 56-65, DOI: 10.3109/21679169.2015.1039575

To link to this article: http://dx.doi.org/10.3109/21679169.2015.1039575

Published online: 28 Apr 2015.

European Journal of Physiotherapy, 2015; 17: 56–65

informa healthcare

REVIEW ARTICLE

Neuroplasticity in action post-stroke: Challenges for physiotherapists

GUNILLA E. FRYKBERG[1] & RAJUL VASA[2]

[1]*Department of Neuroscience/Rehabilitation Medicine, Uppsala University, Sweden, and* [2]*Rajul Vasa Foundation, Center for Brain and Spinal Injury Rehab, Mumbai, India*

Abstract
Knowledge regarding neuroplasticity post-stroke is increasingly expanding. In spite of this, only a few physiotherapy interventions have been able to demonstrate effectiveness in achieving recovery of lost sensorimotor control. The aims of this review article are to highlight and discuss challenges for physiotherapists working with patients post-stroke, to question some current assessment methods and treatment approaches, and to pose critical questions indicating a possible new direction for physiotherapists in stroke rehabilitation. Differentiation between recovery and compensation post-stroke is increasingly being emphasized. Implementation of this goal in the clinic is insufficient, with a lack of assessment tools with potential to discriminate between the concepts. Large-scale reviews are performed without considering whether functional gains are achieved through "more effective" compensatory strategies or through recovery. Cortical plasticity in neurorehabilitation research and voluntary control in contemporary treatment methods are in focus. Challenges for physiotherapists in stroke rehabilitation consist of rethinking, including looking upon the body under the influence of gravity, focusing on implicit factors that impact movement control and developing new assessment tools. The introduction of a new assessment and treatment concept aiming at expanding the boundaries of center of mass movements towards the paretic side is proposed. In conclusion, we need to assume our responsibilities and step forward as the experts in movement science that we have the potential to be.

Key words: *Biomechanics, motor control, neurology, rehabilitation*

Introduction

In the clinic, we observe people afflicted by stroke compensating with the non-paretic side of the body to reach everyday goals. We, as physiotherapists, support the patient in using the paretic side, aiming for true recovery of mobility and selective voluntary control in the process of rehabilitation. However, compensatory movement strategies are often noticed as a result.

From the research point of view, in spite of an increasing amount of research regarding recovery versus compensation [e.g. (1)] and neuroplasticity post-stroke [e.g. (2)], this new knowledge has yet not resulted in evidence-based physiotherapy interventions with proven effects on recovery of lost sensorimotor function (3). It is argued that an in-depth understanding of the underlying mechanisms behind recovery and compensation post-stroke is still lacking and urgently needed in order to develop strategies for therapy-induced recovery (4–7).

Furthermore, two fundamental questions with respect to different motor control theories, namely "What is being controlled?" and "How are these processes organized?", posed in the 1960s (8), have still not been answered. In addition, if and how central concepts such as self-organization of dynamic biological systems, non-linearity and movement variability from one recently developed motor control theory, the dynamic systems theory (DST) (9,10), might be integrated into the development of effective physiotherapeutic interventions post-stroke is still an open question. Altogether, the above implies that new questions need to be asked addressing clinical as well as research issues for physiotherapists in the areas of recovery versus compensation, neuroplasticity post-stroke and the state of the art of

Correspondence: Gunilla Elmgren Frykberg, RPT, PhD, Department of Neuroscience/Rehabilitation Medicine, Uppsala University, SE-751 85 Uppsala, Sweden. Tel: +46 18 611 28 75. E-mail: gunilla.elmgren.frykberg@neuro.uu.se

(Received 15 December 2014; accepted 6 April 2015)

ISSN 2167-9169 print/ISSN 2167-9177 online © 2015 Informa Healthcare
DOI: 10.3109/21679169.2015.1039575

physiotherapy in stroke rehabilitation, irrespective of lesion location, phase post-stroke and age of the patients. In this article, which does not claim to comprehensively cover all aspects of the aforementioned areas, we will highlight experience-based clinical reflections and research results, question some existing assessment methods and treatment approaches, and ask some critical questions which address challenges that physiotherapists involved in stroke rehabilitation are facing today.

Recovery and compensation

In recent years it has become more and more clear that it is critical to distinguish between the concepts of recovery and compensation in stroke rehabilitation (4,5,7,11–13).

In this article, we join the definition formulated by Kitago and colleagues (1):

> We define motor recovery as the re-emergence of movement kinematics similar to those of healthy age-matched controls, resulting from a decrease in impairments, whereas compensation involves the use of the unaffected limb or alternative muscle groups on the affected side to accomplish the task.

Without highlighting the quality of movement it is not possible to distinguish between the two concepts of recovery and compensation (11). It is also generally accepted that the different levels of the International Classification of Functioning (14) should be taken into account when these two concepts are defined and discussed (11).

Still, there seems to be some confusion regarding the definitions of these two concepts post-stroke, which is further illustrated by the use of the concept "functional recovery", indicating improvements in the capability to move and to use the arm/hand in daily life post-stroke (5,15), regardless of the quality of movement.

Likewise, in a recent review including 467 research studies regarding the effect of physiotherapeutic interventions post-stroke, the importance of discriminating between these two concepts was mentioned in the Discussion, aiming at true understanding of what happens in the process after a stroke (7). However, in the same review there was almost no consideration given to whether achieved gains after 53 scrutinized interventions were mediated through compensatory movement strategies or through truly recovered, i.e. typical, movements.

From a clinical perspective, we observe people who have had a stroke standing with asymmetrical weight-bearing, rising up to walk pushing with the non-paretic hand, reaching for objects with atypical movement patterns and walking with specific gait deviations. These atypical movement strategies were described as early as the 1960s by Signe Brunnström, a Swedish physiotherapist (16). In addition, while walking, the person who has had a stroke is often observed to "give" the control over the body's center of mass (COM) to the non-paretic leg and occasionally also to the non-paretic hand with the help of a walking stick. The whole body's COM is considered a key component in balance control and is defined as an abstract point about which the mass of the body is evenly distributed and balanced (17–19).

Similarly, examples from research, using a force platform with separate left/right force sensors, demonstrate compensatory movement strategies illustrated through a significantly smaller contribution from the paretic leg to standing balance, and consequently with greater responsibility taken by the non-paretic leg (20). Furthermore, in the same study the association between weight-bearing and balance contribution was unclear in the stroke group, but shown to be one-to-one when the control subjects consciously distributed their weight unevenly. Results in a study of sit-to-walk in a stroke and a control group, where all subjects succeeded in fulfilling the task, revealed 4.5 times larger braking impulses beneath the buttocks and feet prior to seat-off in the stroke group, in spite of their displaying the same total amount of anterior–posterior force impulses as the controls (21). Similar results have been presented during a reaching task, where all stroke subjects, irrespective of severity of disability, succeeded in accomplishing the task, but with considerably different movement strategies (22). Compensatory strategies have also been demonstrated in studies investigating gait initiation (23) and walking post-stroke (24,25), even independent of walking speed (26). Significant asymmetry effects of walking aids on ground reaction force parameters have been shown, revealing different roles for the non-paretic and paretic lower limb (27).

In this context of recovery and compensation, some concepts from the DST)may be relevant to reflect upon. The DST tries to explain fundamental principles of dynamic systems (10,28,29), such as the brain and nervous system. One fundamental principle is the capability of self-organization, i.e. the potential to create new spatial and/or temporal movement patterns from within as a reaction to changing prerequisites (30), such as a brain lesion. New movement patterns emerge owing to interaction between interrelated subsystems making up the whole system (9). It is essential in DST to suggest an answer regarding the control over the redundant degrees of freedom (DoF) existing within the human body, with all its skeletal bones, joints and muscles, where external (i.e. gravitational) and internal forces acting on the body also need to be considered (28). Coordination of movement is proposed to be the means to achieve mastery of the multitude of DoF and muscles working together in synergy, thus contributing towards solving the DoF problem (28).

Altogether, these theoretical reflections as well as clinical observations and research results, demonstrating compensatory movement strategies in everyday activities post-stroke, challenge us to formulate some new critical questions:

- Does the self-organizing human brain immediately post-injury start acting without waiting for the rehabilitation team to give instructions?
- How does the brain solve the problem of increased DoF when the muscles are flaccid/hypotonic on the paretic side?
- How does the brain deal with gravitational forces to control its COM safety, when half of the body's muscles are not available for generating forces with respect to this control?
- Is there a correlation between the clinical observations of the patient shifting the control for the safety of COM over to the non-paretic side and the self-organizing brain trying to solve its problem of safety?
- Why would the brain choose to group the muscles into flexion and extension synergies [as described by Brunnström (16)] instead of restoring segmental voluntary control?
- Is there a correlation between abnormal synergic grouping and restriction of DoF for the safety of COM?
- Might it be possible to achieve normal synergy grouping if the COM post-stroke is controlled through the paretic limbs and trunk?

Some assumptions regarding recovery and compensation in stroke rehabilitation need to be commented and reflected upon.

It seems to be generally accepted that there is a limited time window within which most of the improvements occur after a brain lesion (31–35). In a large-scale stroke study (36) it was proposed that a probable prognosis of walking ability might be determined as early as 3 weeks post-injury and that one should not expect improvements after 9 weeks. Similarly, recovery of arm function outcome at 6 months has been suggested to be predicted at 4 weeks post-onset (37).

Critical new questions arise here:

- Are these predictions based on what we observe as the brain's spontaneous recovery?
- Are these predictions based on an understanding of the underlying mechanisms behind recovery and compensation?
- How do these predictions influence the mindset of a clinician in his or her goal-setting for the patient?

From clinical experience-based understanding of the mechanisms underlying sensorimotor problems post-stroke and of how to achieve true recovery, this time-limited perspective on recovery after stroke may not be valid. Most important, it is suggested, is understanding "what not to do" with the body and the brain, while these are in constant interaction with gravity.

Another assumption within the area of recovery and compensation is the emphasis in rehabilitation interventions on the *explicit* loss, i.e. voluntary control to be achieved post-stroke. Other questions are raised here:

- What is the priority of the brain post-injury, especially when the muscles on one side of the body are unable to generate forces to combat gravity?
- Would in-depth knowledge about *implicit* control of different paretic body segments to control their own segmental COM as well as the whole body's global COM be of significant importance for true recovery?

A further assumption has been that compensatory movement strategies post-stroke have primarily been supposed to be due to dysfunction mainly in the neural system. However, biomechanical changes in the musculoskeletal system itself, proposed to be separated into viscosity and elasticity of soft tissues (38), also need to be considered (39,40), as well as Bernstein's suggestion of viewing the whole body as a mechanical system (28).

For a long time, it has been a profound assumption that we should allow compensatory movement strategies post-stroke (12). However, this view is now changing and the debate is ongoing as to whether we should prevent atypical movement patterns occurring or not (41,42). A critical question is whether permitting patients early post-stroke to use compensatory movement strategies prevents recovery (4,12,22).Obviously, there are many challenging questions for physiotherapists to deal with in current stroke rehabilitation.

Neuroplasticity

Neuroplasticity within the central nervous system has been recognized for a long time (43), and its importance for assumptions underlying neurological rehabilitation and for recovery post-stroke has been highlighted during the past few decades (44). In this article, we will subscribe to the definition of neuroplasticity suggested by Bethe (43), as "… the ability to adapt to changes and to meet the dangers of life. It is the capacity of the central nervous system to reorganize following insult and to restore adequate function." Whether this reorganization that takes place in the nervous system is adaptive, i.e. associated with improved movement capacity (45), or maladaptive, with deterioration as a result (11,46), needs to be discussed.

One of the principles behind post-injury neuroplasticity is that "behavioral experience" is a very powerful modulator (2). When it comes to physiotherapeutic interventions, this behavioral experience is provided to patients post-stroke through different kinds of sensorimotor training. Whether this motor experience will reshape the brain in adaptive or maladaptive ways is considered to depend on the quantity and quality of the specific treatment (2,4).

Regarding quantity, the dose–response relationship is essential to consider when translating results from animal studies into stroke rehabilitation, as animals accomplish hundreds of repetitions daily (47). Furthermore, constraint-induced movement therapy (CIMT), a treatment approach in stroke rehabilitation that is implemented worldwide, has its origin in studies with non-human primates, and one of its main components is high-repetition doses of training (48). In the study by Birkenmeier et al. (47), the stroke participants performed at least 300 repetitions per therapy hour, without experiencing increased pain or fatigue, and with functional improvements being obtained. Similar results with significant improvements have been demonstrated in recent studies with high-intensity training post-stroke (49,50). However, the critical importance of how to empower patients to take full responsibility for this kind of training in order to achieve permanent improvements in post-stroke rehabilitation remains to be investigated (2,49).

Regarding the quality of the motor experience, studies with rats show that in the absence of training, i.e. due to so-called spontaneous recovery, the animals exhibited compensatory movement patterns at 5 weeks post-onset, revealed through intracortical microstimulation (51). Furthermore, findings from monkeys (52) and rodents (53) suggest that motor activity alone, such as pressing a bar, without acquiring any motor skill does not seem sufficient to promote neurophysiological changes in cortical areas. It seems as if cortical plasticity is skill or learning dependent, and that use itself, in the form of simple repetitions or strength training (54), may not be enough for neuroplastic changes to take place.

A second principle underlying neuroplasticity is that there seems to be a limited time window for recovery mechanisms to operate post-stroke. In the first hours to days, biochemical changes take place aiming at both the limitation of damage in the penumbra region close to the brain injury (55) and the alleviation of diaschisis (4,56). Primarily spontaneous neurological recovery is assumed to contribute to functional improvements within the first weeks post-stroke (32). Furthermore, in the course of the first 10–12 weeks post-injury it is assumed that most of the restitution of impairments is accomplished (57) and that after this period it is mainly compensatory adaptation that takes place (4).

The importance of motivation is considered to be a third principle critical for driving neuroplasticity (3,58,59) and significant functional improvements have been demonstrated in spite of a huge lesion (44).

There is a primary focus on cortical plasticity in many research studies, both in animals and in humans (2). However, in humans, lesions in subcortical areas occur much more frequently than in cortical areas (60). Thus, our knowledge about plastic changes in subcortical networks is limited. Logically, task-specific training with a focus on upper-extremity function involves voluntary control, and thereby cortical plastic changes are of interest. To further the understanding of mechanisms underlying recovery and compensation of upper-limb function post-stroke, a multicenter research program has been implemented in the Netherlands, called the EXPLICIT stroke program [an acronym for EXplaining PLastICITy after stroke (61)]. We propose that an IMPLICIT stroke research program is needed (investigating, for example, how gravity and movements of COM influence body movements and strengthen paretic muscles from within), aiming at understanding how these and other "Invisible" factors have impact on Movement control with the potential to drive neuroPLasticity post-stroke and put the patient on the road to recovery.

Challenging questions for physiotherapists arise with respect to neuroplasticity:

- Should compensatory movement strategies be viewed as maladaptive plasticity?
- Is it possible to reverse spasticity and abnormal synergic grouping by exploiting inter-limb coordination (e.g. generating forces beneath the paretic foot aiming at influencing improvements of the paretic arm) coupled with the brain's priority suggested to be "safety of COM" forced from the paretic side?
- Could such treatment contribute to the much-needed quality of motor experience in order to promote adaptive neuroplastic changes post-stroke?

- Could physiotherapeutic interventions based on the understanding of underlying mechanisms promote true recovery instead of compensation and extend the sensitive period for adaptive improvements post-stroke?

Physiotherapy in stroke rehabilitation: state of the art

The following paragraph addresses critical questions for physiotherapists in current stroke rehabilitation concerning "Where do we stand?" and "Where are we heading?"

Worldwide, an interdisciplinary team approach in stroke rehabilitation is considered optimal in supporting the patient to as full a recovery as possible (5). In this context, physiotherapists are important team members, often with in-depth knowledge of movement analysis, which is necessary in order to observe and analyze how people who have had a stroke use their bodies to reach functional goals in everyday life. In our opinion, this expert knowledge in observatory-based movement analysis (OMA) has the potential to discriminate between typical (i.e. recovered) and atypical (i.e. compensatory) movement patterns, but it does not seem to be optimally utilized in stroke rehabilitation at present.

In assessment, clinical tools currently in use by physiotherapists in stroke rehabilitation, irrespective of addressing arm/hand function, balance or mobility, often report only whether the patient can accomplish the task or not [categorical data, e.g. Stops Walking When Talking, SWWT (62)], or can perform the task better or worse [ordinal data, e.g. Berg Balance Scale, BBS (63)], or just give a single summary value [e.g. the Nine Hole Peg test (64); Timed Up and Go (65); walking speed (66)], without giving any information about the quality with which the task was performed. Furthermore, the focus is most often mainly on the explicit task accomplishment without considering more implicit contributing factors, from the initiation of movement to the end result. Unfortunately, it seems as if physiotherapists today only to a very limited extent use and report structured OMA, with the potential to assess quality of movement over time, even though these tools exist, e.g. Rancho Los Amigos Gait Analysis Form (67). In addition to OMA, there is a need to objectively quantify and describe three-dimensional (3D) kinematics in order to be able to differentiate between recovery and compensation post-stroke (12,68). This can be achieved with portable movement-analysis systems, which is an area currently undergoing rapid development thanks to the miniaturization of accelerometers, gyroscopes and magnetometers [e.g. (69,70)]. In order to explain the dynamics of neural recovery, it has been suggested that serial kinematic measurements should be undertaken early after stroke, where the quality of motor performance is systematically registered (4), which is also implemented in the Dutch EXPLICIT stroke program (61,71).

Challenging questions regarding assessment post-stroke arise here:

- Shall we physiotherapists, for clinical decision-making, continue to be satisfied with insufficient information in evaluating our interventions?
- What benefit do we derive from categorical data, ordinal data or a single value for facilitation of how we should design interventions?

In treatment, the focus in physiotherapeutic interventions in stroke rehabilitation has largely been on muscles, although approaches have changed over the years. During a period from the 1950s to the 1970s, the elusive phenomenon of spasticity was central, and treatment methods, e.g. neurofacilitation approaches, aimed at inhibiting increased muscle tone [e.g. (72)] and promoting muscular improvements on the paretic side (16,73,74).

The muscular focus in stroke rehabilitation has continued, with muscle weakness entering as a main target in stroke rehabilitation, and progressive resistance training has been developed and reported to be successful in maintaining muscle strength (75–77), also at long-term follow-up after 4 years (78). However, the benefit and transfer of maintained muscle strength to other measures of functional activity, such as gait performance, are equivocal (76,78–80).

Questions regarding this muscle focus remain to be answered:

- Are we ready to think beyond muscle inhibition and muscle strengthening?
- How can the limited transfer effects of muscle-strength training to everyday functional activities be explained?

Treatment approaches post-stroke have gradually changed as a result of advances in neuroscience and our extended knowledge of mechanisms for motor control (81). During the 1980s, principles of motor learning, including active cognitive involvement of the patient, were highlighted, and predominantly hands-off training was implemented post-stroke (82).

A further step was taken when the "task-oriented approach" in stroke rehabilitation was developed, which includes considering not only the individual with his or her perceptual, cognitive and neuromusculoskeletal abilities, but also the characteristics of the task and the environment in which the task is to be performed (10). In this treatment model, everyday tasks are repetitively practiced, e.g. reaching out for a glass, grasping it, bringing it to the mouth and drinking, i.e. the focus is on voluntary action.

When the effect of repetitive task training on global, upper and lower limb function post-stroke was evaluated in a Cochrane review, the results demonstrated modest positive changes regarding lower-limb function, some impact on activities of daily living, but no effect on upper-limb motor activities (83). Furthermore, no evidence could be found for lasting improvements after 6 and 12 months. Outcome measures for lower-limb function were, among others, walking distance, walking speed and time to complete sit-to-stand, i.e. single values representing end results, without considering whether the improvements were achieved owing to "more effective" compensatory strategies or to true recovered movement patterns.

CIMT, mentioned above, is a treatment model in stroke rehabilitation which has been in use since the early 1990s (84). Its focus is predominantly on removal of the learned non-use of the paretic arm and hand, with the principles of preventing the use of the non-paretic upper limb in daily activities by use of a sling and glove and massed practice of everyday tasks with the paretic hand/arm (85). There has been extensive evaluation of CIMT. Results from the Extremity Constraint Induced Therapy Evaluation (EXCITE) study, a randomized controlled study with 222 participants, demonstrated significant improvements in daily use of the paretic hand/arm both at 1 year (86) and at 2 year (87) follow-ups. However, the chosen outcome measures did not have the potential to differentiate between recovery and atypical movement patterns, and in a later proof-of-concept study using kinematic measures besides clinical scales it was suggested that the functional improvements achieved through CIMT were mediated through compensatory movement strategies (1,68). The CIMT method has also been scrutinized in a Cochrane review, where moderately positive effects were demonstrated on disability and on arm motor function as well as on arm motor impairments (88). However, many of the 19 studies included had small sample sizes and incomplete information about withdrawals. No evidence of remaining positive effects was found at follow-up after 6 months. Thus, contradictory results exist regarding the efficacy of CIMT.

Here, questions arise with respect to focus on the task:

- Why do we continue to focus on the end result, the accomplishment of the task, when there are so many preparatory steps in the process before the end result?
- Why not consider exploiting gravity to drive true recovery instead of compensation when it remains invariant and cannot be avoided?
- Why not exploit different segments of the body, including the trunk, as a mechanical system aiming at generating forces in specially designed postures, wherein the entire body is in action, with the paretic limbs controlling segmental COMs as well as the whole body's COM?
- Might the temporary and impermanent functional effects as demonstrated in the use of the task-oriented approach be explained partly by the focus being mainly on the end result and partly by a lack of understanding of the underlying mechanisms that contribute to recovery post-stroke?

Efforts have also been put into evaluating the effect of different physiotherapy approaches on functional independence (89,90). The conclusions were that no single treatment approach post-stroke is more effective than any other in achieving "recovery of function" and "mobility" post-stroke. It should be noted that no discrimination between recovery and compensation was discussed in these reviews. To our knowledge, the selected assessment tools and outcome measures (independence in activities of daily living, motor function, balance, gait velocity and length of stay) do not have the potential to relate improvements to either true recovery or compensation.

In recent years, many "new" therapies have been introduced and are under evaluation in stroke rehabilitation, such as virtual reality, robot-assisted arm and gait training, locomotor treadmill training, motor imagery and mirror therapy. The focus regarding the state of the art in physiotherapy in the current article is on commonly used treatment approaches, so the debate is limited to those.

To conclude:

- Where are we physiotherapists heading in stroke rehabilitation?
- Who could be better equipped to understand the underlying mechanisms behind movement problems post-stroke and to find solutions beyond neural control than physiotherapists with expertise in movement science?

Summary

Neuroplasticity in action post-stroke, with respect to both the self-organized brain and physiotherapy interventions post-stroke, with reports in many research studies (20–27), seems to have implied the development of compensatory movement strategies. Thus, there is a need to think beyond the traditional way with its focus on the lesion, on cortical plasticity and on voluntary control for task accomplishments.

The demand to differentiate between recovered and compensated movement strategies in stroke rehabilitation is increasingly being highlighted. However, this critical issue is not yet being put into practice neither in the clinic, where, for example, measurement tools yielding this information are lacking, or in research, where large-scale reviews concerning physiotherapy interventions post-stroke are scrutinized. Furthermore, the knowledge base regarding cortical neuroplasticity is increasingly comprehensive, although information about subcortical, cerebellar and spinal plastic changes post-stroke is lacking.

Physiotherapists worldwide now face the challenge of having to take a stand on whether we should consider the emergence of compensatory movement patterns in subjects who have had stroke as necessary or not, also taking into account the increasing number of reports suggesting that compensation post-stroke may prevent true recovery. Furthermore, large-scale reviews continue to emphasize the urgent need to understand principles driving recovery post-stroke.

So, how can the challenges facing physiotherapists in stroke rehabilitation today be summarized?

First, we need to rethink. There is a need to complement the focus on the neural system in stroke rehabilitation with looking upon the human body as a mechanical system, with the possibility of exploiting gravitational forces. Gravity is suggested to be the most important and critical invariant factor that evokes compensation and also has the potential to drive true sensorimotor recovery post-stroke. It is necessary to understand "what not to do" and "what to do" with the brain and the body, which are in constant interaction with gravity. We need to shift the focus from voluntary control to the "invisible" factors influencing movement control, exemplified by promoting proprioceptive afferent inflow to trigger automatic motor outflow for prioritizing the safety of the COM. This is hypothesized to be a potent modulator to drive and force the brain into adaptive neuroplastic changes on many levels of the nervous system. Furthermore, the consequences of the lesion on the body and on the brain, and not the lesion *per se*, are suggested to be in focus in future physiotherapy clinical practice and in research.

Second, the quality of movements needs to be assessed and documented, and we should not be satisfied with single values as outcome measures. Thus, we need to further develop our expert skill with respect to 3D OMA, so that we can discriminate between true recovery and compensatory movement patterns post-stroke. In addition, close cooperation with bioengineers may be fruitful in order to objectively capture and quantify 3D movements using recently developed portable movement-analysis systems. Furthermore, we need to develop new assessment tools which can capture not only the explicit voluntary control, but also, more importantly, the ability of the patient to control segmental as well as global COM through the paretic side of the body, which is proposed to contribute to the implicit control of movement.

Third, we need to empower and educate the patient and his or her friends and family, as "therapy" needs to be constantly ongoing to have a chance to promote permanent sensorimotor recovery. Empowering the patient will help him or her to feel responsible for the treatment. Patient values as one important component in evidence-based practice need to be highlighted and put into practice.

Fourth, there is a need to introduce a new assessment and treatment concept aiming at expanding the boundaries of COM movements towards the paretic side by using the paretic limbs coupled with the paretic trunk to control the safety of the COM. With this in focus, the ultimate goal would be to put the patient on the road to recovery. This new concept needs to be thoroughly scientifically investigated, before it can be decided whether it can lead to true recovery or not.

We predict that physiotherapists worldwide taking responsibility for their contribution in stroke rehabilitation as experts in movement science will have the potential to direct the patients in how to influence adaptive neuroplasticity in action, turning it into recovery instead of compensation. We also predict the evolution of physiotherapy into a clinical science within the neurorehabilitation sciences.

Declaration of interest: The authors have no conflicts of interest to declare.

References

1. Kitago T, Liang J, Huang VS, Hayes S, Simon P, Tenteromano L, et al. Improvement after constraint-induced movement therapy: recovery of normal motor control or task-specific compensation? Neurorehabil Neural Repair. 2013;27:99–109.
2. Nudo RJ. Recovery after brain injury: mechanisms and principles. Front Hum Neurosci. 2013;7:887.
3. Cramer SC, Sur M, Dobkin BH, O'Brien C, Sanger TD, Trojanowski JQ, et al. Harnessing neuroplasticity for clinical applications. Brain. 2011;134:1591–609.
4. Buma F, Kwakkel G, Ramsey N. Understanding upper limb recovery after stroke. Restor Neurol Neurosci. 2013; 31:707–22.
5. Langhorne P, Bernhardt J, Kwakkel G. Stroke rehabilitation. Lancet. 2011;377:1693–702.

6. Van Peppen RP, Kwakkel G, Wood-Dauphinee S, Hendriks HJ, Van der Wees PJ, Dekker J. The impact of physical therapy on functional outcomes after stroke: what's the evidence? Clin Rehabil. 2004;18:833–62.
7. Veerbeek JM, van Wegen E, van Peppen R, van der Wees PJ, Hendriks E, Rietberg M, et al. What is the evidence for physical therapy poststroke? A systematic review and meta-analysis. PLoS One. 2014;9:e87987.
8. Granit R. Comments on history of motor control. In: Brooks VB, editor. Handbook of physiology. Baltimore, MD: Williams & Wilkins; 1981. p. 1–16.
9. Perry SB. Clinical implications of a dynamic systems theory. Neurol Rep. 1998;22:4–10.
10. Shumway-Cook A, Woollacott MH. Motor control: issues and theories. In: Shumway-Cook A, Woollacott MH, editors. Motor control – translating research into clinical practice. 4th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2012. p. 3–20.
11. Levin MF, Kleim JA, Wolf SL. What do motor "recovery" and "compensation" mean in patients following stroke? Neurorehabil Neural Repair. 2009;23:313–9.
12. Lum PS, Mulroy S, Amdur RL, Requejo P, Prilutsky BI, Dromerick AW. Gains in upper extremity function after stroke via recovery or compensation: potential differential effects on amount of real-world limb use. Top Stroke Rehabil. 2009;16:237–53.
13. Zeiler SR, Krakauer JW. The interaction between training and plasticity in the poststroke brain. Curr Opin Neurol. 2013;26:609–16.
14. World Health Organization. International Classification of Functioning, disability and health, ICF. Geneva: WHO; 2001.
15. Tecchio F, Zappasodi F, Tombini M, Caulo M, Vernieri F, Rossini PM. Interhemispheric asymmetry of primary hand representation and recovery after stroke: a MEG study. Neuroimage. 2007;36:1057–64.
16. Brunnström S. Movement therapy in hemiplegia. London: Harper and Row; 1970.
17. Enoka RM. Neuromechanics of human movement. 4th ed. Champaign, IL: Human Kinetics; 2008.
18. Scholz JP, Schoner G, Hsu WL, Jeka JJ, Horak F, Martin V. Motor equivalent control of the center of mass in response to support surface perturbations. Exp Brain Res. 2007;180:163–79.
19. Winter D. A.B.C. (Anatomy, Biomechanics and Control) of balance during standing and walking. Waterloo: Graphic Services, University of Waterloo; 1995.
20. van Asseldonk EH, Buurke JH, Bloem BR, Renzenbrink GJ, Nene AV, van der Helm FC, et al. Disentangling the contribution of the paretic and non-paretic ankle to balance control in stroke patients. Exp Neurol. 2006;201:441–51.
21. Frykberg GE, Thierfelder T, Aberg AC, Halvorsen K, Borg J, Hirschfeld H. Impact of stroke on anterior–posterior force generation prior to seat-off during sit-to-walk. Gait Posture. 2012;35:56–60.
22. Cirstea MC, Levin MF. Compensatory strategies for reaching in stroke. Brain. 2000;123:940–53.
23. Tokuno CD, Eng JJ. Gait initiation is dependent on the function of the paretic trailing limb in individuals with stroke. Gait Posture. 2006;24:424–8.
24. Bowden MG, Balasubramanian CK, Neptune RR, Kautz SA. Anterior–posterior ground reaction forces as a measure of paretic leg contribution in hemiparetic walking. Stroke. 2006;37:872–6.
25. Stanhope VA, Knarr BA, Reisman DS, Higginson JS. Frontal plane compensatory strategies associated with self-selected walking speed in individuals post-stroke. Clin Biomech (Bristol, Avon). 2014;29:518–22.
26. Chen G, Patten C, Kothari DH, Zajac FE. Gait differences between individuals with post-stroke hemiparesis and non-disabled controls at matched speeds. Gait Posture. 2005;22:51–6.
27. Bacik B, Saulicz E, Gnat R. Ground–foot reaction forces in hemiplegic gait patterns with and without orthopaedic aids. Int J Rehabil Res. 2006;29:255–9.
28. Bernstein N. The coordination and regulation of movements. Oxford: Pergamon Press; 1967.
29. Thelen E, Ulrich BD, Niles D. Bilateral coordination in human infants: stepping on a split-belt treadmill. J Exp Psychol Hum Percept Perform. 1987;13:405–10.
30. Thelen E, Ulrich BD, Niles D. The developmental origins of locomotion. In: Woollacott MH, Shumway-Cook A, editors. Development of posture and gait across the life span. Columbia, SC: University of South Carolina Press; 1989. p. 25–47.
31. Carmichael ST. Cellular and molecular mechanisms of neural repair after stroke: making waves. Ann Neurol. 2006;59:735–42.
32. Kwakkel G, Kollen B, Twisk J. Impact of time on improvement of outcome after stroke. Stroke. 2006;37:2348–53.
33. Nijland RH, van Wegen EE, Harmeling-van der Wel BC, Kwakkel G. Presence of finger extension and shoulder abduction within 72 hours after stroke predicts functional recovery: early prediction of functional outcome after stroke: the EPOS cohort study. Stroke. 2010;41:745–50.
34. Prabhakaran S, Zarahn E, Riley C, Speizer A, Chong JY, Lazar RM, et al. Inter-individual variability in the capacity for motor recovery after ischemic stroke. Neurorehabil Neural Repair. 2008;22:64–71.
35. Stinear CM, Barber PA, Petoe M, Anwar S, Byblow WD. The PREP algorithm predicts potential for upper limb recovery after stroke. Brain. 2012;135:2527–35.
36. Jorgensen HS, Nakayama H, Raaschou HO, Vive-Larsen J, Stoier M, Olsen TS. Outcome and time course of recovery in stroke. Part II: Time course of recovery. The Copenhagen Stroke Study. Arch Phys Med Rehabil. 1995;76:406–12.
37. Kwakkel G, Kollen BJ, van der Grond J, Prevo AJ. Probability of regaining dexterity in the flaccid upper limb: impact of severity of paresis and time since onset in acute stroke. Stroke. 2003;34:2180–6.
38. Lindberg PG, Gaverth J, Islam M, Fagergren A, Borg J, Forssberg H. Validation of a new biomechanical model to measure muscle tone in spastic muscles. Neurorehabil Neural Repair. 2011;25:617–25.
39. Latash ML, Scholz JP, Schoner G. Toward a new theory of motor synergies. Motor Control. 2007;11:276–308.
40. Mirbagheri MM, Tsao C, Rymer WZ. Natural history of neuromuscular properties after stroke: a longitudinal study. J Neurol Neurosurg Psychiatry. 2009;80:1212–7.
41. Kollen B, van de Port I, Lindeman E, Twisk J, Kwakkel G. Predicting improvement in gait after stroke: a longitudinal prospective study. Stroke. 2005;36:2676–80.
42. Krakauer JW, Carmichael ST, Corbett D, Wittenberg GF. Getting neurorehabilitation right: what can be learned from animal models? Neurorehabil Neural Repair. 2012;26:923–31.
43. Bethe A. Plastizität und Zentrenlehre. Handb Norm Path Physiol. 1930;15:1175–220.
44. Bach-y-Rita P. Brain plasticity as a basis for therapeutic procedures. In: Bach-y-Rita P, editor. Recovery of function: theoretical considerations for brain injury rehabilitation. Bern: Hans Huber Publishers; 1980. p. 225–63.
45. Cohen LG, Celnik P, Pascual-Leone A, Corwell B, Falz L, Dambrosia J, et al. Functional relevance of cross-modal plasticity in blind humans. Nature. 1997;389:180–3.
46. Nudo RJ. Plasticity. NeuroRx. 2006;3:420–7.
47. Birkenmeier RL, Prager EM, Lang CE. Translating animal doses of task-specific training to people with chronic stroke

in 1-hour therapy sessions: a proof-of-concept study. Neurorehabil Neural Repair. 2010;24:620–35.
48. Taub E, Uswatte G, Pidikiti R. Constraint-induced movement therapy: a new family of techniques with broad application to physical rehabilitation – a clinical review. J Rehabil Res Dev. 1999;36:237–51.
49. Cooke EV, Mares K, Clark A, Tallis RC, Pomeroy VM. The effects of increased dose of exercise-based therapies to enhance motor recovery after stroke: a systematic review and meta-analysis. BMC Med. 2010;8:60.
50. Hsieh YW, Wu CY, Liao WW, Lin KC, Wu KY, Lee CY. Effects of treatment intensity in upper limb robot-assisted therapy for chronic stroke: a pilot randomized controlled trial. Neurorehabil Neural Repair. 2011;25:503–11.
51. Nishibe M, Barbay S, Guggenmos D, Nudo RJ. Reorganization of motor cortex after controlled cortical impact in rats and implications for functional recovery. J Neurotrauma. 2010;27:2221–32.
52. Plautz EJ, Milliken GW, Nudo RJ. Effects of repetitive motor training on movement representations in adult squirrel monkeys: role of use versus learning. Neurobiol Learn Mem. 2000;74:27–55.
53. Kleim JA, Barbay S, Cooper NR, Hogg TM, Reidel CN, Remple MS, et al. Motor learning-dependent synaptogenesis is localized to functionally reorganized motor cortex. Neurobiol Learn Mem. 2002;77:63–77.
54. Remple MS, Bruneau RM, VandenBerg PM, Goertzen C, Kleim JA. Sensitivity of cortical movement representations to motor experience: evidence that skill learning but not strength training induces cortical reorganization. Behav Brain Res. 2001;123:133–41.
55. Witte OW, Bidmon HJ, Schiene K, Redecker C, Hagemann G. Functional differentiation of multiple perilesional zones after focal cerebral ischemia. J Cereb Blood Flow Metab. 2000;20:1149–65.
56. Feeney DM, Baron JC. Diaschisis. Stroke. 1986;17:817–30.
57. Kwakkel G, Kollen B, Lindeman E. Understanding the pattern of functional recovery after stroke: facts and theories. Restor Neurol Neurosci. 2004;22:281–99.
58. Kleim JA, Jones TA. Principles of experience-dependent neural plasticity: implications for rehabilitation after brain damage. J Speech Lang Hear Res. 2008;51:S225–39.
59. Woldag H, Hummelsheim H. Evidence-based physiotherapeutic concepts for improving arm and hand function in stroke patients: a review. J Neurol. 2002;249:518–28.
60. Carmichael ST. Rodent models of focal stroke: size, mechanism, and purpose. NeuroRx. 2005;2:396–409.
61. Kwakkel G, Meskers CG, van Wegen EE, Lankhorst GJ, Geurts AC, van Kuijk AA, et al. Impact of early applied upper limb stimulation: the EXPLICIT-stroke programme design. BMC Neurol. 2008;8:49.
62. Lundin-Olsson L, Nyberg L, Gustafson Y. "Stops walking when talking" as a predictor of falls in elderly people. Lancet. 1997;349:617.
63. Berg K, Wood-Dauphinée S, Williams JI, Gayton D. Measuring balance in the elderly: preliminary development of an instrument. Physiother Can. 1989;41:304–11.
64. Mathiowetz V, Weber K, Kashman N, Volland G. Adult norms for the Nine Hole Peg Test of finger dexterity. OTJR (Thorofare N J). 1985;5:24–38.
65. Podsiadlo D, Richardson S. The timed "Up & Go": a test of basic functional mobility for frail elderly persons. J Am Geriatr Soc. 1991;39:142–8.
66. Bohannon RW. Comfortable and maximum walking speed of adults aged 20–79 years: reference values and determinants. Age Ageing. 1997;26:15–9.
67. Perry J, Burnfield JM. Gait analysis: normal and pathological function. 2nd ed. Thorofare, NJ: SLACK; 2010.
68. Massie C, Malcolm MP, Greene D, Thaut M. The effects of constraint-induced therapy on kinematic outcomes and compensatory movement patterns: an exploratory study. Arch Phys Med Rehabil. 2009;90:571–9.
69. Zhang JT, Novak AC, Brouwer B, Li Q. Concurrent validation of Xsens MVN measurement of lower limb joint angular kinematics. Physiol Meas. 2013;34:N63–9.
70. Öhberg F, Lundström R, Grip H. Comparative analysis of different adaptive filters for tracking lower segments of a human body using inertial motion sensors. Meas Sci Technol. 2013;24:085703.
71. van Kordelaar J, van Wegen EE, Nijland RH, Daffertshofer A, Kwakkel G. Understanding adaptive motor control of the paretic upper limb early poststroke: the EXPLICIT-stroke program. Neurorehabil Neural Repair. 2013;27:854–63.
72. Bobath B. Adult hemiplegia: evaluation and treatment. 3rd ed. London: Butterworth-Heinemann; 1990.
73. Goff B. Appropriate afferent stimulation. Physiotherapy. 1969;55:9–17.
74. Knott M, Voss DE. Proprioceptive neuromuscular facilitation. 2nd ed. New York: Harper and Row; 1968.
75. Ada L, Dorsch S, Canning CG. Strengthening interventions increase strength and improve activity after stroke: a systematic review. Aust J Physiother. 2006;52:241–8.
76. Bohannon RW. Muscle strength and muscle training after stroke. J Rehabil Med. 2007;39:14–20.
77. Patten C, Lexell J, Brown HE. Weakness and strength training in persons with poststroke hemiplegia: rationale, method, and efficacy. J Rehabil Res Dev. 2004;41:293–312.
78. Flansbjer UB, Lexell J, Brogardh C. Long-term benefits of progressive resistance training in chronic stroke: a 4-year follow-up. J Rehabil Med. 2012;44:218–21.
79. Kim CM, Eng JJ, MacIntyre DL, Dawson AS. Effects of isokinetic strength training on walking in persons with stroke: a double-blind controlled pilot study. J Stroke Cerebrovasc Dis. 2001;10:265–73.
80. Moreland JD, Goldsmith CH, Huijbregts MP, Anderson RE, Prentice DM, Brunton KB, et al. Progressive resistance strengthening exercises after stroke: a single-blind randomized controlled trial. Arch Phys Med Rehabil. 2003;84:1433–40.
81. Horak F. Assumptions underlying motor control for neurologic rehabilitation. In: Lister M, editor. Contemporary management of motor control problems. Proceedings of the II STEP conference. Alexandria, VA: Foundations for Physical Therapy; 1991. p. 11–27
82. Carr JH, Shepherd RB. A motor relearning programme for stroke. London: Heinemann Medical; 1982.
83. French B, Thomas LH, Leathley MJ, Sutton CJ, McAdam J, Forster A, et al. Repetitive task training for improving functional ability after stroke. Cochrane Database Syst Rev. 2007:Cd006073.
84. Taub E, Miller NE, Novack TA, Cook EW III, Fleming WC, Nepomuceno CS, et al. Technique to improve chronic motor deficit after stroke. Arch Phys Med Rehabil. 1993;74:347–54.
85. Miltner WH, Bauder H, Sommer M, Dettmers C, Taub E. Effects of constraint-induced movement therapy on patients with chronic motor deficits after stroke: a replication. Stroke. 1999;30:586–92.
86. Wolf SL, Winstein CJ, Miller JP, Taub E, Uswatte G, Morris D, et al. Effect of constraint-induced movement therapy on upper extremity function 3 to 9 months after stroke: the EXCITE randomized clinical trial. JAMA. 2006;296:2095–104.

87. Wolf SL, Winstein CJ, Miller JP, Thompson PA, Taub E, Uswatte G, et al. Retention of upper limb function in stroke survivors who have received constraint-induced movement therapy: the EXCITE randomised trial. Lancet Neurol. 2008;7:33–40.
88. Sirtori V, Corbetta D, Moja L, Gatti R. Constraint-induced movement therapy for upper extremities in stroke patients. Cochrane Database Syst Rev. 2009:Cd004433.
89. Pollock A, Baer G, Campbell P, Choo PL, Forster A, Morris J, et al. Physical rehabilitation approaches for the recovery of function and mobility following stroke. Cochrane Database Syst Rev. 2014;4:Cd001920.
90. Pollock A, Baer G, Pomeroy V, Langhorne P. Physiotherapy treatment approaches for the recovery of postural control and lower limb function following stroke. Cochrane Database Syst Rev. 2007:Cd001920.