

RESEARCH

Open Access



# Effect of cycling exercise on motor excitability and gait abnormalities in stroke patients

Mohamed S. El-Tamawy<sup>1</sup>, Moshera H. Darwish<sup>2</sup>, Mye A. Basheer<sup>3</sup>, Abdelazim M. Reda<sup>1\*</sup>, Mahmoud Elzanaty<sup>2,4</sup> and Heba A. Khalifa<sup>2,5</sup>

## Abstract

**Background:** The concepts of brain excitability are still re-wiring in response to changes in environment. Ambulation is often limited in stroke patients.

**Objective:** To determine the effect of cycling exercise on motor excitability and consequences on spatiotemporal gait parameters in stroke patients.

**Methods:** Forty male ischemic stroke patients were included; their age ranged from 45 to 60 years. The patients were assigned into two equal groups: control group (GI) and study group (GII). The GI is treated by a design physical therapy program in the form of task-oriented progressive resistance exercise for lower limb muscles, and the GII is treated by the same program in addition to cycling exercise for 30 min. Treatment was conducted three times per week for 10 weeks. The excitability over motor area (Cz) was assessed by the quantitative electroencephalogram (QEEG). The spatiotemporal gait parameters were assessed by the Biodex Gait Trainer 2<sup>TM</sup>.

**Results:** There was a significant increase of speed, step cycle, and step length of the affected side ( $P < 0.05$ ) and a non-significant difference of step length of the non-affected side in the study group compared with that of the control group ( $P > 0.05$ ). There was a significant increase of excitability over motor area (Cz) in the study group compared with that of the control group ( $P < 0.05$ ).

**Conclusion:** Cycling exercise has a positive effect on excitability over motor area of lower limbs and can improve gait parameters in stroke patients.

**Keywords:** Stroke, Cycling exercise, Motor excitability, Gait parameters

## Introduction

Stroke burden in Egypt was found to be significant; epidemiological studies done revealed that the mean and median crude prevalence rates were 721.6/100,000 and 655/100,000, respectively; however, the mean and median crude incidence rates were 187/100,000 and 180.5/100,000, respectively [1].

Stroke is the leading causes of disability in most countries. It is responsible for a large proportion of the

problem of neurologic disorders. It is often disabling [2]. One of the most common complications in patients with stroke is the functional impairment of the lower extremity (LE). The LE motor function plays an important role in daily life activity. Skeletal muscle weakness, abnormal synergies of the muscles, and spasticity are the main causes which may inhibit the selective activation of muscles during walking [3].

The cerebral cortex has a crucial role in controlling cyclical motor tasks. Electroencephalogram (EEG) during pedaling suggested that the activity of the muscles acting during locomotion is under corticospinal control.

\* Correspondence: [abdelazimneuro@gmail.com](mailto:abdelazimneuro@gmail.com)

<sup>1</sup>Department of Neurology, Faculty of Medicine, Cairo University, Cairo, Egypt  
Full list of author information is available at the end of the article

By using a unipolar montage, the recording data was transformed into the mapping program to make spectral analysis of it. The data was recorded in this study from motor area (Cz) [4]. The equation utilized to detect brain activity was mean values of mean frequency of  $[(\delta + \theta) / (\alpha + \beta)]$  ratio [5]. The transitions from extension to flexion (and vice versa) during walking may be the most vulnerable locomotor function to be impaired after stroke [6].

One of the important rehabilitation programs is the leg cycling exercise as it improves the ambulatory function in stroke patients [7]. The reciprocal and rhythmic nature of cycling motion allows patients to generate timely symmetrical and reciprocal forces from both limbs required for locomotion. Cycling exercise improves the neuromuscular control for ambulation. It helps to maintain the functional range of motion of knee and hip joints required for walking. Cycling exercise improves several aspects of strength gain, cycling performance, cardio-respiratory function, and the use of the affected leg in patients with ischemic stroke [8].

The aim of the present study was to determine the effect of cycling exercise on motor excitability and its consequences on the spatiotemporal gait parameters in the stroke patients.

## Methods

Forty male ischemic stroke hemiparetic patients, confirmed by MRI or CT scan (in territory of carotid system), represented the sample of the study; the patients were selected from the Outpatient Clinic of Kasr El Aini, Teaching Hospital, Cairo University and from the Out-Patient Clinic, Faculty of Physical Therapy, Cairo University.

Patients were included with age ranged from 45 to 60; body mass index (BMI) was  $< 30 \text{ kg/m}^2$ . The stroke duration was at least 6 months before starting the study. All patients were able to walk 10 m independently without any assistive device (power grade 4) and the spasticity degree of paretic lower limb muscles ranged from 1 to 1+ according to the Modified Ashworth Scale. Patients with visual, auditory, or vestibular deficits or cognitive impairments; history of prior strokes or other neurological diseases; and significant orthopedic or chronic pain conditions for both lower limbs were excluded.

The patients signed a written consent form. The study protocol was approved by the ethical committee of Faculty of Physical Therapy, Cairo University, Egypt NO: P.T.REC/012/00969 (26/5/2015).

All patients were subjected to thorough clinical neurological assessment. The patients were assigned randomly into two equal groups: control group (GI) and study group (GII). The control group (GI) received a design physiotherapy program in the form of task-oriented

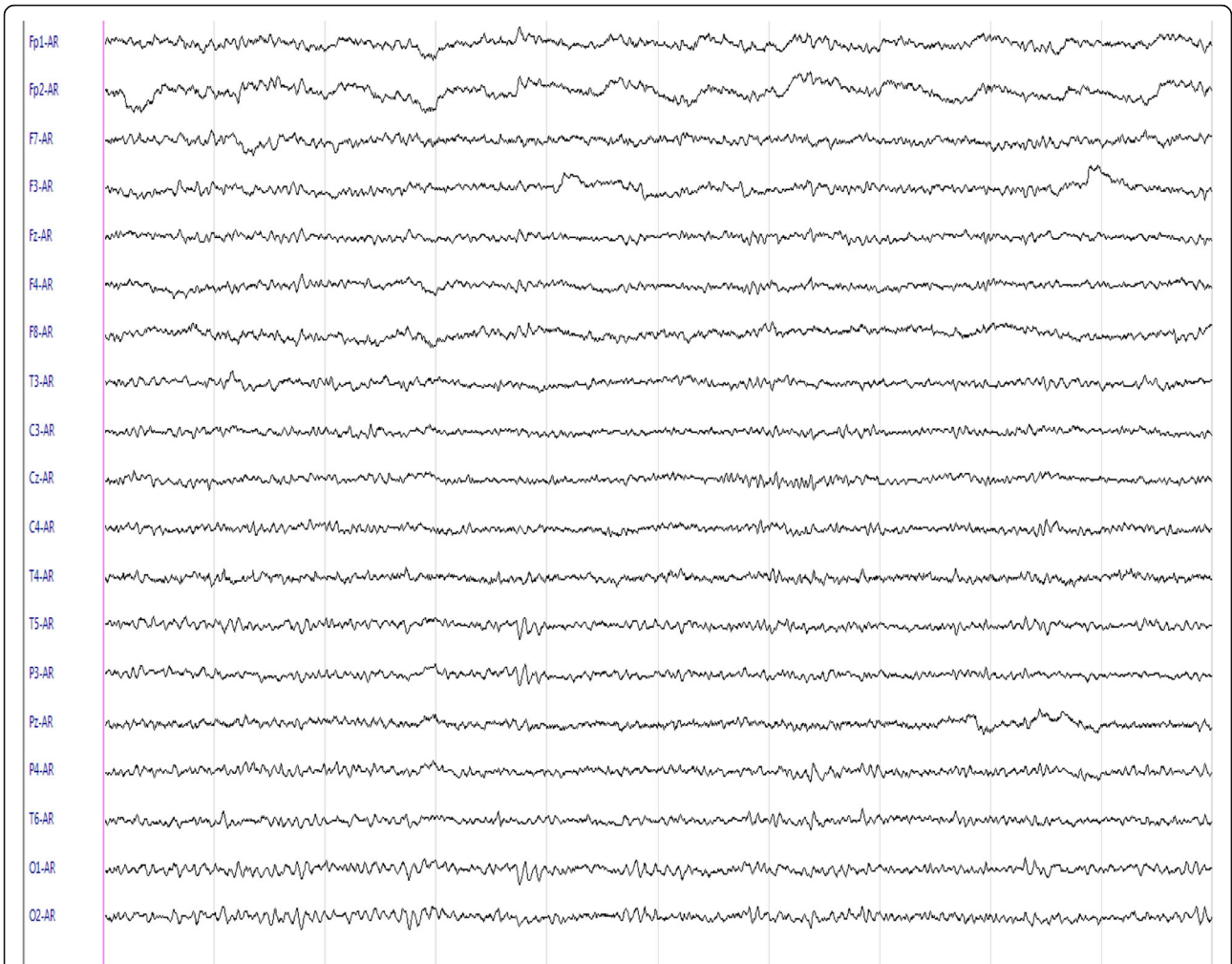
progressive resistance exercise for lower limb muscles with total duration of 1 h for a session. The study group (GII) received the same program as group (GI) for 30 min in addition to cycling exercise using Bicycle ergometer Kettler polo S (Model K7960-700, Germany) for 30 min with a total duration 1 h. The program for both groups was three sessions per week for 10 weeks.

The motor excitability was assessed by the QEEG (EB, Florence, Italy; Mizar –PC Peripheral System CE Version – B9800037800) at a brain lab EEG recorder at Kaser Al Aini Teaching Hospital and Cairo University. The patient was comfortable with the cap on while the clinician was recording EEG on a computer. The patient was asked to close eyes to minimize eye movements and blink artifacts and to prevent any visual feedback. The data was recorded according to the International 10–20 system with Ag/AgCl electrodes, using a unipolar montage. Impedance was kept  $< 5 \text{ kohm}$  to avoid polarization effects (Fig. 1). The recorded data was transformed into quantitative values: quantitative EEG. Various data were available, like brain maps for spectral analysis. However, numerical values of mean frequencies in the motor area (Cz) were the elements chosen for further statistical analysis (Fig. 2) [4]. The equation utilized to detect brain activity was mean values of mean frequency of  $[(\delta + \theta) / (\alpha + \beta)]$  ratio [5]. It was done before the program and after the end of the program for both groups.

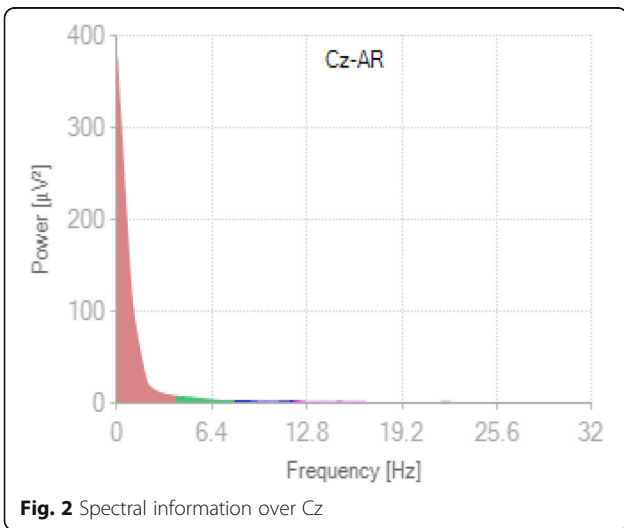
Spatiotemporal parameters of gait were assessed by Biodex Gait Trainer 2<sup>TM</sup> (model NO. 601-1-M90, EN 60601-1 USA). Each patient was instructed to wear suitable clothes and suitable light shoes before assessment. The patient was standing on the treadmill holding on handrails to ensure safety. The speed of treadmill was ramp up slowly to 0.3 m/h. It was then increased gradually to comfortable pace for the patient; then, the data recording was started. Each patient was asked to walk continuously for 3 min, till the evaluation session ended, and the gait trainer slow down gradually until it stopped. Three successive trials were done for each patient. The mean value of each variable of spatiotemporal parameters of gait [walking speed (m/s), step cycle (cycle/s), and step length (m)] were recorded [9]. It was done before the program and after the end of the program for both groups.

## Statistical analysis

Collected data were analyzed by the Statistical Package for Social Studies (SPSS) version 19 for windows (IBM SPSS, Chicago, IL, USA). Descriptive statistics were used in the form of arithmetic mean and standard deviation for all variables. Normality of data was checked using the Shapiro-Wilk test. Unpaired *t* test was used to compare the general chronological features of the patients (age and BMI) of both groups. The mixed design



**Fig. 1** EEG traces with all measurable channels displayed; average reference montage



**Fig. 2** Spectral information over Cz

multivariate analysis of variant test was used to compare all of the tested dependent variables within and between the two groups at two measuring periods (pre- and post-treatment). *P* value was used to indicate the level of significance ( $P < 0.05$  was considered significant).

**Results**

There was no statistically significant difference between both groups regarding the age and body mass index ( $P > 0.05$ ) (Table 1).

Both groups showed significant increase in the mean values of walking speed, step cycle, and step length of the non-affected side post-treatment compared with that of pre-treatment ( $P < 0.05$ ). Moreover, there was a significant increase in the mean value of step length of the affected side and motor excitability over “Cz” only in GII ( $P < 0.05$ ) (Table 2).

Regarding the mean values of pretreatment walking speed, step cycle, step length of the affected side, step

**Table 1** The mean values of age and body mass index in the control and study groups

General characteristics	Control group		t value	P value
	Mean ± SD	Study group Mean ± SD		
Age (years)	53.2 ± 4.11	52.55 ± 4.34	0.48	0.63
Body mass index (kg/m <sup>2</sup> )	27.36 ± 2.72	27.68 ± 2.68	- 0.38	0.71

SD standard deviation, P probability, non-significant ( $P > 0.05$ )

length of the non-affected side, and motor excitability over “Cz,” there was no statistically significant difference between both groups ( $P > 0.05$ ). Post-treatment comparison between both groups revealed significant increase in the mean values of walking speed, step cycle, step length of the affected side, and motor excitability over “Cz” and it was in favor of the study group (GII) ( $P < 0.05$ ). However mean values of step length of the non-affected side showed no significant difference between both groups ( $P > 0.05$ ) (Table 3).

## Discussion

In patients with ischemic stroke, the function of cerebral cortex becomes impaired with preserved spinal cord function. The ability to generate information of the spinal cord required for walking can be utilized through specific movements to reorganize the cortex for walking [10]. The results of the current study showed a significant increase in the mean values of motor excitability over motor area of lower limbs (Cz) in study group comparing to control. These results can be explained from neurophysiological point of view that cycling exercise after stroke can cause gradual shrinking of the lesion volume [11, 12].

Also from neurophysiological point of view, cycling exercise works on neuronal plasticity as it activates both supplementary motor areas and primary motor cortex bilaterally. Rhythmic motor tasks such as pedaling is generated and under control of these higher motor centers [13]. Primary motor area (M1) is a highly plastic region with repeated training causing reorganization

which associated with skill acquisition and motor learning. Facilitation of motor learning induces the motor recovery by directly increasing the excitability in the ipsilesional motor cortex [14]. This explained the increased motor excitability in the study group.

The results of the present study contradicted with the finding of Ambrose et al. [15] who demonstrated that reciprocal exercise do not induce significant alterations in brain activity. The discrepancy between the two studies may be attributed to the frequency of exercise program (once weekly) that may be a decisive factor in altering behavioral performance and hemodynamic activity in cortex.

The results of the current study showed a non-significant change in the mean values of cortical reorganization over the selected brain areas in control group. These results agree with Smith et al. [16] and Dobkin [17], who explained that the designed exercise program alone was insufficient to increase the excitability of output neurons in the primary motor area and other brain areas. Strength training, by contrast, can alter the excitability of motor neurons and induce synaptogenesis within the spinal cord, but does not alter the cortical motor organization.

Cycling exercise increases proteins and genes that protect from oxidative damage, and free radical levels do not significantly elevate [18]. Cycling exercise after stroke reduced DNA fragmentation [19] and caspase-3 [11], and all of which are markers and/or facilitators of

**Table 2** Comparison of spatiotemporal gait parameters and motor excitability over Cz pre- and post-treatment within each group

	Control group			Study group		
	Pre-treatment (mean ± SD)	Post-treatment (mean ± SD)	P value	Pre-treatment (mean ± SD)	Post-treatment (mean ± SD)	P value
Walking speed (m/s)	0.55 ± 0.08	0.61 ± 0.11	0.001*	0.54 ± 0.06	0.68 ± 0.08	0.0001*
Step cycle (cycle/s)	0.55 ± 0.1	0.62 ± 0.12	0.02*	0.54 ± 0.13	0.72 ± 0.19	0.0001*
Step length (m)						
Affected side	0.38 ± 0.1	0.41 ± 0.08	0.24	0.37 ± 0.09	0.52 ± 0.18	0.0001*
Non-affected side	0.51 ± 0.1	0.56 ± 0.09	0.04*	0.50 ± 0.12	0.60 ± 0.2	0.0001*
Cz	3.9 ± 0.38	3.93 ± 0.24	0.79	3.91 ± 0.6	4.19 ± 0.47	0.01*

SD standard deviation

\*Significant ( $P < 0.05$ )

**Table 3** Comparison of spatiotemporal gait parameters and motor excitability over Cz pre- and post-treatment between both groups

	Pre-treatment			Post-treatment		
	Control group	Study group	<i>P</i> value	Control group	Study group	<i>P</i> value
Walking speed (m/s)	0.55 ± 0.08	0.54 ± 0.06	0.58	0.61 ± 0.11	0.68 ± 0.08	0.03*
Step cycle (cycle/s)	0.55 ± 0.1	0.54 ± 0.13	0.76	0.62 ± 0.12	0.72 ± 0.19	0.04*
Step length (m)						
Affected side	0.38 ± 0.1	0.37 ± 0.09	0.65	0.41 ± 0.08	0.52 ± 0.18	0.03*
Non-affected side	0.51 ± 0.1	0.50 ± 0.12	0.78	0.56 ± 0.09	0.60 ± 0.2	0.41
Cz	3.9 ± 0.38	3.91 ± 0.6	0.95	3.93 ± 0.24	4.19 ± 0.47	0.03*

\*Significant ( $P < 0.05$ )

apoptotic cell death [20]. Cycling exercise stimulates several mechanisms of recovery, such as angiogenesis, neurogenesis, and synaptogenesis, which contribute to neuronal and synaptic plasticity [21]. Moderate to high intensity of cycling exercise after focal ischemia resulted in increased angiogenesis-promoting proteins in the cortex surrounding the lesion [22] and striatum [13, 23].

The results of the current study showed a significant increase in the mean values of gait parameters in the study group comparing to control. These results can be explained from neurophysiological point of view that gait results from dynamic interactions between a central program and feedback mechanisms. The muscles and skin afferents as well as some senses (vision, audition, vestibular) generate the feedback that dynamically adapts the gait pattern to the environmental requirement. Proprioceptive inputs can adjust the degree and timing of activity of the muscles to the gait speed. Stimulation of descending pathways may affect gait pattern in particular phases of step cycle [24].

The superior improvement of gait spatiotemporal parameters in the study group could be explained as cycling exercise may improve gait deficits in stroke patients through several mechanisms: (a) it acts as a pseudo walking task-oriented exercise standing [25]; (b) it facilitates muscle control and improves muscle strength of the lower limbs, so it allows putting more weight on the affected leg while standing [26]; (c) it provides adequate sensory input from vestibular system, feet, and neck which may stimulate the spinal locomotor circuitry (central pattern generator) leading to a regular walking pattern [27]; (d) it facilitates the frontal-lobe cognitive strategies by externally cueing the sequential movement [28]; and (e) it improves cardiopulmonary capacity and metabolic walking economy and reduces the effort of sub-maximal workloads [29].

The spinal cord generate the basic motor pattern for stepping, while various brain regions, including cerebral motor cortex, brain stem, and cerebellum, control fine walking [30]. The spinal cord has central pattern generators (CPGs) that are networks of nerve cells that

generate movements and enclose the information necessary to activate different motor neurons in the suitable order and intensity to generate motor patterns. These networks have been proposed to be “innate” although “perfected and adapted by experience.” The three principles that characterize CPGs are as follows: (I) the capacity to generate intrinsic pattern of rhythmic activity regardless the sensory inputs, (II) the presence of a developmentally defined neuronal circuit, and (III) the modulatory influences from central and peripheral inputs [31]. The CPG functions mainly in guiding post-lesional neuronal plasticity. Over ground walking, a spinal pattern generator seems to be sufficient. Supraspinal control is mandatory to provide both the drive for locomotion and the coordination to negotiate a complex environment [32].

From biomechanical point of view, the improvements of gait parameters in study group could be explained by good stability around both knee and hip that increases stability in standing. The patient was able to take longer stride, which makes it easier for the ankle plantar flexors to push off actively. The increased stride length, speed, and push off correspond well with the increase in muscle strength around the hips, knee, and ankle. This may be mediated by strengthening exercise that emphasizes specific training of motor control in activities and represents a shift away from facilitation of movement and exercise therapy [33, 34].

Strengthening exercise provides also feedback from the entire lower extremity and increases stimulation of the mechanoreceptors around the joints and firing of muscle spindles. In addition, gait function of stroke patients improve through motor re-learning by increased neuroplasticity and brain reorganization in response to repetition of the reciprocal task [35].

The results of the present study disagreed with the results of Globas et al. [36] who revealed that cycling exercises had non-significant alterations in gait parameters. The discrepancy between results of the current study and results of Globas et al.’s study may be due to the inherent nature of mobility measure used. The small sample size in combination with strict inclusion and

exclusion criteria might have selected a healthier cohort with higher mobility than the average person with chronic stroke.

## Conclusion

It could be concluded that cycling exercise has a positive effect on excitability over motor area of lower limbs and can improve gait parameters in stroke patients.

## Abbreviations

BMI: Body mass index; CPG: Central pattern generator; CZ: Central presentation of motor area (lower limbs); EEG: Electroencephalogram; LE: Lower extremity; M1: Primary motor cortex; QEEG: Quantitative electroencephalogram; SPSS: Statistical Package for Social Studies

## Acknowledgements

The authors acknowledge subjects for their participation and cooperation in this study.

## Authors' contributions

MSE, MHD, and MAB: research idea, data acquisition, data analysis and interpretation, and manuscript writing and reviewing. AMR and HAK: data acquisition, data analysis and interpretation, and manuscript writing and reviewing. ME: data acquisition and data analysis and interpretation. All authors have read and approved the manuscript.

## Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

## Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly available due to current Cairo University regulations and Egyptian legislation but are available from the corresponding author on reasonable request and after institutional approval.

## Ethics approval and consent to participate

The aim and procedures of the study were explained to every participant, and an informed written consent was obtained from all participants before being enrolled in the study. The study was approved by the ethical committee of Department of Physical Therapy for Neuromuscular Disorders and its Surgery, Faculty of Physical Therapy, Cairo University: NO:P.T.REC/012/00969 (26/5/2015).

## Consent for publication

Not applicable.

## Competing interests

The authors declare that we have no competing interests (financial and non-financial). We declare that the research was conducted in absence of any commercial relationships that could be constructed as a potential conflict of interest.

## Author details

<sup>1</sup>Department of Neurology, Faculty of Medicine, Cairo University, Cairo, Egypt. <sup>2</sup>Department of Physical Therapy for the Neuromuscular Disorder and its Surgery, Faculty of Physical Therapy, Cairo University, Cairo, Egypt. <sup>3</sup>Department of Clinical Neurophysiology, Faculty of Medicine, Cairo University, Cairo, Egypt. <sup>4</sup>Department of Physical Therapy for the Neuromuscular Disorder and its Surgery, Faculty of Physical Therapy, Deraya University, New Minya, Egypt. <sup>5</sup>Physical Therapy, Department of Physical Therapy for the Neuromuscular Disorder and its Surgery, Faculty of Physical Therapy, Badr University in Cairo (BUC), Cairo, Egypt.

Received: 14 April 2020 Accepted: 16 October 2020

Published online: 07 January 2021

## References

1. Abd-Allah F, Khedr E, Oraby MI, Bedair AS, Georgy SS, Moustafa RR. Stroke burden in Egypt: data from five epidemiological studies. *Int J Neurosci*. 2018;128(8):765–71.
2. Jemal A, Ward E, Hao Y, Thun M. Trends in the leading causes of death in the United States, 1970–2002. *JAMA*. 2005;294(10):1255–9.
3. Yang HC, Lee CL, Lin R, Hsu MJ, Chen CH, Lin JH, et al. Effect of biofeedback cycling training on functional recovery and walking ability of lower extremity in patients with stroke. *Kaohsiung J Med Sci*. 2014;30(1):35–42.
4. Jain S, Gourab K, Schindler-Ivens S, Schmit BD. EEG during pedaling: evidence for cortical control of locomotor tasks. *Clin Neurophysiol*. 2013;124(2):379–90.
5. Sheorajpanday RV, Nagels G, Weeren AJ, van Putten MJ, De Deyn PP. Quantitative EEG in ischemic stroke: correlation with functional status after 6 months. *Clin Neurophysiol*. 2011;122(5):874–83.
6. Jin H, Jiang Y, Wei Q, Wang B, Ma G. Intensive aerobic cycling training with lower limb weights in Chinese patients with chronic stroke: discordance between improved cardiovascular fitness and walking ability. *Disabil Rehabil*. 2012;34(19):1665–71.
7. Hwang S, Lee HM, Cherng RJ, Chen JJ. Electromyographic analysis of locomotion for healthy and hemiparetic subjects—study of performance variability and rail effect on treadmill. *Gait Posture*. 2003;18(1):1–2.
8. Sibley KM, Tang A, Brooks D, Brown DA, Mclroy WE. Feasibility of +adapted aerobic cycle ergometry tasks to encourage paretic limb use after stroke: a case series. *J Neurol Phys Ther*. 2008;32(2):80–7.
9. Druzbicki M, Guzik A, Przsada G, Kwolek A, Brzozowska-Magoń A. Efficacy of gait training using a treadmill with and without visual biofeedback in patients after stroke: a randomized study. *J Rehabil Med*. 2015;47(5):419–25.
10. Verma R, Arya KN, Sharma P, Garg RK. Understanding gait control in post-stroke: implications for management. *J Bodyw Mov Ther*. 2012;16(1):14–21.
11. Zhang L, Hu X, Luo J, Li L, Chen X, Huang R, et al. Physical exercise improves functional recovery through mitigation of autophagy, attenuation of apoptosis and enhancement of neurogenesis after MCAO in rats. *BMC Neurosci*. 2013;14(1):46.
12. Sakakima H, Khan M, Dhammu TS, Shunmugavel A, Yoshida Y, Singh I, et al. Stimulation of functional recovery via the mechanisms of neurorepair by S-nitrosoglutathione and motor exercise in a rat model of transient cerebral ischemia and reperfusion. *Restor Neurol Neurosci*. 2012;30(5):383–96.
13. Christensen LO, Johannsen P, Sinkjaer T, Petersen N, Pyndt HS, Nielsen JB. Cerebral activation during bicycle movements in man. *Exp Brain Res*. 2000;135(1):66–72.
14. Nudo RJ, Milliken GW, Jenkins WM, Merzenich MM. Use-dependent alterations of movement representations in primary motor cortex of adult squirrel monkeys. *J Neurosci*. 1996;16:785–807.
15. Ambrose T, Nagamatsu LS, Voss MW, Khan KM, Handy TC. Resistance training and functional plasticity of the aging brain: a 12-month randomized controlled trial. *Neurobiol Aging*. 2012;33(8):1690–8.
16. Smith A, Goldsworthy MR, Garside T, Wood FM, Ridding MC. The influence of a single bout of aerobic exercise on short-interval intracortical excitability. *Exp Brain Res*. 2014;232:1875–82.
17. Dobkin BH. Training and exercise to drive poststroke recovery. *Nat Clin Pract Neurol*. 2008;4(2):76–85.
18. Cechetti F, Worm PV, Elsner VR, Bertoldi K, Sanches E, Ben J, et al. Forced treadmill exercise prevents oxidative stress and memory deficits following chronic cerebral hypoperfusion in the rat. *Neurobiol Learn Mem*. 2012;97(1):90–6.
19. Lee HH, Shin MS, Kim YS, Yang HY, Chang HK, Lee TH, et al. Early treadmill exercise decreases intrastriatal hemorrhage-induced neuronal cell death and increases cell proliferation in the dentate gyrus of streptozotocin-induced hyperglycemic rats. *J Diabetes Complications*. 2005;19(6):339–46.
20. Luo CX, Jiang J, Zhou QG, Zhu XJ, Wang W, Zhang ZJ, et al. Voluntary exercise-induced neurogenesis in the posts ischemic dentate gyrus is associated with spatial memory recovery from stroke. *J Neurosci Res*. 2007;85(8):1637–46.
21. Ergul A, Alhusban A, Fagan SC. Angiogenesis: a harmonized target for recovery after stroke. *Stroke*. 2012;43(8):2270–4.
22. Ma Y, He M, Qiang L. Exercise therapy downregulates the overexpression of TLR4, TLR2, MyD88 and NF-κB after cerebral ischemia in rats. *Int J Mol Sci*. 2013;14(2):3718–33.

23. Yang YR, Chang HC, Wang PS, Wang RY. Motor performance improved by exercises in cerebral ischemic rats. *J Mot Behav*. 2012;44(2):97–103.
24. Rossignol S, Dubuc R, Gossard JP. Dynamic sensorimotor interactions in locomotion. *Physiol Rev*. 2006;86(1):89–154.
25. Katz-Leurer M, Sender I, Keren O, et al. The influence of early cycling training on balance in stroke patients at the subacute stage. Results of a preliminary trial. *Clin Rehabil*. 2006;20:398–405.
26. Kim S, Cho H, Kim Y, Lee S. Effects of stationary cycling exercise on the balance and gait abilities of chronic stroke patients. *J Phys Ther Sci*. 2015; 27(11):3529–31.
27. Klarner T, Barss TS, Sun Y, Kaupp C, Zehr EP. Preservation of common rhythmic locomotor control despite weakened supraspinal regulation after stroke. *Front Integr Neurosci*. 2014;8(95):1–9.
28. Moriya M, Aoki C, Sakatani K. Effects of physical exercise on working memory and prefrontal cortex function in post-stroke patients. *Adv Exp Med Biol*. 2016;923:203–8.
29. Ofori E, Frimpong E, Ademiluyi A, Olawale O. Ergometer cycling improves the ambulatory function and cardiovascular fitness of stroke patients—a randomized controlled trial. *J Phys Ther Sci*. 2019;31(3):211–6.
30. Takakusaki K. Neurophysiology of gait: from the spinal cord to the frontal lobe. *Mov Disord*. 2013;28(11):1483–91.
31. Grillner S. The motor infrastructure: from ion channels to neuronal networks. *Nat Rev Neurosci*. 2003;4(7):573.
32. Field-Fote EC, Dietz V. Single joint perturbation during gait: preserved compensatory response pattern in spinal cord injured subjects. *Clin Neurophysiol*. 2007;118(7):1607–16.
33. Salem Y, Godwin EM. Effects of task-oriented training on mobility function in children with cerebral palsy. *NeuroRehabilitation*. 2009;24(4):307–13.
34. Manns PJ. Commentary to: task-specific and impairment-based training improve walking ability in stroke. *J Physiother*. 2014;60(2):110.
35. Lee NK, Kwon JW, Son SM, Kang KW, Kim K, Hyun-Nam S. The effects of closed and open kinetic chain exercises on lower limb muscle activity and balance in stroke survivors. *NeuroRehabilitation*. 2013;33(1):177–83.
36. Globas C, Becker C, Cerny J, Lam JM, Lindemann U, Forrester LW, et al. Chronic stroke survivors benefit from high-intensity aerobic treadmill exercise: a randomized control trial. *Neurorehabil Neural Repair*. 2012;26(1): 85–95.

## Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Submit your manuscript to a SpringerOpen<sup>®</sup> journal and benefit from:

- Convenient online submission
- Rigorous peer review
- Open access: articles freely available online
- High visibility within the field
- Retaining the copyright to your article

---

Submit your next manuscript at ► [springeropen.com](https://www.springeropen.com)

---