# Electrical Stimulation of the Posterior Spinal Cord Horns and its Effect on Gait Kinematics

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**Summary**

Spinal cord stimulation (SCS) is often used as therapy for some difficult-to-treat painful conditions. Its analgesic effect is well known, but less studied are its effects on motor function. Single case reports have described improved motor function in patients implanted with neuromodulation for painful conditions. The exact mechanism of action underlying this reported improvement also is not known. This study included 15 participants who had received SCS implantation for persistent spinal pain syndrome and did not have any condition that otherwise limited their locomotion. Hypothesizing that stimulation would affect their gait parameters, we recorded 12 attempts to walk barefoot at each patient's preferred speed, six before stimulation (with pain) and six after stimulation (with reduced pain perception). Kinematic analysis was performed using 10 infrared cameras and force platforms. Retroreflective markers and clusters were attached to the participants for these recordings and the data processed using software. We found significant improvements in gait acceleration, stride lengthening, and range of motion of the pelvis and hip joints. SCS may affect locomotion in the long term in ways that could be associated with reduced fall risk, suggesting benefits for a broader patient population, such as those with Parkinson's disease. Further investigations of motor changes in patients with implanted SCS are needed.

**Key words**

Spinal cord stimulation • Gait • Kinematics • Pain • Locomotion • Parkinson´s disease • Neuromodulation

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## Introduction

The analgesic effect of SCS is well known, but less is known about its effects on motor function. In two case reports of patients who received SCS implantation for Persistent Spinal Pain Syndrome (PSPS), Buonocore *et al.* described improvement in motor function, but with only minimal and transient pain relief [1,2]. Improvements in motor function also have been reported in patients with cerebral palsy, dystonia, multiple sclerosis, torticollis, and traumatic spinal cord injury. In a study following 735 patients implanted with SCS for various motor disorders, Waltz *et al.* found improvements in walking, activities of daily living (ADL), and hand dexterity, reduced frequency of spasms, improved balance and stability, and reduced tremor [3]. Rijken's study investigated the effect of SCS on balance control and gait in PSPS patients who underwent SCS. Gait analysis was performed using a Vicon eight-camera system and a treadmill. The authors did not report any changes in stride length or frequency or significant changes in lower limb joint kinematics [4]. Wilson, in

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a similar study with natural walking on the floor, described significant changes in hip ROM [5]. Yozu, in turn, demonstrated significantly increased step frequency, walking speed, and increased hip ROM in a patient with thalamic pain after SCS implantation [6]. One possible explanation could be a direct effect of SCS on motor neurons through facilitation of motor neurons located deep below the site of stimulation in the posterior horns of the spinal cord [7]. In addition to effects on local and integrated spinal circuits, some studies have described effects on multiple projection bundles to the brainstem, cerebellum, basal ganglia, thalamus and cortical areas [8]. A supporting factor may also be the location of the electrodes in the epidural space of the thoracic spine where, due to the anatomical conditions of the fibers passing through, SCS can modulate deeper fiber layers and a wider range of ascending pathways. There is also an increased stimulation threshold at this level of the spinal cord. The nuclei that collateralize and transmit afferents in the dorsal column and spinocerebellar tracts are located here, and these directly reach various structures in the brainstem, cerebellum and diencephalon. Pain relief induced by SCS also may partially mediate motor improvement in patients undergoing stimulation for pain. Motor function also improved in patients with spinal cord injury (SCI). In subjects with functionally complete sensory and motor paralysis, after electrical epidural stimulation of the lumbosacral spinal cord with regular rehabilitation, the ability to maintain full standing with own weight, increased muscle activation, and improved standing balance and trunk stability in sitting were observed. Restoration of voluntary movement is also possible [9]. Following injury, there may be combinations of unidentified descending anatomical connections that may have a supraspinal effect on the excitability of the spinal circuits beneath the lesion [10]. However, residual neural activity is unable to reach threshold levels for activation. Electrical epidural stimulation enhances the efficacy of residual descending signals from supraspinal motor areas through both stimulation of dorsal roots of afferent sensory neurons that go into synapse with interneurons integrating proprioceptive input and through direct activation of efferent motor neurons. The reorganization of the nervous system after SCI also plays a role [9].

The lumbar spine plays a vital role in connecting the upper and lower body, facilitating the transmission of forces and coordinating all movements. Chronic Lower back pain (LBP) affects motor function of the trunk and lower limbs [11). Patients with LBP have impaired motor control and impaired proprioception, limiting their ability to adapt their gait to changing circumstances and to deal with unexpected obstacles. They often compensate by walking more slowly and less flexibly. In addition, this response to pain affects postural stability, pelvic–spinal coordination, joint range of motion (ROM), and level of movement pattern variability. Gait can be uneven and stride time and stride length reduced [11]. Reduced ROM of the pelvis, spine, and hip joint have been reported, as has disruption of the coordination patterns on the pelvis–spine axis [12]. Because of a lower level of variability, patients with LBP tend to be more susceptible to injury [13]. These LBP-related changes can be observed during normal daily activities but become more pronounced with increased effort, such as cycling. The protective adjustments to gait and movement result from efforts to control pain, such as by reducing muscle activity or joint ROM. Increased activity of the erector spinae muscles in response to painful conditions has been described and inferred to stabilize the spine and inhibit further stress on structures [12]. In addition to these mechanical responses, restriction of movement and continuous activity can lead to the accumulation of metabolic wastes, which may contribute to the chronicity of pain [14]. Such pain adaptation is thus likely a protective strategy against further pain and injury, but in the long term, it can have negative consequences [15].

Gait analysis is commonly used to assess the relationship between LBP and altered motor control. One of the most commonly impaired ADL in patients with LBP is walking. This complex movement requires integration of systems (visual, vestibular, musculoskeletal), high motor control, and precise and coordinated limb movements. Normally, walking is a highly flexible and adaptive activity that is constantly changing to meet internal and environmental demands. In normal gait, coordinated trunk and pelvic rotation patterns and muscle activity are important for maintaining dynamic balance, as well as for reducing energy expenditure and effectively addressing disturbances during locomotion [16]. It has been proposed that back pain leads to a disturbance in the perception of limb position and movement, altering gait coordination and affecting gait parameters [17].

The aim of our study was to compare the kinematic parameters of gait in participants with implanted SCS before and after electrical stimulation of the posterior spinal cord horns. We hypothesized that

electrical stimulation would lead to increased gait speed and thus to changes in gait kinematics.

## Methods

This prospective observational study was approved by the Ethics Committee of the University of Ostrava and the University Hospital. The selected participants were followed for a long time in the Pain Clinic of the University Hospital, and all participants signed informed consent. For inclusion in the study, the following criteria had to be met: ability to walk without crutches and a diagnosis of PSPS with SCS for chronic intractable pain. The time since stimulator implantation at the time of measurement ranged from 7 to 2 years (mean 4 years). In all participants, thoracic electrodes were implanted percutaneously and connected to the generator subcutaneously (Fig. 1). Stimulation parameters were set individually by the technician after implantation and were adjusted, if necessary, by the technician at subsequent regular follow-up visits according to the patient's needs. In the group of participants studied, the frequency was between 40 and 60 Hz, the pulse width between 200 and 450 μs, and the amplitude between 3 and 8 V. The participants were subsequently followed up in the pain clinic every 3 months. Subjectively, all participants reported an improvement in analgesia (decrease in VAS) on average by 4 orders of magnitude and quality of life (QoL) after implantation, and most reported a reduction in analgesics. Average sleep time improved by 2 h. Participants were excluded if they had diabetic polyneuropathy, rheumatoid arthritis, ankylosing spondyloarthritis, hip osteoarthritis, systemic lupus erythematosus, lower extremity ischemic disease, active psychiatric disorder, or any other significant condition that would affect pain perception. The total cohort consisted of 15 participants (2M; 13F) with a mean age of 48.6 years.

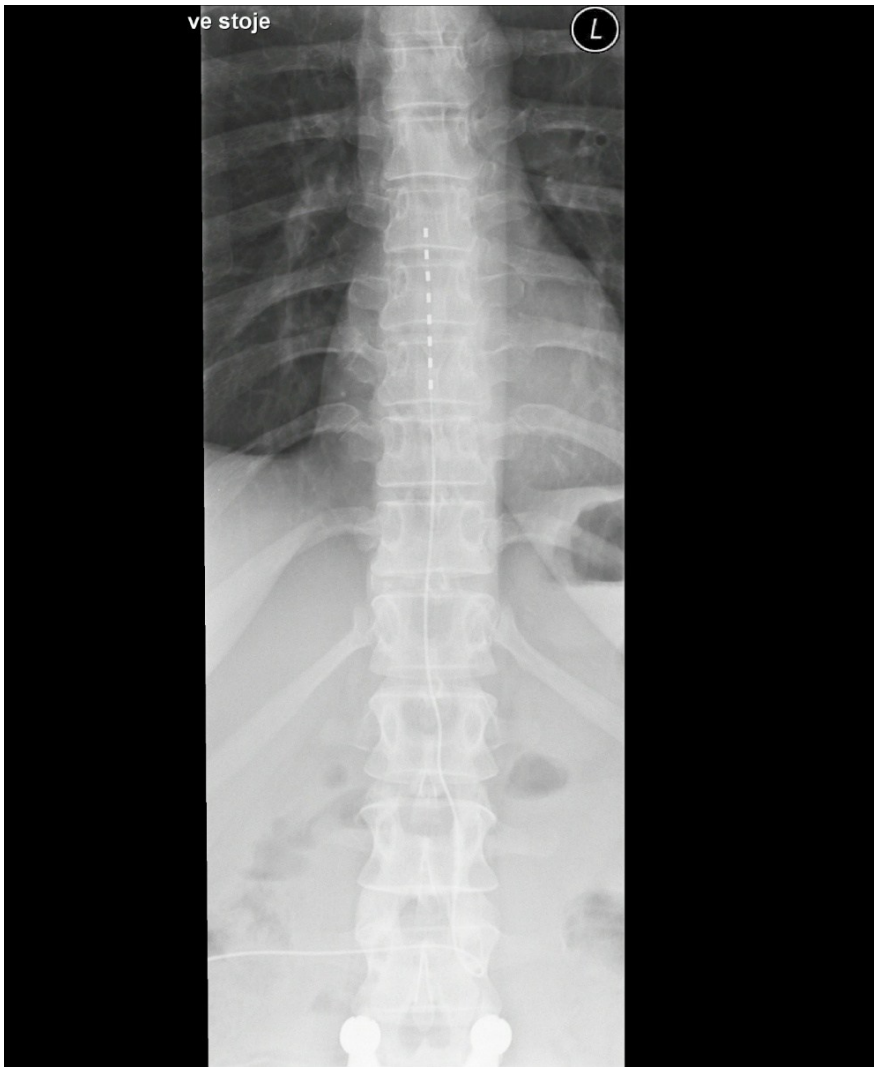



**Fig. 1.** Placement of the electrode in the thoracic epidural space at the level of Th7-Th9. L = left side. "Ve stoje" means standing.

Gait was recorded using 10 cameras motion capture system (9× Oqus 700+, 1× Oqus 510+, Qualisys, Inc., Gothenburg, Sweden). Three force platforms (Kistler 9286AA, 9281CA, and 9287CCAQ02, Kistler Instruments AG, Winterthur, Switzerland) embedded in a walkway 10 m in length were used to analyze reaction force data. The frequency of the captured kinematic data was set to 240 Hz and 2160 Hz.

Participants performed 12 valid barefoot walking trials, at their own preferred speed. Of these, six walking trials were performed before stimulation and six trials after stimulation. Retroreflective markers were placed on the pelvis and lower limbs. The beginning and end of the walkway was marked with cones. Participants were asked to walk naturally in the marked area. An attempt was considered valid if the patient stepped with the whole right foot on the first force platform. Measurements were always performed in the afternoon, and participants were instructed not to stimulate at all or to do so as little as possible on the day of measurement. 13 participants did not stimulate at all on the day of measurement, 2 stimulated in the morning. All participants reported increased pain perception as assessed by VAS. In addition, seven participants used higher doses of additional oral analgesia especially non-steroidal anti-inflammatory drugs or tramadol. Tonic stimulation was performed individually according to the patient's needs and lasted 10-20 min.

Kinematic data were processed using QTM software (Track Manager; Qualisys, Sweden, Göteborg) and Visual3D software (C-Motion, Germantown, Kentucky, KY, USA). Marker trajectories were filtered using a fourth-order Butterworth low-pass filter with a cut-off frequency of 12 Hz (kinematic) and 40 Hz (kinetic).

The spatiotemporal parameters of gait, kinematics of movement in the joints of the lower limb and pelvis, and reaction forces on the mat during the standing phase of one step cycle were monitored.

*Statistical analysis*

Comparison of parameters before and after stimulation using paired *t*-test was performed using IBM SPSS Statistics 24.0 software. The level of statistical significance was set at $p<0.05$. The results were then assessed for substantive significance of the differences in means using the Effect of Size (ES). Cohen's d, which determines substantive significance, was expressed according to Hopkins (2002): 0-0.2 = negligible effect; 0.2-0.6 = small effect; 0.6-1.2 = moderate effect; 1.2-2.0 = large effect; 2.0-4.0 = very large effect; and 4.0 and above = excellent.

## Results

Table 1 shows the differences in spatio-temporal parameters before and after stimulation. Walking speed increased after stimulation, from a mean (± standard deviation) of 0.88±0.17 m/s before stimulation to 0.98±0.16 m/s ($P<0.05$) afterward. The stride length also was greater after stimulation, increasing from 1.07±0.14 m before to 1.13±0.11 m ($P<0.05$) after. The other parameters remained unchanged.

Table 2 shows the results for ROM. In the hip joint, ROM increased in the sagittal plane bilaterally after stimulation ($P<0.05$). The range of pelvic rotation in the transverse plane also increased after stimulation ($P<0.05$). ROM in the pelvic sagittal and frontal planes did not change significantly after stimulation.

## Discussion

*Walking speed and stride length*

Slower gait reflects the presence of pain and may also indicate an attempt to reduce pain by restricting spinal movements [16]. Slow walking is associated with instability and increases the risk of falling [18]. Globally in 2018, falls were the second leading cause of death from accidental or unintentional injuries, according to the World Health Organization. Falls often lead to adverse health outcomes, resulting in injuries, limitations on ADL and socialization, transition to long-term care, and impaired QoL. In older adults, falls can cause fractures of the femoral neck, increasing morbidity and mortality. According to an experimental study, walking speed significantly influences the direction of fall and the point of impact [19]. Higher velocities lead to forward falls, and lower velocities tend to lead to sideways and lateral falls, and thus to hip compromise. Falling sideways increases the risk of hip by three- to five-fold compared to falling in other directions, and a direct impact to the hip or its surroundings increases the risk of fracture by 21- to 49-fold [19]. Other studies consider stride variability as a stronger indicator of falls [20]. Patients with LBP have a higher degree of step-to-step variability, leading to increased fluctuations in dynamic pelvic and trunk oscillations and less optimal gait patterns [21]. In the preferred gait pattern, there is a relationship between

**Table 1.** Spatiotemporal differences during walking, before and after stimulation.

| *Variable* | **Before stimulation** | **After stimulation** | **ES$^{Sig}$** |
|---|---|---|---|
| *Speed (m/s)* | 0.88 ± 0.17 | 0.98 ± 0.16 | 0.61* |
| *Stride length (m)* | 1.07 ± 0.14 | 1.13 ± 0.11 | 0.48* |
| *Stride width (m)* | 0.14 ± 0.04 | 0.13 ± 0.04 | 0.25 |
| *Cycle time (s)* | 1.19 ± 0.1 | 1.17 ± 0.11 | 0.19 |
| *Left step length (m)* | 0.53 ± 0.07 | 0.56 ± 0.06 | 0.46 |
| *Right step length (m)* | 0.55 ± 0.08 | 0.58 ± 0.07 | 0.4 |
| *Left step time (s)* | 0.59 ± 0.05 | 0.59 ± 0.06 | 0 |
| *Right step time (s)* | 0.60 ± 0.05 | 0.59 ± 0.06 | 0.18 |

* $P<0.05$.

**Table 2.** Hip and pelvis range of motion differences during walking, before and after stimulation.

| *Variable* | **Without stimulation** | **With stimulation** | **ES$^{Sig}$** |
|---|---|---|---|
| *Hip left sagittal ROM (°)* | 30.15 ± 6.62 | 31.38 ± 6.01 | 0.19* |
| *Hip right sagittal ROM (°)* | 30.34 ± 7.71 | 32.28 ± 6.59 | 0.27* |
| *Pelvis sagittal ROM (°)* | 10.30 ± 4.31 | 9.60 ± 3.50 | 0.18 |
| *Pelvis frontal ROM (°)* | 9.20 ± 3.45 | 9.07 ± 3.43 | 0.04 |
| *Pelvis transversal ROM (°)* | 129.14 ± 38.28 | 163.52 ± 14.60 | 1.19* |

ROM = range of motion, * $P<0.05$.

walking speed and stride length. Stability increases with gait speed and depends on gait speed [22], and speed probably also affects coordination. Lower speeds are more demanding on the motor control system and as a result, the neuromuscular system must exert more effort to maintain dynamic balance during a slower gait. A slower walking speeds, a shorter stride, a wider base and a longer part of the cycle in double leg support make the gait more unstable, predisposing walkers to increased risk of loss of balance and falls [22]. Research confirms that the slow gait that patients with LBP favor is associated with an inability to adapt trunk-pelvic coordination and erector spinae muscles to changes in speed [23]. Other factors influencing gait speed include weight, height, gender, proprioception, non-dominant hip abduction strength, and the cognitive, visual and vestibular systems. Although various reference limits for optimal walking speed can be found in the literature, there are no cut-off points for speeds that lead to a division between falling and non-falling. Moreover, walking speed alone cannot be assessed without context with other variables.

*ROM of the lumbar spine and pelvis and hip joints*

People with LBP have reduced ROM in the lumbar spine in all directions [24]. Increased pelvic rotation in the transverse plane contributes to increased stride length with increasing walking speed in healthy individuals [25]. In patients with LBP, trunk and pelvic movements in the transverse plane are impaired, with significantly less movement between the trunk and pelvis compared to healthy controls [26]. There is also increased variability in pelvic rotations independent of speed [23], probably because of increased spinal stiffness. During walking, these patients have increased activity of the erector spinae muscles during the swing phase, although normally these muscles are not active during this phase [16]. This may be a protective strategy to avoid painful movement and/or to compensate for bony changes in the spine to prevent further injury or pain irritation. According to a review study by Avman *et al.*, patients with LBP during walking have decreased hip ROM in both the transverse and frontal planes and have less hip internal rotation compared to healthy controls [27]. In the gait cycle, a lateral weight shift occurs before step initiation. Sufficient weight shifting can maintain a stable posture on one leg during the support phase, allowing the

other leg to be released for stepping. Studies have shown that at this point, there is a significant reduction in ROM of the ipsilateral hip joint in the transverse plane and the ipsilateral knee joint in the coronal plane in patients with LBP [28]. These patients also have reduced proprioception [28]. Somatosensory perception from the lower limbs is an essential resource in maintaining postural stability. Restriction of hip ROM, reduced proprioception perception, and promotion of spinal stiffness may lead to a conservative postural control strategy aimed at reducing the potential risk of postural instability in gait [29]. It is likely that any disturbed movement of the hip joint in any plane could affect the rest of the kinematic chain and may contribute to altered coordination of the lumbo-pelvic-hip complex during functional movement, resulting in increased and/or abnormal mechanical forces acting on the lumbar spine [27]. As individuals with LBP are less likely to move, ROM of the hip joint is important for their adequate function.

Patients with LBP have impaired gait because of the desire to reduce pain by restricting spinal movement and because of the fear of pain. In our patients, some kinematic values improved after stimulation. There was a significant increase in velocity, an increase in stride lengthening, and a significant increase in pelvic ROM in the transverse plane and hip ROM in the sagittal plane. Increased speed and stride lengthening may lead to increased stability and improved coordination, and increasing pelvic ROM in the transverse plane also contributes. Increasing hip joint ROM is protective for proper gait, as well, and can lead to a reduced risk of falls and the associated negative consequences for the patient and society. Previous studies have already confirmed that patients with LBP have persistent altered patterns in gait kinetics and kinematics. We speculate that patients with SCS implantation may benefit in the long term by changing kinematic patterns towards more optimal gait stereotypes, but longitudinal studies are lacking. Previous studies focusing on gait measurement in patients with back pain have shown conflicting results. The limitations of these studies are the small sample size of patients and different measurement methodologies. Rijken's study of the effect of SCS on gait does not describe an improvement in gait speed or stride length. However, the measurements were performed on a treadmill, where the results may be somewhat distorted due to the adaptation of gait to the speed generated by the treadmill, which may lead to differences compared to natural gait. Wilson, on the other hand, demonstrated altered patterns of range of motion in the hip joint and small differences in lateral movement of the upper torso in those patients who did not benefit from SCS implantation. This is consistent with the claim that patients with pain show greater gait instability and try to avoid pain with compensatory strategies aimed at stabilizing the lumbar spine. The increase in ROM in the hip joint was also confirmed by our measurements. However, both previous studies performed measurements within fourteen days of implantation, which may not have provided sufficient time for adaptation to the stimulation or the manifestation of effects. The average implantation in our patients was four years. In agreement with our study, Yozu described a significant increase in walking frequency and speed, as well as an increase in hip ROM, after SCS. The different results of the aforementioned studies in gait kinematics may stem from the different levels of motor performance of the participants studied. The effect of SCS on walking may be more evident in participants with more severe gait impairment than in those with a lesser degree of gait impairment. Medication use is one of the major risk factors for falls. Antidepressants and benzodiazepines decrease walking speed, stride length, level of alertness, reaction time, and attention [30]. Patients with pain most commonly take opioids and other drugs with sedative effect. Reduction of medication after SCS introduction has been demonstrated more than once [31] and is another factor that contributes to improved locomotion in patients with LBP. In line with the analgesic effect of SCS, this study contributes to the understanding of the management of motor changes in patients with back pain. These findings also may benefit patients in the field of rehabilitation. Movement therapy, uniquely designed for each patient, may improve the accuracy of spinal movements and induce specific musculoskeletal changes, such as altering movement patterns and promoting pain-free movement patterns [32]. In patients with LBP without neuromodulation, rehabilitation leads to decreased pain, increased pelvic strength, favorable changes in trunk and pelvic ROM, and improved stability [33].

The limitation of the study is the lack of a control group and the differentiation of the effect of SCS on walking. The small number of included patients is also a limitation of our work. The absence of a sham-stimulated control group limits the ability to distinguish true neuromodulatory effects from placebo or expected effects. Seven participants who did not use a stimulator on the day of measurement and had increased pain

perception used additional analgesics. Thus, their pain perception was reduced even without the use of stimulation, but there was still an improvement in some of the kinematic parameters observed after stimulation. We do not believe that supplementary support, particularly including nonsteroidal anti-inflammatory drugs and tramadol, which patients use chronically, thereby somehow affecting the kinematics. All participants in the study had a stimulator with tonic stimulation triggered by a self-controller and were aware of the stimulation through paresthesia. A theoretical solution in the future may be to measure patients before and after SCS implantation. There are now methods of SCS that do not induce paresthesia, such as burst, which could make measurements more accurate. Other limitation of this work is the small sample of patients and the gender imbalance (2 males and 13 females) and the wide age range. Some authors have reported no significant differences in gait parameters in LBP patients compared with healthy controls of the same sex and similar height and age, but others describe shorter stride length in women, slower walking speed and shorter stride in the elderly, and faster walking speed and longer stride those of greater height. However, there are differences between men and women in walking in a healthy population in the ROM of the lower limb joints [34]. More research is needed to describe and elucidate the effects of neuromodulation on locomotion in various stratified patient groups.

If a direct effect of SCS on motor function is confirmed, Parkinson's patients could also benefit. In the advanced stage of the disease, people with Parkinson's suffer from axial symptoms, poor posture, gait disturbances (lower speed and shorter steps), freezing of gait (FoG) and postural instability, all of which reduce their ADL and QoL [35]. In addition, higher instability leads to a higher frequency of falls and thus increased morbidity and mortality. Patients with Parkinson's disease also suffer from pain significantly more often. Previous research has confirmed the involvement of the dopaminergic system in pain processing. These patients appear to have a reduced pain threshold because of abnormalities of the dopaminergic system in the basal ganglia, and they most commonly experience back and lower extremity pain [36]. Antiparkinsonian medications, especially L-dopa and deep brain stimulation, improve motor fluctuations and pain, but the efficacy of this therapy decreases with disease progression. Nevertheless, improvements in QoL can be observed in patients with deep brain stimulation even 15 years after its application [37], although intolerable side effects occur with disease progression and increasing doses. Agari and Date conducted a study on 15 patients with Parkinson's disease who were introduced to SCS for low back and leg pain. In addition to pain relief, these patients experienced improvements in postural stability, gait, posture, and body rigidity, as well as in ADL and QoL [38]. Others have reported similar results and the possible therapeutic potential of SCS [39]. Moreover, newer stimulation regimens do not induce paresthesia and, according to a recently published study, may also improve emotional experience [40]. Some studies have not shown improved locomotion [41], however, although authors of recent reviews have suggested that SCS may provide significant motor and analgesic benefits to patients with Parkinson's disease [42]. The exact efficacy of SCS on motor function in Parkinson's disease patients remains uncertain. Previous research should be taken with caution for several reasons, including significant heterogeneity and small numbers of included patients. The improvement in some gait parameters induced by SCS, such as stride length, speed or FoG, suggests that SCS probably acts on suprasegmental circuits by stimulating ascending fibers [43]. Neuromodulation appears to increase neuronal firing in the primary motor cortex and reduce pathological corticostriatal synchronous low-frequency oscillatory activity, which may restore physiological aspects of the neuronal circuits involved in gait initiation [44], as confirmed in animal models of Parkinson's disease [45].

## Conclusions

Patients with SCS implantation showed significant post-stimulation increases in gait speed, stride lengthening, ROM of the pelvis in the transverse plane, and ROM of both hip joints in the sagittal plane.

## Conflict of Interest

There is no conflict of interest.

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