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Influence of sensory integration training on sensory motor functions in patients with thalamic syndrome

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Abstract

Introduction. Thalamic stroke causes impairment in sensory motor functions; these changes in perception lead to pain and deteriorate postural stability. The goal of this study was to investigate the influence of sensory integration training on pain and posture stability in patients with thalamic syndrome.
Methods. Overall, 30 patients of both sexes, aged 45–65 years, diagnosed with a thalamic stroke were randomly allocated into 2 groups. The control group received selected sensory training of the conventional physical therapy program and the study group received sensory integration training with the Biodex Balance System beside the conventional physical therapy program.
Results. The intra-group comparisons with MANOVA revealed statistically insignificant differences in pain, as well as overall, anteroposterior (forward and backward), and mediolateral (right and left) limits of stability after the treatment in the control group (p-value of 0.180, 0.301, 0.792, 0.247, 0.381, and 0.847, respectively). In the study group, there was a statistically significant decrease in pain and improvement in overall, anteroposterior (forward and backward), and mediolateral (right and left) limits of stability after the treatment (p-value of 0.01 for pain and 0.000 for each of the 5 limit of stability variables, respectively). The inter-group comparisons showed statistically significant differences in favour of the study group (p = 0.000).
Conclusions. Adding the sensory integration training program was effective to decrease pain and improve posture stability in patients with thalamic syndrome.
Key words: sensory integration training, pain, posture stability, thalamic syndrome

Introduction

The somatosensory deficit is the most frequent disorder observed in cerebral injuries. Somatic sensation debilitates in 37% of patients with injured right hemisphere and 25% of those with injured left hemisphere. The most serious problems of somatosensory deficit include impairment in tactile recognition and manipulation of objects, danger of burns or other injuries to the insensate limb, impairment of motor control of the affected limb, deficits in controlling the level of force of the hand during grasping, and poor posture stability both in an upright position and during ambulation. The somatosensory decay causes the patient's functional dependence in activities of daily living [1].

Patients with thalamic syndrome reliably express disappointment with sensory recovery. Up to 89% of individuals with hemiparesis exhibit sensory defects when examined for touch, temperature, weight, texture, and shape discrimination. Although sensory dysfunction predicts the extent of motor recovery, thalamic syndrome rehabilitation often focuses on motor impairment and ignores sensory dysfunction [2].

Numerous studies employing functional magnetic resonance imaging have documented that neural plasticity is associated with motor recovery; however, few studies have suggested that neural reorganization is related to the recovery of sensory function. The sensory component of the superior thalamic radiations incorporates afferent connections to the somatosensory cortex and the functional aspect of the corticospinal tract [2–4].

The recovery of thalamic syndrome was related to the improvement in sensory function with an enhancement of somatosensory cortex of the ipsilesional hemisphere, while balance training activated changes in the ventral premotor and parietal cortex of the contralateral hemisphere. Unfortunately, the sensory function was not measured, and the limited knowledge of the neural reorganization that accompanies sensory recovery after thalamic stroke points to the need for research in this area [4].

There are countless clinical and neuroimaging reports considering motor manifestations while the quantity of studies following somatosensory symptoms after thalamic stroke and their recovery is fairly little [4–6].

Thalamic pain relates to the site of the lesion and is distinct from other painful conditions (such as shoulder pain). It typically involves the spinothalamic and thalamocortical pathways from hemispheric lesions; as a result, patients complain about sharping, stabbing, or burning pain and encounter hyperesthesia and allodynia [6].

Postural sway in individuals with somatosensory loss is significant owing to disrupting postural control, which increases the risk of falling. Patients lose the centre of pressure and the centre of mass information for controlling the posture instance, with impaired ability to remain standing on feet with greater posture sway [7].

Effective balance training can reduce sensory abnormalities and improve context-specific instabilities of postural control in patients with thalamic stroke. For improving standing postural control, there are subclinical constraints with

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the guidance effect of external visual feedback concerning the posture stability training [6].

Coordination between different body parts or muscle groups is necessary to control the multi-joint movement in a fluent manner. This synergy might be deteriorated by factors such as pain and posture instability in different neurological problems [8].

Sensory integration training emphasizes postural control and progressive challenges to the sensorimotor system to restore normal motor programs that stimulate Aα nerve fibres, responsible for proprioception, and inhibit C fibres, responsible for pain [9].

So, was there an influence of adding sensory integration training on pain and posture stability in patients with thalamic syndrome? The purpose of this study was to investigate the impact of sensory integration training on pain and posture stability in patients with thalamic syndrome. It was hypothesized that there would be no influence of adding sensory integration training on pain and posture stability in patients with thalamic syndrome.

Subjects and methods

Study design

A randomized control trial of pre- and post-experimental design was conducted in the outpatient clinic of the Faculty of Physical Therapy, Modern University for Technology and Information in the period from April 2019 to June 2019. Anonymity and confidentiality of all procedures were assured in compliance with relevant laws and institutional guidelines.

Participants

The study involved 30 referred patients with thalamic syndrome after 6–12 months of injury, aged 45–65 years, of both sexes (21 males and 9 females). The inclusion criteria were as follows: lateral thalamic infarction within the right hemisphere due to occlusion of the proximal posterior cerebral artery, somatosensory abnormality due to thalamic syndrome confirmed with a brain computed tomography scan at the beginning of injury and 2 weeks later, hemi-sensory loss at the non-dominant side confirmed by right/left hand Hit-the-Dot test (Appendix I), somatosensory deficit causing significant debilitation in activities of daily living confirmed by tests for superficial sensation (moderate to severe level of pain according to the Patient-Reported Outcomes Measurement Information System [PROMIS]), motor deficit of Brunnstrom stage IV (mild impairment of posture stability), and ability to walk with or without an assistive device. Clinically, all recruited patients suffered from pain and posture instability and all had controlled blood glucose level, haemoglobin A1c value of 6.5–7% or lower, and blood pressure of 120–140/80–90 mm Hg.

The exclusion criteria involved a previous cerebrovascular attack, sensory impairment because of peripheral vascular disease or neuropathy, visual disturbances, balance disturbances rather than thalamic syndrome (e.g. ear problems, labyrinthitis, diabetic neuropathy), cardiac problems, cognition problems (Mini-Mental State Examination, cut-off score of 24), psychiatric disorders, gross motor deficits or limb apraxia, history of pedal ulcer, amputation, advanced arthritis, osteoporosis, malunion fractures, obesity (body mass index over 30 kg/m^2), and nerve root compression (radiculopathy).

Instruments

All patients underwent a pre-treatment and post-treatment assessment to measure pain by using the PROMIS scale [10]. PROMIS instruments demonstrated validity and adequate test-retest reliability, which means that they can be used for clinical and research outcomes [11]. Limits of stability (LOS) were measured with the Biodex Balance System (Biodex Medical Systems Inc., Shirley, NY, USA) (Figure 1). The Bio-

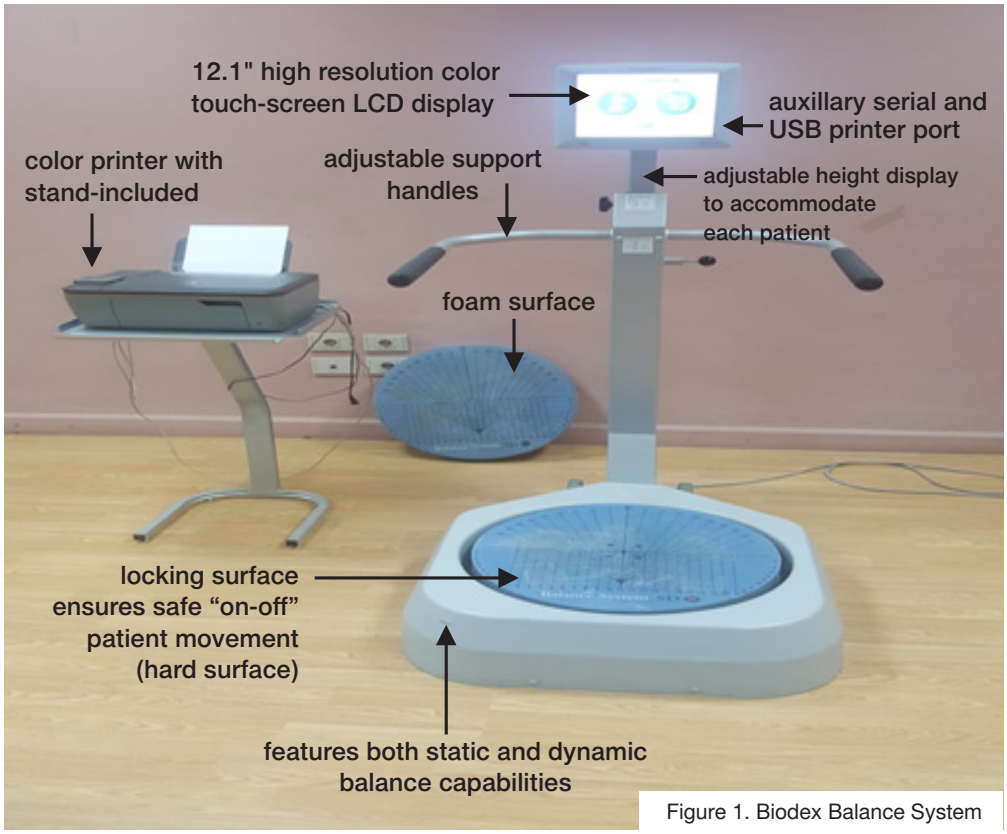


Figure 1. Biodex Balance System

dex Balance System has been shown to be a reliable and valid postural stability assessment and treatment tool [12]. The device consists of a circular foot platform of 21.5 cm diameter that permits 20° tilting in all directions, support rails (height-adjustable), a display screen (height-adjustable), and a printer. The instrument offers 12 dynamic levels of measurement plus locked static measurements [13].

Procedures

Testing procedures

1. PROMIS: The patients were asked to rate their pain intensity and mark it on a scale from 1 (representing no pain) to 5 (representing very severe pain) (Figure 2).

2. Biodex Balance System: Calibration was performed before the beginning of the testing procedures (Appendix II). Each patient was asked to stand barefoot on the platform of the Biodex Balance System. LOS testing was selected, the participant's name, age, and height were entered, and the 8th level of stability was adjusted for 10 seconds (test period). The foot angle for each patient was detected by using the alphanumeric grid on the foot platform (25–30°). The subjects were instructed to hold the platform at a stable level to measure their ability to shift the centre of gravity overall, as well as in the anteroposterior (forward and backward) and mediolateral (right and left) directions. Three trials were obtained for each measurement and the mean was obtained by a LOS test. A report was generated and printed for each trial, including overall, anteroposterior (forward and backward), and mediolateral (right and left) LOS. Decreasing LOS number indicates considerable motion and high amount of sway, meaning lower posture stability.

Intervention procedures

1. Selected sensory training of the conventional physical therapy program: The control group (11 males and 4 females) received object recognition, tactile discrimination, joint position sense, weight discrimination, and active range of motion exercises for 1 hour. The treatment duration was 8 weeks (24 sessions, 3 times per week) [15] (Table 1).

2. Sensory integration training: The study group (10 males and 5 females) received training on the Biodex Balance System for 10 minutes, repeated 3 times (with a total duration of the session of about 30 minutes), beside the conventional physical therapy program (1 hour; the total duration of treatment equalled 90 minutes). Postural stability training simulates specific movement patterns or strategies by placing markers on specific locations on the screen grid. The platform stability was set to level 8 (more stable). The foot angle for each patient was detected by using the alphanumeric grid on the foot platform (15–20°). All subjects were standing on both feet and grasped the handrails. Once the platform moved, the participant was instructed to achieve a centred position. The hand support was released gradually, and the patient continued the training without support. The individuals attempted to touch targets 9 times using an on-screen cursor manoeuvred by their legs on the device platform. The treatment duration was 8 weeks (24 sessions, 3 times per week).

Statistical analysis

All statistical calculations were carried out with the IBM SPSS version 22 computer program (IBM Corporation, USA). The sample size calculation was performed by using the G*Power software (version 3.0.10). Pain was chosen as the primary outcome measure. A generated sample size of at least 12 patients per group was required. Allowing for a 20% dropout rate, it was necessary to reach a total sample of

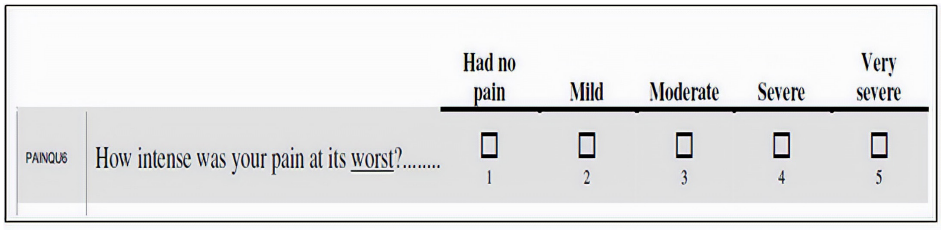


Figure 2. Patient-Reported Outcomes Measurement Information System (adapted from [14])

Table 1. The selected sensory training

Selected sensory training	Description
Object recognition	The blindfolded patient was requested to perform 3 tasks: manipulate a target object and discriminate it visually among 3 objects; manipulate a group of small objects (e.g., rice, bolts, stones) and discriminate them visually among the 3 groups; and manipulate 2 objects simultaneously with the affected and unaffected hand and then report whether the 2 objects were the same or different
Tactile discrimination	The blindfolded patient was asked to detect different textures of a touched point at any area of the affected limb by using 3 tactile discrimination tasks: sandpaper surfaces of different grains, surfaces made of different materials (e.g., rubber, cloth, paper), and grating orientation
Joint position sense	The patient put their affected arm inside a wooden box (20 × 40 × 40 cm) that was open at opposite ends. The proximal segments of the joint (i.e., the forearm in the case of the wrist) were stabilized inside the box. The therapist moved the patient's wrist or metacarpophalangeal joints in different angular positions. The patient was to actively reproduce the same position as shown by the therapist
Weight discrimination	The blindfolded patient weighed an object by the affected hand. Then, they weighed 3 objects with the unaffected hand and chose which of them corresponded in weight to the previous object
Active range of motion	Exercises for shoulder, elbow, radioulnar, wrist, fingers, hip, knee, ankle, foot, and toe movements

30 individuals. Levene's test showed that all data were homogenous. The Shapiro-Wilk test revealed that data concerning pain were not normally distributed, so nonparametric tests were used (Wilcoxon signed-rank and Mann-Whitney tests), while data concerning LOS were normally distributed, so a parametric test was applied (MANOVA). Spearman correlation was determined between pain and LOS in the study group. The value of $p < 0.05$ was considered statistically significant.

Ethical approval

The research related to human use has complied with all the relevant national regulations and institutional policies, has followed the tenets of the Declaration of Helsinki, and has been approved by the Ethics Committee of the Faculty of Physical Therapy, Cairo University, Egypt (approval No: P.T.REC/012/002307) and registered in the Pan African Clinical Trials Registry (No.: 201908825773318).

Informed consent

Informed consent has been obtained from all individuals included in this study.

Results

All patients were randomly assigned to 2 groups by using closed envelopes (control group: $n = 15$, study group: $n = 15$) (Figure 3).

General demographic data

The age and body mass index of patients in the control and study groups did not differ significantly (p-value of 0.793 and 0.434, respectively). The numbers of males to females in the control and study groups were 11:4 and 10:5, respectively (Table 2, Figure 4).

Pain

The intra-group comparisons showed a statistically insignificant difference in the control group ($p = 0.180$) and a statistically significant decrease in the study group ($p = 0.01$).

Table 2. General demographic data

	Age (years)	Body mass index (kg/m^2)
Control group	61.8 ± 1.12	28.907 ± 0.754
Study group	62.27 ± 1.31	29.767 ± 0.779
t	0.270	0.789
p	0.793	0.434

Sex					
Number	Males	Females	Percentage (%)	Males	Females
	11	4		73.33	26.67
	10	5		66.67	33.33
χ^2 statistics	χ^2	0.16			
	p	0.69			

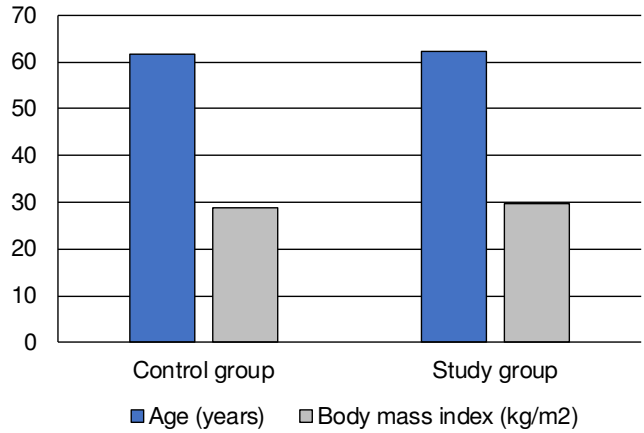


Figure 4. Patients' age and body mass index

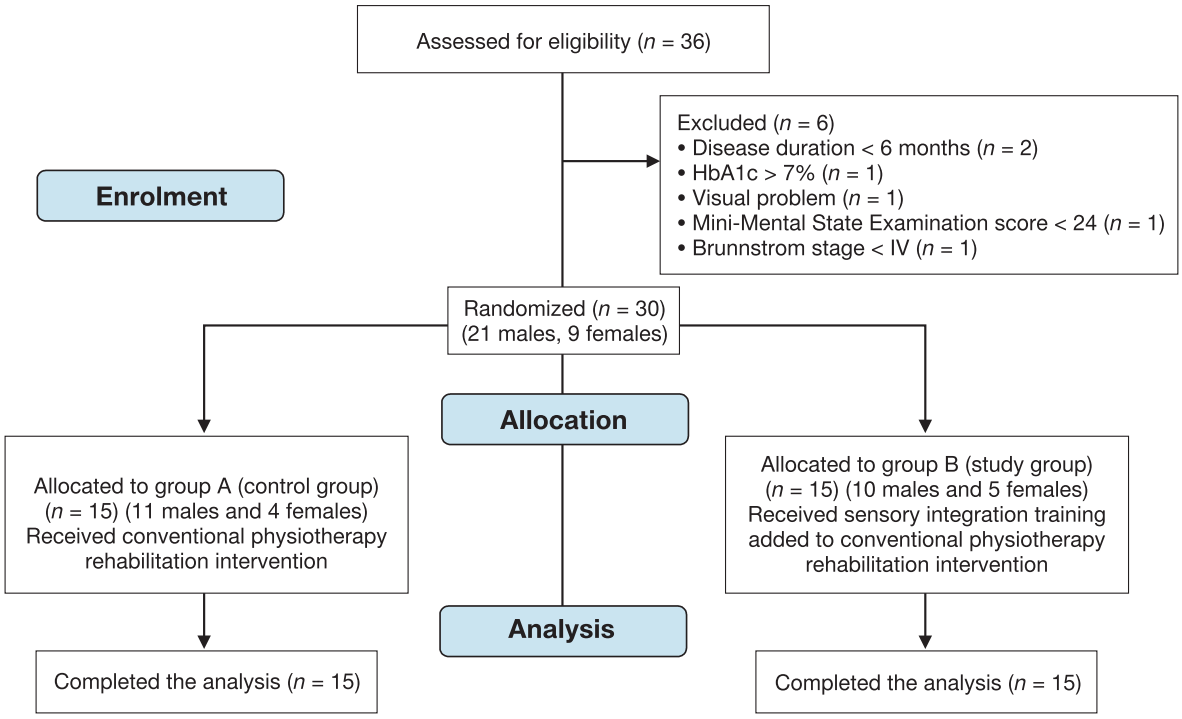
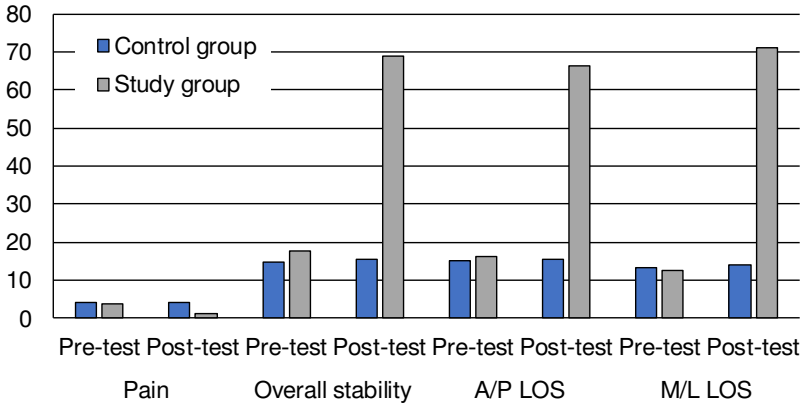


Figure 3. Flow chart for participants

Table 3. Within- and between-group comparison of pain values

	Before	After	Mean difference	CI	*Z*	*p*
Control group	4.200 ± 0.192	4.067 ± 0.182	0.09	0.54–3.4	1.342	0.180
Study group	3.867 ± 0.236	1.2000 ± 0.107	1.73	0.88–4.79	03.473	0.01*
Z	1.346	4.882				
p	0.233	0.000*				

Z – Wilcoxon signed-rank (within-group) and Mann-Whitney (between-group) tests
* significant: $p < 0.05$



A/P – anteroposterior
M/L – mediolateral
LOS – limits of stability

Figure 5. Comparison of results between the control and the study groups

Table 4A. The overall limit of stability

	Overall stability				
	Before	After	Mean difference	CI	*p* (within-group)
Control group	14.87 ± 1.41	15.60 ± 1.59	10.4	1.04–3.5	0.301
Study group	17.67 ± 1.15	68.93 ± 1.53	26	24.86–27.32	0.000*
p (between-group)	0.644	0.000*			

* significant: $p < 0.05$

Table 4B. The anteroposterior (forward and backward) limit of stability

	Anteroposterior stability									
	Forward stability					Backward stability				
	Before	After	Mean difference	CI	*p* (within-group)	Before	After	Mean difference	CI	*p* (within-group)
Control group	15.00 ± 1.76	15.53 ± 1.93	0.33	1.69–2.35	0.792	12.07 ± 1.61	12.80 ± 1.66	0.4	1.04–3.5	0.247
Study group	16.40 ± 1.74	66.53 ± 2.18	29.4	27.82–30.98	0.000*	12.47 ± 1.28	62.73 ± 1.68	26.57	24.55–28.59	0.000*
p (between-group)	0.696	0.000*				0.465	0.000*			

* significant: $p < 0.05$

Table 4C. The mediolateral (right and left) limit of stability

	Mediolateral stability									
	Right stability					Left stability				
	Before	After	Mean difference	CI	*p* (within-group)	Before	After	Mean difference	CI	*p* (within-group)
Control group	13.20 ± 1.11	14.1 ± 1.32	0.45	1.04–3.5	0.381	14.6 ± 1.39	15.6 ± 1.57	0.5	1.6–5.27	0.847
Study group	12.53 ± 0.888	71.1 ± 1.91	28.77	26.77–30.76	0.000*	15 ± 1.41	76.1 ± 1.72	30	28.56–31.44	0.000*
p (between-group)	0.642	0.000*				0.78	0.00*			

* significant: $p < 0.05$

The inter-group comparisons revealed a statistically insignificant difference before the intervention and a highly significant decrease after the intervention in favour of the study group (*p*-value of 0.23 and 0.000, respectively) (Table 3, Figure 5).

Limits of stability

The intra-group comparisons showed statistically insignificant differences in overall, anteroposterior (forward and backward), and mediolateral (right and left) LOS (*p*-value of 0.301, 0.792, 0.247, 0.381, and 0.847, respectively) in the control group. There was a highly statistically significant increase of LOS ($p = 0.000$) in the study group.

The inter-group comparisons revealed statistically insignificant differences before the treatment (*p*-value of 0.644, 0.696, 0.465, 0.642, and 0.784, respectively) and a highly statistically significant increase after the treatment in favour of the study group ($p = 0.000$) (Table 4A–C, Figure 5).

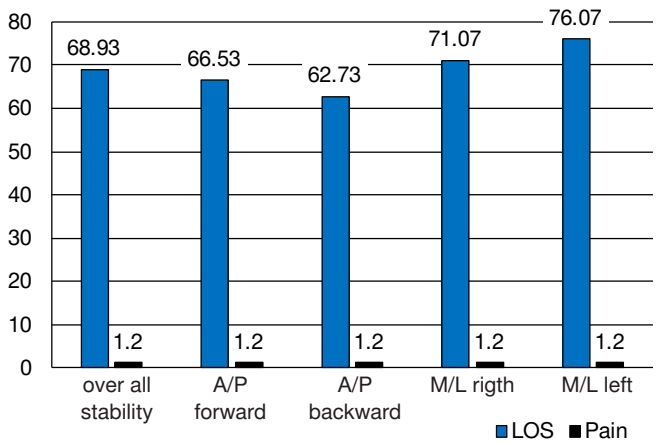
Correlation

There was a strong inverse correlation between pain and LOS after the intervention ($p < 0.05$) (Table 5, Figure 6).

Table 5. Correlation between pain and limit of stability after the treatment

Limit of stability		Correlation coefficient	Pain
Overall stability		*R*	62.8%
		p	0.000*
Anteroposterior stability	Forward stability	*R*	59%
		p	0.001*
	Backward stability	*R*	61.3%
		p	0.000*
Mediolateral stability	Right stability	*R*	51.9%
		p	0.004*
	Left stability	*R*	52.8%
		p	0.043*

* significant: $p < 0.05$



LOS – limits of stability, A/P – anteroposterior, M/L – mediolateral

Figure 6. Correlation between pain and limit of stability after the intervention

Discussion

After a thalamic stroke, conservative treatment methods such as exercise therapy and pharmacotherapy are used. Those techniques increase mobilization and reduce pain, as well as improve functional status and mental state [16]. Thalamic syndrome recovery interventions keep concentrating on motor dysfunction, disregarding somatosensory impairment [17]. Somatosensory deficit regularly causes debilitation in movement control owing to its influence on perception [18]. It was enlisted that changes of sensory function are common after thalamic stroke, with a variable detailed pervasiveness extending from 11% to 85%, and usually these modifications of perception result in pain [19]. This was supported by the measured baseline magnitudes of pain in both the control and the study group (3.867 ± 0.236 and 4.2 ± 0.192, respectively).

The central post-stroke pain (CPSP) disorder is less responsive to physiotherapy and requires a pharmacological method using amitriptyline, gabapentin, or pregabalin [20]. This was observed in post-intervention measures for pain in the control group ($p = 0.18$), while in the study group, there was a significant decrease ($p = 0.01$); therefore, the first hypothesis was rejected because the sensory integration training decreased CPSP.

The pathophysiological components underlying the progression of CPSP were due to a reduction in spontaneous discharge of injured neurons in the thalamus or the cortex and declined proprioceptive input by visual data [21]. The pathophysiology of CPSP is vague and involves various mechanisms in the thalamus, including the deafferentation of afferent pathways (sensory loss), hyper-excitation of segment (hyperesthesia), and affection of the spinothalamic tract (diminished superficial sensation of pain and temperature) [22].

These mechanisms are exacerbated by excitotoxic and provocative changes brought about by the thalamic syndrome, leading to pain regardless of whether it is activated by a noxious stimulus. This was affirmed by the pre-intervention measures for both the control and the study group, which differed insignificantly [23] ($p = 0.233$), while the difference between the post-treatment measures was highly significant ($p = 0.000$). This confirmed the superiority of sensory integration training in regaining the altered balance between inhibition and facilitation of sensory-motor brain areas, which has been proposed as a possible underlying mechanism of CPSP [24].

In particular, injury of the lateral thalamus has been identified as one of the most common causes of CPSP. A lesion of the lateral thalamus and lenticular nucleus could induce an alteration in the functional connectivity between basal ganglia and primary/secondary somatosensory cortices which are involved in pain intensity and perception, sensory discrimination, and nociceptive information processing [25]. Sensory integration training also encouraged the brain to continuously match between visual and kinaesthetic inputs during movements through linking what is seen with what is felt [26]. The combination of visual feedback and proprioceptive awareness would lead the central nervous system to reach a sensory harmony, which would contribute to pain reduction [27, 28].

A review of thalamic syndrome identified a need for well-designed studies for sensory rehabilitation [29]. Although sensory training had commonly focused only on sensation, without attention to motor recovery [30], the clearest consequences of somatosensory deficit are an impairment of motor control

in the affected limbs [31] and poor posture stability [32]. That was illustrated in the baseline overall, anteroposterior (forward and backward), and mediolateral (right and left) stability in both the control and the study group (17.67 ± 1.15, 16.40 ± 1.74, 12.47 ± 1.28, 12.533 ± 0.888, 15 ± 1.41; and 14.87 ± 141, 15 ± 1.76, 12.07 ± 1.61, 13.2 ± 1.11, 14.67 ± 1.39, respectively).

Several studies have shown that a somatosensory deficit exerts a negative effect on the functional outcome of patients with hemiplegia and prolongs their rehabilitation treatment. Bobath and Brunnstrom centred on motor re-learning, not sensory re-education, by techniques developed to deal with motor loss as the cardinal problem during stroke recovery. Until now, despite the clinical importance of somatosensory deficiency, little consideration has been given to the recovery of somatosensory function [33–36].

Both human and animal examinations have shown that somatosensory structures in the cerebrum exhibit a high level of plasticity and a rehabilitation program aimed to alleviate somatosensory deficits and related motor disabilities can prompt critical practical enhancements [37, 38]. This was confirmed by the results for pain in the study group and the statistically significant inter-group difference (p = 0.000) with a high effect size of 94.7%.

Earlier rehabilitation of a somatosensory deficit in patients with thalamic injury recovered the proprioceptive and quantitative feedback abilities [39]. This was confirmed by the statistically significant differences between the groups (p = 0.000) with a high effect size of 88.9% in favour of the study group. The identified recovery of perception was associated with increased activation in the primary somatosensory cortex of the ipsilesional hemisphere [40].

In turn, the countless tests for deep sensation revealed that when patients with thalamic syndrome were treated to improve the proprioceptive sensation, the progress was not confined to proprioception but related to the motor function as well [41, 42]. So, it was not surprising that all patients in the study group experienced a highly significant improvement in LOS (p = 0.000). A combination of many somatosensory sub-modalities and motor outputs was essential in the rehabilitation of patients with thalamic syndrome, which was reflected in a strong direct correlation between LOS and pain after the treatment (p = 0.000).

Diffusion tensor tractography (DTT), which is a method of modelling white matter connections in the human brain in vivo, has suggested that a loss of connectivity occurs as a result of degeneration in axons at the site of the lesion and distally to it after thalamic stroke. In a consequence, DTT has been used primarily in cross-sectional studies to explore the relationship between infarct locations and sensorimotor pathways, as well as to quantify the damage of the corticospinal tract. Nevertheless, the sensory functions related to structural changes in thalamic stroke were unclear in DTT [43, 44].

Moreover, several studies confirmed a strong correlation between the structural integrity of the corticospinal tract and post-stroke motor function; yet, there is little direct evidence of white matter remodelling after stroke [45, 46]. Finally, the present study demonstrated that patients with thalamic syndrome accomplished a statistically significant reduction of pain and significant improvement in all directions of posture stability. It becomes clear that the integrity of the somatosensory system is essential to motor recovery after stroke owing to the tight connection between motor control and somatosensory function. These results point to the importance of devoting more attention to the rehabilitation of patients with thalamic syndrome because sensory integration training was an effective modality to reduce pain and improve posture stability. So, the hypothesis of no influence of adding sensory integration training on pain and posture stability in patients with thalamic syndrome was rejected.

Limitations

The authors are aware of the study limitations. This examination was constrained by diminished patients' capacity to complete the treatment procedures, as the individuals were all of a sudden feeling migraine, inconvenience concentrating, blurred vision, or fatigue (powerless and tired inclination). The psycho-physiological burden at the time of examination and training was assumed to be the same for patients all over the study. Also, the sample size may be considered as a limitation.

Conclusions

The implemented sensory integration training program was effective to decrease pain and improve posture stability in patients with thalamic syndrome.

Recommendations for future research

Further studies are needed to examine the effect of sensory integration training on the risk of falling in patients with thalamic syndrome by using the Biodex Balance System, as well as to evaluate nerve conduction and electromyographic outcomes before and after sensory integration training.

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Disclosure statement

No author has any financial interest or received any financial benefit from this research.

Conflict of interest

The authors state no conflict of interest.

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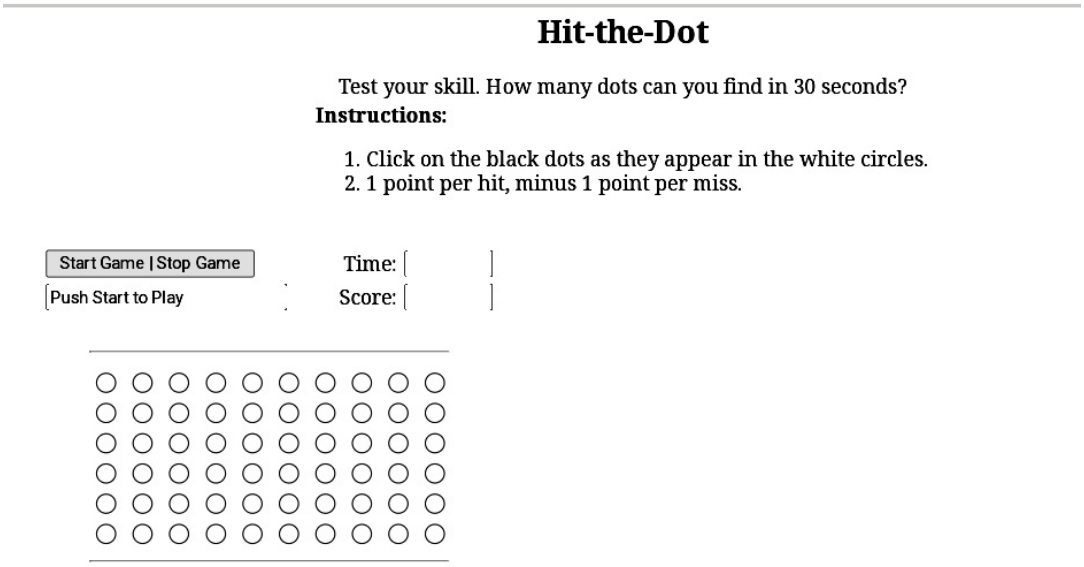
Appendix I

Hit the dot test



Appendix II

Biodex Balance System calibration

A. Platform actuator calibration

1. Tap the touch screen to turn on the display.
2. Touch <UTILITIES> on the screen. Note: The next step requires pressing the hidden keypads in the upper right and left corners of the display's touch screen.
3. Touch top-right corner, top-left corner, top-right corner successively, to access the <SYSTEM MAINTENANCE> menu on the display.
4. Touch <SYSTEM MAINTENANCE>.
5. Touch <DIAGNOSTICS>.
6. Touch <PLATFORM ACTUATOR> on the screen.
7. Press the <DOWN ARROW> on the screen until the motor stalls (bottoms out). Then look at the READING in the bottom right of screen and bring motor up 3 digits.
8. Touch <SET NEW MIN> on the screen to calibrate level 1.
9. Touch and hold the <UP> arrow on the screen until the motor stalls.
10. Touch <SET NEW LOCKED> on the screen to calibrate the locked position.
11. Touch <COMPENSATION> at the bottom of the screen and listen for the actuator. The 'arrow' indicator will move also.
12. Touch <BACK> on the screen 4 times to exit and return to the main screen.

B. Tilt sensor calibration procedure

1. Install the platform if it is not already installed.
2. Tap the touch screen to turn on the display.
3. Touch 'UTILITIES' on the screen. Note: The next step requires pressing the hidden keypads in the upper right and left corners of the display's touch screen.
4. Touch top-right corner, top-left corner, top-right corner successively, to access the 'SYSTEM MAINTENANCE' menu on the display.
5. Touch 'SYSTEM MAINTENANCE'.
6. Touch DIAGNOSTICS.
7. Touch 'TILT SENSOR' to release the platform.
8. Depress the platform to the maximum 9 o'clock position. The 'LEFT (min)' window will turn green signifying proper calibration.
9. Depress the platform to the maximum 3 o'clock position. The 'RIGHT (max)' window will turn green signifying proper calibration.
10. Depress the platform to the maximum 6 o'clock position. The 'BACKWARD (min)' window will turn green signifying proper calibration.
11. Depress the platform to the maximum 12 o'clock position. The 'FORWARD (max)' window will turn green signifying proper calibration.
12. YOU MUST press the BACK key 4 times to save and exit the calibration screen.