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Presence of Ideomotor Apraxia in Stroke Patients with Pusher Syndrome

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Abstract. [Purpose] Pusher syndrome, which is a disorder of postural balance that occurs in hemiparetic stroke patients, is characterized by a particular tendency to strongly push toward the hemiparetic side. The purpose of this study was to investigate whether stroke patients with pusher syndrome have ideomotor apraxic behavior. [Subjects] Fifteen stroke patients with pusher syndrome and 31 stroke patients without pusher syndrome were recruited. [Methods] All subjects were tested with two tests assessing ideomotor apraxia of movements of the upper and lower limbs. Each test included 12 items of movements, which required the subjects to reproduce movements by imitation after presentation. [Results] Patients with pusher syndrome had significantly lower ideomotor apraxia scores in all of the upper and lower limbs than patients without pusher syndrome. A significant difference was observed between the two groups in the existence of neglect. [Conclusion] We found that patients with pusher syndrome had more severe apraxic disorder in all of the upper and lower limbs than patients without pusher syndrome. Pusher syndrome may be attributable to disabilities in motor planning and execution, which are required to compensate for the partial damage to the postural control system.
Key words: Stroke patients, Ideomotor apraxia, Pusher syndrome

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INTRODUCTION

Postural impairment is one of the leading causes of disturbances in physical activities of daily life of hemiparetic stroke patients[1,2)]. Some patients show a peculiar postural dysfunction of actively pushing away toward the hemiparetic side and resisting any attempt at passive correction while in a sitting or standing posture. This pathological behavior of postural control is called pusher syndrome (PS), and it needs long-term hospitalization due to poor prognosis[3–6)]. The incidence of PS is estimated to be over 5% of the stroke population and 10% of stroke patients admitted for rehabilitation[3,7)]. Some clinicians and researchers have assumed that the symptom might be caused by spatial neglect, perceptual disorder of body orientation, anosognosia, aphasia, ideomotor apraxia, and so forth[8–12)]. Recently, it has been suggested that PS is caused by a distortion of two vertical references relative to earth-verticality (e.g, the visual vertical and postural vertical)[8,13)]. This implies that patients with PS have no motor plan for the adaptive postural strategy, compensating for mismatch between impaired postural verticality and intact visual verticality. With respect to motor planning disorder, PS might be related to ideomotor apraxic behavior. According to Davies' clinical observations[4)], crucial motor symptoms related to ideomotor apraxia were detected in patients with PS, such as clumsy movements of the unaffected hand and motor learning disability in activities of daily living. Although ideomotor apraxic behavior is clinically observed in patients with PS, there have been few studies that have investigated the relationship of PS and ideomotor apraxic symptoms. Therefore, we set out to investigate whether ideomotor apraxic symptoms are observed in stroke patients with PS in a comparison with stroke patients without PS.

SUBJECTS AND METHODS

Fifty patients with hemiplegia due to cerebral infarct or hemorrhage participated in the study. The patients were divided into 19 hemiparetic patients (13 men, 6 women, age: 70.00 ± 10.85) with PS and 31 hemiparetic patients (8 men, 23 women, age: 63.75 ± 10.87) without PS, according to the cutoff criterion for diagnosing PS. Inclusion criteria for

Table 1. Comparison of demographic and clinical characteristics between the pusher group and the non-pusher group.

	Pusher group	Non-pusher group
Demographic characteristics		
Sex (M/F)	19 (8/11)	31(19/12)
Age	70.0 ± 10.9	63.8 ± 10.9
Onset time	16.7 ± 13.3	15.0 ± 14.0
Lesion side (RBD/LBD)	14/5	16/15
Education	6.5 ± 5.2	7.3 ± 5.5
Clinical characteristics		
MMSE	23.6 ± 3.7	25.4 ± 4.7
Total MI	84.2 ± 23.3	94.8 ± 25.8
Somatosensory (I/A)	4/15	12/19
Kinesthesia (I/A)	3/16	13/18
Neglect (+/–)	10/9 *	0/31
Aphasia (+/–)	3/16	5/26

*: $p<0.05$, M: male, F: female, MMSE: Mini-mental status examination, MI: Motricity index, RBD: right brain damage, LBD: left brain damage, I: intact, A: absent, Neglect (+/–) or aphasia (+/–) indicates patients with symptoms present or not. All values presented as mean ± standard deviation.

patients included the following: first ever stoke, which was confirmed by a neuroradiologist; right-handedness as verified by the modified Edinburgh Handedness Inventory[14], no comprehensive language disorder, no severe cognitive problems (above 20 points on the mini-mental status examination), and (5) no history of psychiatric or neurologic disease. All of the stroke patients understood the purpose of this study and provided their written informed consent prior to their participation in the study in accordance with the ethical standards of the Declaration of Helsinki. This protocol was approved by the Institutional Review Board of Yeungnam University Hospital. The demographic data for the patients are summarized in Table 1.

Contraversive pushing was assessed by the standardized Scale for Contraversive Pushing (SCP)[13]. This scale may help to diagnose and quantify the behavior of patients with stroke and left or right brain damage who demonstrate PS. Based on Davies' criteria[4], the SCP comprises 3 parts: the symmetry of spontaneous body posture, the use of the non-paretic arm and/or leg to increase pushing force by abduction and extension of the extremities, and the resistance to passive correction of the tilted posture. Each item was assessed in both the sitting and standing positions. In our study, we used the cut-off criteria of the pusher, suggested by Baccini et al.[15], who assigned a diagnosis of PS to all participants with an SCP score of greater than 0 in each SCP score section, leading to a total SCP score of 1.75. The reliability and validity of the SCP is well established[16].

Ideomotor apraxia was tested with the movements of the arm and leg. Each test was composed of 12 items demonstrated once by the examiner, and then the participants were invited to reproduce the movement by imitation immediately after presentation. Participants were instructed to use the non-paretic limbs. Each item was scored as pass or fail according to a set of rules derived from a previous study[17]. Failed items were demonstrated two further times; only the items that were consistently failed were scored 0, items that were successfully recreated in any of the three attempts were scored 1. The total score of each test ranged from 0 to 12. The ideomotor apraxia test assessing the arm was taken from De Renzi and Faglioni[18], and the test for the leg was taken from Ambrosoni et al[19]. The test items for the arm ideomotor apraxia are as follows. 1) Arm is raised laterally, perpendicular to the body. The open hand is swept from one side to the other and brought, palm down, into contact with the opposite shoulder. 2) Open palm is slapped against the back of the neck. 3) Hand is placed open, palm down, under the chin. 4) Saluting. 5) Hand is held like a tube against the mouth. Patient blows through it. 6) Raise the hand, palm open forward, as for the sign to stop. 7) Closed fist, thump sideways on table, Open hand, slap palm down on table. 8) Fist on the forehead and then on the mouth. 9) Fingertips and thumb tip together in ring, all touching forehead. Hand moves out from forehead, rotating and opening wide as it moves. 10) Cross yourself. 11) Hand perpendicular to the body, fingers downwards. Hit forehead three times. 12) Send a kiss. Fingertips together in ring on the mouth. Hand opens wide as it moves out. The test items for the leg ideomotor apraxia are as follows. 1) Slide leg forward. 2) Slide leg backward. 3) Kick forward. 4) Cross legs whilst seated. 5) Put one foot in front of the other touching. 6) Pretend to extinguish a cigarette with your foot. 7) Trace a cross on the floor using your foot. 8) Place one foot above the other. 9) Trace an anti-clockwise circle on the floor using your foot. 10) Place the inner side of your foot on the floor. 11) Place your toe then your heel on the floor. 12) Place the external edge of your foot on the floor.

The Motricity Index (MI), with a maximum score of 100, was used to measure of motor function[20]. The MI of the affected extremities was measured twice: at the onset of stroke and at 6 months after stroke onset. The reliability and validity of the MI is well established[20]. The evaluator of clinical data including the SCP was blinded to the ideomotor

Table 2. Scores on the scale for contraversive pushing (SCP) and ideomotor apraxia for the pusher group and the non-pusher group.

		Pusher group	Non-pusher group
SCP	Posture	1.1 ± 0.5*	0.1 ± 0.2
	Extension	1.3 ± 0.5*	0.4 ± 0.6
	Resistance	1.8 ± 0.4*	0.2 ± 0.5
	Total	4.1 ± 1.1*	0.7 ± 1.0
Apraxia	Upper limb	8.2 ± 3.1*	10.9 ± 1.9
	Lower limb	7.6 ± 3.6*	10.6 ± 2.4
	Total	15.8 ± 5.8*	21.5 ± 3.8

*: $p<0.05$. All values presented as mean ± standard deviation.

apraxia data, and the evaluator of the ideomotor apraxia was blinded to the clinical data.

Data were analyzed with the Mann-Whitney U test in order to compare the SCP scores, the ideomotor apraxia scores, and demographic and clinical variables between the pusher group and the non-pusher group. A chi-square test was used to analyze the distribution of sex, lesion side, and other clinical values between the two groups. All statistical analyses were performed using PASW 18.0 (SPSS Inc, Chicago, IL, USA), and $p<0.05$ was used as the criterion for statistical significance.

RESULTS

Table 1 shows demographic and clinical characteristics of the two groups. The demographic characteristics of the two groups were similar with respect to age, onset time, lesion side, and education. In addition, there were no significant differences between the two groups in terms of the mini-mental status examination and MI scores. Presence of loss of somatosensory and kinesthesia were not significantly different between the two groups. The presence of aphasia in both groups was similar, but the existence of neglect was significantly different ($p<0.05$) between the two groups.

Table 2 shows the mean scores of the SCP and ideomotor apraxia tests of the two groups. In the pusher group, the SCP total scores were significantly higher ($p<0.05$), and the detailed item posture, extension, and resistance scores were significantly higher than those of in the non-pusher group ($p<0.05$). For the ideomotor apraxia assessment, the scores of the pusher group were lower those of the non-pusher group for the upper limb, lower limb, and total score. In particular, the total scores including upper and lower limb scores were significantly lower than those of the non-pusher group ($p<0.05$). The correlation between SCP scores and ideomotor apraxia scores was not significant ($r = 0.11$, $p = 0.72$).

DISCUSSION

In the current study, we found that the PS group had more severe ideomotor apraxic disorder in all of the upper and lower limbs than the non-PS group. This main finding suggests that stroke patients with PS have difficulty establishing a motor strategy necessary to perform motor plan and execution. Several clinical studies have revealed that motor symptoms related to ideomotor apraxia are detected in patients with PS[4,7,11]. According to Davies' clinical observations[4], patients with PS had considerable difficulty in learning purposeful movement and performing common activities of daily living. Moreover, performance of skilled tasks using the non-affected limb appeared clumsy. Cardoen and Santens[11] reported that gait apraxia was observed in two of their patients with PS. Based on these clinical observations, only one systemic investigation has looked for a causal relationship between PS and neuropsychological factors[3]. According to that study, it was reported that neuropsychological symptoms, such as ideomotor apraxia, were not the cause of PS. These conflicting findings may be attributable to the fact that an ideomotor apraxia test tool of proven validity and reliability was used. In order to screen for ideomotor apraxia, some simple items were tested, e.g., ask to point, wave, and salute. Therefore, there is a possibility that subtle apraxic symptoms in patients with PS were not detected. In our study, we adopted the ideomotor apraxia test because of its proven validity and reliability and because it assesses the symptoms of each upper and lower limb. Accordingly, our findings of ideomotor apraxic behavior in PS may be due to the sensitivity of the apraxia assessment tool. To our knowledge, ours is the first study to suggest that stroke patients with PS have ideomotor apraxic behavior in all of the upper and lower limbs. In addition, neglect also has been suggested as one of the possible causes. Similar to our findings, the distribution of neglect was significantly different between pusher patients and non-pusher patients. However, because some cases of pusher patients were associated with neglect, this symptom itself could be the cause of PS.

Possible mechanisms underlying PS have been presented in the studies of Karnath et al. and Perennou et al[8,13]. Karnath et al.[13] explained that the prominent factor in PS is an altered perception of the body's orientation in relation to gravity. For instance, patients with PS have an upright body orientation tilted 18° to the ipsilesional side, whereas they show undamaged neural processing for visual and vestibular inputs, which determine visual verticality. Accordingly, PS was considered to be related to abnormal postural control

that is caused by dissociation of two perceptual systems, i.e., disturbed upright body orientation and intact visual vertical orientation[5,20,21]. In addition, Perennou et al.[8] speculated that exaggerated sensory feedback from the affected side led PS patients to reflexively compensate for a false feeling of leaning toward the unaffected side. Commonly stroke patients without PS have some degree of ability to compensate for postural control by integrating their residual sensory and perceptual modalities, since essential elements for postural control, such as sensory, proprioception, vestibular function, vertical orientations, and so forth, may be partially damaged. However, it is thought that patients with PS lose their abilities of motor planning and execution, which are required to compensate for the partial damage to the postural control system. These disabilities of motor formulation are similar to ideomotor apraxic behavior, which is defined as a disorder of motor planning which may be acquired or developmental[18]. In addition, ideomotor apraxia is characterized by the loss of the ability to execute or carry out learned purposeful movements. Therefore, we believed that ideomotor apraxia might be one of the neuropsychological factors that cause and contribute to the deterioration in PS. However, some of our patients with PS had higher ideomotor apraxia scores than the average, which was shown in patients without PS. Thus, apraxia is not necessarily a critical factor in PS, and PS might be caused by the interaction of various neuropsychological symptoms.

PS contributes to a deterioration of functional abilities in hemiparetic stroke patients, especially with respect to postural control and gait function. Prior studies have reported that patients with PS take 3.6 weeks longer for therapeutic interventions to reach the same functional outcome than patients without PS[3,6,22]. Treatments based on scientific evidence can reduce the recovery period for prognostic function outcome, which leads to increased economic efficiency. Therefore, information for therapeutic interventions for PS is an important issue in the field of physical therapy. We have shown that patients with PS have dysfunctions of motor planning and execution for their upper and lower limbs. These findings suggest that therapeutic treatment for apraxic behavior is necessary to improve the functional ability of patients with PS. This current study was not designed to elucidate the crucial factors that cause PS. Further studies with larger sample sizes are required to identify the causes of the neuropsychological and movement related factors that influence PS.

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