Neurology Asia 2020; 25(4) : 459 – 465

Does the presence of ideomotor apraxia in stroke patients adversely affect rehabilitation outcomes? A prospective study

[1]Sacide Nur Saracgil Cosar *MD*, [1]Selin Ozen *MBBS BSc*, [2]Selin Taflan Selcuk *MD*, [1]Oya Umit Yemisci *MD*

[1]Department of Physical Medicine and Rehabilitation, Baskent University Faculty of Medicine; [2]Dr. Abdurrahman Yurtaslan Ankara Oncology Education and Research Hospital, Ankara, Turkey

Abstract

Background and Objectives: Ideomotor apraxia is an inability to perform a gesture following a verbal command despite having intact knowledge of the task. The presence of ideomotor apraxia may negatively affect functional outcome of stroke patients. The aim of this study was to evaluate the effect of ideomotor apraxia on rehabilitation outcomes following first ever stroke. *Methods:* A cross sectional observational study of 35 stroke patients admitted to a rehabilitation unit for treatment. Patients were evaluated for ideomotor apraxia using the ideomotor apraxia test. Function was assessed before and after treatment using the Functional Independence Measure, motricity index and functional ambulation scale. Cognitive function was evaluated using the Functional Independence Measure and mini mental state examination. *Results:* Ideomotor apraxia was present in 31.4% of patients. Mean Functional Independence Measure motor and cognitive scores of apraxic patients on admission and at discharge were lower than those of non-apraxic patients, ($p<0.05$). The cognitive and total Functional Independence Measure scores and motricity scores in the ideomotor apraxia group on discharge had not reached the admission values of the non-ideomotor apraxia group.
Conclusion: The presence of ideomotor apraxia following stroke has a negative impact on overall function, both before and after rehabilitation, when compared to stroke patients without ideomotor apraxia. Stroke patients must be assessed for ideomotor apraxia prior to commencement of a rehabilitation program in order to guide treatment and determine realistic treatment goals.

Keywords: Apraxia, ideomotor, stroke, recovery of function

INTRODUCTION

Apraxia is the inability to carry out previously learned, purposeful, skilled movements despite intact sensory, motor, and language functions, motivation, memory and comprehension.[1] The phenomenon of apraxia was first described in a stroke patient by Hugo Liepmann who observed that the patient was able to carry out spontaneous movements, such as using a spoon, but could not perform simple gestures following a verbal command.[2] Liepmann concluded that apraxia occurred due to a deficiency in motor planning and went onto classify apraxia into its subtypes: ideomotor, ideational, and limb kinetic. The most widely recognized type of apraxia is ideomotor apraxia (IMA) and is commonly seen in patients with stroke or neurodegenerative disease.[3] IMA can be defined as an inability to perform a gesture following a verbal command despite having intact knowledge of the task.[4] Patients know what to do in order to perform the task but not how to execute it. IMA is also related to cognitive insufficiency in the declaration of movement knowledge suitable for the object[5], disturbance in solving a mechanical problem[6], inadequacy in motor planning[7,8] and difficulty in learning new gestures.[9] IMA is commonly associated with damage to the parietal association areas[10] , less frequently with lesions of the premotor cortex and supplementary motor area, and usually with disruption of the intrahemispheric white matter bundle which interconnects them, as well as with basal ganglion and thalamic damage.[11,12]

Address correspondence to: Selin Ozen, Baskent University Faculty of Medicine, Department of Physical Medicine and Rehabilitation, Maresal Fevzi Cakmak Cad. 5. Sok. No: 48, Bahcelievler, Ankara, 06490. Turkey. Tel: +90 312 212 6650, E-mail: drselinozen@gmail.com

Date of Submission: 5 May 2020; Date of Acceptance: 22 June 2020

IMA is seen in both neurodegenerative disease and following a stroke with a prevalence of 28-57% in the presence of a left hemispheric and 0-34% in right hemispheric cerebrovascular lesions.[13] In studies to date, IMA has been found to adversely affect functional independence and so activities of daily living (ADL).[14,15,16] A more recent study found that the presence of IMA following stroke resulted in a significantly lower level of functional independence even after a period of inpatient rehabilitation. In this study IMA was also significantly associated with the presence of neglect, lower mini mental state examination (MMSE) scores and total anterior circulation ischaemia.[17]

The aim of this cross sectional observational study was to evaluate the relationship between patient demographics, stroke aetiology and IMA and to determine the effect of IMA on inpatient rehabilitation outcomes in terms of function following a first time stroke.

METHODS

Design and participants

Sixty consecutive first ever cerebrovascular stroke patients admitted to the tertiary inpatient rehabilitation clinic of Baskent University Faculty of Medicine, Ankara, Turkey, within a 6 month period were evaluated for study inclusion. A total of 35 patients were included in the study. All patients signed a written informed consent form before their inclusion in the study. In cases in which the patient could not comprehend the details of the informed consent form due to their levels of cognition, verbal consent was also obtained from their next of kin. Inclusion criteria included: (1) A first time diagnosis of stroke as defined by the World Health Organization: 'a vascular lesion of the brain that results in rapidly developing clinical signs or focal or global loss of brain function that persists for at least 24 hours or longer or leads to death, with no apparent cause other than being of vascular origin.'[18] In all patients, the diagnosis was confirmed by computed tomography or magnetic resonance imaging. (2) Ability to engage in rehabilitation; (3) Ability to speak Turkish; (4) A minimum level of primary school education. Exclusion criteria included: (1) Previous history of stroke; (2) Presence of bihemispheric stroke; (3) Previous episode of rehabilitation for the current stroke; (4) Medical instability; (5) History of other neurological or psychiatric illness; (6) Presence of visual defects; (7) Presence of aphasia; (8) Presence of a learning disorder.

All patients who met the inclusion criteria of the study were evaluated for the presence of IMA using the Ideomotor Apraxia Test (IAT).[19] The IAT uses both transitive (movements involving tools) and intransitive (communicative or gestural) movements to evaluate for the presence of IMA. The validity and reliability of the Turkish version of the IAT in stroke patients has been shown.[20] The Turkish IAT is comprised of four categories, each containing five items: Facial (put out your tongue, close your eyes, whistle, sniff a flower, blow out a match); Upper extremity (make a fist, salute, wave goodbye, scratch your head, snap your fingers); Instrumental (use a comb, use a toothbrush, use a spoon to eat, use a hammer, use a key); and Complex (pretend to drive a car, pretend to knock at the door, pretend to fold a newspaper, pretend to light a cigarette, pretend to play the Turkish lute). The cut-off value for a diagnosis of IMA using the Turkish version of the IAT is 51.56; therefore in this study, patients with scores lower than or equal to this value were accepted as apraxic.

All patients were included in the stroke rehabilitation program consisting of: (1) Five one- hour-long sessions per week of individualized physical therapy (PT) with a physiotherapist who was blind to the presence/absence of a diagnosis of IMA; (2) Seven one-hour-long sessions per week of individualized occupational therapy activities mainly aimed at treating the upper extremities (including upper body ergometry, and task based exercises); (3) Psychosocial counseling; (4) Speech and language therapy where necessary. All treatments occurred under the supervision of the same Physical and Rehabilitation Medicine (PRM) specialist. Physical therapy included neurofacilitation techniques, range of motion exercises, progressive resistance exercises, balance, coordination and ambulation training. The duration and intensity of therapy was similar for all patients. None of the treatments were aimed at treating IMA in particular. The duration of treatment was not predefined.

The study was approved by Baskent University Institutional Review Board and Ethics Committee (study number KA03/106) and carried out according to the institutional guidelines and the principles of the Decleration of Helsinki. Written informed consent was obtained from each patient prior to study inclusion.

Outcome measures

All the patients' demographic details, time since stroke onset, side of lesion, dominant or non-dominant hemisphere involvement and details of stroke etiology were recorded. Characteristics and functional outcomes of apraxic and non-apraxic patients were compared.

All outcome measures were evaluated by the same PRM specialist. The primary outcome measure was the Functional Independence Measure (FIM) which was evaluated on admission and prior to discharge. FIM is evaluated in all stroke inpatients before treatment commencement, at standard intervals during the patients' hospital stay and prior to discharge as part of the standard practice of the rehabilitation centre. The FIM is an eighteen item seven level ordinal scale of physical, psychological and social function.[21] This tool is used to assess a patient's level of function as well as change in patient status in response to rehabilitation or medical intervention and is one of the most widely used standardized functional outcome measures in medical rehabilitation.[22] In this study the Turkish version of the FIM was used.[23] Thirteen of the eighteen items of the FIM assess motor function and provide a 'motor score', the remaining five questions assess communication and social cognition and provide a 'cognition score'. Each item is scored from 1=complete dependence of task to 7=complete independence of task. The lowest possible attainable total score is 18 and the highest possible attainable total score is 126. The higher the score the higher the level of independence. When considering the sub scores, the lowest possible attainable motor score is 13 and the highest is 91. The lowest possible attainable cognition sub score is 5 and the highest is 35. Functional gain was recorded as the difference between the FIM score on admission and the FIM score at discharge.

The secondary outcome measure included the motricity index which was evaluated on patient admission and prior to discharge. The motricity is used to evaluate limb motor function and has good validity and reliability in stroke patients.[24] The test consists of six items: pinch grip, elbow flexion, shoulder abduction, ankle dorsiflexion, knee extension and hip flexion which all receive a score between 0 (no movement) to five (normal). A total 'arm score' and total 'leg score' are obtained as well as a 'side score' which is the sum of the arm and leg scores, divided by two. One point may be added to each limb score giving a maximum score of 100.

Pre-existing co-morbidities were scored according to the Charlson index.[25] This index takes into account both the number and seriousness of co-morbidities. Myocardial infarction, congestive heart failure, peripheral vascular disease, dementia, chronic pulmonary disease, connective tissue disease, ulcer disease, mild liver disease and diabetes are each assigned a score of 1. Hemiplegia, moderate and severe renal disease, diabetes with end-stage organ damage, tumors, leukemia, and lymphoma are each assigned a score of 2. Moderate and severe liver diseases are each scored as 3, and metastatic solid tumor and acquired immune deficiency syndrome are each assigned a score of 6.

The Mini Mental State Examination (MMSE) was applied in order to assess cognitive function.[26] The reliability and validity of the Turkish version of the MMSE in the diagnosis of mild dementia in the Turkish population has been shown.[27] The MMSE is divided into two sections; the first section requires vocal responses only and addresses orientation, memory, and attention, the maximum score is 21. The second part tests the patient's ability to name, follow verbal and written commands, write a sentence spontaneously and copy a complex polygon. The functional ambulation scale (FAS) was used to measure the ambulatory status of the patients at discharge.[28] The FAS is composed of six levels, from zero to five. While zero indicates that the patient is bedbound, five indicates fully independent ambulation. This classification also takes into account the patient's ability to walk on a flat or non-flat surface, up a hill or stairs, the use of ancillary equipment and the amount of support required for ambulation.

Statistical Analysis

Statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) program for Windows, version 13.0. Descriptive analyses were presented using means and standard deviations for normally distributed variables, medians (minimum-maximum) for the non-normally distributed variables and percentages for ordinal variables. The independent sample t-test was used for intergroup comparison of the normally distributed parametric variables and the Mann-Whitney U test was used for intergroup comparison of the non-normally distributed qualitative variables. The Chi-square test was used to evaluate categorical variables. A p value less than 0.05 was considered statistically significant.

RESULTS

In total 35 patients were included in this cross sectional observational study. IMA was present in 11 (31.4%) of the patients. The mean age of the study participants was 62.46 ± 13.65 years. Baseline characteristics of both groups have been given in Table 1. A flowchart of the recruitment and flow of patients through the study is depicted in Figure 1. The mean Charlson comorbidity index score was 2.91±0.22 in the apraxic patient group, and 2.81 ± 0.22 in the non-apraxic group (p=0.954).

Ten (55.6%) of the patients with left hemisphere lesions and 1 (5.9%) of the patients with a right hemisphere lesion had IMA according to the IAT (X2 =10.01, p=0.002). No significant differences were found with respect to age, sex, and stroke aetiology between apraxic and non-apraxic patients. There was no significant difference in time since stroke onset, length of hospital stay, MMSE scores between those with IMA and those without.

The admission and discharge values of the outcome measures in those with/without IMA according to the IAT are shown in Table 2. The mean total FIM scores of apraxic patients on admission and at discharge were found to be lower than those of non-apraxic patients, and there was a statistically significant difference between the two groups (p<0.0001 on admission and p=0.002 on discharge). Similar findings were present when the motor and cognition subsections of the FIM were studied. There was no between- group difference in FIM gain scores at the end of the treatment program.

When comparing the IMA group with the non-IMA group, there was no significant difference in FAS on discharge (p>0.05). The mean IAT score was 32.83 ± 26.14 in the FAS 0-2 group and 56.37 ±5.80 in the FAS 5 group (p=0.019). The Motricity score was higher in the non-IMA group when compared to the IMA group on admission to hospital (43.91±33.87 versus 22.45±16.22). Motricity scores increased in both the IMA and non-IMA group with treatment. However, there was no significant difference in between-group scores on discharge (0.066).

DISCUSSION

Lesions of the left hemisphere have been largely regarded as the main cause of apraxia, but IMA is also seen following right hemisphere cerebrovascular accidents. The results of this study were consistent with the majority of studies to date[13,16,29]; significantly more patients with left sided lesions had IMA when compared to those with right lesions (p=0.002). However, the small sample size in this study makes it difficult to draw firm conclusions regarding presence of IMA and lesion location. A recent study by Civelek *et al.*, 2015 in which patients were assessed for IMA using the IAT, reported that a similar proportion

Table 1: Demographic and clinical characteristics of the apraxic and nonapraxic patients

	All Patients n=35	Apraxic n=11	Nonapraxic n=24	P
Age, mean years ± SD	62.46 ± 13.65	64.73± 7.92	61.42 ± 15.64	0.983
Gender				
Male n (%)	19 (54.3)	6 (54.5)	13 (54.2)	0.762
Type of stroke				
Haemorrhage n(%)/ Infarction n (%)	8 (22.9)/27 (77.1)	1 (9.1)/10 (90.9)	7 (29.2)/17 (70.8)	0.189
Hemisphere damage				
Right n(%) /Left n(%)	17 (48.6) /18 (51.4)	1 (9.1)/10 (90.9)	16 (66.7)/ 8 (33.3)	0.002*
Time since stroke onset (mean days ± SD)		35.81 ± 33.72	58.41 ± 53.67	0.177
LOS (mean days ± SD)		47.72 ± 30.19	45.58 ± 24.93	0.826
MMSE score		20.71 ± 5.08	22.30 ± 5.00	0.469

*p<0.05
SD: Standard deviation. LOS: length of stay. MMSE: Mini mental status examination.

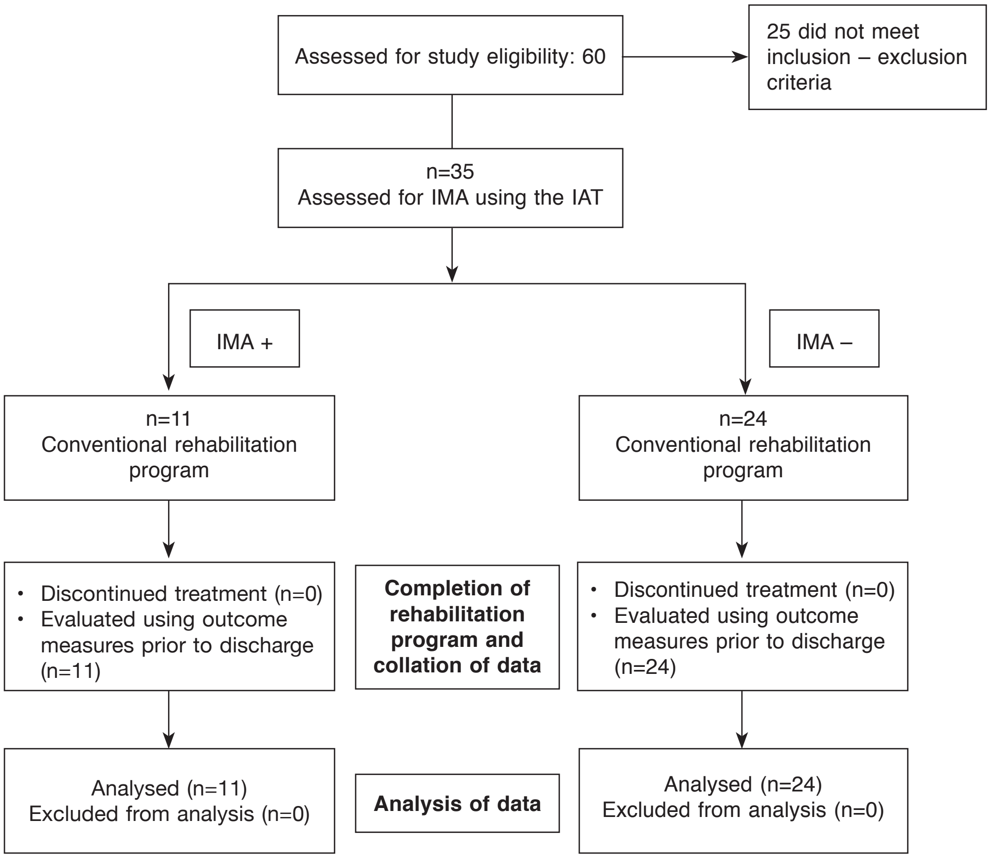

Figure 1. Flow of participants through the study

of those with left sided and right sided stroke had IMA.[17] Again the sample size was small in this study also.

In the present study, no relationship was found between the prevalence of apraxia and the patients' demographic details, stroke etiology or presence of pre-existing co-morbidities. This is in keeping with previous studies.[16,17] The difference in FAS

Table 2: Mean values of FIM and MI of apraxic and nonapraxic patients before and after treatment

	Apraxic* (n=11)	Nonapraxic† (n=24)	P
FIM motor score ± SD (admission)	22.27 ± 10.45	43.70 ± 21.20	<0.001 ‡
FIM motor score ± SD (discharge)	44.72 ± 15.06	61.91 ± 16.76	0.007 ‡
FIM cognition score ± SD (admission)	22 ± 10.04	33.12 ± 3.41	0.002 ‡
FIM cognition score ± SD (discharge)	26.36 ± 8.73	33.58 ± 2.24	0.011 §
Total FIM score ± SD (admission)	44.27 ± 18.21	77.66 ± 23.34	<0.001 ‡
Total FIM score ± SD (discharge)	70.63 ± 20.96	96.20 ± 18.19	0.002 ‡
FIM gain in score ± SD	26.36 ± 23.56	18.54 ± 17.55	0.280
MI score ± SD (admission)	22.45 ± 16.22	43.91 ± 33.87	0.210
MI score ± SD (discharge)	37.45 ± 18.99	54.77 ± 27.27	0.066

*Ideomotor Apraxia Test (IAT) score ≤ 51.56
† Ideomotor Apraxia Test (IAT) score > 51.56
‡ p<0.001, §p<0.05
SD: Standard deviation, FIM: functional independence measurement. MI: motricity index. SD: Standard deviation.

grading in those with IMA and those without was not significant. However, those with a lower grade of functional ambulation also had a lower IAT score. The correlation between the presence of IMA and low FAS is difficult to decipher as, again, lesion location and size may also have affected the level of mobility.

In the present study, contrary to the findings of previous studies[13,15], there was no significant difference in MMSE in those with IMA when compared to those without. This may be due to the MMSE evaluating cognitive functions such as orientation, memory, attention and calculation which may not be affected by the presence of the IMA. In contrast, the cognition score of the FIM was significantly lower in those with IMA both on admission and discharge (p=0.007 and p=0.002 respectively). The cognitive component of the FIM evaluates social interaction, problem solving and memory disturbances, all of which are likely to be affected by the presence of IMA.

Admission motor and cognition FIM scores are important predictors of the functional status of the patient on discharge.[30] The motor component of the FIM and total FIM scores were significantly lower in the apraxic patients compared to those without IMA, both on admission and at discharge. Indeed, the cognition and total FIM scores and motricity scores in the IMA group on discharge had not reached the admission values of the non-IMA group. Interestingly, the gain in FIM score of the IMA patients was more than in the non-IMA group, although the difference was non significant. This may suggest that IMA patients may benefit more from rehabilitation therapy. Therefore it is reasonable to suggest that rehabilitative treatment in stroke patients with IMA should include a special focus on techniques aimed at treating IMA. One study which included a 'behavioral training program' comprised of gesture production exercises in aphasic apraxic stroke patients showed that both praxis and ADLs improved in those who received conventional treatment for aphasia.[31] However, the long term benefits of such treatments remain unknown.

Noninvasive brain stimulation maybe another useful technique in treating both motor loss and apraxia following stroke due to its presumed excitatory/inhibitory influence on cortical plasticity.[32] Transcranial direct current stimulation (tDCS), repetitive transcranial magnetic stimulation (rTMS) and paired associative stimulation have been employed in treating various neurological disorders[33,34], however their impact on cognitive disorders remains largely unknown.

Limitations to the study include 1) Patients were not assessed for the presence of neglect. Neglect is a pathology of spatial cognition and is present in 13-85% of patients with right brain damage.[35] Furthermore, in one study as many as 90% of the stroke patients with neglect were also apraxic.[36] Therefore, in this study, the presence of neglect may have affected the rehabilitation outcomes. 2) Mechanism of stroke and stroke severity may also affect rehabilitation outcomes therefore in future studies such factors may also be taken into consideration and investigated. 3) The limited number of patients included in the study inhibited a regression analysis of the impact of various factors, including a diagnosis of IMA, on functional outcome.

In conclusion, the presence of IMA following stroke is common and results in significantly poorer overall function, both before and after rehabilitation, when compared to stroke patients without IMA. Therefore, even though the presence of apraxia may not always be immediately evident, it is important to assess stroke patients, and those with neurodegenerative disease, for IMA prior to commencement of the rehabilitation program in order to guide the patient's treatment accordingly and determine realistic treatment goals. Future studies should focus on investigating the effects of specific IMA rehabilitation techniques, including the role of noninvasive brain stimulation, on functional and cognitive outcome.

DISCLOSURE

Financial support: This study was supported by Baskent University Research Fund.

Conflict of interest: None

REFERENCES

1. Heilman KM, Watson RT, Rothi LG. Disorders of skilled movements: limb apraxia. In: Feinberg TE, ed: Behavioural Neurology and Neuropsychology. New York: McGraw- Hill Co., 1997: 227-35.
2. Goldenberg G. Apraxia and brain: Life and work of Hugo Liepmann. *Cortex* 2003; 39(3): 509-24.
3. Heilman KM, Rothi LG. Apraxia. In: Heilman KM, Valenstein E, eds:Clinical Neuropsychology. New York: Oxford University Press, 2003: 215-35.
4. Park JE. Apraxia: Review and update. *J Clin Neurol* 2017; 13(4): 317-24.
5. Buxbaum LJ, Saffran EM. Knowledge of object manipulation and object function: dissociations in apraxic and nonapraxic subjects. *Brain Lang* 2002; 82: 179-99.
6. Spatt J, Bak T, Bozeat S, PattersonK, Hodges

J. Apraxia, mechanical problem solving and semantic knowledge: contibutions to object usage in corticobasal degeneration. *J Neurol* 2002; 249: 601-8.
7. Harrington DL, Haaland KY. Motor sequencing with left hemisphere damage: are some cognitive deficits specific to limb apraxia? *Brain.* 1992; 115: 857-74.
8. Clark MA, Merians AS, Kothari A *et al*. Spatial planning deficits in limb apraxia. *Brain* 1994; 117: 1093-06.
9. Faglioni P, Basso A, Botti C, Aglioti S, Saetti C. Gestural learning in apraxia. In: Jeannerod M, ed: Attention and performance XIII. Hillsdale: Lawrence Erlbaum, 1990: 837-56.
10. Buxbaum LJ, Randerath J. Limb apraxia and the left parietal lobe. In: Vallar G, Branch Coslett H, eds: Handbook of Clinical Neurology. Amsterdam: Elsevier BV, 2018: 2(151): 349-63.
11. Wheaton LA, Hallett M. Ideomotor apraxia: a review. *J Neurol Sci* 2007; 15, 260 (1-2): 1-10.
12. Rothi LJ, Ochipa C. A cognitive neuropsychological model of limb praxis. *Cognit Neuropsychol* 1991; 8: 443-58.
13. Donkervoot M, Dekker J, van den Ende E, Stehmann-Saris JC, Deelman BG. Prevalence of apraxia among patients with a first left hemisphere stroke in rehabilitation centres and nursing homes. *Clin Rehabil* 2000; 14(2): 130-6.
14. van Heugten CM, Dekker J, Deelman BG, van Dijk AJ, Stehmann-Saris FC, Kinebanian A. Measuring disabilities in stroke patients with apraxia: a validation study of an observational method. *Neuropsychol Rehabil* 2000; 10: 401-14.
15. Hanna-Pladdy B, Heilman KM, Foundas AL. Ecological implications of ideomotor apraxia: evidence from physical activities of daily living. *Neurology* 2003; 11(60): 487-90.
16. Unsal-DelialiogluS, Kurt M, Kaya K,Culha C, Ozel S. Effects of ideomotor apraxia on functional outcomes in patients with right hemiplegia. *Int J Rehab Res* 2008; 31(2): 177-80.
17. Civelek GM, Atalay A, Turhan N. Association of ideomotor apraxia with lesion site etiology, neglect and functional independence in patients with first ever stroke. *Topics in Stroke Rehabilitation*. 2015; 22(2): 94-100.
18. Hatano S. Experience from a multi centre stroke register: a preliminary report. *Bull World Health Organ* 1976; 54: 541-53.
19. Kertesz A, Ferro JM. Lesion and location in ideomotor apraxia. *Brain* 1984; 107: 921-33.
20. Kurtulus K, Delialioglu Unsal S, Kurt M, Altınok N, Ozel S. Evaluation of ideomotor apraxia in patients with stroke: A study of reliability and validity. *J Rehabil Med* 2006; 38: 108-12.
21. Granger CV, Gresham GE. New developments in functional assessment. *Phys Med Rehabil Clin N Am* 1993; 4: 417-99.
22. Dromerick AW, Edwards DF, Diringer MN. Sensitivity to changes in disability after stroke: acomparison of four scales useful in clinical trials. *J Rehabil Res Dev* 2003; 40: 1-8.
23. Kucukdeveci AA, Yavuzer G, Elhan AH, Sonel B, Tennant A. Adaptation of the functional independence measure for use in Turkey. *Clin Rehabil* 2001; 15: 311-9.
24. Collin C, Wade D. Assessing motor impairment after stroke: a pilot reliability study. *J Neurol Neurosurg Psychiatry* 1990; 53: 576-79.
25. Charlson ME, Pompei P, Ales KL, MacKenzie R. A new method of classification of prognostic comorbidity for longitudinal studies: development and validation. *J Chron Dis* 1987; 40:373-83.
26. Folstein MF, Folstein SE. ‘ Mini mental state’. A practical method for grading the cognitive state of patients for clinician. *J Pshchiatr Res* 1997; 12: 189-98.
27. Gungen C, Ertan T, Eker E, Yasar R, Engin F. Standardize Mini Mental Test’in Türk Toplumunda Hafif Demans Tanısında Geçerlik ve Güvenilirliği. *Türk Psikiyatri Dergisi* 2002; 13(4): 273-81.
28. Collen FM, Wade DT, Bradshaw CM. Mobility after stroke: reliability of measures of impairement and disability. *Int Disabil Stud* 1990; 12(1): 6-9.
29. Zwinkels A, Geusgens C, van de Sande P, van Heugten C. Assessment of apraxia: inter-rater reliability of a new apraxia test, association between apraxia and other cognitive test, association between apraxia and other cognitive deficits and prevalence of apraxia in a rehabilitation setting. *Clin Rehabil* 2004: 18(7); 819-27.
30. Ng YS, Stein J, Ning M, Black-Schaffer RM. Comparison of clinical characteristics and functional outcomes of ischemic stroke in different vascular territories. *Stroke* 2007; 38(8): 2309-14.
31. Smania N, Aglioti SM, Girardi F, *et al*. Rehabilitation of limb apraxia improves daily life activites in patients with stroke. *Neurology* 2006; 67: 2050-52.
32. Nowak DA, Grefkes C, Ameli M, Fink GR. Interhemispheric competition after stroke: brain stimulation to enhance recovery of function of the affected hand. *Neurorehabil Neural Repair* 2009; 23: 641-56.
33. Lefaucher JP, Andre-Obadia N, Antal A, *et al*. Evidence based guidelines on the therapeutic use of repetitive transcranial magnetic stimulation (rTMS). *Clin Neurophysiol* 2014; 125: 2150-206.
34. Lefaucher JP, Antal A, Ayache SS, *et al*. Evidence based guidelines on the therapeutic use of transcranial direct current stimulation (tDCS). *Clin Neurophysiol* 2017; 128: 56-92.
35. Sinanovic O. Neuropsychology of acute stroke. *Psychiatr Danub* 2010; 22(2):278-81.
36. Linden T, Samuelsson H, Skoog I, Blomstrand C. Visual neglect and cognitive impairment in elderly patients late after stroke. *Acta Neurol Scand* 2005; 111(3): 163-8.