# Investigation of relation between apraxia type and lesion localization

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*Both left and right hemisphere lesions may cause apraxia, although it has been claimed that types of apractic disturbances are associated with lesion in particular areas of the cerebral cortex.*

*It is impossible to understand existence of the apraxia and its forms, without performing tests. Apraxia may occur without clinical or other sign of cortical dysfunction in the neurological examination.*

*In this study by using different tests apraxia has been investigated in 50 patients with right hemisphere lesions, in 50 patients with left hemisphere lesions, and in 30 normal controls. Apraxia was frequently seen in association with left-sided postrolandic lesions including lesions in the parietal lobe, and was seen together with aphasia in 23 percent and hemispatial neglect in 21 percent.*

*In conclusion apraxia seemed to be involved with right hemisphere lesions (constructional) and left hemisphere lesions (ideomotor, ideational, constructional, buccofacial) and deep subcortical structures of intrahemispheric connections and midline lesions result in apraxia as well (constructional, ideomotor). [Turk J Med Res 1993; 11(1): 44-50]*

KeyWord: Apraxia

Praxia; which is one of the highest brain function is a rather elementary subject than the other cerebral activities. Apraxia is defined as the defect that takes place in the dexterous and swift motions (1,2). Although the patients don't have weakness, sensorial lost, defects in tonus, posture, audition and comprehension, they can not perform the simple and complicated target directed movements that they had done previously in a plan and ordered manner (2-6).

In 1900, apraxia is firstly defined and systematically analysed in a patient who can not use his left hand perfectly and having a left hemisphere and pons lesion (3,7). Although a century has passed; neither the pathophysiological properties are understood nor the standardization to tests that are applied to patients are finished and the signs of the lesion localization, apraxia types and cortical defects are still in a cause.

The lesions which occures on the different parts of the cerebrum may also cause to the defects in the target accordance motion. However the lesions which are located in motor, premotor cortex, corpus callosum, angular and supramarginal girus, subcortical regions are thought to be responsible from apraxia (3,5,6,8). Although apraxia due to the left hemisphere lesion is rarely observed, it can also be caused from the right hemisphere lesion. The type of apraxia can be classified according to the latéralisation and localization (5,6,8).

The satisfactory response to the verbal orders without any defect in planning the movements but defects in starting and performing are the signs of idiomotor apraxia (3,6,8-10). In the angular and submarginal girus lesions the application of the orders and mimics can't be done but the submarginal girus anterior mediated lesions are characterized by the defects in performing the programmed movements (11,12). Right parietal lobe and corpus callosum lesions are also defined (6,10,13). In the ideational apraxia, the patient can perform the simple movements but the defects in planning and formulation cause to the disturbance of the combined series movements (3,6,14). The lesions in dominant hemisphere, intrahemispheric junctions and arcuate fasiculus are thought to be responsible from this type of apraxia (1,15). Buccofacial apraxia is characterized by the defects in the larynx supported motions. Parietal, anterior pericilvian area, anterior insula and temporal girus lesions cause this

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type of apraxia 92,16). In some researches oral apraxia is reported as an aphasia or a subgroup of idiomotor apraxia (17,18).

The lesion location in junction between the motion programming and performing centers causes to the disturbance of series movements leads to constructional apraxia (3,4,7,19.20). In this case the 3 dimention concept is lost, and the combined motions can't be performed (21,22). Generally the right hemisphere lesions are thought to be the reason of this type of apraxia furthermore dominant and nondominant parietal lobe, corpus callosum dysfunctions can be the reason (21,23,24). Another type of apraxia is Limb-Kinetic Apraxia. Which is characterized by defects in the motions which are experienced by distal muscles (25,26). Sometimes it is mentioned as aphasic agraphy (27,28).

When the lesions are localized in the praxia and language centers or areas close to these regions apraxia is observed together with the aphasia in a range between twenty to sixty (20%-60%) percent. An apractic patient may have 10-20% agnozia, 30-40% agraphy, 20-30% visuaconstructive defects and 40-50% cortical sense lost (30-33). Because of the anatomophysiological properties apraxia and aphasia exist at the same time which creates difficuilties (34).

In order to correctly detect the type of apraxia and differing it from aphasia neurological examinations are required also the tests specified for apraxia must be used.

The aim of this study is to investigate the relation between apraxia type and lesion localization on the patients having apraxia caused by hemisphere lesion.

## MATERIALS AND METHODS

100 patients who applied to the Neurology Department of Gevher Nesibe Hospital and 30 healthy volunteers were subjected in this study with low literate status and keen on using their right hands.

Patients with consciousness problems that would cause an obstracle to cooperation, defects in comprehension, audition and generalized defects in tonus, posture weakness and sensorial lost where excluded. For the hemiparesia case the other extremities were used in the application of the tests. All the patients were neurologically examined and after the first seventh day the tests applied. From the ethical point of view the permition was asked to every patient and required information were given.

In order to determine the lesion localization computerized brain tomography and electroencephelography were used. Some patients had cerebral angiography and lumbar punction tests. Magnetic resonance imaging examination was asked from an another medical center.

While determining the type apraxia modified tests Were used (3,35-37). The applied tests were consisted of eight main groups each having 10 subgroup. The first one was the application of the orally directed simple movement ("Acknowledge a salute!") and the other tests were as; buccofacial and extremite pandomimes ("Follow me!" "Do the same!") defining the series movements ("Take the pills out of a box, pull them to a spoon and drink"), drawing simple figures or copying ("Place a dot to the center of a circle") and the patients were asked to place an object in space, draw a figure with its 3 dimention finally the combined skill tests were applied ("Draw a car, a man, a house .... etc"). The classical scoring was used (37).

In addition to the above mentioned tests; aphasia, hemispatial neglect and right hemisphere functions were aso tested (41-44). According to the clinical and laboratory results patients having bilateral hemisphere lesions, cortical atrophies, or significant decrease in the sensorial, visual sense were all excluded. The study population were randomly selected among the patients and 50% had only right, and the rest had only left hemisphere lesions.

The statistical tests were used in order to compare the study and control groups.

## RESULTS

Sixty-two (62%) male and thirty - eight (38%) female adult patients (Mean age: 48) were investigated in this study.

The control group was consisted of 30 healthy people as 20 male (66.7%) an 10 female (33.3%) adults (Mean age: 42).

According to the clinical and laboratory examination results 4 patients had cerebral malignancies (4%), 1 had inflammation (1%) and 95 patients had cerebrovascular problem (95%). 57 (60%) cerebral infarct, 36(37.9%) cerebral hematoma and 2 (2.1%) heamoragic infarct cases were observed.

Apraxia was observed in 18 (35.29%) right, 33 (64.71%) left hemisphere lesions as the result of the applied tests. 14 (27.4%) idiomotor, 7 (13.72%) ideational, 27 (52.95%) constructional and 3 (5.88%) buccofacial apraxias formed the 51 cases in our study (Table 1).

Left hemisphere lesions were responsible from all of the ideational and buccofacial apraxias. However most of the idiomotor (78.58%) apraxias were induced by the left hemisphere lesions. These results were statistically significant but, the differences between the constructional apraxias localized either right or left hemisphere were statistically insignificant ($p>0.05$) (55.5% right, 44.5% left hemisphere constructional apraxias were studied) (Table 1).

At the end of the tests applied it was observed that 51 (51%) patients had apraxia, 28 (28%) had aphasia and 23 (23%) had hemispatial and visuoconstructional neglect (Table 2).

Table 1. The assessment of the aphasia tests in patients and investigation of the relationship hemisphere lateralization

| Apraxia Type | Patients | | Right Hemisphere Lesion | | Left Hemishere LesiuD. | | | |
|---|---|---|---|---|---|---|---|---|
| | n | % | n | % | n | % | $x^2$ | P |
| Ideomotor | 14 | 27.5 | 3 | 21.4 | 11 | 78.6 | 4.57 | <0.05 |
| Ideational | 7 | 13.7 | – | – | 7 | 100 | 5.12 | <0.05 |
| Constructional | 27 | 52.9 | 15 | 55.5 | 12 | 44.5 | 0.33 | >0.05 |
| Buccofacial | 3 | 5.88 | | – | 3 | 100 | 4.96 | <0.05 |
| Total | 51 | 100 | 18 | | 33 | | | |

Table 2. The comparison of lesion localization with aphasia, apraxia and visuospatial neglect tests

| Lesion Localization | Patients | | ADraxia | | ADhasia | | YisuQSDatiaLnealect | |
|---|---|---|---|---|---|---|---|---|
| | n | % | n | % | n | % | n | % |
| Cerebral cortex | | | | | | | | |
| Frontal lobe | 7 | 7.0 | — | — | 2 | 28.5 | — | — |
| Parietal lobe | 11 | 11.0 | 7 | 63.6 | — | — | 4 | 36.3 |
| Temporal lobe | 9 | 9.0 | 2 | 22.2 | 2 | 22.2 | — | |
| Occpital lobe *M -* | 1 | 1.0 | — | — | — | | 1 | 100 |
| Frontoparietal lobe | 6 | 6.0 | 4 | 66.6 | 1 | 16.6 | 1 | 16.6 |
| Frontotemporal lobe | 6 | 6.0 | 2 | 33 | 5 | 83.3 | — | — |
| Frontoparietotemporal | 11 | 11.0 | 9 | 81.8 | 6 | 54.5 | 5 | 45.4 |
| Parietotemporal | 8 | 8.0 | 6 | 75 | 1 | 12.5 | 3 | 37.5 |
| Temporoaccipital | 2 | 2.0 | — | »±/inu?' '< | — | | 1 | 50 |
| Parietooccipital | 1 | 1.0 | 1 | 100 | — | — | 1 | 100 |
| Subcortical, deep localization | | | | | | | | |
| **Basal ganglions** | 5 | 5.0 | | | | | | |
| **Corpus callosum** | 2 | 2.0 | 1 | 50 | — | — | — | — |
| Cortical, subcortical mid line structures | | | | | | | | |
| Parietotemporal | | | | | | | | |
| Thalamus, internal capsul | 7 | 7.0 | 6 | 85.7 | 1 | 14.2 | 2 | 28.4 |
| Temporal, basal gang | 2 | 2.0 | 1 | 50 | 1 | 50 | — | — |
| Temporal, callosal | 2 | 2.0 | 1 | 50 | — | — | — | — |
| Frontotemporal, basal gang | 4 | 4.0 | 1 | 25 | 1 | 50 | — | — |
| Frontoparietotemporal | | | | | | | | |
| thalamus, callosal | 8 | 8.0 | 6 | 75 | 5 | 62.5 | 4 | 50 |
| Frontoparietal, basal gang | 5 | 5.0 | 4 | 80 | 3 | 60 | 1 | 20 |
| Subcortical, Brain stem | | | | | | | | |
| Pons | 1 | 1.0 | — | — | — | — | — | — |
| Bulbus | 1 | 1.0 | — | — | — | — | — | — |
| Pontocerebellar, subcortical | 1 | 1.0 | — | | — | — | — | — |
| Total | 100 | 100 | 51 | | 28 | | 23 | |

When the apraxia and other test results were compared with the lesion localization, most of the apraxias were rised due to the defects in the parietal lobe, but they were in the mild form. While the lesions localized in the parietal and subcortical regions didn't cause aphasia the frontal and temporal localized ones caused aphasia. There was also a close relation between the apraxia and hemispatial neglect phenomena in terms of lesion localization (Table 2).

All left hemisphere localized lesions caused aphasia and 23 (23%) patients had both aphasia together with apraxia 9 of the patients had only aphasia. Sixteen of the twenty-three patients had right, 6 had left hemisphere lesion. Except the two patients who had optical and temporooccipital lesions and all of the rest had apraxia 21% of the cases had apraxia and hemispatial neglect together and 13% had all of them (aphasia, apraxia and hemispatial neglect).

While the right hemisphere lesions could be characterized by the defects in drawings of the left parts, in hemispatial and visuospatial neglect models difficulties exist in the skill tests. The left hemisphere lesions

caused difficulties in performing the orally directed orders and unskilled combined motions.

In figures 1 and 2 the tomographic aspects of two patients having right and left hemisphere lesions.

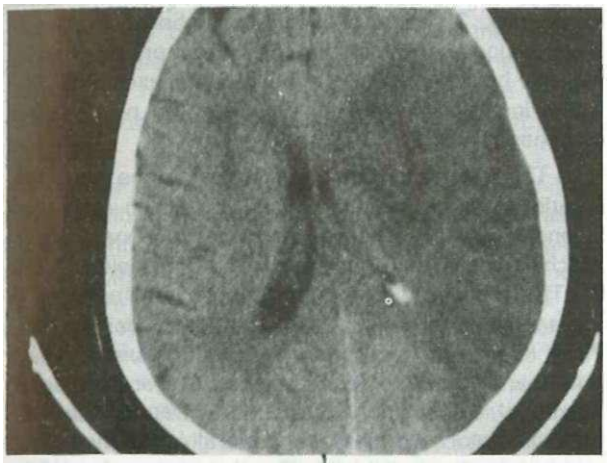

**Figure 1.** Tomographic aspect of the patient with apraxia and aphasia in the left hemisphere widespread lesion.

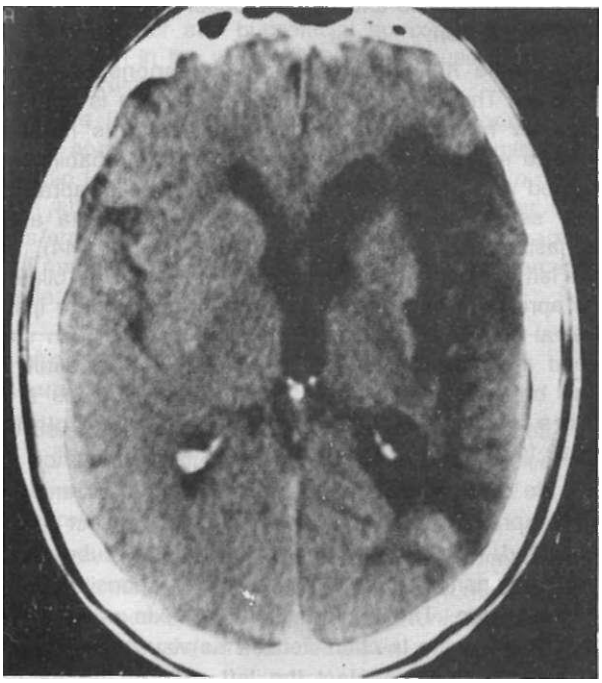

**Figure 2.** Computed tomographic scan showing of the patient with apraxia in the right hemisphere lesion.

## DISCUSSION

Idiomotor, ideational, constructional and buccofacial are the four different clinics of apraxia (2,3,5,8,14). In some reports the terms such as frontal, language and walking apraxia are used (2,6,24,26,28). Most of them can be classified as idiomotor and oral apraxia (12,13).

The classification of the apraxia hasn't finished yet, because of the problems in the lesion localization application of the tests and also there are some problems in differing from other cerebral activities. The left hemisphere is responsible from the control of verbal, nonverbal and coordinated motions (2,5,10,29) whereas the right hand side is devoted for the construction, topographic orientation, verbal and nonverbal emotional functions (23,24,31,32,42,43,46-59).

When apraxia takes place defect in coordinated and simple motor motion are observed by constructional and neglect phenomena. The functional defect that can be formed in one of the two hemisphere causes apraxia (20,22,26). In the lesions located on the left hand side apraxia and aphasia exist together so differing apraxia from aphasia becomes a problem. Apraxia due to the right hemisphere lesions cause constructional defects, hemispatial neglect. Moreover the center responsible from memory is also located on the right side (5,11,15,23,46,50,51). Therefore the acustic stimulus reachs to the temporal lobe of the related center and validated by the previous knowledge, while the optical cortex meanwhile by the help of arcuat faciculus Wernicke region is also activated. So any defect in the mentioned pathway will result as apraxia (3). Also the lesions in; motor and sensorial regions, subcortical deep localization and the sysetms taht get in touch in the inter and intrahemispheric communication will cause apraxia (Figure 3,4).

In order to understand the lesion localizations apraxia regions are examined (3,4,6). It was observed that anterior lesions were the main reason of idiomotor and oral apraxia. Motor apraxia is characterized with the unskilled motions (6,11,12,15). Posterior lesions are the main source of the ideational apraxia however it is very difficult to distinguish from the sensorial aphasia. They have similiar symptoms such as the defects in performing the simple and combined movements (14,17). Cortical and subcortical lesions are characterized by the defects in the combined motions which is named as constructional apraxia (1,3,7,19,22).

Generally apraxia is caused by the lesions located in the left parietal lobe and particular areas. As previously described the lesions located in the left parietal lobe (premotor complex, frontal operculum, preventicular white mater) cause idiomotor and buccofacial apraxia and lesions in the superior temporal and temporoparietal region are common for ideational apraxia. Constructional apraxia is the only one which is mediated from the right hemisphere lesions. However the lesions located in the corpus callosum and other subcortical region without only lateralization can cause idiomotor apraxia (3,6,7,11,17).

Right hemisphere lesions are thought to be responsible from the constructional apraxia by lateralization (4,19,21,22). Furthermore the lesions occupied in the cerebrum may also cause apraxia (3,7,20). The percentage of the right and left hemisphere lesions are

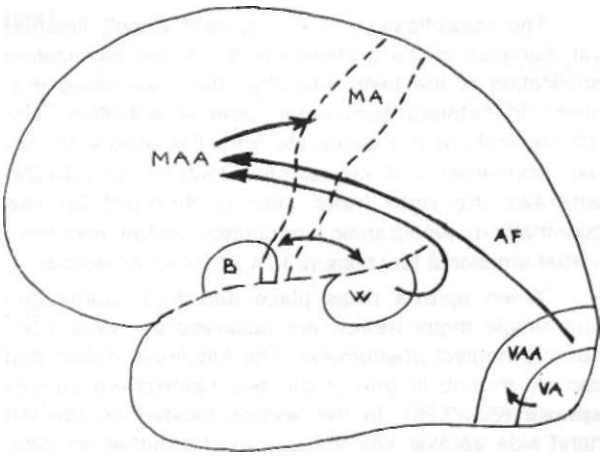


Figure 3. Language and praxia areas and connections. Lateral view of the left side of the brain. Geshwid's Schema.
MA: Motor Area, MAA: Motor Association Area,
B: Broca Area, W: Wernicke Area, VA: Visual Area,
VAA: Visual Association Area, AF: Arcuate Fasciculus

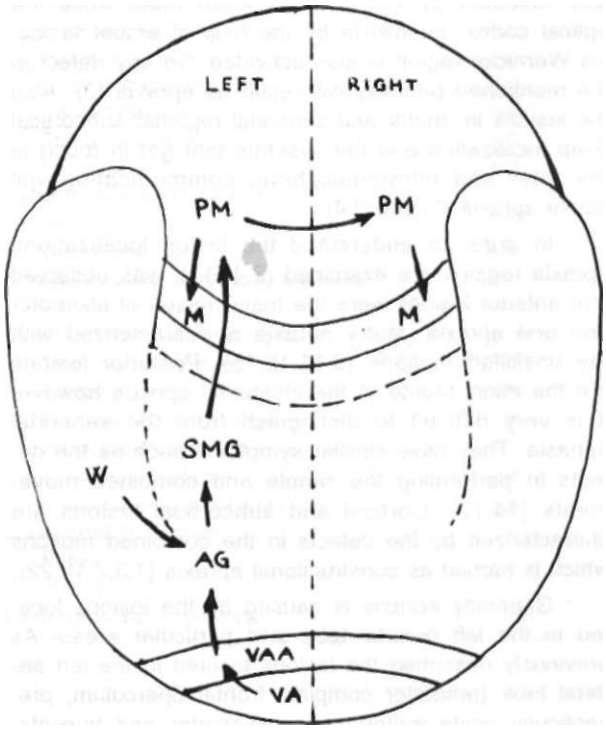


**Figure** 4. View from top of the brain. The arrows indicate major connections of the areas shown. Author's Schema.
M: Motor Area, PM: Premotor Area, AG: Angulare Gyrus,
SMG: Supramarginal Gyrus, VA: Visual Area,
VAA: Visual Association Area

not strictly determined (4,19,21,22,42). In our study most of the apraxias were constructional and left hemisphere mediated (67.41 %). All of the ideational, buccofacial and constructional apraxias were characterized by the patients with left hemisphere defects (44.5% left hemisphere), while only 35.29% was due to the right hemisphere lesions. The percentage of our results is low with respect to the literature but we only dealed with the cerebral hemisphere lesions and subjected the patients having lateralization at their neurological experience. 3 patients with pontocerebellar and brain stem lesion were included and the apraxias which couldn't be differed from aphasia were excluded. The lesion localization were suitable with the previous researches (Table 2).

The lesions localized near parietal lobe and frontoparietotemporal regions caused apraxia in a high incidence. One of the cases had constructional apraxia which is caused by the corpus callosum lesion (Table 2). The probability of existing apraxia and aphasia at the sametime is about twenty to seventy percent (20-70%) in the left hemisphere lesions because the language and target directed motions have anatomic and functional combinations (2,5,8,16,17,29). Buccofacial and idiomotor apraxias are generally observed with motor aphasias but the ideational apraxias are observed with sensorial aphasia. In our study 23 patients (23%) with apraxia and aphasia observed, with left hemisphere lesion.

Single hemisphere lesion is responsible from aphasia. Therefore the incidence of apraxia is high in patients having aphasia. However patients having apraxia may not have aphasia. The dual probability is related with the lesion localization and type of apraxia. The severe forms are observed when apraxia and aphasia exist together (2,3,5,8,16,17,34,41,42,44). In the left hemisphere lesions patients failed in the bilateral apraxia tests, however in the right hemisphere ipsilateral control doesn't exist therefore patients can suceed the apraxia test (3,17,24,50,52). As a result it can be concluded that, left hemisphere lesions are more complicated and have a severe clinic (5,10,12,29).

In 21% apraxia and hemispatial neglect and in 13% apraxia, aphasia and hemispatial neglect were observed. The wide lesions located in the subcortical and frontoparietotemporal region were responsible from this situation. The constructional apraxia caused by right hemisphere is characterized as visuopatial defect and the patients neglect the left side of the paper while drawing pictures (10,12,19,22,26). In the left hemisphere defects; it is in the form of apraxia defect (10,12,19,22,26).

A hemisphere with a lesion which causes hemispatial neglect phenomenon effects the opposite side of the body (3,4,24,27,41,48). The neglect phenomenon can easily be confused with other clinics such as; hemiacinezy, visual auditor agnozia (16,21,27,31,41,42, 43,49,51,52). It can be observed as a neglect to every type of sensorial stimulus.

The neglect phenomena are more common due to the right hemisphere lesions and for every sense

the ratio of neglect phenomena is defined (24,27,28, 42,47,48,50,52). It can exist with apraxia but generally observed with agnosia and visuospatial defects (47,48,51,52). The neglect phenomenon can not be explained by the defects in hemisphere so the cerebral activities have to be controlled. Although the related center is located in the right hemisphere, the lesion localization is not that much important because of the cortical and subcortical hemispheric junctions (43,50,51).

At the end of our study we have observed that patients with right hemisphere lesion have difficulties in skilled works. Most of them had visuospatial neglect but could perform the simple movements and pandomimes easily. In contrast, patients with left sided lesions had difficulties in performing simple and combined movements, b[illegible] they were able to correct their mistakes in repetitive motions while the patients with right hemisphere lesion couldn't. Moreover they were weak in performing hand target motions, and defects in bilateral apraxia tests. The right hemisphere lesions didn't cause apraxia in the ipsilateral extremities but bilateral apraxia. Focal lesion specificity was observed in the idiomotor and buccofacial apraxia and specific lateralization in the wide lesion which cause ideational and constructional apraxia was observed.

The lesions in right and left hemisphere may cause apraxia in a nigh incidence of left hemisphere specific tests for apraxia are required in order to differ from aphasia and neglect phenomenon. Further studies are required by the position emission tomography, and magnetic resonance imaging in order to determine the lesion localization.

## Apraksi tipi ve lezyon lokalizasyonu arasındaki ilişkilerin incelenmesi

*Serebral korteksin özel sahalarının lezyonlarında apraksik bozuklukların değişik tipleri tarif edilmekle beraber sağ ve sol hemisfer lezyonlarının ikisi de apraksi yapabilir.*

*Testler uygulanmadan apraksi ve formlarının varlığını anlamak mümkün değildir. Apraksi nörolojik muayenede klinik ve diğer serebral fonksiyon bozukluklarını göstermeden de olabilir.*

*Bu çalışmada 50 sağ ve 50 sol hemisfer lezyonu olan hasta ile 30 kontrol grubunda değişik testler uygulanarak apraksi araştırıldı. Apraksi sıklıkla sol parietal lobu da içine alan postrolandik lezyonlarında görüldü ve afazi ile %23, hemispasyal ihmal fenomen/eriyle %21 oranında birlikte bulundu. Sonuç olarak apraksi sağ hemisfer (konstrüksiyonel) ve sol hemisfer (idiomotor, ideasyonal, bukkofasiyal, konstrüksiyonel) lezyonlarında görülmüştür. Intrahemisferik bağlantıları sağlayan derin subkortikal strüktür ve orta hat yapıların lezyonlarında da apraksiler oluştu (konstrüksiyonel, idiomotor). [Türk Tıp Yaştırma 1993; 11(1); 44-50]*

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