Apraxia is the disorder of skilled movement

Apraxia is defined as a "disorder of skilled movement not caused by weakness, akinesia, deafferentation, abnormal tone or posture, movement disorders such as tremors or chorea, intellectual deterioration, poor comprehension, or uncooperativeness" (heilman and rothi, 1993 as cited in adriani et al., 2007, p. 61). In simple words apraxia can be referred to as motor agnosia (Chawla.J., 2009). Apraxics are not partially paralytic but they lack information about how to perform skilled movements (Chawla.J.,2009). The following essay is structured as follows. It begins with a short description of apraxia , its types, then it views the existing models apraxia which is followed by understanding the laterality of lesions in apraxia. After that it tries to understand the complex nature of apraxia then it argues for a multidisciplinary approach to study apraxia followed by diagnosis, treatment, prognosis and special concerns .

Apraxia is a disorder of motor control which can be referred neither to "elemental" motor deficits nor to general cognitive impairment" (Geschwind&Damasio, 1985; Heilman&Rothi, 1993).

Apraxia is a syndrome reflecting motor system dysfunction at the cortical level, exclusive of primary motor cortex. In planning movements, previously learned, stored complex representations of skilled movements are used. These 3-dimensional, supramodal codes, also called representations or movement formulae, are stored in the inferior parietal lobule of the left hemisphere. Diseases that involve this part of the brain, including strokes, dementias, and tumors, can cause loss of knowledge about how to perform skilled movements.

Apraxia can occur with lesions in other locations as well. Information contained in praxis representations is transcoded into innervatory patterns by the premotor cortices, including the supplementary motor area (SMA) and possibly the convexity of the premotor cortex; the information is then transmitted to the primary motor cortex and a movement is performed. Lesions of the SMA or other premotor cortices also can cause apraxia; in this case, knowledge about movement is still present, but the ability to perform movement is absent.

Apraxia also occurs with lesions of the corpus callosum, such as tumors or anterior cerebral artery strokes. Although the corpus callosum is not known to be involved directly in the performance of skilled movements, it contains crossing fibers from the right hemisphere to the premotor cortex. This type of apraxia represents a classic disconnection syndrome; patients with callosal apraxia typically are apractic only with the left hand.

Apraxia has a neurological cause that localizes fairly well to the left inferior parietal lobule, frontal lobes (especially the premotor cortex, supplementary motor area, and convexity), or corpus callosum. Any disease of these areas can cause apraxia, although

stroke and dementia are the most common causes. Interestingly, callosal apraxia is rare after callosotomy and is much more common with anterior cerebral artery strokes or tumors. Apraxia can be related to specific neural substrate that causes the disorder, for example following subcortical lesions in corticobasal degeneration (Merrians et al., 1999 as cited in Petreska et al.,2007).

Types of Apraxia

The following types of apraxia have been described in this section.

Ideational Apraxia –It was first assesed by performing purposive sequences of actions that requires to follow a correct sequence of processes for various objects (preparing tea) (Poeck .,1983). Ideational apraxia is a larger deficit that concerns the evocation of single actions. In this view , complex sequences of multiple objects are simply more suitable to reveal the deficit , possibly because of the heavier load placed on memory and attentional resources (De Renzi and Lucchelli, 1988).

Conceptual apraxia is defined as the loss of knowledge about tools and movements associated with their use. Patients having parietal lesions may have conceptual apraxia. This is attributed to faulty transcoding of the "innervatory patterns" in the motor cortex (Chawla,J.,2009). This apraxia is often observed in alzheimer's disease.

Ideomotor Apraxia – The concept of ideational apraxia was proposed by Liepmann. The neural correlates of ideomotor apraxia have been thought to be located in the left parietal lobe but some studies have claimed other region (Makuuchi et al., 2005). Lesion studies have reported defective imitation and movements executed by verbal commands (ideomotor praxis) after left parietal damage (Ghika et al., 1998 as cited in Makuuchi et al.,2005). A recent lesion study found maximum overlap of lesions in patients with ideomotor apraxia in the left intraparietal sulcus (Harrington et al., as cited in Makuuchi et al.,2005).

Conduction Apraxia – It is the syndrome of superior performance on verbal command than on immitation (Ochipa et al.,1994 as cited in Petreska et al.,2007). The opposite pattern has also been observed i.e., very poor performance on verbal command that improved on immitation or when seeing the object (Heilman.,1973 ; Merians et al., 1997 as cited in Petreska et al., 2007). The selective inability to imitate with normal performance was termed visuo – imitative – apraxia (Merians et al.,1997).

Constructional Apraxia - Constructional apraxics are unable to draw objects, copy figures simultaneously. It is an outcome of damage not only to the dominant but non dominant hemisphere as well. Hence, constructional apraxia appears to reflect the loss of bilaterally distributed components for constructive planning and the perceptual processing of categorical and coordinate spatial relations (Platz and Mauritz., 1995 ; Laeng.,2006 as cited in Petreska et al., 2007).

Apraxia of speech is a subtype of kinetic or verbal apraxia. It often occurs following damage to Broca's area (retrieved from http://www.answers.com/topic/apraxia on May 2, 2010). Usually apraxia of speech doesn't occur with aphasia. Notable symptoms of speech apraxia are "dysfluent articulation problems", as they grope to posture correctly sequential tongue, lip, and jaw movements during speech activities, numerous but "variable articulatory errors" characterized by false starts, re-starts, sound substitutions, sound and word repetitions, and overall slow rate of speech (Retrieved from http://www.answers.com/topic/apraxia on May 2, 2010).

Existing Models of Apraxia

Five diifferent neuropsychological views has been observed for apraxia. They are:

Liepmann's Model – This model dates back to 100 years ago. He belived to execute a movement , the spatio temporal image of the movement is transformed in to "innervatory patterns" that yield "positioning of the limbs according to directional ideas" (Jacobs et al.,1999 as cited in Petreska et al., 2007). Liepmann distinguished between 3 forms of apraxia namely, "ideational" . "ideomotor" and "limb kinetic."

Disconnection Model by Geschwind – This model says that verbal command for movement is comprehended in Wernicke's area and transferred to the ipsilateral motor and premotor areas that control the movement of the left hand (Clark et al.,1994 as cited in Petreska et al., 2007). This model cannot explain impaired immitation or impaired object use since they do not require a verbal command (Rothi et al.,1991 as cited in Petreska et al.,2007).

Representational Model by Heilman and Rothi (1993) - According to this, apraxia is a gesture production deficit that may result from the destruction of the spatiotemporal representations of learned movements stored in the left parietal lobule. The model attempted to distinguish between dysfunction caused by destruction of parietal lobes and deficit which would result from the disconnection of the parietal areas from the frontal motor areas (Heilman et al.,1982 as cited in Petreska et al.,2007). This explains why patients with a gesture production deficit with anterior and posterior lesions perform differently on tasks of gesture discrimination, gesture recognition and novel gesture learning.

Cognitive Model of Limb Praxis by Roy and Square (1985) – It basically involves two limb systems namely the conceptual system and production system (Petreska et al.,2007). This model predicts three patterns of impairement (Heath et al.,2001 as cited in Petreska et al.,2007) namely "pantomime deficit" , "deficit in imitation" and "concurrent impairement" (Petreska , et al., 2007, p.67).

Multi – modular Model by Rothi (1991) - To address the issue of modality specific dissociation themulti modular model has input that is selective according to the modality, a specific "action semantics system" dissociable from other semantics system,

an "action reception lexicon" that communicates with an "action production lexicon" and a separate "non lexical route" for the imitation of novel and meaningless gestures (Rothi et al., 1997 as cited in Petreska et al.,2007,p.67).

This model was extended by Cubelli in 2000 and two new dimensions "visuomotor conversion mechanism and "gestural buffer" were added. Buxbaum and others (2000) further extended the model based on their observation. According to them, a unitary set of representations named "body schema" calculates and updates the dynamic positions of the body parts relative to one another. Importantly, this is a common processing stage between "lexical" and "non lexical route" and hence serves meaningful and meaningless actions.

Existing models of apraxia fail to account for differential performance in imitation of hand postures and finger configuration (Goldenberg and Hagmann.,1997 ac cited in Petreska et al.,2007,p.69). Moreover, in a study of ideomotor apraxia data was provided which was compatible with the influential "mirror neuron hypothesis" (Buxbaum et al.,2005 as cited in Petreska et al.,2007,p.69). Apraxia models cannot easily be reconciled with this hypothesis which is based on the neurophysiological observations from the monkey brain (Rizzolatti and Craighero.,2004 as cited in Petreska et al.,2007,p.69). The representations in "mirror neuron hypothesis" serve action recognition and action production tasks, thus it can be said that perception of movement is constrained by its executional knowledge (Petreska, et al., 2007,p.69). Realated to apraxia, the "mirror neuron hypothesis" questions the seperation of "input" and "output lexicon" (Koski et al.,2002 as cited in Petreska et al.,2007,p.69).

Laterality of Lesion in Apraxia

Several studies have failed to find a consistent association between the locus of lesions within a hemisphere and the severity of apraxia (Basso et al.,1980 as cited in Petreska et al.,2007,p.70). Moreover areas in apraxia can also be damaged in non apraxic patients (Petreska et al.,2007,p.70). Apraxic deficits are most common after a parietal and frontal lesion however, apraxic deficits were also observed temporal, occipital and subcortical damage (Goldenberg.,1995 as cited in Petreska et al.,2007,p.70). Moreover, lesion in ideomotor and motor apraxias were observed following lesions in left inferior parietal and the left dorsolateral frontal lobes (Haaland et al.,2000 as cited in Petreska et al.,2007,p.70). For example, several studies suggested that angular and supramarginal gyri of the inferior parietal lobule are critical in visuo imitative apraxia (Goeldenberg et al., 2001 as cited in Petreska et al., 2007, p. 71) and ideomotor limb apraxia (Haaland et al.,1999 as cited in Petreska et al., 2007,p.71).

Furthermore, premotor lesions i.e. lesions till supplementary motor area were affected by bimanual actions (Halsband et al., 2001) and transitive actions (Watson et al., 1986).

It has been difficult to disentangle between the specific contributions of the parietal and frontal lobe and frontal lobe cortices as lesions in these areas lead to similar deficits

(Haaland et al., 1999). For instance, target and spatial errors are related to posterior lesions only (Goldenberg and Karanath,2006) though internal hand configuration errors were present in patients with and anterior and posterior lesions (Haaland et al.,2000). Moreover, patients with posterior lesions only had problems in discriminating between correctly and incorrectly performed actions and in recognizing appropriate hand postures (Halsband et al., 2001).

Apraxia can also develop following subcortical lesions (Graham et al., 1999). Though in this case it has two possibilities whether apraxia originates from lesions in basal ganglia which are well connected to superior parietal lobe, premotor and supplementary motor areas (Jacobs et al., 1999) or from the fronto parietal connections which form the white matter (Pramstaller and rsden.,1996).

Failure to find clear correlations between specific lesion loci and different apraxic deficits argues for a widespread cortical and subcortical representation of praxis, distributed across specialized neural systems (Leiguarda and Marsden,2000). However it is believed that that a selective damage to one of the systems may produce a particular pattern of errors tightly related to a subtype of apraxia.

Complex Nature of Apraxia

Apraxia refers to the impairment of Praxis system after a brain lesion. Studies on apraxia have separately tackled the faulty execution of many types of gestures of various end effectors in different types of modalities (Petreska et al., 2007,p.74). The multidimensional aspect of the varying parameters has given rise to the problem of systematicity in apraxia assessment. Moreover, it poses a problem in the coherent interpretation of the disorder.

It is very likely that mechanisms of imitation and execution of movements vary according to the type of action that is imitated or executed (Schnideret

al.,1997). This points out to the fact that different categories of action require the use of separate systems at some stage of processing, but the level of separation between the representations behind each kind of action is still very unclear. Petreska and others have argued for understanding of a particular gesture in terms of brain processes and resources when compared to other gesture.

There is also a cloud of misunderstanding between meaningful and meaningless gestures. The reproduction of a recognized meaningful gesture appear to be entirely based on the internal representation of a gesture though the knowledge of a learned skill is preferable retrieved from memory than being constructed (Halsband et al.,2001). However, reproduction of a meaningless gesture involves a close visual tracking of the imatee's body configuration and is modeled by a "body schema" (Buxbaum et al.,2000).

The double dissociation between imitation of meaningful and meaningless gestures points out towards the existence of separate processing systems which is not addressed by any of the existing models of apraxia. Meaningless actions involve novel motor sequences that can be analyzed and constructed from the existing movements (Koski et al.,2001).

Identifying the overlap of these processes can provide a clearer framework for interpreting the patient's performance and would simplify the analysis of the lesion correlates. Similarly, kinematic measures of pointing movement was correlated to gesture imitation, suggesting kinematic deficits observed during pointing movements are generalized to more global aiming movements (Hermsdorfer et al., 2003). Accordingly, gesture imitation is to believed to depend upon the same cognitive mechanisms as reaching and grasping (Haaland et al.,2000), however the level and extent of interplay is not clear. There is a possibility of the underlying representations being componential. For example, separate hand posture and representations for transitive gestures (Buxbaum et al.,2005).

Overcoming the complexity of Apraxia

One way to cope with the complex nature of apraxia is to become even more precise and systematic in assessing apraxia. Unfortunately, the qualitative measures of the errors such as kinematic measures of the movement trajectory are frequently missing or given in a pure statistical fashion. Petreska and others believe that the inability to distinguish between different types of errors related to different types of gestures has prevented the researchers till now to discover the precise neuroanatomical correlates of apraxia to accurately identify the brain lesion in apraxia. Thus the types and analysis of apraxic errors need to be improved. Petreska and colleagues suggest extensive categorization of errors and their characteristics via kinematic methods. Moreover, errors should be reported in relation to the exact movement and not only specific condition tested.

It is also put forward that apraxia assessment should try to integrate more tasks of motor learning as apraxics may also be deficient in learning new motor tasks (Heilman et al.,1975).

Furthermore, modeling research may prove to be very useful to gain some insights into the details and potential implementation of human praxis. The differences and similarities between reaching to body centered versus peripersonal cues would become evident through the development of corresponding algorithm as they would be explicitly computed. Computational models of motor control in humans and robots often provide solid foundations that can help us to ground the vast amount of neuro scientific data that is collected today (Schaal and Schweighofer,2005).

Petreska and Billiard.,2006 gave a neurocomputational model that accounts for the colossal apraxic deficits observed in a seminal experimental study of imitation of

meaningless gestures (Goldenberg et al.,2001). In a neurocomputational model one has to take into account the computational principles of movement that reproduce the behavioral and kinematic results of the patient.

The model of Petreska basically combines two computational methods for unsupervised learning applied to a series of neural networks. The model suggests neuroanatomical substrates for the performance of apraxic patients in all conditions of Goldenberg's experiment. Petreska and colleagues believe that their model is compatible with the view of integrating knowledge from different lines of research and also happens to be a good way to probe our understanding for apraxia.

Diagnosis of Apraxia

During testing, patients are asked to do or imitate commonly learned tasks such as saluting, combing hair, opening a lock with a key, striking and blowing out a match, stopping or starting to walk, using a screwdriver or scissors, inhaling deeply and holding a breath. Additionally, Strength and range of motion can be assessed to exclude motor weakness and musculoskeletal abnormalities as symptoms. Neuropsychologic testing or assessment may help identify more subtle apraxias.

Caregivers should be asked about the patient's ability to do activities of daily living, especially those that involve household tools (eg, correct and safe use of eating utensils, toothbrush, kitchen utensils to prepare a meal, hammer, and scissors) and writing.

Brain imaging is required to diagnose and characterize central lesions (eg, infarct, hemorrhage, mass, focal atrophy).

Neuropsychological tests of sensory perceptual and motor functions are typically administered as part of more extensive batteries, providing evaluation of tactile, visual , auditory and motor functions. The most widely used of these tests are components of Halstead-Reitan or Luria-Nebraska batteries. These tests are administered to detect impairement of vision, touch , movement or hearing with brain dysfunction. They are of particular value in lateralization of brain lesion accompalished by comparing the two sides of the body or visual fields (Goldstein and Beers , 2004, p.318).

Treatment

There is no specific medical treatment. Physical and occupational therapy can improve functioning but is more often useful for making the environment safer and for providing devices that help patients circumvent the primary deficit.(Goldenberg 1998; Sundet 1988). Research into the different

therapeutic interventions available to treat apraxia is limited.

Types of interventions include: • strategy training in daily living activities: this technique teaches internal (for example, the patient is taught to verbalise and implement the task steps at the same time) or external (for

example, when aids are used to overcome a functional barrier) compensatory strategies that enable a functional task to be completed.

These strategies will not have been used prior to the stroke (Van Heugten 1998)

• sensory stimulation: stimulations including deep pressure, sharp and soft touch are applied to the patients' limbs (Butler 1994);

• proprioceptive stimulation: the patient leans on and puts weight through their upper and lower limbs;

• cueing, verbal or physical prompts: given to enable each stage of the task to be completed;

• chaining (forward or backward): the task is broken down into its component parts. Using backward chaining the task is completed with facilitation from the therapist apart from the final component, which the patient carries out unaided. If successful next time further steps are introduced. Forward chaining is the reverse of backward chaining;

• normal movement approaches: the therapist facilitates the

body through normal movement patterns

Conclusion

The inability to execute purposeful and previously learned motor tasks, despite physical ability and willingness, is called Apraxia. This is a manifestation of brain damage. Diagnosis is clinical, often including neuropsychologic testing, with brain imaging (eg, CT, MRI). Prognosis depends on the cause and extent of damage and patient age. There is no specific treatment, but rehabilitation, may improve the patient's condition.