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Neuropsychological Deficits in Initial Parkinson's Disease

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1. Introduction

Parkinson's disease (PD), is a neurodegenerative illness, producing movement disorders, although it is also associated to cognitive deficit and emotional and behavioral alterations (Fuiza & Mayán, 2005; Vera-Cuesta et al., 2006), being prominent the presence of neuropsychological deficits among the majority of PD patients.

The neuropsychological disorders can be observed from the very initial phases of the illness, nevertheless, the results obtained from studies of these deficits are often confusing and little clear. These contradictory results are due to diverse factors, which mainly are: the heterogeneity of the samples used in the studies, the absence of consensus during the tests and to the lack of clarity at the time of using diverse terms as specific cognitive alterations, mild cognitive impairment and dementia.

During the last years there is an increased interest in studying and describing cognitive deficit associated with PD (Aarsland et al., 2003; Giannaula, 2010; Locascio, Corkin & Growdon, 2003; Ostrosky-Solís, 2000; Perea-Bartolomé, 2001; Weintraub et al., 2004). This interest is justified if keeping numbers in mind. It is saying, the totality of PD patients show alterations from initial phases in tasks requiring attention, visuospatial, mnesic o executive functions. Also, about 20% to 30% of the patients with PD reach dementia along the evolution of the illness (Caixeta & Vieira, 2008; Halvorsen & Tynes, 2007; Hilker et al, 2005).

It has been emphasized in diverse studies that the diagnosis of dementia in PD is often underestimated (Caixeta & Vieira, 2008; Halvorsen & Tynes, 2007; Hobson & Meara, 2004). Therefore, long term studies to be carried out with such patients will be needed in order to evaluate the type and scope of cognitive deficit associated with the PD. Caviness et al. (2007) carried out a study among 86 patients, showing the presence of signs of cognitive deterioration. In particular, they found that 62% of the participants were cognitive intactly, 21% were fulfilling criteria of mild cognitive impairment and only 17% had symptoms compatible with dementia according to DSM IV. The study results also proved that patients with mild cognitive impairment were characterized for having, principally, executive deficit and alterations of memory, especially in tasks of free delayed memory and working memory. Therefore, the above mentioned study emphasizes the need to detect signs of cognitive deterioration in PD patients from the very beginning of the illness, that is to say, to detect the early neuropsychological deficits, and to supervise the evolution and course of these cognitive deficits.

In the same line, Taylor et al. (2008) carried out a longitudinal study in a sample of newly diagnosed patients of PD, analyzing the relation between the appearance of dementia depending on variables such as alterations to movement and attention related deficit. They found that patients suffering major postural instability since the beginning of the illness, and those with attention deficits, had a significant probability of developing dementia. All of these patients had deficits in tasks such simple reaction time tasks, tasks of election and of attention related control. In addition to the executive, attention related and mnesic deficit, as possible risk factors of the development of dementia in PD patients (Taylor et al., 2008). It has been proved that other factors exist, precipitating the development of dementia, such as: alteration of the visuospatial function, advanced age, increased development of movement related symptoms, presence of emotional alterations (depression and anxiety) and psychotic disorders (Halvorsen & Tynes, 2007).

It has been observed that the prevalence of dementia depended on the studied population, on the definition of dementia elected and on the methods used to diagnose it (Caviness et al., 2007). The dementia observed in PD patients usually appears approximately in 20-30% of them, being more frequent in those with major age, depressive symptoms, and in those with severe movement related deficit (Vera-Cuesta et al., 2006).

But it is important that various recent investigations on neuropsychological deficit in PD have verified that the most frequent cognitive alteration in the debut of the illness it is not the presence of a mild cognitive impairment, nor of dementia, but the presence of one or several cognitive deficits which do not affect the complex and instrumental activities in daily life (Elgh et al., 2009; Giannaula, 2010; Halvorsen y Tysnes, 2007; Verbaan et al, 2007; Zgaljardic et al. 2003). The above mentioned cognitive deficits without interference in the activities of the daily life have been found from initial phases in patients de novo, that is to say in those without pharmacological treatment. In particular, the studies emphasize on the presence of specific cognitive deficit of executive type (deficit in planning, sequence, abstract reasoning and verbal fluency), visuospatial deficits and, also, difficulties in the mnesic function have been found in the delayed recall of information and in certain aspects of the implicit memory (Higginson et al., 2005; Kemps et al., 2005; Locascio, Corkin & Growdon, 2003; Muslimovic et al., 2005).

On the other hand, through neuroimaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), it has been proved that the deficit in executive functions and in working memory observed in the initial phases would depend partly on the alteration of frontostriatal circuits that connect the basal ganglions with the prefrontal dorsolateral cortex (Bruck et al., 2005; Pillon et al, 2000). That is to say, in patients in initial phases a hypo-activation of prefrontal dorsolaterals areas have been found during execution of tasks of executive function (Caballol, Martí & Tolosa, 2007; Castiello et al., 2009). Recent studies have found that these executive deficit partially decline with restoring dopaminergic medication, which has led to suggest the importance of the dopaminergic system in the genesis of this deficit (Nobili et al, 2009). These finds suggest that the neuropsychological deficits observed in initial phases of the illness must be identified and treated as soon as possible, since these measures might help to delay the appearance and severity of cognitive deterioration and they would also improve the quality of life for patients (Halvorsen & Tynes, 2007; Mamikonian et al, 2009; Zgaljardic et al., 2003). The study and management of cognitive alterations, with behavioral and emotional disturbances, is determinate because implies a serious limitation in the execution of daily activities for patients, as well as in their social and familiar relations (Dubois et al, 2007).

The aim of the present chapter is to describe the main neuropsychological alterations from initial phases of the illness in PD patients. In particular, we will focus on the deficits produced in attention, visuospatial, executive and mnemonic functions. We will comment on the results obtained in several studies based on neuropsychological tests, related to the fronto-subcortical cognitive impairment in patients in initial phases of the illness, according to the scale of Hoehn and Yahr (Fahn, 1967). Also, we will give a few small recommendations on how these deficits can be rehabilitated, that is to say, on how to carrying out the necessary neuropsychological intervention in order to postpone the appearance and severity of cognitive disorders and thus to improve the quality of life of patients.

2. Main neuropsychological deficits in initial Parkinson's disease

2.1 Attention deficits

Attentional deficits in patients with PD are related from the initial stages of the disease, and affect tasks involving simple reaction times and those that require attentional control and, therefore, require a flexible distribution of attentional resources (Caviness et al, 2007). That means, in these patients there is an increase in reaction time and a general slowing of information processing, a typical phenomenon observed in the PD bradyphrenia. This slowness may be increased due to a consequence of any extra effort of the cognitive processes involved, and of the association of complex of motor and cognitive responses, and finally this slowness may be caused also by the process of selecting the appropriate response (Caviness et al, 2007, Nobili et al, 2009; Ostrosky-Solís, 2001).

It has been suggested that, through the connections between the basal ganglia and prefrontal cortex, basal ganglia will regulate the main functions of this part of the cerebral cortex, based on the anterior and posterior attentional systems (Posner & DiGirolamo, 1998). That means, the *posterior attentional network*, which depends on the posterior parietal cortex, superior colliculus, the pulvinar thalamic nuclei and inferior temporal lobes, control the focused attention, visual orientation and recognition of objects and attributes located in space. However, the *anterior attentional network*, which depends on the proper functioning of the frontal lobe and cingulate areas, would be responsible for selective attention, attentional control and the initiation and inhibition of responses (Posner & DiGirolamo, 1998). From early stages of the disease, there is a change in both attentional systems (Alonso et al., 2003), especially in the anterior attentional system (Lewis et al, 2003). Patients had deficits in shifting attention to different regions of visual space, difficulties in directing the attentional system into different areas of the environment and to manage these resources based on the objectives. Generally, we can say that patients, from initial stages, show alterations in shifting the focus of attention and in inhibition of both motor and cognitive programs in a short time interval (Alonso et al, 2003).

In our research with patients in early stages (Muñiz Casado & Rodríguez Fernández, 2007), we observe that attentional tasks are affected since the debut of the disease in line with the results obtained by Owen (2004), Owen et al. (1992) and Zgaljardic et al. (2006). More specifically, we found out that patients have a *general slowing of information processing* (Caviness et al., 2007, Nobili et al., 2009; Ostrosky-Solís, 2001) that, in our case, is evaluated through:

- increasing the time spent in the copy of the Rey-Osterrieth Complex Figure Test;

- more time than the controls participants in the TMT (Trail Making Test) performance, both Part A and Part B;
- less scores in the Digit Symbol subtest of the WAIS (Wechsler Adults Intelligence Scale), and
- decrease the speed of auditory information processing in the test PASAT (Paced Auditory Serial Addition Test).

All these changes indicate that since the onset of the disease, there is decrease in the speed of information processing.

On the other hand, if we look at the performance on the Digit Span subtest of the WAIS (Wechsler Adults Intelligence Scale), we found significant differences in the backward and the total score. That means that our patients have an attentional span similar to the one of the control group participants, but have more difficulties in the inverse series, which may point to a *deficit in the distribution of attentional resources*. Furthermore, using the Stroop test, patients with initial PD have a lower score than controls in reading words and reading the color film, probably because of the processing speed deficit, already discussed, but also because of a deficit in *attentional control to inhibition the incoming information that is not relevant to the task* and the resistance to a new task, since we found significant differences in the Stroop interference. These data are consistent with those of Owen (2004), who found in newly diagnosed patients the presence of attentional deficits in tasks involving selective control and inhibition of irrelevant information, such as in the Stroop test.

The deficits of attentional control in initial PD have been demonstrated using different tests. On one site, the *problems presented in the cognitive flexibility* to produce fewer scores than the controls on the PASAT (Paced Auditory Serial Addition Test). Through the PASAT we can appreciate a deficit in the flexible distribution of resources at the time to sustain attention throughout the test, which brings us to conclude that our patients have *difficulties in sustained attention tasks* that require a flexible distribution of attentional resources. On the other site, the mistakes made in Part B of TMT (Trail Making Test), show alterations in their ability to alternate between two sequences, a fact that does not happen in Part A of this test in which there is a simple sequence and patients do not make mistakes, having a similar performance to that of the controls. In addition, our patients had fewer scores than the controls in a cancellation task, which shows: a *deficit in changing the focus of attention* to different regions of visual space, alterations in directing the attentional system into different areas of the space and a *challenge to manage these resources* based on the demands required. On the other hand, the sustained attention task CPT (Continuous Performance Test), although without significant differences in the hits relative to controls, patients make more false positives, which suggests a difficulty of stopping and controlling unsuitable responses and even some difficulty in keeping the attention on a monotonous task like this.

As a summary, as other authors have found (Caviness et al, 2007, Alonso et al, 2003; Fuiza & Mayán, 2005; Owen, 2004), from the initial stages, our patients show alterations in attentional focus change and inhibition of both cognitive and motor programs. If we focus on this last point, we have observed in the different sections of a test GO / NO GO, which are the change of hand position and point of opposing reactions, those are significant differences although are not statistically significant.

About cognitive **rehabilitation** of attention is important that the patient, firstly, choose the time and place to enable him to be concentrate and free from stimuli that may interfere, and create a daily routine of mental stimulation. To improve the processing speed issues it is

recommended to monitor the execution time of the exercises of attention, so this will allow him to compare his own performance on different occasions (using the same exercises at first and then similar tasks but with different stimuli).

In order to improve *sustained attention*, the patient will increase the duration of the exercises, beginning with short exercises, and as he feels less fatigue and reduce mistakes at the end of the exercise, he will progress to longer duration exercises. That means, when the patient will reduce his mistakes, he can gradually increase the complexity of the task.

To improve his *control attentional* ability in tasks involving selective and divided attention it is recommended that he starts with exercises that involve the detection of a single stimulus and it is different from the distracting ones, this way he is working his *selective attention* at a simple level. Once dominated the ability to choose a stimulus among several, it will progressively increase the difficulty to find a stimulus among several ones so similar and even will increase the number of stimuli to be search and the number of distractors. At last, to improve the patient's *divided attention*, once able to concentrate in a relax environment (sustained attention), he needs to direct his attentional focus inhibiting other stimuli (selective attention), the next step is when the patient perform two simple similar tasks simultaneously but in different sensory modalities. For example, a visual cancellation task with an auditory selective attention task, and then little by little, it will increase the degree of difficulty.

2.2 Visuospatial and visuoperceptual deficits

From the beginning of the illness the patients present visuospatial deficits in tasks of spatial location, implying dimensional positioning of objects and integrating them coherently in the space (Galtier et al., 2009; Levin, Tomer & Rey, 1992; Owen, 2004; Sánchez-Rodríguez, 2002). Functional neuroimaging studies have found a metabolic reduction in the frontal and parieto-occipital cortex in initial PD patients, associated with the poor execution of visuospatial tasks (Kemps et al., 2005).

The same studies (Levin, Tomer & King, 1992; Kemps et al., 2005) show that the facial recognition is the visual perceptive function which is the earliest that became affected. Also, the studies emphasize on deficit in visuoconstructive tasks like the copy of the Rey-Osterrieth Complex Figure Test (Cooper et al., 1991; Owen, 2004). This alteration becomes clearer with the further illness evolution (Caixeta & Vieira, 2008). The patients showed deficit in some of the subtests of the manipulative scale according to WAIS (Wechsler Adults Intelligence Scale), such as: picture completion, blocks design and object assembly (Levin, Tomer & Rey, 1992; Dubois & Pillon, 1997).

Regarding the visuoperceptual and visuospatial deficits in their patients in diverse stages of the illness, Levin, Tomer and King (1992) came to the following conclusions:

- With regard to the control group initial patients were showing similar results in tasks evaluating the skills to carry out mental rotations of objects and reconstruction of pieces to form a meaningful object. Meanwhile PD patients in groups of moderate and advanced evolution of the disease were presenting deficits in these areas. Also an increase in the deterioration of the judgment of linear orientation was observed in patients with more years of PD evolution.
- Facial recognition and the constructive praxis of drawings and complex models were altered from the beginning of the illness and deteriorate as the illness develops.

- The patients of the initial group presented worse results in the face recognition with regard to the control group. Nevertheless, it is necessary to consider such a task, in addition to evaluating perceptive and facial components as they are discriminated by different characteristics of positioning and shades or the vision of dimensional contrast between different aspects, it also implies analytic skills, based on the reasoning and judgments of deduction, which may be depended on some executive aspects (Possin et al., 2008). Certain studies have found complex visuospatial alterations when executive aspects increase, such as planning, sequences and generation of a movement plan (Kemps et al., 2005; Possin et al., 2008; Rudkin, Pearson & Logie, 2007).

The studies that we are carrying out coincide in this sense with the majority of previous investigations, which have demonstrated that the facial recognition is the first visual perceptive function that is altered from the debut of the disease (Levin, Tomer & Rey, 1992; Kemps et al, 2005). In particular, our patients obtained worse results than the control group in all the components of Benton's facial recognition test, presenting a deficit in the specific skill to identify and discriminate unfamiliar human faces.

Also we have found difficulties in visual constructive tasks such the copy of the complex figure of Rey-Osterrieth, as well as other studies (Cooper et al., 1991; Owen, 2004). Nevertheless, these studies were including patients of initial and moderate stages, and our study was including only initial patients. We have found that they present alteration in the copy of the Rey-Osterrieth Complex Figure Test that, which implies visualperceptive and visualconstructive components in addition to executive elements.

Finally, in the picture completion of subtest of WAIS (Wechsler Adults Intelligence Scale) our patients present deficit in the perception of difference in relevant details from the irrelevant ones and in the visual organization, results that could stand in the same line as the found by other authors (Levin, Tomer & Rey, 1992; Dubois & Pillon, 1997; Owen, 2004).

To **rehability** visuoperceptual, spatial and constructive deficits of patients, we propose, first of all, to assure an improvement in their attention related functions. For the rehabilitation of the *visuoconstructive alterations*, the recommendation is to start fine motricity exercises, in which the patient with a colored pencil follows numerated points of a drawing until finally managing to complete all the points and to identify the drawing. The aim is to start with simple and two-dimensional drawings, getting to complex drawings and three-dimensional figures. Next, the patient will be proposed to reproduce drawings of different grade of difficulty, firstly with a patter and later without it. Finally, he will be asked to complete incomplete drawings or such containing perceptive intrusive elements.

To improve his *visuoperceptive capacity*, he will be shown incomplete drawings, and he will have to identify the lacking element. On the other hand, he will be shown fragments of drawings and before the drawing is completed, the patient will have to identify it from the incomplete degraded versions of it. He will be shown stimuli in different tonalities of color and grades of lighting, so that he could identify them correctly. To increase even further his dimensional skills, he will be presented tasks in which he will have to indicate the position of the object, or tests in which he would have to mentally rotate drawings to identify them perceptively.

2.3 Executive function deficits

In many cases the deficits found at the beginning of the PD in executive functions has explained the changes in other cognitive areas (Caviness et al, 2007, Halvorsen & Tynes, 2007, Lewis et al, 2003, Nobili et al, 2009; Poussin et al., 2008, Vera-Cuesta et al., 2006).

Executive functions have been considered as those higher cognitive processes which associate ideas, movements and actions to execute complex behaviors and enable human adaptation to its environment (Fuster, 2007; Stuss & Alexander, 2000; Tirapu-Ustárrroz, Muñoz-Céspedes & Pelegrin-Valero, 2002). From the neuroanatomical point of view, it has been associated mainly to the dorsolateral prefrontal cortex (Sawamoto et al., 2007; Tinaz, Schendan & Stern, 2008; Tirapu-Ustárrroz & Muñoz-Céspedes, 2005). Therefore, the dorsolateral circuit is related to executive functions as planning, manipulation of information in working memory, concept formation and mental flexibility (Caviness et al., 2007). However, throughout the prefrontal cortex and its various cortical and subcortical connections (mainly with the basal ganglia) will influence the functioning of executive functions.

Using the Wisconsin Card Sorting Test (WCST) has been found a reduction in the number of categories produced by these patients from initial stages, and a high number of perseverative mistakes (Cooper, Sagar & Sullivan, 1993, Dubois et al., 2007; Lewis et al., 2003; Ostrosky-Solís, 2000, Vera-Cuesta et al., 2006). PD patients have difficulties with the organization, the management and the replacement of some concepts with others more innovative and adaptive, so that show *problems in guided tasks only with internal search key*, highlighting the *difficulties in developing their own strategies* which guide their own behavior, causing a deficit in the generation of new concepts or sets, in the change and planning for them and in the search for the rule involved in the solution of the task (Mckinlay et al., 2008).

On the other hand, we know that executive control is concerned with: planning, control of the plans involved in the task, monitoring the inhibition of irrelevant information and to manage mental flexibility to change the focus of attention. These cognitive functions are involved in tasks such as: the TMT (Trail Making Test), verbal fluency task, the Mental Control subtest of the WMS (Wechsler Memory Scales), the Stroop test and the PASAT (Paced Auditory Serial Addition Test). Patients with initial stages of PD show alterations in most of these tests (Bruck et al., 2005, Lewis et al. 2003, Williams-Gray et al., 2007). Thus there have been found evidence of deficits in the maintenance of a sequence in a flexible way, as it happens in the Trail Making Test (Cools et al., 2001, Williams-Gray et al., 2007).

It has been also found that from early stages, PD patients often have difficulties in verbal fluency task, of both phonological and semantic nature, which means that their production of words, compared to controls, is fewer and they use less access strategies to the lexical and semantic stores, showing a lack of monitoring of information. These results suggest that the alteration of verbal fluency, found in patients with *de novo*, involves a dysexecutive impairment more than a problem of semantic memory (Henry & Crawford, 2004, Williams-Gray et al., 2007; Zec et al., 1999).

Our results have shown, once again, that patients from initial stages show difficulties in executive tasks. In brief, our patients have deficits in executive processes as following:

- Mental flexibility and capacity inhibition to the interference of other not relevant issues (statement based on the results of the Stroop test).
- Generation of new concepts, abstract reasoning and change of set (according to the results of the Wisconsin Card Sorting Test (WCST) and the Similarities subtest of the WAIS (Wechsler Adults Intelligence Scale)).
- Ability to initiate and maintain a verbal task, sequencing and planning the execution of it (according to the results of the verbal fluency task (semantic and phonetic)).

- Ability to plan strategies and to solve problems (evidence obtained from the results of the Hanoi Tower and in the copy of the Rey-Osterrieth Complex Figure Test).

To **rehabilitation** his executive functions, firstly it is recommended to improve the patient's ability to *initiate the execution of tasks by himself*. For this, it is proposed to improve the occurrence of new ideas or the skill to invent a short story, thinking of dishes that normally do not cook or drawing pictures without a model.

In order to stimulate the categorization and abstract reasoning is suggested to perform tasks involving sorting and classifying items into categories, such as classifying countries by continents, organizing the closet by clothes for different seasons or preparing shopping list according to different types of food. To stimulate the *mental flexibility* and the *regulation of behavior*, it can be proposed different table games like cards or dominoes. With these tasks, the patient not only has to implement his plans and sequence the steps to reach them, but also has to be flexible and able to adapt to other people and circumstances involved in the game, as well as stimulate social aspects such as empathy and other social skills.

Finally, to improve *planning* and *sequencing of steps* to take in a task, we ask him to write and list the necessary steps in order to paint a wall, to prepare a birthday party or a holiday.

2.4 Memory deficits

Often PD patients have memory deficits on tasks involving spatial working memory, implicit learning of sequences, learning pairs of related words and visuospatial learning (Brown et al., 2003, Galtier et al, 2009; Higginson et al., 2005; Verbaan et al, 2007; Zizak et al, 2005).

Regarding to declarative *long-term memory*, patients often reduce performance as the disease progresses, of both episodic and semantic memory (Aarsland, Zaccai & Brayne, 2005; Caballol, Marti & Tolosa, 2007, Lewis et al., 2005). The specific alterations of these patients, especially at early stages, of both verbal and visuospatial material, probably because of a deficit in attentional and organizational control (Pillon et al, 1998, Nobili et al, 2009). It has shown that patients keep their storage capacity but often they have problems with *encoding and recovering of information* due to executive alterations (Janvin et al., 2003).

Several studies show that some aspects of *working memory* are more impaired than others (Higginson et al., 2005, Moustafa, Sherman & Frank, 2008, Williams-Gray et al., 2007). There has been found that patients, in early and moderate stages without dementia, show deficits on tests of visuospatial working memory (Galtier et al, 2009; Poussin et al., 2008), while their performance in a similar test of verbal working memory was preserved (Galtier et al, 2009; Owen, 2004). Therefore, patients show a deficit in *spatial working memory*, and, in part, this alteration reflects an executive deficit rather than a pure memory deficit, since patients *de novo* and without medication can have difficulties in visuospatial tasks involving strategic processes, organization and active manipulation of information stored temporarily (Rudkin, Pearson & Logie, 2007; Sawamoto et al, 2007). However, the Lewis et al. (2003) working team found out deficits in *verbal working memory* task in their initial patients, which involved manipulation of information (to sort submitted letters by certain rules), although the same patients kept intact their execution when only they were asked to maintain and recover the letters.

Regarding to alterations in *implicit memory*, we know that patients can learn new concepts, cognitive and motor, but with remarkable slowness (Owen et al., 1998). Muslimovic and colleagues (2007) suggest that patients have a deficit in implicit tests, which involve a

sequential component like the paradigm of serial reaction time, but are able to learn the skill, although with more difficulty than controls. However, the acquisition of more perceptive skills such as mirror reading or repetitive motor skills that require less planning and organization are usually preserved in patients (Ferraro, Ballot & Connor, 1993). That means that patients, from early stages, often keep preserved their performance over the control participants in the paradigm of priming or facilitation effect (Chenery, Angwin & Copland, 2008; Muslimovic et al., 2007).

According to our results, patients with initial PD have a reduced *short-term memory* compared with controls (evaluated by the Digit Span subtest of the WAIS (Wechsler Adults Intelligence Scale)). Regarding the declarative *long-term memory* (evaluated by TAVEC which is the Spanish version of the CVLT (California Verbal Learning Test)), patients recover fewer words in free recall tasks, in both short and long term, and they benefit less from semantic clustering than the controls. They also have perseverations in the long-term free recall, which may indicate a lack of monitoring when information is recovered. Furthermore they show the phenomenon of interference, in both the free recall test of the intrusive list (proactive interference) and the short-term memory of the first list (retroactive interference), and recognize fewer words than controls. However, we observed no differences in discriminability index, in the response bias, or in the commission of false positives during recognition. Therefore, patients keep what they have learned when distractors appear, and also their hits in the recognition are independent of the chance and are related to what they learned and stored.

Generally, our patients are able to store information and keep it but in a disorganized way. We have noticed that they learn new information but slower and with lower performance level than the controls, that means, they have fewer scores in memory tests, with and without clues, and in the recognition test. Probably, as other authors have found (Stefanova et al., 2001; Weintraub et al., 2004; Verbaan et al., 2007; Zizak et al., 2005), this deficit is due to inefficient use of strategies during the encoding and the storage, which involves a lower recovery because of the inefficiency in the planning and the organization of information. Other studies have found that executive deficits in patients with early PD such as lack of planning, the use of strategies and the phenomenon of interference, together with working memory deficits (as found in test and subtest Letters and Numbers of the WAIS (Wechsler Adults Intelligence Scale)), could be the main reason of some of the alterations seen in short term and long term memory, in verbal and visual modalities (Cooper et al., 1991; Muñiz Casado & Rodríguez Fernández, 2007; Pillon et al., 1998)).

We have found in our initial patients a deficit in verbal working memory, using the subtest Digit Span (in backward form), Letters and Numbers and Arithmetic of the WAIS (Wechsler Adults Intelligence Scale). These results are consistent with those obtained by other authors, testing the verbal working memory, even in patients with newly diagnosed disease (Higginson, 2001; Lewis et al., 2003).

For the **rehabilitation** of the memory functions in patients, and taking into account that many of their difficulties in this area are explained by attention and executive deficits, we propose the following strategies to:

- focus on relevant information.
- associate the information with already known material by the patient.
- organize information to remember through the use of categories at the time of encode and store the information.

- use two different ways, verbal and visual, to recover previously stored information.
- perform exercises in semantic verbal fluency: naming in particular tense examples of a certain category.

3. Conclusions

Cognitive impairment is considered a common feature of this disease. The majority of PD patients have developed some early mild cognitive deficits in the course of their disease. Several studies demonstrated that between 20% and 50% PD patients will develop Mild Cognitive Impairment (MCI), that means, many patients will have executive deficits, memory impairment, visuospatial deficits and other cognitive and emotional symptoms at lower level than it occurs in patients with dementia, but these deficits also damage their daily activities.

Factors such as the patient age at the beginning of the disease, the educational level, the occupation, the presence of emotional disorders, the asymmetry of the symptoms at the onset of illness, relevant motor symptoms, the appropriate response to drug therapy and other types of personal and family history, will influence the cognitive deficits presented by the patient. That is to say, cognitive alterations in these patients are a powerful predictor of their quality of life.

Along the chapter we explain and describe the cognitive deficits in initial stages of PD, and how those deficits should be assessed and treated in order to prevent the appearance of one probably MCI or dementia. Specifically, we describe attentional, visuospatial, executive and memory deficits in early PD. We consider necessary to asses and identify cognitive and emotional deficits in PD patients because those kinds of deficits may cause impairments in the social and daily activities of the patients. Besides, these neuropsychological deficits may be treated and prevented in order to improve the quality of life in those PD patients and, indirectly, the burden of their caregivers will diminish.

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5. References

- Aarsland D, Andersen K, Larsen JP, Lolk A, & Kragh-Sørensen P. (2003). Prevalence and characteristics of dementia in Parkinson disease. An 8-year prospective study. *Arch Neurol*, 60: 387-92.
- Alonso PE, Esteban E, Trujillo MC, Fernández GE, Roussó VT & Cordero EA. (2003). Alteraciones específicas de la atención en estadios tempranos de la Enfermedad de Parkinson. *Rev Neurol*, 36(11):1015 – 18.
- Brown RG, Jahanshahi M, Limousin-Dowsey P, Thomas D, Quinn NP & Rothwell JC. (2003). Pallidotomy and incidental sequence learning in Parkinson's disease. *Neuro Report*, 14, 21-4.

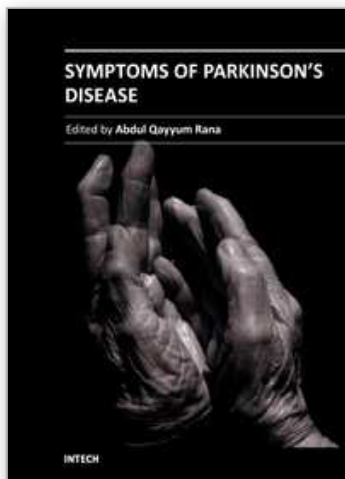
- Bruck A, Aalto S, Nurmi E, Bergman J & Rinne JO. (2005). Cortical 6-[18F]fluoro-L-dopa uptake and frontal cognitive functions in early Parkinson's disease. *Neurobiol Aging*, 26:891-898.
- Butter T, Van den Hout A, Matthews F, Larsen J, Brayne C & Aarsland D. (2008). Dementia and survival in Parkinson disease. *Neurology*, 70, 1017-22.
- Caballol N, Martí M & Tolosa E. (2007). Cognitive dysfunction and dementia in Parkinson disease. *Mov Disord*, 22(Suppl 17): 358-66.
- Caixeta, L. & Vieira, R.T. (2008). Dementia in Parkinson's disease. *Rev Bras Psiquiatr*, 30(4): 375-83.
- Castiello U, Ansuini C, Bulgheroni M, Scaravilli T & Nicoletti R. (2009). Visuomotor priming effects in Parkinson's disease patients depend on the match between the observed and the executed action. *Neuropsychologia*, 47(3): 835-42.
- Caviness JN, Driver-Dunckley E, Connor DJ, Sabbagh MN, Hentz JG, Noble B, Evidente VG, Shill HA & Adler CH. (2007). Defining mild cognitive impairment in Parkinson's disease. *Mov Disord*, 22(9):1272-7.
- Chenery HJ, Angwin AJ & Copland DA. (2008). The basal ganglia circuits, dopamine, and ambiguous word processing: a neurobiological account of priming studies in Parkinson's disease. *J Int Neuropsychol Soc.*, 14(3):351-64.
- Cools R, Barker RA, Sahakian BJ & Robbins TW. (2001). Mechanisms of cognitive set flexibility in Parkinson's disease. *Brain*, 124, 2503-2512.
- Cooper JA, Sagar HJ, Jordan N, Harvey NS & Sullivan EV. (1991). Cognitive impairment in early, untreated Parkinson's disease and its relationship to motor disability. *Brain*, 114, 2095-122.
- Cooper JA, Sagar HJ & Sullivan EV. (1993). Short-term memory and temporal ordering in early Parkinson's disease: effects of disease chronicity and medication. *Neuropsychologia*, 31, 933-49.
- Dubois B, Burn D, Goetz C, Aarsland D, Brown R, Broe G, et al. (2007). Diagnostic procedures for Parkinson's disease dementia: recommendations from the Movement Disorders Society Task Force. *Mov Disord*, 22: 2314-24.
- Dubois B, Pillon B (1997). Cognitive deficits in Parkinson's disease. *J Neurol*, 244, 2-8.
- Elgh H, Domellöf M, Linder J, Edström M, Stenlund H & Forsgren L. (2009). Cognitive function in early Parkinson's disease: a population-based study. *European Journal of Neurology*, 16(12) : 1278-1284.
- Ferraro FR, Balota DA & Connor LT. (1993). Implicit memory and the formation of new associations in nondemented Parkinson's disease individuals and individuals with senile dementia of the Alzheimer type: a serial reaction time (SRT) investigation. *Brain Cogn*, 21, 163-80.
- Fuiza, MJ & Mayán, JM. (2003). Características de la enfermedad de Parkinson. M.J. Fiuza & J.M. Mayán (eds). *¿Qué es el Parkinson? Guía de tratamiento para el lenguaje, el habla y la voz*. Pirámide, Madrid, 2005.
- Fuster, J. M. (2007). Jackson and the frontal executive hierarchy. *International Journal of Psychophysiology*, 64(1), 106-107.
- Galtier I, Nieto A, Barroso J & Lorenzo JN. (2009). Deterioro del aprendizaje visoespacial en la enfermedad de Parkinson. *Psicothema*, 21(1), 21-26.
- Giannaula, R (2010). Alteraciones cognitivas y demencia en la enfermedad de Parkinson. *Revista Neurologia*, 50(1), 13-16.

- Halvorsen, O & Tysnes, OB (2007). Dementia in Parkinson's disease. *Tidssk Nor Laegeforen*, 127(22): 2973-4.
- Henry JD & Crawford JR. (2004). Verbal fluency deficits in Parkinson's disease: a meta-analysis. *J Int Neuropsychol Soc*, 10, 608-622.
- Higginson CI, Wheelock VL, Carroll KE & Sigvardt KA. (2005). Recognition memory in Parkinson's disease with and without dementia: evidence inconsistent with the retrieval deficit hypothesis. *J Clin Neuropsychol*. 27(4): 516-28.
- Hilker R, Thomas A, Klein J, Weisenbach S, Kalbe E, Burghaus L, et al. (2005). Dementia in Parkinson disease. Functional Imaging of cholinergic and dopaminergic pathways. *Neurology*, 65: 1716-22.
- Hobson, P. & Meara, J. (2004). Risk and incidence of dementia in a cohort of older subjects with Parkinson's disease in the United Kingdom. *Mov Disord*, 19:1043-1049.
- Janvin C, Aarsland D, Larsen JP & Hugdahl K. (2003). Neuropsychological profile of patients with Parkinson's disease without dementia. *Dement Geriatr Cogn Disord*, 15, 126-131.
- Kemps E, Szmalec A, Vandierendonck A & Crevits L. (2005). Visuo-spatial processing in Parkinson's disease: evidence for diminished visuo-spatial sketch pad and central executive resources. *Parkinsonism Relat Disorders*. 11(3): 181-6.
- Levin BE, Tomer R, Rey GJ. (1992). Cognitive impairments in Parkinson's disease. *Neurol Clin.*, 10, 471-85.
- Lewis SJ, Dove A, Robbins TW, Barker RA & Owen AM. (2003). Cognitive impairments in early Parkinson's disease are accompanied by reductions in activity in frontostriatal neural circuitry. *J Neurosci*, 23: 6351-6.
- Lewis SJ, Slabosz A, Robbins TW, Barker RA & Owen AM. (2005). Dopaminergic basis for deficits in working memory but not attentional set-shifting in Parkinson's disease. *Neuropsychologia*, 43(6), 823-32.
- Locascio, JJ, Corkin, S & Growdon, JH (2003). Relation between characteristics of Parkinson's disease and cognitive decline. *J Clin Exp Neuropsychol*, 25: 49-109.
- Mamikonian E, Moberg P, Siderowf A, Duda J, Have T, Hurtig H, et al. (2009). Mild cognitive impairment is common in Parkinson's disease patients with normal Mini-Mental State examination (MMSE) scores. *Parkinsonism Relat Disord*, 15: 226-31.
- Martínez-Martín P. (2006). Repercusiones sobre la calidad de vida del deterioro cognitivo en la enfermedad de Parkinson. *Revista de Neurología*, 43: 168-172.
- McKinlay A, Kaller CP, Grace RC, Dalrymple-Alford JC, Anderson TJ, Fink J & Roger D. (2008). Planning in Parkinson's disease: a matter of problem structure?. *Neuropsychologia*, 46(1): 384-9.
- Moustafa AA, Sherman SJ & Frank MJ. (2008). A dopaminergic basis for working memory, learning and attentional shifting in Parkinsonism. *Neuropsychologia*, 46(13): 3144-56.
- Muñiz Casado, J & Rodríguez Fernández, R. (2007). Déficit de memoria en pacientes con enfermedad de Parkinson inicial. *Mapfre Medicina*, 18(1), 39-45.
- Muslimovic D, Post B, Speelman JD & Schmand B. (2005). Cognitive profile of patients with newly diagnosed Parkinson's disease. *Neurology*, 65: 1239-45.
- Muslimovic D, Post B, Speelman JD & Schmand B. (2007). Motor procedural learning in Parkinson's disease. *Brain*, 130(11), 2887-97.
- Nobili F, Abbruzzese G, Morbelli S, Marchese R, Girtler N, Dessi B, Brugnolo A, Canepa C, Drosos GC, Sambucetti G & Rodríguez G. (2009). Amnestic mild cognitive

- impairment in Parkinson's disease: a brain perfusion SPECT study. *Mov Disorders*, 24(3): 414-21.
- Ostrosky-Solis, F. (2000). Características neuropsicológicas de la enfermedad de Parkinson. *Revista de Neurología*, 30(8): 788-796.
- Owen, AM (2004). Cognitive dysfunction in Parkinson's disease: the role of frontostriatal circuitry. *Neuroscientist*, 10(6):525-37
- Owen AM, Doyon J, Dagher A, Sadikot A & Evans AC. (1998). Abnormal basal ganglia outflow in Parkinson's disease identified with PET. Implications for higher cortical functions. *Brain*, 121, 949-65.
- Owen AM, James M, Leigh PN, Summers BA, Marsden CD, Quinn NP, et al. (1992). Frontostriatal cognitive deficits at different stages of Parkinson's disease. *Brain*, 115: 1727-51.
- Perea-Bartolomé, M.V. (2001). Deterioro cognitivo en la enfermedad de Parkinson. *Revista de Neurología*, 32(12): 1182-1187.
- Pillon, B., Ardouin, C., Damier, P., Krack, P. & Houeto, L. (2000). Neuropsychological changes between off and on STN or Gpi stimulation in Parkinson's disease. *Neurology*, 55: 411-418.
- Pillon, B., Bernard, D., Vidailhet, M., Bonnet, A.M., Hahn-Barma, V. & Dubois, B. (1998). Is impaired memory for spatial location in Parkinson's disease domain specific or dependent on "strategic" processes?. *Neuropsychologia*, 36(1), 1-9.
- Posner M.I. & Digirolamo, G.J. (1998). Executive attention: Conflict, target detection and cognitive control. En R. Parasuraman (Ed.), *The attentive brain* (pp. 401-423). Cambridge: MIT Press.
- Possin KL, Filoteo JV, Song DD & Salmon DP. (2008). Spatial and object working memory deficits in Parkinson's disease are due to impairment in different underlying processes. *Neuropsychology*, 22(5), 585-95.
- Rudkin SJ, Pearson DG & Logie RG. (2007). Executive processes in visual and spatial working memory tasks. *Q J Exp Psychol*, 60(1), 79-100.
- Sánchez-Rodríguez, J. L. (2002). Déficit neuropsicológicos en la enfermedad de Parkinson. *Revista de Neurología*, 35(4), 310-317.
- Sawamoto N, Honda M, Hanakawa T, Aso T, Inoue M & Toyoda H. (2007). Cognitive slowing in Parkinson disease is accompanied by hypofunctioning of the striatum. *Neurology*, 68, 1062-8.
- Stefanova, E.D., Kostie, V.S., Ziropadja, L.J., Ocic, G.G. & Markovic, M. (2001). Declarative memory in early Parkinson's disease: Serial position learning effects. *Journal of Clinical and Experimental Neuropsychology*, 23(5), 581-591.
- Stuss, D. T., & Alexander, M. P. (2000). Executive functions and the frontal lobes: A conceptual view. *Psychological Research*, 63, 289-298.
- Taylor JP, Rowan EN, Lett D, O'Brien JT, Mckeith IG & Burn DJ. (2008). Poor attentional function predicts cognitive decline in patients with non-demented Parkinson's disease independent of motor phenotype. *J Neurol Neurosurg Psychiatry*, 79(12): 1318-23.
- Tinaz S, Schendan HE & Stern CE. (2008). Fronto-striatal deficit in Parkinson's disease during semantic event sequencing. *Neurobiol aging*, 29(3), 397-407.
- Tirapu-Ustárriz J, Muñoz-Céspedes JM. (2005). Memoria y funciones ejecutivas. *Rev Neurol*, 41, 475-484.

- Tirapu-Ustárrroz, J., Muñoz-Céspedes, J. M., & Pelegrín-Valero, C. (2002). Funciones ejecutivas: Necesidad de una integración conceptual. *Revista de Neurología*, 34(7), 673-685.
- Vera-Cuesta, H. Vera-Acosta, H., Álvarez-González, Fernández-Maderos, I. & Casabona-Fernández, E. (2006). Disfunción frontal en la enfermedad de Parkinson idiopática. *Revista de Neurología*, 42(2): 76- 84.
- Verbaan D, Marinus J, Visser M, Van Rooden SM, Stiggelbout AM, Middelkoop HA & Van Hilten JJ. (2007). Cognitive impairment in Parkinson's disease. *J Neurol Neurosurg Psychiatry*, 78 (11): 1182-7.
- Weintraub D, Moberg PJ, Culbertson WC, Duda JE & Stern MB. (2004). Evidence for impaired encoding and retrieval memory profiles in Parkinson disease. *Cog Behav Neurol*, 17(4): 195-200.
- Williams-Gray CH, Foltynie T, Brayne CE, Robbins TW & Barker RA. (2007). Evolution of cognitive dysfunction in an incident Parkinson's disease cohort. *Brain*, 130(7), 1787-98.
- Zec RF, Landreth ES, Fritz S, Grames E, Hasara A, Fraizer W, et al. (1999). A comparison of phonemic, semantic, and alternating word fluency in Parkinson's disease. *Arch Clin Neuropsychol*, 14, 255-264.
- Zgaljardic DJ, Borod JC, Foldi NS & Mattis PJ. (2003). A review of the cognitive and behavioural sequelae of Parkinson's disease: relationship to frontostriatal circuitry. *Cogn Behav Neurol.*, 16(4):193-21.
- Zizak VS, Filoteo JV, Possin KL, Lucas JA, Rilling LM, Davis JD, Peavy G, Wong A & Salmon DP. (2005). The ubiquity of memory retrieval deficits in patients with frontal-striatal dysfunction. *Cogn Behav Neurol*, 18(4), 198-205.

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This book about Parkinson's disease provides a detailed account of various aspects of this complicated neurological condition. Although most of the important motor and non-motor symptoms of Parkinson's disease have been discussed in this book, but in particular a detailed account has been provided about the most disabling symptoms such as dementia, depression, and other psychiatric as well as gastrointestinal symptoms. The mechanisms responsible for the development of these symptoms have also been discussed. Not only the clinicians may benefit from this book but also basic scientists can get enough information from the various chapters which have been written by well known faculty.

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