

Parkinson's Disease and Frontal Lobe Dysfunction. Review

Dr. Julio López Argüelles^{1*}, Lic Aleima B Rodriguez Carbajal², Dr. Leydi M. Sosa Aguila³

¹Department of Neurology, Ambulatory specialized center Héroes de Playa Girón, University Hospital Gustavo Aldereguía Lima, Cienfuegos, Cuba.

²Department of Neurology, University Hospital Gustavo Aldereguía Lima, Cienfuegos, Cuba. ³Genetic Department, University Hospital Paquito González Cueto, Cienfuegos, Cuba. *julito.arguelles@gmail.com*

*Corresponding Author: Julio López Argüelles, Neurology Department, University Hospital Gustavo Aldereguía Lima, Cienfuegos, Cuba.

Abstract

The frontal lobes occupy a third of the cerebral cortex in humans. The frontal lobe represents the most important neuroanatómica basis of the human complex behavior, where there are represented the larger differences that differentiate us from the rest of animals. It has been demonstrated that the signs of frontal dysfunction are well represented in dementias of subcortical pattern, whose prototype well can be Parkinson's disease. in Parkinson's disease the degenerative process involves the frontal lobes with different areas involved as the motor and premotor cortex, and dorsolateral and cingular areas, resulting in the appearance of classical frontal syndromes, furthermore demonstrates the affectation of different ways and neurotransmitters involved.

Keywords: Parkinson's disease, frontal dysfunction, disexecutive syndrome.

INTRODUCTION

The frontal lobes occupy a third of the cerebral cortex in humans. The prefrontal cortex is the frontal region previous to the primary and premotora motor cortex. It is a structure that has increased of size with the phylogenetic development (8.5% of the total of the cerebral cortex in lemures, 11.5% in macacos, 17% in the chimpanzees, and 29% in humans) and it is heterogeneous from the anatomical and functional standpoint. The frontal lobe represents the most important neuroanatómica basis of the human complex behavior, where there are represented the larger differences that differentiate us from the rest of animals. This lobe is related to the selective attention, the motor programming, the initiative, the articulation of the language, the conscience, the moral and ethical behavior, the reasoning and the organization and execution of the mobile behavior [1;2].

The frontal lobe is the anatomical substratum for the executive functions that are the ones that make it possible for us to direct our behavior toward an end and include: the attention, planning, sequencing, and

reorientation on our acts. They are the ones in charge of taking the information on all the other structures and coordinating them in order to act jointly. They are very implied in the components motivational (motivation) and behavioral (behavior) of the subject; which means that if a harm is caused in this structure it can happen that the subject maintains an appearance of normalcy upon no being mobile deficits, of speech, of memory or including reasoning; existing however an important deficit in the social and behavioral capacities [3].

It has been demonstrated that the signs of frontal dysfunction are well represented in dementias of subcortical pattern, whose prototype well can be Parkinson's disease. Research on the neuroanatomic substratum of these alterations might follow that are related to anatomofuncionals disorders of the frontal lobes, where exists the possibility of compensating the nigroestriatal deficit and strengthening the frontal activity by other not as affected ways and, with this, to reestablish at least partially the motor behavior [4].

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DISCUSSION

The effects of an injury of the frontal lobe on the behavior vary in relation to the size and of the localization of the physical defect. The small injuries do not tend to cause notable changes in the behavior if they only affect a side of the brain, although they sometimes cause convulsions. The large injuries of the back of the lobes frontal can cause apathy, lack of attention, indifference and, sometimes, incontinence. The people who present major alterations further towards the lateral part which are previous or of the frontal lobes tend to be distracted easily, feel euphoric without apparent reason, are argumentativas, vulgar, and rough; furthermore, maybe not be aware of the results of their behavior [3;5].

There are 5 recognized frontosubcorticals circuits: one "motor", that is originated in the motor supplementary area; an "oculomotor" circuit, that departs from area 8, and three more than depart from the different regions of the prefrontal cortex (dorsolateral, orbitofrontal and of the previous girdle) [2].

Different cognitive, behavioral, and emotional profiles are related to these circuits. The dorsolateral prefrontal injuries produce deficit in the verbal and non-verbal creep, reduce the ability to solve problems and to alternate between cognitive categories, in addition reduce the learning and the recovery of the information. The orbitofrontals injuries cause lack of inhibition and irritability. Injuries in the frontal/ previous medial girdle result in apathy and reduction of the initiative [6].

The framework of the frontal-subcorticales systems provides a basis in order to understand the functions of the frontal lobe. First, injuries in different frontal regions can cause cognitive and behavioral changes. Second, the functions of the frontal lobe are not under a single anatomical jurisdiction in the frontal cortex.

Parkinson's disease is one of the neurological diseases that induce dysfunction of these fronto-subcorticals circuits, producing as a consequence in the patient [4;7]:

DORSOLATERAL SYNDROME

Occurs a cognitive alteration as a result of a disorder in the following functions:

Executive functions: group of cognitive functions that are used to coordinate basic cognitive capacities,

emotions and for the regulation of behavioral responses regarding different environmental demands. Work memory: It is the information that a person is capable of maintaining "online" and that it is going to need short-term, while it carries out a given action (short-term memory). Memory alterations. Deficit in the motor programming, in the motor performance, and in the inhibitory behavior control. Reduction of the verbal and non-verbal fluidity. Alteration of the behavior: the patients with dorsolateral injury tend to appear apathetic, slow, demotivated, distracted, dependents of the environment, with difficulties in the attention, lack curiosity. With left injuries, depression is a frequent symptom. Temporary event ordering: difficulties in ordering the events in the time, or to follow a sequence, both verbal and motor. Disorders in the resolution of problems and decision-making.

Orbitofrontal Syndrome

It joins together with lack of inhibition, inappropriate behaviors, irritability, emotional lability, and difficulties in responding to social signs.

SUBCORTICAL DISEXECUTIVE SYNDROME

Is caused by damage in to the subcorticales structures of white or gray substance. When the damage is moderated, these changes are presented as a disexecutive attentional deficit, but when the damage is severe and persistent constitutes the Subcortical dementia syndrome: Slowed down thinking. Memory: Poor in processes of coding, low recovery of the information with good performance in the phase on recognition. Executive Functions: poverty in the resolution of problems. Affection: apathy, depression.

CONCLUSION

with the previous review becomes evident as in Parkinson's disease the degenerative process involves the frontal lobes with different areas involved as the motor and premotor cortex, and dorsolateral and cingular areas, resulting in the appearance of classical frontal syndromes, furthermore demonstrates the affectation of different ways and neurotransmitters involved.

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