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Organic personality disorder: conceptual review and research strategies

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The historical review of «psychiatric personality disorders» reveals the lack of convergence of those disorders with the organic personality disorder (OPD). Only the psychopathy concept has been used as a psychopathological phenotype for one of the groups of OPD, the so-called «pseudopsychopaths». These patients have been described from the beginning of the XXth century under the heading of «frontal lobe syndrome». It was only with the development of the psychiatric nosologies, towards the middle of the XXth century, that the term «organic personality disorder» started to be used.

The accumulation of knowledge about the different prefrontal areas and the development of neuropsychological models that try to explain social behavior have opened new ways of understanding this syndrome. The orbitofrontal cortex has been identified as one of the key structures in behavioral and emotional regulation. Recognition of emotions in voices and faces, empathy, appreciation of humor, tasks that show «theory of mind» are some of the dimensions included in the examination of the non-cognitive functions of the prefrontal cortex.

Key words:
Organic personality disorder. Frontal lobe syndrome. Prefrontal cortex. Emotion. Empathy. «Theory of mind».

Actas Esp Psiquiatr 2007;35(2):115-121

El trastorno orgánico de la personalidad: análisis conceptual y estrategias para la investigación

La revisión histórica del concepto de trastorno de la personalidad en psiquiatría pone de manifiesto que dicha categoría no converge ni da pie al concepto actual de trastorno orgánico de la personalidad (TOP); solamente el concepto de psicopatía sirve de viñeta fenomenológica que será utilizada para la descripción de algunos de los TOP como «seudopsicopáticos». Estos pacientes han sido descritos desde principios del siglo XX bajo el paraguas del síndrome del lóbulo frontal; a mediados del siglo XX, con el desarrollo de las nosologías psiquiátricas se adoptó el término trastorno orgánico de la personalidad para caracterizarlos.

El fraccionamiento de la corteza prefrontal y el desarrollo de modelos neuropsicológicos que tratan de explicar la conducta social han abierto nuevas posibilidades de exploración y comprensión de esta patología. La distinción anatomofuncional entre corteza ventral y corteza dorsolateral señala a la primera como responsable de la modulación emocional y conductual. La evaluación del reconocimiento de emociones en voces y caras, de la empatía, del sentido del humor y de la «teoría de la mente» son algunas de las nuevas herramientas de que disponemos para explorar las funciones no cognitivas de la corteza prefrontal.

Palabras clave:
Trastorno orgánico de personalidad. Síndrome del lóbulo frontal. Córtex prefrontral. Emoción. Empatía. «Teoría de la mente».

INTRODUCTION

The tenth edition of the International Classification of Disease (ICD-10) includes «organic personality disorder (OPD)» (F07.0) among its list of diagnoses[1]. The equivalent category in the DSM-IV receives the name of «change of personality due to medical disease» (310.1)[2].

The definition of this disorder is not simple. In the ICD-10, reference is made to specific alterations of the emotions, impulses, needs and more complex cognitive processes such as activity planning and forecast of social and personal consequences. In the DSM-IV description, the most frequent changes are mentioned as «affective instability, poor impulse control, outbursts of aggression or rage, apathy, suspiciousness or paranoid ideation». The subtypes (labile, disinhibited, aggressive, apathetic, paranoid) appear in both classifications, although only in the version called «research criteria» in the ICD-10. Both its provisionality and

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the need to investigate the validity of the different clinical profiles proposed are stressed in this version. Both nosologic systems stress the need for evidence of brain disease to make the diagnosis. The DSM-IV also includes a criterion referring to the necessary impact of the picture in form of significant occupational or social impairment.

In spite of the clear recognition that this diagnostic category has in the psychiatric nosologies and in the scales used to evaluate post-traumatic sequels[3], the research done on it is very limited. In this review, a conceptual framework is proposed for investigation on the organic personality disorder. The authors are psychiatrists and neuropsychologists who work in multidisciplinary services monographically dedicated to rehabilitation and evaluation of patients with acquired brain damage. The objectives of this reflection are the following:

- Initiate a conceptual analysis of the organic personality disorder as a diagnostic category.
- Review the alternative forms of approaching the description and typing of these patients developed by psychiatrists, neurologists and neuropsychologists.
- Based on this revision, suggest research strategies that may contribute to the advance of knowledge in this area.

PERSONALITY AND ITS DISORDERS IN PSYCHIATRIC NOSOLOGY

«Personality», «constitution», «character», «temperament», «self», «oneself», are some of the terms used to refer to the psychological characteristics that underline the behavior of each person and makes him/her a psychologically unique and differentiated individual. The detailed review of the history of each one of them goes beyond the aims of this study, but may be consulted in the work of Berrios[4,5].

The term «personality» was dominant and this originated to a great degree in the work of Kurt Schneider entitled «Die psychopathischen Persönlichkeiten»[6]. The repercussion of this work condemned the terms «constitution» and «temperament» to a certain ostracism. The latter term continues to be used and refers to the aspects contributing to shape the personality and that are more biologically determined, almost always by genetic inheritance.

The origin of personality disorder as diagnostic category

During the XVIII century, mental diseases were considered as intellect disorders. It was Pinel[7] who introduced the concept of mental disorder with conservation of the intellect, thus breaking with the purely intellectualistic view of madness. Manie sans *delire*[*] describes an affective disorder (with intact intellect), characterized by fury, aggressiveness, emotional instability and social maladjustment[4,8]. Esquirol[9] adhered to this reflection, proposing the category of monomania that implied partial alteration of reason, emotion and will.

This conceptual definition reflects something that was occurring on the diagnostic level, differentiation between abnormalities of behavior and other forms of «madness» such as schizophrenic psychosis, mania, depression or dementia. Two centuries later, psychopathy was constituted regarding this diagnostic group.

Towards a modern concept of psychopathy

Craft[10] proposed the first operative definition of psychopathy as an antisocial disorder in which the primary traits were absence of emotion in regards to others and tendency to act impulsively. The secondary traits included aggressiveness, lack of shame or remorse, incapacity to learn from experience and reduction of motivation. In this first definition, the extraordinary phenomenological similarity with the present OPD criteria stands out. Practically the only difference is that a demonstrated organic lesion is not mentioned as cause of the clinical syndrome. A few years later, Cleckley[11] established sixteen criteria in his definition of psychopathy, most of which have been included in the diagnostic criteria for the Antisocial Personality Disorder (301.7) of the DSM-IV.

Psychopathy and organicity

While the history of personality disorders, and more specifically that of antisocial disorder, does not lead us towards the OPD category, antisocial patients with a history of criminality have been studied under the hypothesis of the existence of prefrontal dysfunction[12-14]. Comparison between convicts with psychopathy and prisoners without psychopathy provides significant differences in tests sensitive to ventral frontal functioning (go/no go tasks, odor identification tasks), while no differences are observed in tests that assess dorsolateral functioning or retrorolandic functions[15]. The presence of orbitofrontal dysfunction, accompanying the psychopathic behavior profile, and in absence of evidence of brain injury, explains the diffuse and heterogeneous concept of «minimum cerebral dysfunction» as hypothetical etiological substrate of the mentioned dysfunction and its behavioral correlates.

*With *manie*, Pinel is not referring to the present concept of mania but rather to a persistent state of florid psychosis characterized by aggressiveness and fury. *Délire* also is not equivalent to the present delusion, but rather refers to a disorder of intellect, emotion or will, but rather, in this period, especially of reasoning.

Apathy: concept and history

From the phenomenological point of view, in addition to disinhibited and pseudopsychopathic behavior, apathy is the other great symptom or syndrome that fills the organic personality disorder with content. The term «apathy» gives the name to one of the subtypes and significantly contributes to the global concept.

Its history is dilated. It was introduced into psychiatric literature at the beginning of the XIX century with the meaning of indifference due to the disappearance of emotions. Until the first World War, lack of will, called «abulia», and lack of emotions or «apathy» were considered as different and dissociable symptoms. The disappearance of the concept of apathy as loss of emotions caused a restriction of the use of this term in the frontal lobe syndrome scope. Resurgence of it in the 1970's and 1980's gave rise to a single mixed concept of apathy in which both concepts are combined[16]. Its usage within the frontal lobe syndrome has a close relationship that has given rise to its prominent role in the present concept of organic personality disorder.

THE FRONTAL LOBE SYNDROME (FLS)

The study of the personality disorders in patients with frontal lesions greatly owes its development to the case of Phineas Gage[17-19]. In 1848, a bar crossed through his head and injured prefrontal areas of both hemispheres. John Martyn Harlow, the physician who saw him, described a surprising recovery with preservation of the intellectual functions, but the personality change caught his attention. «Gage's behavior was now characterized by irritability, childlessness, obstinacy, lack of consideration towards others, impatience and lack of impulse control».

The evidence that linked prefrontal lesion with personality disorder began to be clearly seen at the beginning of the XX century, giving rise to the use of the term «frontal lobe syndrome». In his discussion on the frontal lobe symptoms, Moritz Jastrowitz[20] described a type of dementia in patients with frontal tumors that was characterized by a rare and cheerful agitation that he named with the term «moria». He stressed the inconstancy of the symptoms, the difficulty to describe them and the fact that only some patients with frontal lesions had it. The syndrome could also be seen in the initial phases of general paralysis, in dementias and in alcoholic patients.

Oppenheim knew the work of Jastrowitz when he published his article on the clinical manifestations of brain tumors[21]. In the section on frontal tumors, he indicated «psychic abnormality» as a sign with diagnostic value of location. Instead of speaking of «moria» or insipidness, he described a special «addiction to trivial jokes having a sarcastic nature» that he called *Witzelsucht*. The four patients who had this picture had right frontal lobe tumors, three of them with invasion to mesial and basal regions. Welt, who published a case of a depressed frontal fracture with behavior change that abated after a few months, indicated that the orbital disease was a necessary but not sufficient condition for the debut of a personality disorder[22]. In his review of the literature, an overwhelming majority of the cases with this presentation had orbital damage, normally documented by necropsy. However, on the other hand, many other cases with orbital lesion did not have this personality disorder.

During the first half of the XX century, there was access to a large number of cases of prefrontal lesion, many of them as a consequence of war wounds. The association between prefrontal cortex lesion and personality disorder was reinforced, but the diversity of the clinical presentations has entailed a serious difficulty since then for the formation of theories on psychic functions or disturbed basic cognitive functions and the reactive or adaptive mechanisms they give rise to.

The impossibility of forming a syndromic definition with a small number of characteristic symptoms became the main guarantee against the disappearance of the term «frontal lobe syndrome.» The miscellaneous behavior, affective and cognitive disorders that may occur in association with prefrontal lesion continues to be known with an anatomical coined term instead of being substituted by another name closer to the phenomenological. As Duffy and Campbell accurately stated, this is probably the explanation which, in the first place, may be called an «inherited anachronism of a strict and old-fashioned localization» (p. 379)[23].

FRACTIONING OF THE PREFRONTAL CORTEX AND ITS SYNDROMES

Stuss et al. have recently summarized their work on patients with lesions located in different prefrontal regions[24]. The results are the product of simultaneously refining the differentiation of cognitive processes and location of lesions in the front cortex. From this anatomical-functional point of view, there were four resulting large frontal subdivisions, left and right dorsolateral, a single inferior medial region and another superior medial region.

Emotional and/or behavior disorders are especially present when the lesions affect the inferior medial region (traditionally known as orbitofrontal or ventral bilateral; a neurobiological substrate of the inhibition and reward processes, essential for behavioral self-regulation[19,25]. Damasio's hypothesis of somatic marker indicates the orbitofrontal region as responsible for the formation of emotional biases acquired by conditioning[18], that orient future responses to reexperience of a stimulus or situation.

Cummings also dissected the prefrontal cortex, describing five frontal-subcortical circuits, three of which are related with behavior syndromes[26,27]:

- A prefrontal dorsolateral circuit, whose lesion gives rise to a dysexecutive syndrome.
- A lateral orbitofrontal circuit, whose lesion gives rise to a syndrome that includes disinhibition, lack of concern, irritability, euphoria, lack of tact, emotional lability and behavior dependent on external clues.
- Anterior cingulate circuit, related with a syndrome characterized by apathy and akinetic mutism in the severest cases.

All these authors have gone deeper into and somehow overcome the original formulation of the executive functions proposed by Lezak[28]: «capacities that permit an effective, creative and socially accepted behavior.» Their contributions suggest that behavior disorders characteristics of FLS are the result of failures, among others, in the emotion-situation-consequence association systems, of non-recognition of emotions in the other, of the equivalents to the behaviors used in social situations, and of disruptions in mechanisms of initiation to action[29]. The generic term of executive functions has been substituted or complemented by another series of concepts among which that of social cognition stands out at present.

RESEARCH STRATEGIES WITHIN NEUROPSYCHIATRY

Before formulating the research strategies, it may be useful to consider three of the confounding sources that have harmed the progress of knowledge in this area. Probably, the most important is the identification of concepts that belong to different levels of analysis of reality: anatomic, neuropsychologic, psychopathologic and even nosologic. The most obvious is the indiscriminated use of the terms «frontal functions» and «executive functions»[30]. There is much evidence of executive function disorders, or at least of very poor performance in the tests designed to measure these functions, in patients with lesions in other areas of the brain[31], and that disorders secondary to frontal lesions are difficult to classify as dysexecutive syndromes: oculomotor disorders, apathy, self-consciousness disorders. The terms «frontal lobe syndrome,» «organic personality disorder» and dysexecutive syndrome» are also used as pseudosynonymous. Although the overlapping between them is undeniable[32], it is important to recognize that their conceptual limits differ as they are marked by the level of analysis in which they are generated: anatomic for the «frontal lobe syndrome,» psychopathological and nosological for the «organic personality disorder» and neuropsychological for the «dysexecutive syndrome.»

The second scope of confusion refers to the multiple terms, sometimes interchangeable, that are used in the study of the organic personality changes: antisocial behavior[12], psychopathy[15], social and moral behavior[32], neurobehavioral symptoms[33], characterological changes[34], psychosocial adjustment[35], psychosocial functioning, personality disorders[36] and organic personality disorder[37]. This makes defining syndromes and conceptual and terminologic consensus difficult.

Fractioning course of the prefrontal region on the one hand, the recent development of the neuropsychological models of social behavior (occupied by the lax concept of executive functions until recently) and the phenomenological variability of the organic personality syndromes have complicated the study of the relationship between these three levels of knowledge. Figure 1 attempts to represent the conceptual course that is presently occurring (fig. 1).

Finally, the psychopathological study of these patients uses personality concept as a reference. This is a complex psychological construct that includes volitive, emotional and cognitive components that articulate and generate usual behavior patterns. The relationship between personality and neurobiological substrates is confusing. When we speak of a personality disorder, the situation is very different from when we must face the patient with a memory, speech or face recognition disorder. In the latter situations, the relationship between neuroanatomy, neuropsychology of normal functioning and psychopathological alteration is much better defined than in the case of personality disorders. In the future, it is foreseeable that the personality construct may be explained on another analysis level by means of mediating variables that have a more direct relationship with neurobiology. Investigation in this area will

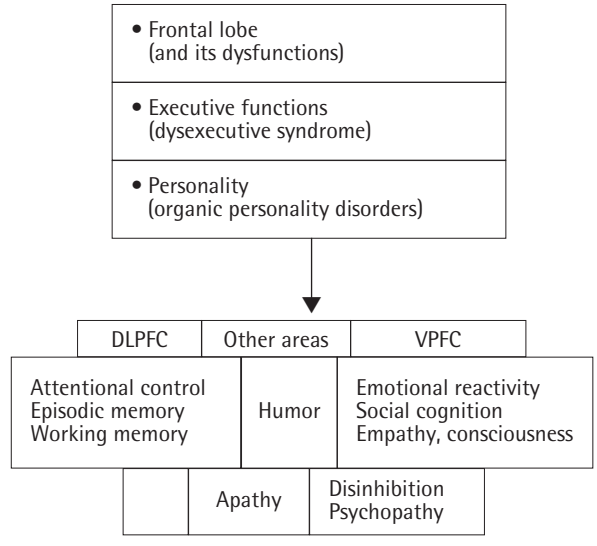

Figure 1 *Representation of the evolution from a model that establishes an identity relationship between the anatomic, neuropsychological and psycho(patho)logical level towards another in which each one of these levels has evolved, has been subdivided, and in which the relationship between levels is complicated. DLPFC: dorsolateral prefrontal cortex; VPFC: ventral prefrontal cortex.*

then be more fruitful. Concepts such as impulsiveness, apathy, empathy, theory of mind may be needed to occupy this intermediate analysis level between personality and brain.

Psychiatry focuses its interest on treatment, relief and care of patients with mental disorders. This should thus be the axis of any investigational activity. To be able to fulfill this aspiration, descriptive and diagnostic tools that make it possible to measure the reality observed must be developed and it must be assessed if the pharmacological, neuropsychological, psychotherapeutical or psychosocial strategies used are of any use. On the contrary to depression or anxiety or to the universe of the psychotic, the scales developed to assess behavioral and personality changes secondary to brain lesion are limited[38]. Scales that overlap with the OPD but that are not constructed from this reference framework have been published[39]. Consequently, desirable diagnostic refinement is not occurring due to the absence of empiric psychopathologic data.

Research strategies that are suggested distinguish between single case studies and studies of series of patients.

Single case studies

These are detailed longitudinal descriptions that document, in the first place, the natural history of the psychopathology of patients who, having suffered any brain lesion, come to the clinician basically with a personality change. As far as possible, in addition to the etiology, an attempt is made to document the site and extension of the lesion by structural or functional neuroimaging techniques. A third type of information having maximum interest is that shaped by the results of tests that measure attention, executive functioning and the new tests designed to capture information on neuropsychology of social behavior: theory of the mind, recognition of emotions in faces and voices, and grasping of humor, irony or ambiguous sentences.

These three types of variables - psychopathological, neuropsychological and neuroanatomical - may be studied alone or in combination, ideally as a longitudinal study, that can document changes in each one of them and in the relationship they have between them.

Comparison between patients having very different psychopathological profile (for example, apathic versus uninhibited) could explain if there are differentiated neuropsychological mechanisms and neuroanatomic substrates for these types of behavioral changes.

This method can also study response to specific therapeutic interventions, both pharmacological and psychotherapeutical[32,40,41]. Any strict study on therapeutic efficacy with larger samples should have previously undergone successful single case trials.

Studies of series of patients

The study with large samples of patients allows for the validation of global psychopathological scales of OPD or psychopathological dimensions of interest: disinhibition, apathy, design or adaptation of this type of instruments must include psychometric studies of validity, reliability and sensitivity to change. The samples also make it possible to study comorbidity between subtypes and their relationship with etiology (basically traumatic, vascular and tumoral), site and neuropsychological performance.

CONCLUSIONS

OPD is a diagnostic category that has been scarcely investigated in psychiatry. Its nosological location is differentiated from the non-organic base personality disorders. This is a reflection of a different historic development in which the etiological variable (known organicity) has been the dominant nosologic criterion in detriment of the phenomenological criterion.

Personality disorders began their differentiation at the beginning of the XIX century when mental diseases with conservation of intellect began to be recognized. Different authors stressed will, impulse or emotion disorders. The psychopathy concept has served as a reference to the modern formulation of personality disorders.

The relationship between frontal lesions and behavior or personality disorders was clearly established at the end of the XIX century. Variability in the clinical presentation, from absence of disorder to different behavioral, emotional and cognitive disorders, has made it impossible to disregard the term «frontal lobe syndrome» and definitively substitute it with another or others closer to the clinical typing. In the middle of the XX century, psychiatric nosological systems began to be developed and the frontal lobe syndrome ended up being translated as «organic personality disorder». This term has not reached the generalized acceptation that other psychiatric categories such as schizophrenia or bipolar disorder, for example, have had. Other disciplines continue to refer to these patients as persons who have a frontal lobe syndrome, prefrontal syndrome, neurobehavioral disorder or dysexecutive syndrome.

To a large degree, the study of these patients has been done by neurologists and neuropsychologists in recent decades. They have followed two different, but convergent, lines of work: on the one hand, those who have tried to identify clinical syndromes that correspond with lesions in different areas or circuits that involve the prefrontal region; on the other, those who stress the need to propose new neuropsychological models that may explain the main social behavior disorders. A present review of these theoretical models can be consulted in Sánchez Cubillo et al.[29].

Finally, it is difficult to venture a future to this diagnostic category. The proposal of diagnostic subtypes of OPD, prefrontal syndromes and the advances in their neuropsychological understanding seem to announce a differentiation in subsyndromes more than an integration with the remaining personality disorders. The neurobiology of the personality is a task that has hardly been initiated and its development also depends on the nosological course of its disorders. It is desirable for psychiatry to join the investigator efforts of other disciplines, since its psychopathological contribution may be extraordinarily useful for the progress of knowledge in this area.

ACKNOWLEDGEMENTS

The authors thank the Education, Universities and Research Department of the Basque Government and Bilbao Bizkaia Kutxa for its financing.

REFERENCES

1. OMS. CIE-10: Trastornos mentales y del comportamiento. Criterios diagnósticos de investigación. Madrid: Meditor, 1994.
2. APA. DSM-IV: Manual diagnóstico y estadístico de los trastornos mentales. Barcelona: Masson, 1995.
3. Quemada JI, Hormaechea JA, Muñoz Céspedes JM. La peritación psiquiátrica y neuropsicológica del daño cerebral traumático y la Ley 30/95. Actas Esp Psiquiatr 2003;31:353-60.
4. Berrios GE. Personality and its disorders. En: Berrios GE, editor. The history of mental symptoms. Cambridge: University Press, 1996; p. 419-42.
5. Berrios G, Porter R. A history of clinical psychiatry. The origin and history of psychiatric disorders. London: The Athlone Press, 1995.
6. Schneider K. Psychopathic personalities. London: Casell, 1950.
7. Pinel P. Traité Médico-philosophique sur l´alienation mentale, 2nd ed. París: Brosson, 1809.
8. Sass H, Herpertz S. Personality disorders: clinical section. In: Berrios G, Porter R, editores. A history of clinical psychiatry. London: The Athlone Press 1995; p. 633-44.
9. Esquirol E. Des Maladies Mentales Considerées sous les Rapports Médical. Hygienique et Médico-legal. París: Baillière, 1838.
10. Craft M. Psychopathic disorders and their assessment. Oxford: Permagon Press, 1966.
11. Cleckley H. The mask of sanity: an attempt to clarify some issues about the so-called psychopathic personality. San Luis: Mosby, 1976.
12. Kandel E, Freed D. Frontal-lobe dysfunction and antisocial behavior: a review. J Clin Psychol 1989;45:404-13.
13. Prins H. Offenders, deviants, or patients? An introduction to the study of socio-forensic problems. London: Tavistock Publications, 1980.
14. Jurado MA, Junqué C. Psicopatía y neuropsicología del córtex prefrontal. Actas Luso Esp Neurol Psiquiatr Cienc Afines 1996; 24:148-55.
15. Lapierre D, Braun CM, Hodgins S. Ventral frontal deficits in psychopathy: neuropsychological test findings. Neuropsychologia 1995;33:139-51.
16. Sanchez P, Quemada JI. Trastorno organico de la personalidad. Aspectos conceptuales y diagnósticos. Rev Neurol 2000; 30:772-8.
17. Stuss DT, Gow CA, Hetherington CR. «No longer Gage»: frontal lobe dysfunction and emotional changes. J Consult Clin Psychol 1992;60:349-59.
18. Damasio AR. El error de Descartes. Barcelona: Crítica, 1996.
19. Harlow JM. Recovery from the passage of an iron bar through the head. Massachusetts Medical Society 1868;2:327-47.
20. Jastrowitz M. Beiträge zur Localisation im Grosshirn und über deren praktische Verwerthung. Dtsch Med Wochenschr 1888;14:81-3.
21. Oppenheim H. Zur Pathologie der Gehirngeschwulste. Arch Psychiatr 1890;21:560-78.
22. Welt L. Über Charakterveränderungen des Menschen infolge von Läsionen des Strirnhirns. Dtsch Arch Klin Med 1888;42:339-90.
23. Duffy JD, Campbell JJ, 3rd. The regional prefrontal syndromes: a theoretical and clinical overview. J Neuropsychiatry Clin Neurosci 1994;6:379-87.
24. Stuss DT, Alexander MP, Floden D, Binns MA, Levine B, Mcintosh AR, et al. Fractionation and localization of distinct frontal lobe processes: evidence from focal lesions in humans. In: Stuss DT, Knight RT, editores. Principles of frontal lobe function. New York: Oxford University Press, 2002; p. 392-407.
25. Eslinger PJ, Damasio AR. Severe disturbance of higher cognition after bilateral frontal lobe ablation: patient EVR. Neurology 1985;35:1731-41.
26. Cummings JL. Frontal-subcortical circuits and human behavior. Arch Neurol 1993;50:873-80.
27. Cummings JL. Frontal-subcortical circuits and human behavior. J Psychosom Res 1998;44:627-8.
28. Lezak MD. Neuropsychological assessment. 2nd ed. New York: Oxford University Press, 1983.
29. Sánchez-Cubillo I, Muñoz-Céspedes JM, Quemada JI. ¿Qué procesos cognitivos están implicados en el trastorno orgánico de personalidad? An Psicol 2004;20:273-87.
30. Stuss DT, Alexander MP. Executive functions and the frontal lobes: a conceptual view. Psychol Res 2000;63:289-98.
31. Barcelo F, Santomé A. Revisión crítica del test de clasificación de cartas de Wisconsin como indicador de disfunción frontal. Rev Neurol 2000;30:855-64.
32. Anderson SW, Bechara A, Damasio H, Tranel D, Damasio AR. Impairment of social and moral behavior related to early damage in human prefrontal cortex. Nat Neurosci 1999;2:1032-7.
33. Deb S, Lyons I, Koutzoukis C. Neurobehavioural symptoms one year after a head injury. Br J Psychiatry 1999;174:360-5.
34. Tate RL. Executive dysfunction and characterological changes after traumatic brain injury: two sides of the same coin? Cortex 1999;35:39-55.
35. Tate RL, Broe GA. Psychosocial adjustment after traumatic brain injury: what are the important variables? Psychol Med 1999; 29:713-25.
36. Prigatano GP. Personality disturbances associated with traumatic brain injury. J Consult Clin Psychol 1992;60:360-8.

37. Franulic A, Horta E, Maturana R, Scherpenisse J, Carbonell C. Organic personality disorder after traumatic brain injury: cognitive, anatomic and psychosocial factors. A 6 month follow-up. Brain Inj 2000;14:431-9.
38. Marin RS, Biedrzycki RC, Firinciogullari S. Reliability and validity of the Apathy Evaluation Scale. Psychiatry Res 1991;38: 143-62.
39. Chow TW. Personality in frontal lobe disorders. Curr Psychiatry Rep 2000;2:446-51.
40. Lough S, Hodges JR. Measuring and modifying abnormal social cognition in frontal variant frontotemporal dementia. J Psychosom Res 2002;53:639-46.
41. Salloway SP. Diagnosis and treatment of patients with «frontal lobe» syndromes. N Neuropsychiatry Clin Neurosci 1994;6:388-98.