Impulsivity: historical and conceptual review

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Impulsividad: revisión histórica y conceptual

Summary

There is great confusion about the concept of impulsivity despite its important clinical use. Limits with others symptoms or behaviors, like aggressiveness, are imprecise, and we find no general agreement on the definition of impulsivity in the present literature. From the point of view of neurosciences, adequate identification and measurement of a symptom as impulsivity could increase the validity of syndromic diagnoses, improve the development of animal models and genetic studies, and improve the treatment of several psychiatric disorders. Based on this perspective, the overall goals of this review article are: a) *to establish the historical evolution of term, concept and behavior that we presently identify as impulsivity, starting from the classical psychopathology of the will;* b) *to review several concepts of impulsivity described in the scientific literature, and* c) *to provide basic guidelines for an operative definition, just as some authors have proposed, to create research lines and understand various discoveries that basic sciences have contributed.*

Key words: *Impulsivity. Will. Aggressivity.*

Resumen

A pesar de su amplio uso clínico existe una gran confusión en torno al concepto de impulsividad. Los límites con otros síntomas o conductas, como la agresividad, son imprecisos y no encontramos en la literatura actual una definición aceptada por consenso. Desde el campo de las neurociencias, la identificación y medición adecuadas de un síntoma como la impulsividad aumentaría la validez de los diagnósticos sincrónicos, facilitaría el desarrollo de modelos animales e investigaciones genéticas y mejoraría el abordaje terapéutico de diversos trastornos. A partir de esta perspectiva, los objetivos planteados en la presente revisión son: a) *establecer la evolución histórica del término, concepto y conducta que actualmente denominamos impulsividad a partir de la clásica psicopatología de la volutad;* b) *revisar los diversos conceptos de impulsividad descritos en la literatura científica, y* c) *señalar las directrices básicas para una definición operativa, tal y como varios autores han propuesto, para crear diferentes líneas de investigación y comprender los diversos hallazgos que han aportado las ciencias básicas.*

Palabras clave: *Impulsividad. Voluntad. Agresividad.*

INTRODUCCIÓN

The term *impulsivity* is widely used in daily life and refers to a relevant concept in the psychiatric practice. Its extensive use contrasts with the fact that few studies have tried to clarify the role of impulsivity in mental illnesses and with the absence of a definition accepted by consensus, thus impulsivity or lack of control of impulses may refer to psychopathologically unequal phenomena.

Impulsivity is defined in different psychiatry dictionaries as a rapid action with no thought or previous conscious judgement[1], as a behavior without adequate thought[2], or as a tendency to «go on to action» with less previous reflection than most of the individuals with the same knowledge or skills[3], and we find it in Psychiatric Classification Manuals as a part of the diagnostic criteria of several psychiatric disorders[4], such as personality disorders (borderline and antisocial) or epilepsy, and as a symptom of such diverse entities as disorders due to substance abuse, ludopathy, hyperactivity disorders or mania.

Within the field of neurosciences, it is recognized that adequate identification and measurement of a symptom, such as impulsivity, would increase the validity of syndromic diagnoses, would facilitate the development of animal models and genetic research (if we consider the possibility that the symptoms may correspond better with the phenotypes than with the syndromes) and probably would improve the therapeutic approach of these disorders, since it is a fact that the present treatments act more on a symptomatic level than a syndromic one. However, the approach to the concept of impulsivity both from a longitudinal (historic) as well as cross-sectional (based on the present knowledge) point of view poses a series of problems such as: *a)* confusion regarding the

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concept of impulsivity; *b)* unclear limits with other symptoms and even with behaviors considered «normal», and *c)* and limited specificity.

From this perspective, the objectives of our article are: *a)* establish the historical evolution of the term, concept and behavior that we presently call impulsivity; *b)* review the different concepts of impulsivity described in the scientific literature, limiting the abuse of this term, and *c)* mention the basic guidelines for an operative definition, as several authors have proposed, to create different research lines and understand the different findings that basic sciences have contributed.

«IMPULSION» AND WILL, HISTORIC EVOLUTION OF THE CONCEPT OF «IMPULSION»

General aspects

The term *impulsivity* originates from its Latin root *impulsus,* a participle of *impéllere,* that means hit, press or push[5]. The present word, impulsivity, was imported from the language of the French mecanicists, however, it refers to primitive behaviors, conducts, *cravings* and appetites whose knowledge escapes the control of the will. During the XIX century, disorders of the will due to excess were called *impulsive* states and the concept of impulsion was used to refer to all the forms of paroxystic, sterotypal and (apparently) involuntary actions[6].

From the classical epoch to the end of the XIX century, *will* (as a philosophical and psychological concept) was the fundamental axis of Western thinking. The concept of *will* was developed in the *classical epoch* as a semantic combination of impulse, instinct, tendency, desire, object and inclination. The Greeks had no separate notion of will and considered it as a human action integrated into the intellect and emotions. Aristotle proposed that appetite and will were involved in action, although in comparison with pure appetites, will was a rational and calculated activity, a view that can be found centuries later in the works of Kant.

On the contrary to this integrated view developed by the Greeks, Jewish-Christian tradition emphasizes the distinction between a deliberate faculty (decision making) and an executive faculty (will). Thus San Agustin suggested that will was an independent power and John Duns Scotus believed that it was the engine of all mental faculties.

The Jewish-Christian perception that the will was an independent mental function predominated over the Greek concept of an action that was totally integrated with feelings and the intellect. During the XIX century, clinical phenomena such as abulia, impulses and obsessions were conceptualized as the result of the pathological changes of the will, attracting the interest of many and relevant authors, until towards the end of that century when these disorders were adopted, due to the decline of the will, as an explanatory and descriptive concept. Will as an independent faculty of the mind began to be attacked and this decline was supported by psychoanalysis and conductism.

In spite of its absence in most of the present classifications and studies, we consider it is well to remember the evolution experienced by psychopathology of the disorders of will during the XIX century before approaching the concept of impulsivity and its disorders, since the first references to the concept that we aim to analyze are found in the pathology of the will[7].

The will and its disorders during the XIX century

The fundamental school in the construction of descriptive psychopathology of the XIX century, *psychology of faculties,* was responsible for the fragmentation of the psychopathological categories into a triple division of mental phenomena: emotions, reasoning and will (subdivided in turn into abulia, lethargy, and excess of impulse). Since then, the idea that mental health depended on the free exercise of the will can be found in various psychiatric writings of this century. For example, in the *Magazin zur Erfahrungsseelenkunde*, Maimon wrote: «Health of the mind consists in a state in which the will is free and can exercise its function without an obstacle. No state in which this does not occur can be considered as mental disorder».

However, the greatest propelling force of the disorders of the will during this period was Matthey. Andre Matthey proposed the first classification of the disorders of the will[8]. In 1816, he coined the term *pathomanie* («perversion of the will and of the natural inclinations, without apparent injury of the intellectual functions») and described four forms of alterations of the will:

1. Impulses without insanity (*fureur sans délire*), *involuntary impulses that involve furious acts without insanity*, that were subdivided into *Tigridomanie* («irresistible inclination to shed blood of fellow men with no reason and without mental illness») and *folie raisonnante* («aggression that is only exercised against inanimate objects»).
2. *Uiphobie* («irresistible aversion or antipathy towards one's own children»).
3. *Klopémanie* («irresistible impulse to steal without being poor or to get involved in shameful acts»).
4. *Melancolie suicide* («disposition to commit suicide without deterioration of reasoning»).

Two decades later, Esquirol discussed the pathological changes of the will in relationship with general pathology[9], and created the clinical category of *monomania,* «a chronic disease of the brain, without fever, characterized by an injury to intelligence (*intellectual monomania*), to affection (*affective monomania),* or to the will (*instinctive monomania*).» Influenced by the french spiritualists and by the roman-catholic philosophy, this author differentiated between a state of reasoning and moral decisions, and one's own will, although during the second half of the XIX century, Despine denied the existence of volitional monomania.

A few years later, in 1840, Marc[10] published *De la folie, considéré dans ses rapports avec les questions médico-judiciaires* in which he classified disorders of the will into primary and secondary.

However, the most important writer on diseases of the will after Matthey was Ernest Billod (1847), a follower of the eclectic philosophy of Victor Cousin. He emphasized the importance of studying disorders of the will, following the line of Esquirol and Marc, considering that they deserved the same recognition as the disorders of the intellect and emotions. Billod[11] distinguished between volitional defects of degree and quality, establishing that the relationship of the will with the other faculties is produced by a «balancing effect»: when the will decreases, the force of other faculties increases. The role of the will would consist in balancing force and resistance.

Even though interest on disorders of the will has begun to decrease since the middle of the XIX century, two contributions in the second half of that century can be stressed: in 1867, Griesinger[12] divided mental disorders into emotional and intellectual, including the will in the latter, and stating that impulses in the mental patient represent an altered will; and in 1883 Ribot published *Maladies de la volonté*[13] in which he analyzed the will as a fact, without entering into its causes or into the subject of freedom. This author approached the study of pathological will through the study of the will described as a psychophysiological phenomenon (that responds to the evolutionist laws) composed of two elements: tendency toward action or inhibition (external influences) and character (internal influences). Ribot classified the disorders of the will in:

1. Disorders due to absence of impulse (abulia, agoraphobia, doubt related insanity, psychic paralysis).
2. Disorders due to excess of impulse (kleptomania, pyromania, suicidal mania, etc.).

In both groups, power of control is reduced, however in the latter, inhibition also fails. Ribot considers power of inhibiting as a superior degree in the evolution of the will. Thus, in the latter group, intellectual function is poor and instincts gain what rational activity loses.

Once the XX century began, disorders of the will lose their central role, and progressively stop being a subject of interest for investigators. At present, there are few papers and publications that treat this subject, although some authors defend the usefulness of reconsidering this concept. Thus, for example, Foulquie[14] recovers the ideas of several XIX century authors and defines the concept of will as the faculty of deciding and acting, but as a balance between power of impulsion and power of inhibition, classifying its disorders in:

1. Hyperbulias («excess of will»).
2. Hypobulias or abulias (difficulty or impossibility to make a decision).
3. Parabulias (involves a contradictory attitude that makes the individual incline towards that which he does not want).

Development of the concept of impulsivity in the XIX century

During the XIX century, disorders of the will due to excess were called impulsive states and following this volitional theory, extensive terminology was used to refer to them:

- Pinel (1809): *manie sans délire*.
- Bourdin and Prichard (1896): *moral insanity*.
- Esquirol (1838): *monomanie instinctive*.
- Brierre de Boismont: *folie d'action*.
- Magnan: *syndromes épisodiques de la folie des dégéneres*.
- Dagonet (1870) and Clouston: *folie impulsive*.
- Pitres and Regis: *impulsion*.

Impulsion was used both in a descriptive as well as an explanatory way, being considered in turn as an internal generator and as a reaction to external events, the endogeneous view, and the cases (that were later called impulsive or instinctive) described by Pinel as *manie sans délire* and by Georget as *impetuosité de penchans* being the first to appear.

With his *folie impulsive*, Dagonet[15] refers to an «irresistible and involuntary act, self-imposed in the mind as occurs with hallucinations» (this included phobias, homicidal and suicidal attempts, manic behavior, hypochondriac concerns and epilepsy). This view was criticized as being contaminated by religious and moral connotations, the viewpoint of Magnan being apparently less religious. He believed that the impulsions were the result of the combination of «a rapid explosion of energy and a type of volitional control. A way of brain activity that causes actions that cannot be foreseen».

Considering this view as simplistic, Bourdin suggested the following subdivision: impulsions can be conscious, unconscious, false or good combinations of them all. Conscious impulsions are secondary to obsessions, unconscious impulsions are to a succession of fleeting ideas with no trace of memory (as in epilepsy); pseudoimpulsions arise from the combination of hallucination and delirium (typical of insanity) and finally, typical mixed impulsions of hysteria. In the Bourdin study, impulsions lost their moral connotations and their mysterious irresistibility and began to become incorporated into the field of mental disorders. Pitres and Regis completed this process.

Thus, the concept of impulsion was used to refer to all the forms of paroxystic, stereotyped and (apparently) involuntary actions, the term of *monomania* evolving to *Folie impulsive* and then to the disorder of the control of impulses.

In any event, the debate underlying this terminological confusion during the entire XIX century is the nature of the disorders derived from excess of impulse, specifically, if these only represented a disorder of the will or if they involved alterations of other faculties. For authors such as Dagonet, Pinel or Esquirol, the pathology of impulses represented a pure volitional disorder (with

the rest of the faculties conserved, it being possible for logical and adequate reasoning to exist), while others such as *Griesinger* stated that there was a baseline intellectual or cognitive defect that meant the abolition of free will, and Ribot who defended the existence of a poor or unstable intellectual adaptation.

During the last years of the XIX century, some authors had already begun to state that the disorders of the impulses did not always involve deterioration of the will: in 1894, Legrain already questioned if there was really a disease of the will; in 1904, Clouston defined pathological impulse as a loss of capacity of control in upper brain zones or as an excess of energy in other zones, and, in the latter case, he did not consider that the will was altered. Graphically, it is described in the following way: *The horseman may be so weak that he cannot control well-trained horses, or the horses can be so difficult to ride that the horseman cannot lead them*.

The volitional theory was abandoned to give way to the emotional theory of impulses. Thus, in psychoanalysis, these disorders were conceptualized in terms of the principle of pleasure and reality (*impulse*: disposition towards the action in order to decrease a state of increased stress caused by the accumulation of pulsions or by the decrease of the defenses of the self against them); in the behavioral theories, they were explained in terms of gratification and decrease of anxiety.

The concept of impulsivity in the XX century

As we have seen, the concept of impulsivity is fundamentally developed in the XIX century, supported, in turn, by a concept such as the will that is difficult to make operative, given its important moral connotations. It is this same concept that has been maintained in many of the definitions handled during this century by different authors and that has served as the basis for some studies that have tried to apply measurement scales or even more sophisticated methods, such as neuroimaging tests.

Several authors tried to integrate varied theoretical constructs (including the will) and clinical observation during the last century. In this way, impulsivity was defined as: tendency to respond rapidly and without previous reflection, the impulsive individuals finding it difficult to restrict their own behavior (Murray, 1938)[16]; in terms of feelings of fury and aggressiveness, expressed by homicidal, suicidal or sexual aggression behaviors (Monroe, 1970)[17]; or related with incapacity to maintain attention (Douglas, 1972)[18]. In 1983, Eysenck[19] described a scale of impulsivity as part of the dimension of extroversion, while Barret and Patton[20] defined impulsivity as an act without adequate reflection, accepting risks and trying to obtain the objectives quickly. On their part Lorr and Wunderlich (1985)[21] concluded that there were two principal components in information processing: *a)* resistance to desires against surrendering to these desires or impulses, and *b)* immediate response to the stimuli compared to planning of the actions previously.

Following the works of Frosch and Wortis (1954)[22], as well as the plan of the DSM-III-R classification, Stein et al. (1995)describe pyromania, kleptomania, addictions, perversions, some sexual disorders, and bulimia, as impulse disorders. Among these disorders, they also include suicidal threats and self-mutilating behaviors. By extension, some disorders of the personality, such as antisocial and borderline personality, would reflect disorders of control of impulses. These examples reflect the psychiatric concept of impulsivity as a behavioral aspect of disorders of several types, that have various implications as: *a)* impulsivity can consist of a certain number of components or subfactors, such as scarce control on affections; *b)* impulsivity should be considered as a trait or characteristic of personality more than as an isolated event in the life of an individual, and *c)* by extension, if impulsivity is a trait, efforts should be aimed at determining possible cerebral mechanisms and genetic sources.

Continuing along the previously described line, Plutchik (1980, 1989, 1990)[23] described the interaction of impulsivity with other personality traits in his psychoevolutive theory, that assumes the existence of eight emotional dimensions systematically related in eight clusters of personality traits, through a complex circular model. An important application of this theory is that impulsivity should not be considered alone in regards to other traits that the individual has, thus for example, a person who shows high impulsivity but also high capacity of control could express elevated levels of conflict but not demonstrate disruptive behaviors. These ideas are reflected, in turn, in the model that Hollander described as opposing forces.

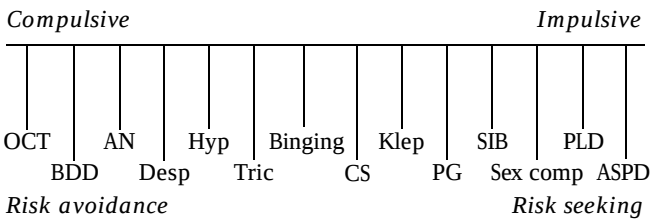
Neurobiological investigations have focused on compulsive pathology and, by extension, have contributed to defining the differences between «impulsive» and «compulsive»[24,25].

According to a *symptomatic/diagnostic perspective* in which the external behaviors define the pictures, the term impulsive appears in the DSM-IV and in the ICD-10 applied to the so-called Disorders of control of impulses[26,27] (ludopathy, kleptomania, pyromania, impulsive sex, etc.). The term «compulsive» is reserved for obsessive-compulsive pathology. Other disorders, such as those related with consumption of toxic substances and uncontrolled intake, are included in other sections (substance use disorders and eating behavior disorders).

According to a *pathogenic perspective*, the existence of a compulsive-impulsive spectrum, in which all these disorders have the same basic nature, related with the obsessive pathology and with the alterations of serotonin alterations[24], has been proposed (table 1).

Between both the symptomatic and pathogenic positions, phenomenological, neurochemical, pharmacological, neuroanatomical and temperamental knowledge seems to indicate that most of these pictures can be defined as compulsive or impulsive pathologies, there also being a solid natural difference between both types (table 2).

TABLE 1. Personality differences: dimensional aspects of the obsessive compulsive spectrum disorders



OCT: obsessive compulsive disorder; BDD: body dysmorphic disorder; AN: anorexia nervosa; Desp: depersonalization disorder; Hyp: hypocondriasis; Tric: trichotiolomania; Binging (of food); CC: compulsive shopping; Klep: kleptomania; PG: pathological gambling; SIB: self-injury behavior; Sex comp: sexual compulsions; PBD: personality borderline disorder; ASPD: antisocial personality disorder

In their last studies published on the concept of impulsivity, Moeller and Barrat[28] proposed defining impulsivity as a predisposition towards internal or external stimuli without considering the negative consequences of these reactions, considering the following as key points:

1. Predisposition, since they consider that there is a behavior pattern more than an individual act.
2. Rapid and scarce planning, that substantially differentiates impulsivity from compulsive behaviors in which planning occurs before the behavior.
3. Non-assessment of the consequences, which involves risks, although they frequently do not coincide with the types of risks that are related with sensation seeking.

The authors state that these aspects of impulsivity are important because they can be related with the last findings in the biological substrate of aggressive behaviors and thus in the treatment of impulsivity.

CONCEPT OF IMPULSIVITY FROM THE NEUROSCIENCES FIELD

During the last two decades, neurochemical and neuroanatomical investigations, that have attempted to cla-

TABLE 2. Differences impulsion vs compulsion

Impulsion	*Compulsion*
Phenomenological differences	
Pleasant	Unpleasant
Objetive: to satisfy	Objetive: reduce anxiety
Stimulating behavior	Anxious behavior
Egosyntonic	Egodystonic, absurd for patient
Psudoresistance	Resistance
Shame linked to the sensation of lack of control, but not to the nature of the behavior itself	Shame linked to the performance or ritual, that the patient considers ridiculous and illogical
Intrusiveness*	Intrusiveness
Neurochemical difference	
Serotoninergic alterations	*Serotoninergic alterations*
m-cpp ($5HT_{1D}$ agonist) → euphoria. Il suggests hypoactivated serotoninergic receptor (Hollander et al.,1994)	m-cpp → increase of compulsions. Suggests hyperactivated receptor (Hollander et al., 1992)
Platelet MAO low in ludopathic and bulimic subjects (Carrasco et al., 2000)	Non-clarifying data on platelet MAO, although in general high of normal
Alteracions in other neurotransmitters	
Noradrenaline (Coccaro et al., 1991) Dopamine (Linnoila, 1983) Acetylcoline (Steinberg et al, 1997)	
Pharmacological differences	
Response with SSRI (bulimia, ludopathy), rapid in 10-15 days, but tends to lose consistency	Responses with SSRI, slow reduction of the symptoms (4-8 weeks), but it is maintained in a lasting way
Neuroanatomical differences	
No orbitofrontal hyperactivity has been demonstrated**	Cognitive dysfunctions suggests insufficiencies in corticofrontal functioning (Gastó, 1995)***

*At times, the behavior is hidden as it is inadequate, but it is not considered ridiculous[31]. **Preliminary data on global hypofunction of prefrontal areas (Intrator et al.), together with anxiety levels that reflect hypofunction in certain limbic areas. ***Hypothesis: the pathogeny of the compulsive symptoms would be related with alterations of the basal ganglia and their connections with prefrontal areas, together with hyperactivation of limbic areas related with anxiety.

rify the knowledge of the neurobiological bases of impulsivity, have multiplied[21,31]. Among these, studies with animal models, the use of various pharmacological tools that manipulate specific neurotransmission pathways (to induce or block behaviors) and non-invasive imaging techniques can be mentioned.

Most of the studies have focused on aggressive or suicidal behaviors. This is reasonable as a first approach, however it cannot be obviated that in many cases aggressiveness is not synonymous of impulsivity and that, in any event, it is only one of its possible manifestations[32].

When animal models are used to study impulsive behaviors, those used most have been those that induce aggressive behaviors in rodents, it being possible that they are modifications of behavior causing offensive aggressiveness (isolation models, resident-intruder paradigm or territorial aggression model, maternal aggressiveness) or defensive aggressiveness (footshock model, pain induced aggressiveness, etc.) and systems of cerebral manipulation, either pharmacological or surgical, such as lesions of the olfactory bulb.

However, aggressiveness in humans does not seem to closely correlate with any of these models. In an attempt to assimilate these types of animal aggressiveness with the human ones, some authors postulate an identification of predatory behavior with planned aggressiveness in humans (non-impulsive aggressiveness) and affective aggressive behavior with impulsive aggressiveness[33].

In a first approach to the physiopathology of aggressiveness, reference should be made to many neuroanatomical studies that have tried to clarify the cerebral anatomical location of these behaviors in both humans as well as in animals. Twenty five years ago, Goldstein[34] stated that aggressive behaviors in the human were caused by alterations in the limbic area and in the frontal-temporal lobes. In another classic study, Ursin[35] established the possible brain sites of offensive as well as defensive or predatory aggressive behavior in the rat, in addition to indicating other zones of the brain that exercised a modulating role in these behaviors.

Authors such as Herbert[36] emphasize the important role of the amygdala as a modulator of aggressive behaviors, generally through excitatory type connections. Thus, discrete lesions in the basolateral nucleus of the amygdala lead to a significant decrease in aggressive behaviors. On the other hand, authors such as Carrasco and Saiz[37] found that lesions in the orbitofrontal area, either due to traumatisms or tumors, caused impulsive episodes of aggressiveness, while it has been verified in later studies that specific lesions in the ventromedial areas of the hypothalamus also cause spontaneous aggressions.

Although many other neuroanatomical studies that supply specific data on possible locations of impulsive aggressive behaviors can be mentioned, we are still far from being able to offer a cerebral map that locates the anatomical bases of these behaviors and the different hypotheses posed only offer partial explanations. What is indisputable is the importance of these and other studies in the need to search for a complete explanation that includes neuroanatomical findings together with those supplied by genetics and neurochemistry. In regards to the latter, it has been proposed that the biological mechanism of these aggressive behaviors could be due to a lack of restraint in the control mechanisms of impulses possibly caused by a deficit in the serotoninergic functionalism. This is the hypothesis that is most greatly accepted at present, although it seems to be improbable that a single central neurotransmission system is involved in this type of disorder. In fact, the most recent theories propose a reduction of serotoninergic functionalism[38], together with hyperactivity of the central systems of noradrenergic and dopaminergic neurotransmission as basic neurochemical foundation of impulsive aggressiveness. In addition, other neurochemical systems, such as cholinergic, gabaergic, opioidergic or glutamatergic, could be involved in this type of behaviors, at least in animal models[39].

Serotoninergic neurotransmission, aggressiveness and impulsivity

As has been previously mentioned, the serotoninergic neurotransmission system has been the most widely studied when approaching aggressive behaviors in different models, and by extension, the system about which there is the greatest knowledge as a neurobiological base of impulsivity. Given the limitations of the present study, it is aimed to make a short review of the most relevant studies to offer a general viewpoint of what the many different investigations in this field could contribute to a complete concept of impulsivity.

The serotoninergic system is phylogenetically very old[40]. It exists in most of the invertebrate organisms and is involved in the regulation of important basal functions. One of the organisms studied most is the *Aplysia californica*, a marine mollusk. Kandel and Schwartz (1982)[41] reported that the response of retraction of its gill, a clearly motor process, was mediated by serotonin release.

From the ontogenic perspective, it must be considered that the 5-HT systems are not totally developed at birth (Azmitia and Whitaker, 1991)[42]. In addition, the receptors where the molecules of serotonin act have different sensitivity levels at birth and in the adult stage.

For this and other reasons, the serotoninergic system has been outlined as a key element in conducts considered as primitive, universal and in some way efficacious ones from the evolutionist point of view, as is the case of impulsive behaviors. And although we should not forget that attributing a behavior to a single neurotransmission system is a simplification, this can be useful to try to find pharmacological interventions as in the case of the H2 agonists in peptic ulcer or Beta blockers in the treatment of HBP.

The many studies that have tried to relate impulsive behaviors with serotoninergic dysfunction have used several parameters, as can be seen in table 3.

TABLE 3. Parameters used int he assessment of the ser otoniner gic systems

Serotonin levels (5-HT) in brain
5-hidroxy-indolacetic acid levels (5-HIAA) in CSF (final metabolic product of serotonin)
Monoamino-oxidasa (MAO) activity in platelets (indirect measurement of brain serotoninergic functionalism)
Binding of radioligands in brain
Density of 5-HT receptors in central nervous system
Response to agents that examine serotoninergic functionalism
Indirect assessment of serotoninergic functionalism by neuroendocrine tests

Studying the cerebral levels of 5-HT, Coccaro[43] observed that a decrease in these levels in the limbic-hypothalamus system was associated with suicidal or impulsive type aggressive behaviors in individuals with affective or personality disorders.

In regards to the study of the 5-HIAA levels in CSF with aggressive or violent behaviors, two studies are already considered as classics in this material, one performed by Linnoila et al. and another by Virkkunen et al., in the University of Helsinki. Linnoila et al., in 1983[44] studied this biochemical marker in the CSF of 36 male murderers who were divided into two groups: nine patients classified as non-impulsive (when there was clear premeditation in their violent acts) and 27 as impulsive (in whose attacks there was no provocation and they did not know their victims, and who fulfilled DSM-III criteria of intermittent explosive disorder or an explosive disorder of the personality). The results of the study showed lower levels in the impulsive group patients and even more in those who had committed more than one crime. On their part, Virkkunen et al., in 1987[45], carried out an assay of the levels of metabolites of different monoamines in 20 males with a background of impulsive like pyromania in comparison with two other groups; 20 violent patients with a history of marked aggressiveness and 10 control patients. The results of the study showed lower levels of 5-HIAA and of the metabolite of noradrenaline 3-methoxy-4-hydroxy-phenyl glycol (MHPG) in CSF in the group of pyromaniacs than in the two other groups. On the contrary, the levels of homovalinic acid (HVA), a metabolite of dopamine, were greater in the pyromaniac group. These results stress the close relationship between serotoninergic functionalism and aggressive behaviors in patients with a deficit type disorder in the control of impulses.

All the data commented on indicate that the reduction of the 5-HIAA rates in the CSF specifically correlates with impulsive type aggressiveness and not with premeditated one, so that some authors have proposed these biochemical changes as a biological marker of impulsive aggressiveness.

In regards to monoamine-oxidase (MAO) activity in platelets, a decrease of this activity has been described in violent individuals[46], as well as in patients with impulse control disorders. Brunner et al.[47] published the case of a Dutch family in which 14 members were arrested due to continuous violent acts, among them murder attempts, violation and pyromania. In this family, a genetic mutation linked to the X chromosome that caused an alteration in the functionality of the MAO-A enzyme was detected and this, in turn, caused a serotoninergic dysfunction, manifested by an increase of the urinary concentration of the substrates of this enzyme and a decrease in 5-HIAA concentration in CSF.

The findings provided through the response to agents that examine serotoninergic functionalism are based on the fact that pharmacological disturbance of a specific system can reveal information regarding the functional integrity of both an altered system directly as well as of the systems that modulate or are modulated by it.

Experimental studies in animals performed by Porsolt[48] verify that a wide range of aggressive behaviors could converge in the hypothalamic area and amygdala, this being susceptible to manipulation with serotoninergic agents. In fact, the serotoninergic system modulates the functionalism of the central catecholaminergic system, which is directly responsible for certain aggressive behaviors by an exaggerated response to external stimuli. Following these postulates, it has been observed that: subjecting the experimental animal to tryptophan deficient diets, very similar behaviors to those observed after injuring the nucleus of the raphe are induced. This structure is very rich in neuronal serotoninergic somas, while diets rich in tryptophan or in 5-hydroxytryptophan inhibit muricidal behaviors to a certain degree[49].

The use of pharmacological agents with activity on the 5-HT receptors or on other structures of the serotoninergic synapsis have also confirmed the facts previously described, as is the case of buspirone, a non-benzodiazepinic anxiolytic agent of the azaspirodecanedione family, partial 5-HT_{1A} agonist, that has been demonstrated to have an anti-aggressive effect[50-52].

Considering the importance of the 5-HT_1 receptors in the control of aggressiveness, a new group of serotoninergics, specifically designed for the treatment of aggressiveness, called «serenoids», has been developed since the middle of the 1980's[53]. These drugs, derived from phenylpiperazines, are characterized by their agonist action of the 5-$HT_{1A/1B}$ receptors. However, their excellent preclinical profile was not verified in the clinical development phase. Coccaro et al.[54] demonstrated in the human that desensitization of 5-HT_{1A} receptors in the limbic-hypothalamic system was associated with an increase in impulsive type aggressive behavior in patients with personality disorder. On the other hand, suppression of the gene that expresses the 5-HT_{1B} receptor in the mouse causes an increase in attack behaviors in certain animal models. This has led the 5-$HT_{1A/1B}$ receptors to be considered the true modulators of aggressive behaviors. The data existing on the possible role of other 5-HT receptors in the modulation of aggressive behaviors are not so eloquent.

Another group of drugs with serotoninergic activity that have been widely studied in animal models are anti-

depressant agents, especially selective serotonin reuptake inhibitors (SSRI), above all fluoxetine, observing a reduction in aggressive behavior in several species and with the use of different animal models[55-56]. These results have also been observed with other SSRI agents such as fluvoxamine, sertraline and paroxetine, and with the nordrenalina and serotonine reuptake inhibitor, venlafaxine.

The results published by Mitchell and Redfern[57], who stated that the SSRI and other antidepressants only had an anti-aggressive action in rats subjected to resident -intruder paradigm in acute administration while chronic administration caused, on the contrary, a significant increase in aggressive behavior, are controversial.

Other neurotransmission systems involved in impulsive aggressiveness

Biochemical observations, both in the experimental animal as well as clinically have shown elevated levels of MHPG, a metabolite of noradrenaline, in the urine of suicides[58] and in the CSF of military personnel with accentuated aggressive behaviors, as well as indirect modifications of the noradrenergic functionalism in certain patients (Coccaro et al. observed an increase in the growth hormone response to the administration of clonidine, an alpha-2 adrenergic agonist, in patients with impulsive aggressiveness)[59].

Recently, a relationship has been established between the development of aggressive behaviors and catechol-O-methyl-transferase (COMT), an enzyme responsible for some metabolic pathways of noradrenaline. Strous et al.[60] have found that schizophrenic patients who phenotypically express a low activity variant of COMT show greater risk of presenting aggressiveness than those homocygotic individuals for the allele of the high activity COMT.

Siever and Davis[61] propose that the involvement of the different neurotransmission systems could give rise to different clinical manifestations in the expression of aggressiveness. Thus, serotoninergic hypofunction would be associated more to impulsive type aggressive behaviors than to premeditated ones, while noradrenergic activity would mark the direction of the aggressiveness, so that when this system is hyperfunctioning, aggressiveness would be directed towards the external environment, and when this is hypofunctioning, violence would be directed towards the individual him/herself, as occurs in aggressive patients with risk of suicide.

The noradrenergic system seems to also modulate dopaminergic activity. Furthermore, brain areas that are so important in the genesis of aggressive behaviors, as the amygdala, are extremely rich in dopaminergic neurons. Thus, the dopaminergic system has been involved in the origin of aggressiveness, in addition to other neurotransmission systems, such as the opioidergic, cholinergic and gabaergic ones, together with sexual hormones, corticosteroids, glucose metabolism and cholesterol levels.

Relationship of the intraneuronal neurotransmission systems

The most recent information indicates that the relationships between some of the neurotransmission systems commented on previously may have common converging points in the intracellular transduction chains linked to their respective receptors. Although in the specific case of aggressiveness, these mechanisms still have not been studied, some interesting data have been supplied on certain disorders of impulse control as well as with the use of some therapeutic tools used in the management of aggressiveness as is the case of the antidepressants, lithium salts, anti-seizure or antipsychotic agents[62], which suggest the possible participation of these systems in the origin of some types of aggressive behaviors.

According to different hypotheses, most of the antidepressants, regardless of the family to which they belong, as well as other psychodrugs such as lithium, carbamazepine or valproic acid, agents that are also effective in the management of aggressiveness, would share a series of mechanisms of common intraneuronal action far from the receptors of the neuronal surface. Corroboration of the involvement of these intracellular loci in the etiology of aggressiveness is a future subject that should be mastered by the present neuroscience.

Genetics and impulsivity

Several studies have shown the relationship of aggressiveness with the Y chromosome, since patients diagnosed of the XYY syndrome not only show low intellectual coefficient but also normally aggressive behavior[63].

Nielsen et al.[64] found a close correlation between impulsive type aggressive individuals with genetic polymorphism in the expression of the gene of triptophan hydroxylase and low levels of 5-HIAA in CSF. This fact involves a clear relationship between the gene that limits the passage of serotonine synthesis and the explosive aggressive behaviors. In addition, lower risk of suicide by violent methods in women is known, a fact that could be genetically related with higher levels of 5-HIAA in CSF and greater serotoninergic activity than in the man[65].

Genetic studies comparing monocygotic and dicygotic twins, adoption studies and other methods have revealed various contributions of inherence to impulsive behavior[66-69]. Gottesman[70] compared monocygotic and dicygotic twins with the MMPI, finding that the monocygotics were more similar in self-control in addition to other items of that scale. In another study between pairs of twins, Eaves et al.[71] revealed that impatience and scarce planning have an important genetic component, as did Eysenck[19]. However, a specific pattern of inheritance or its contribution to other key elements such as education has not been described.

CONCLUSIONS AND DIRECTIONS TOWARDS FUTURE INVESTIGATIONS

As we have pointed out at the onset of this present review, in spite of the wide use and management of the concept of impulsivity, no definition has been accepted by consensus, not even in the DSM-IV itself that mentions the term as a diagnostic criterion of several disorders[71].

After verifying the numerous and diverse definitions that have been published in the literature, we understand that the difficulty when operatively defining impulsivity comes from the fact that most of its definitions are based, in turn, on other concepts having limited scientific value, such as the will, so that attempts to apply measurement scales, develop animal models or perform pharmacological or genetic investigations cannot generate coherent results.

In spite of the advances in neurobiology or other basic sciences, we continue to use a term that appeared in the XIX century that refers to a qualitative disorder of the will when the pathology of the will has disappeared from most of the reference books presently used in psychiatry. In addition, it is not possible to establish a correlation between the original term, the concept (to which it referred to in one period with certain beliefs and knowledge) and varied behaviors that have been integrated in different syndromic groups that probably share a common biological substrate.

In spite of everything, we feel that it is essential to recover the study of impulsivity, since it is a problem that occurs daily in the common clinical practice, with important legal, social and management repercussions that are found both pharmacologically as well as psychotherapeutically.

There is a wide field of investigation, with promising biological findings, aimed not only at the field of aggressive behaviors but also at finding impulsivity indexes that integrate behavioral, biological and social aspects. However, it is necessary to abandon vague definitions, based on theoretical speculations, and to try to indicate the principal aspects that we want to assess, unifying them to obtain a definition of impulsivity as a symptom, which is not obstructed by rigid nosological outlines.

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