



International Journal of Psychology and Psychoanalysis

LETTER TO THE EDITOR

Tourette's Syndrome: A Search for the Etiological Component and for the Improvement of the Effectiveness of the Treatment

Santiago Aliaga*

Department of Neuroscientist, Physiologist, Psychologist and Ph.D. in Psychosomatic Medicine and Psychoanalysis, California-CA, USA



*Corresponding author: Santiago Aliaga, Department of Neuroscientist, Physiologist, Psychologist and Ph.D. in Psychosomatic Medicine and Psychoanalysis, Private Research, Analysis of the Understanding of the Mental Health in the Different Countries of America and Europe, California-CA, USA, E-mail: maiconleali@hotmail.com

Introduction

Characterized by repeated contractions of muscle groups, tics are associated with psychic tensions that must be alleviated by persistent motor actions. In this disorder, even knowing that the movements happen, the individual does not strive to prevent these events from manifesting. In summary we can talk about two fundamental types of tics [1]; 1) *Motor tic* (muscular contractions); 2) *Phonic tic* (vocalizations). The most frequent cases are among the motor tics [2], being the contraction of the facial muscles, elevation of the eyebrows, persistent olfaction, gesticulations, elevation of the shoulder and successive movements of the head.

The problem of lack of adequate understanding of the cerebral origin of this disorder has generated a great deviation of consensus from the world scientific community regarding the etiological bases of the famous discovery of Tourette, a French physician who published nine cases of the disorder in 1885, termed as *maladie des tics convulsifs avec coprolalie* ("compulsive tics disease with coprolalia"). Between the two theoretical strands in the field of neurological clinic, a logical and well justified path between the two must be pointed out. If on the one hand we speak of an organic origin of the disorder, on the other we speak of a manifestation of a psychological nature. But it is not an honest attitude to describe a pathophysiological basis for the persistence of motor acts or psychopathological basis for the psychic tensions that produce them, without an adequate theoretical design and well-defined research indicators.

It is within this context that two intrinsic relations of the disorder are framed, which can help in the search for plausible answers and to extend the horizon of the treatments. One of the relationships to be considered is *anxiety*. Stress has often been linked to tics due to increased seizures in cases of anxiety caused by external pressures or during episodes of nervousness. Another relationship to be considered is *obsessive compulsive disorder* (OCD). This relationship is justified by the fact that many individuals diagnosed with tics have had symptoms such as depression, social phobia, panic disorder, dysmorphic disorder, generalized mania and drug and/or alcohol use, such behaviors that frequently appear in patients with OCD. However, these relationships do not imply that all patients with OCD have tics or vice versa [3]. It is important to be clear that these relationships are part of a methodological research work focused on the search for an etiological explanation for the disorder.

Based on the two relations mentioned here, it is necessary to talk about the neural bases of the motor functions and their psychic correlates. The Primary Motor Cortex (PMC) produces motor actions and the neural circuits establish intrinsic patterns between neurochemical events and psychological processes related to bodily acts. An act or the accomplishment of a task can immediately associate itself with a specific idea, expectation or sensation, according to the person's memories. In the skin and in the muscles, the *mechanical-electrical transduction* mediated by fibers type A, la



Citation: Aliaga S (2018) Tourette's Syndrome: A Search for the Etiological Component and for the Improvement of the Effectiveness of the Treatment. Int J Psychol Psychoanal 4:029. doi.org/10.23937/2572-4037.1510029

Accepted: June 11, 2018; **Published:** June 13, 2018

Copyright: © 2018 Aliaga S. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

and II, produces the somesthetic perception of vibratory pressure and proprioception. Since the entire cerebral cortex is mapped according to the different parts of the body, movements and the perception of movements must occur simultaneously, so that the individual can intervene in the motor act itself to adapt it to the demands of the environment [4]. Thus individual actions can be controlled according to the bodily sensations that are being continuously integrated into the brain. This fact brings us very close to the true etiology of Tourette's syndrome, considering that there is a very close relationship between sensations and motor acts, and that anxious states increase the seizures. This condition makes it perfectly acceptable that the origin of the disorder is in the disproportion of the intensity of association between motor action and the psychic or emotional tension of the individual presenting it.

It is not, therefore, a flaw in a neural circuit as some neurologists claim, just as it is not just about psychic events such as stress or intense emotional states, as most psychiatrists claim. It turns out that the triggering agent of tics has been confused with the aggravating factor of the disorder, a fact that has contributed to the misconception of many medical literature on this topic in mental health [5]. First, there is an exacerbation of the neurochemical action of the brain in the face of the induction of voluntary movements, which leads to the almost automatic production of muscular contractions, followed by the tension release through somesthetic perception of the same. Neurological studies of brain activity in patients with OCD have shown that there is excessive functionality of certain areas of the brain, indicating a *disproportion of associative intensity between the induction of motor acts and the psychic representation of the body segments*, where tic attacks may appear. Another important fact is that the neurotransmitter function of 5-hydroxytryptamine (5-HT) in the brain is also associated with the production of the symptoms of compulsive disorder, among which are the onset of tics. (Other neurotransmitters such as *dopamine* and *norepinephrine* have also been considered in certain studies, but did not lead to a definitive conclusion).

Individuals with the above characteristics are more predisposed to these types of disorders, in which the high intensity with which they react to stress is always a very strong characteristic. Given the relationship of the tics disorder with compulsive symptoms, it can be understood that the disorder should not be attributed to a hereditary failure of certain neuronal or axonic structures, just as we should not limit the origin of the problem to merely psychiatric conditions. So far, no genetic modification has been found, by which we may regard the disorder as unquestionably hereditary. (There are Tourette's syndrome patients whose family genealogies did not present any other confirmed cases.) What may be related to heredity is the likelihood of developing an *extreme endogenous representation of motor actions*

in the brain of the person with the syndrome, not the alleged defects of the neural structures involved in motor control (many assume abnormalities of the connections with the nuclei of the base, which together with the cerebellum, control the actions produced by the PMC) [6]. This is where *we must discern very well between structural abnormality and neurotransmitter hypersensitivity*. In contact with the external environment and internal sensations, the brain reacts irregularly, attempting a non-ordinary adaptation, creating compulsive neural and psychic demands [7,8]. This means that disorder can be triggered even though neurotransmitter rates are normal in the brain. After reaching adulthood, the brain adapts to neurochemical events and synaptic function becomes less intense, causing a decrease in tic episodes. However, in cases where the increase in presynaptic release becomes significant, the brain can adapt itself by learning to perform intense neurochemical processes, leading to increased tics.

Therefore, it is more honest to speak of the *inheritance of a genetic pre-disposition in responding intensely to the environment associated with an abnormality of the adaptive functions of the brain*, which is expressed through the deviant production of psychomotricity. (It is important to remember that compulsive vocalizations may also be present in the syndrome, which implies that the issue is not simply a failure of movement control. The disorder involves a *spectrum of individual relationship with the stimuli of the environment and with perception of the body itself, in the contact with subjective experiences*.) In the medium and long term, considering the "failure of the brain circuit" in the neurochemical sense of the term becomes plausible, given the relevance of the neurotransmitter intensity to which the brain has been subjected for years. This fact is sufficient to adapt the cerebral cortex to intense neurochemical events which, for a considerable time, cause the CNS to react more intensely than necessary to different situations.

Likewise, body sensations and memories of muscle contractions become persistent psychological ways of experiencing somesthetic perceptions through repeated (or manic) movements of the different bodily regions mapped in the brain. In this case, motor acts become the way in which the individual relates compulsively to his own bodily sensations. These processes, therefore, constitute the aggravating factors of the disorder, since the excessive reactions of the person to certain situations of stress produce intense thoughts, that in turn increase more the anxiety, taking place a vicious circle of difficult reversion [9]. This fact implies that the tics are triggered by the genetic predisposition of the individual, whose intensity varies from person to person. The origin of the disorder should no longer be justified on the basis of the intensity of neural reaction associated with 5-HT only (for example, the data reflect little precision in [10]), because there are many people with elevated

levels of this neurotransmitter who do not have symptoms of tics or OCD. 5-HT may increase in certain people with such predisposition, as well as amplify a predisposition already existing in others. So, there are people with tics or OCD who do not have high rates of 5-HT in the brain. This clinical condition shows that the treatment of tics may involve a pharmacological spectrum associated with psychotherapy.

The idea “dubious effectiveness of psychotherapeutic treatment” [11] is the result of a lack of understanding of the clinical reality of the disorder, especially in relation to the psychic mechanisms of anxiety production, which should be the focus of the study, in order to guide the patient about the control. The use of the drug and the personal understanding of the manifestations of tension contribute to the patient’s understanding of their real mental condition and, consequently, helps to suppress the cause of persistent unpleasant motor effects.

As mentioned earlier, many cases of tics reach the stage of *mania*, where muscle contractions identify with an implicit memory of repetitive motor acts aiming at the constant experience of somesthetic perception of the muscle groups that are being activated, according to the individual level of neuropsychological and neuropsychiatric involvement of the patient (the process of motor reaction to the environment becomes an addiction of neuromotor expression for certain bodily acts). In this case the tics constitute a process of tensional release of *topographic origin* (cerebral mapping of the body segments), whose motor induction is involuntary at the beginning (many patients of the disorder think that the tics are totally voluntary acts due to the difficulty of distinguishing between the motor commands and the mechanoreceptor processes involved in the simultaneous perception of muscle contraction). Here the object of primordial inquiry is the *manic repetition of the memory of the somesthetic perception*, in which is the “significant source” of the obsessive motor actions of the individual.

After the analysis presented here we can argue that the treatment of Tourette’s syndrome should include concomitant pharmacological and psychotherapeutic techniques. In this clinical modality, there is a positive relationship between the pharmacological prescription and the psychotherapeutic method (according to clinical specifications for both, given the characteristics and different intensities of the disorder). In many cases, the use of medications is not necessary. But in all cases, it is very important to consider psychological intervention, to define individual performance assessments in various life contexts, to teach that sensations are perceptions related to the meaningful expressions of the body. Obviously, this concept should be emphasized in psychotherapeutic treatment according to the frequency of tic manifestations and the level of bodily acts of compulsion.

Contrary to the doubt of the neurological clinic regarding the efficacy of psychotherapy, pharmacological administration should be adequately adjusted to the intensity of compulsions and, from that, to establish a compatible psychotherapeutic line in the clinical approach. The idea of ineffectiveness of psychotherapy finds precedents in the fact that its accomplishment has not counted on a previous medical direction. An integrated work is needed between neurological and psychotherapeutic treatment. While the pharmacological treatment adjusts to the intensity of the disorder, psychotherapy investigates the intrinsic motives of the psychic evolution of the tension levels that give rise to such intensity. This integrated procedure in mental health enables the patient to better understand their own mental condition and behavioral tendency, such as they are in the complex constitution of the etiology of tics. In addition, this proposal is an invitation to the patient to reflect on their real needs and to take action in accordance with their personal expectations.

This implies that there are not only limiting factors in the understanding of the syndrome and the responses to treatment (between doctors and psychologists), but also limiting factors in understanding the treatment itself (patients). The behavioral and mental difficulties of the patient extend to the field of Psychology. Consequently, a more comprehensive clinical posture is required from Neurology and Psychiatry in relation to the study of the intrinsic mechanisms present in the origin and intensification of tics. It is in this line of research that the studies should be leveraged, in search of better models of treatment that integrate psychotherapy and pharmacology in a single approach. While medications reduce levels of anxiety, psychotherapy teaches the patient how to act and think about times when seizures tend to reappear.

References

1. Yorston G, Hindley N (1998) Étude sur une affection nerveuse caractérisée par l’incoordination motrice accompagnée d’écholalie et de coprolalie. *Hist Psychiatry* 9: 97-101.
2. Jankovic J (2001) Tourette’s syndrome-Review article. *The New England Journal of Medicine* 16: 1184-1192.
3. Lebowitz ER, Motlagh MG, Katsoyich L, King RA, Lombroso PJ, et al. (2012) Tourette syndrome in youth with and without obsessive compulsive disorder and attention deficit hyperactivity disorder. *Eur Child Adolesc Psychiatry* 21: 451-457.
4. Belluscio BA, Jin L, Watters V, Lee TH, Hallett M (2011) Sensory sensitivity to external stimuli in Tourette Syndrome patients. *Mov Disord* 26: 2538-2543.
5. Scahill L, Woods DW, Himle MB, Peterson AL, Wilhelm S, et al. (2013) Current controversies on the role of behavior therapy in Tourette Syndrome. *Mov Disord* 28: 1179-1183.
6. Singer HS (2013) The Neurochemistry of Tourette Syndrome. In: Martino D, Leckman JF, Tourette Syndrome. Oxford University Press, New York, 276-300.
7. Masselink B, Singer HS (2014) Tourette’s Disorder. *Ency-*

- lopedia of Clinical Psychology, 1-6.
8. McGuire JF, Piacentini J, Brennan EA, Lewin AB, Murphy TK, et al. (2014) A meta-analysis of behavior therapy for Tourette Syndrome. *J Psychiatr Res* 50: 106-112.
 9. Walkup JT, Ferrão Y, Leckman JF, Stein DJ, Singer H (2010) Tic disorders: Some key issues for DSM-V. *Depress Anxiety* 27: 600-610.
 10. Singer HS (2001) The treatment of tics. *Curr Neurol Neurosci Rep* 1: 195-202.
 11. Maurice Victor, Allan H Ropper (2002) Adams and Victor's Manual of neurology. The McGraw-Hill Companies 7: 70-72.