

Abnormal Movements in Psychiatry: Semiological Description and Therapeutic Proposal

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Abstract

Review Article

The classic distinction between psychiatry and neurology is blurring in part because of a better understanding of neuropsychiatric disorders in neurological diseases. Certain structures, including the basal ganglia, are involved in both motor and behavioral processes. Parkinson's disease and Tourette's disease are good examples of these two facets. Other movement pathologies are rarer or less known. Abnormal movements are symptoms that are frequently found in psychiatry, and are most often secondary to the use of an antipsychotic treatment. However, there is no easy-to-use tool to characterize abnormal movements or to adopt an unequivocal course of action, whereas the identification of the movement, its analysis and the delay of management can condition the prognosis. Through three clinical vignettes, this work will allow us to describe semiologically the abnormal movements most frequently found in psychiatry and to propose an optimal management of these movements as soon as they occur and probably to reduce the misuse of anticholinergic treatment.

Keywords: Abnormal movements, psychiatry, semiological characterization, dyskinesia, antipsychotic.

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I. INTRODUCTION

Abnormal movements are symptoms frequently found in psychiatry, most often of iatrogenic origin (secondary to antipsychotic treatment) 1. They can also be caused by a dysfunction, a lesion or a degenerative pathology affecting the motor circuits of the basal ganglia, also called the extrapyramidal system 2. Other etiologies can also be evoked.

The basal ganglia are responsible for the programming and automatic execution of learned motor sequences. Abnormal movements correspond to a disorder in the programming and/or execution of movement during these different stages. They are characterized by the fact that they are hardly or not at all controlled by the will and the fact that it occurs in the absence of paralysis.

The positive diagnosis of abnormal movements is purely clinical, but the etiological diagnosis sometimes requires additional biological, radiological or even electro-physiological examinations.

Regarding the context of this work, the interest is focused on precise and clear terminologies allowing

clarifying the abnormal movements in order to integrate them in the current psychiatric semiology.

II. Clinical Vignettes

1. Clinical vignette N° 1

Mrs F. is 65 years old, divorced, mother of 3 children. She has a history of obsessive-compulsive disorder since she was 17 years old and a mother with Huntington's disease.

She was hospitalized in the psychiatric ward for management of mood sadness, insomnia and verbalization of suicidal ideations.

In 2013, and following the death of her husband, the patient would have moved with her children into the deceased's home. However, since her children work all day long, she spent her days alone, went out shopping, traveled and she reported that she felt very well.

The onset of her disorder dates back to the year of 2018, when the patient would have started to present sudden, involuntary movements especially on the

fingers, she would make facial grimacing, her voice would also have changed, she reports that she felt a generalized fatigue.

She consulted a neurologist who put her on Risperidone at a rate of 1.5 mg/d. The patient then became sad, insomniac and having little appetite. She stopped taking the Risperidone, claiming that it was responsible for her insomnia.

The day before her admission, she had a crying crisis, started to verbalize suicidal ideations by saying that she wanted to die and that it was her only wish, which required her hospitalization at the psychiatric hospital Ar-razi in Salé.

The psychiatric interview on admission revealed a motorically calm patient with facial myoclonia and a depressive syndrome.

The standard workup came back without any particularities. A neurological opinion with neurological examination and neurocognitive tests were in favor of Huntington's disease.

Given the thymic improvement, after a 35-day hospital stay, she was stabilized on Escitalopram 20 mg/d, Quetiapine 300 mg/d and Alprazolam 1mg/d.

2. Clinical vignette N° 2:

Mr A. is 22 years old, single, with no notable history.

He was hospitalized at the psychiatric hospital Ar-razi in Salé for the management of a tendency to run away and aggressiveness within the framework of a schizophrenia evolving since 1 year.

The patient consulted 1 year before his admission to the hospital and was put on Olanzapine with good improvement of his clinical condition.

The treatment was stopped after 2 months, a period of time after which he became isolated, incurious, persecuted against those around him, refusing to share family meals, fearing to be bewitched, which motivated a consultation where the patient was put on Risperidone 4 mg/d with regression of the persecution. The treatment was then stopped by the patient because of a significant weight gain that was not quantified.

20 days before his admission, his condition worsened, he became hallucinated, soliloquizing, verbalizing persecution, he remained alone in a family apartment from which he has escaped and then found himself in a police station from where he was admitted to the Ar-razi hospital in Salé.

The admission interview revealed a calm, reticent patient who expressed himself with

mannerisms, his thoughts were not very penetrable, he adopted attitudes of listening.

A pre-therapeutic assessment was requested and returned without any particularities. The patient was put on Amisulpride 600 mg/d.

The patient presented 20 days after the beginning of the treatment a segmental rigidity at rest in all the directions of the passive movement, which yielded by jerks (phenomenon of the cogwheel). The patient appeared neuroleptic with trunk flexed forward, limbs in half-flexion. The CPK assay was requested and came back at 1277 IU/l with normal constants.

Therefore, it was decided that the extrapyramidal rigidity he presented was secondary to the Amisulpride intake, hence its discontinuation, and the patient was put on rehydration (2L of water/day by mouth) and on Benzodiazepines (15 mg of Diazepam in degression during 10 days) until the normalization of CPK. Clozapine was subsequently considered.

His clinical condition improved and he was discharged from the hospital after 64 days on Clozapine 300 mg/d.

3. Clinical vignette N° 3

Mr O. is 24 years old; single, with a history of problematic use of tobacco and Trihexyphenidyl hydrochloride at a rate of 50 mg/d.

He was admitted to our institution for the management of heteroaggressivity, verbalization of delirious and obscene remarks, and clastic crises within the framework of a schizophrenia evolving for 4 years.

The patient had already been hospitalized in a psychiatric ward where the diagnosis of schizophrenia had been made and after a 6-week stay, he was stabilized on Amisulpride 800 mg/d and Chlorpromazine 300 mg/d.

9 months before his admission, following a therapeutic deviation, he would have become more and more persecuted against his entourage and hallucinated. This decompensation was accompanied by extremely violent heteroaggressive acts, which led the family to leave the home and leave him in the company of his older brother, who was unable to manage his crises, hence his hospitalization.

On admission, the patient was calm with a delusional persecution syndrome, a hallucinatory syndrome with emotional blunting and disturbed sleep.

The patient was put on 10mg/d of Olanzapine and Diazepam in degression. During his stay, he presented with abnormal, involuntary, repetitive

movements involving the trunk and limbs of choreoathetosis type.

A brain MRI was done, which came back normal. A neurology opinion was done and the movements were qualified as late dyskinesias secondary to the antipsychotic drug. The course of action was to reduce the dose of the antipsychotic.

In the absence of improvement, we had to switch to Clozapine which allowed a clear improvement of his clinical condition and of the abnormal movements and this under a dose of Clozapine 300 mg/d in association with Propranolol and vitamin E.

III. Semiological description of the different types of abnormal movements

1. Hypokinetic movements

A. Akinesia

This is the reduction and especially the slow initiation of gestures of progressive installation. Movements are rare, slow, and sometimes impossible.

It manifests itself especially for automatic movements with an alteration of walking (walking with small steps where the initiation of walking is difficult and loss of arm swing) and by a frozen face with a loss of automatic movements of the face usually accompanying emotions or speech and a disappearance or rarity of blinking.

The akinesia is inconsistent over time with circadian fluctuations and is increased or triggered by emotion, fatigue.

B. Extrapyramidal rigidity

Extrapyramidal rigidity or hypertonia sets in gradually; it corresponds to the exaggerated and permanent increase in muscle tone of a muscle at rest.

It is present in all directions of passive movement, also in the flexors and extensors, and in the entire range of motion. It is plastic, compared to a lead pipe, and yields in jerks called the cogwheel phenomenon.

Rigidity predominates in the flexors explaining the general attitude of parkinsonians, with the trunk flexed forward and the limbs in half-flexion.

C. Cataplexy

This is the term given to sudden muscle weakness triggered by strong emotions such as laughter, anger and surprise. The loss of muscle tone can range from just noticeable weakness in the facial muscles to weakness in the knees to total collapse to the floor.

Speech may be slurred and vision impaired (double vision, inability to concentrate) while hearing and consciousness are not impaired.

When cataplexy is present, it is extremely rare that it is an isolated symptom; most who have typical cataplexy will also have symptoms of narcolepsy.

D. Catalepsy

Catalepsy refers to a condition that is characterized by so-called plastic muscle rigidity. It is defined by a punctual loss of voluntary contraction of the muscles of the body. The attitude of the cataleptic is similar to the position of statues. The body stops in the middle of a movement and is frozen during its action.

The person concerned cannot move for a period of time ranging from a few minutes to several hours or even days. In some cases of catalepsy, a third person may move a limb of the patient, such as the arm, and place it in a certain position, which is then held and frozen.

2. Hyperkinetic movements

A. Akathisia

Akathisia is characterized by subjective complaints of impatience and at least one of the following observed movements: impatient movements or swaying of the legs while sitting, rocking of one foot over the other or stomping while standing, need to walk to relieve impatience, inability to sit or stand still for several minutes.

Typically, symptoms occur within 4 weeks of starting or increasing doses of neuroleptic therapy and may sometimes follow a decrease in medication taken to treat or prevent acute extrapyramidal symptoms (e.g., anticholinergics).

B. Athetosis

By definition, this is the inability to maintain a stable position. Athetosis is clinically characterized by a slow, twisting, arrhythmic, continuous and reptatory movement.

This movement is mainly distal, predominantly in the extremities, and oscillates between hyperextension and flexion. It can also affect the trunk and occasionally the face.

It is often increased by activation of another part of the body, stress, fatigue, sensory stimuli and intellectual activities. However, it disappears during sleep.

C. Ballism

Ballism, which literally means "throwing", is an involuntary movement of a limb, extremely abrupt, explosive, dramatic, arrhythmic and very ample, with a tendency to flex and roll the limb on its axis, readily

triggered by an action and increased by stress, predominating at the root of the limbs.

When only one limb is affected, we speak of monoballism and of hemiballism when one hemibody is involved (the most frequent situation) 3.

D. Chorea

This is a term derived from the Greek meaning dancing. Choreic movements are arrhythmic, abrupt, unpredictable, most often generalized, interfering with normal voluntary movement. They are classically accentuated by stress, fatigue and attention, diminished in a calm environment and absent during sleep 3.

There is a notable impairment of voluntary movement with difficulty in initiating movement. The face is typically the site of grimaces that can modify the physiognomy but also sometimes of an impairment of phonation or swallowing.

At the level of the upper limbs, these are flexion-extension movements of one or more fingers, pronation-supination movements of the forearms, and shoulder shrugs.

At the level of the lower limbs, the involuntary movements are generally less intense but they can nevertheless hinder walking, which is bouncy, giving it a dancing appearance.

Chorea is constantly associated with a very clear hypotonia, explaining the amplitude of the movements, the decrease of the gripping strength and the fatigability.

The osteotendinous reflexes are generally wide and pendulous.

E. Dystonia

Dystonia is clinically defined by the occurrence of sustained involuntary muscle contractions, generating repetitive twisting movements and/or abnormal postures, of agonist and antagonist muscle groups, typically reproductive in the same patient 4.

We speak of acute dystonia when these contractions are brief (a few seconds), prolonged (a few minutes) or sustained (a few hours) and of chronic dystonia when these contractions are permanent, accompanied by disability and pain, often unbearable. Classically, they disappear during sleep and are favored by action, posture maintenance and stress.

F. Myoclonus

Myoclonus are sudden and brief muscular contractions, characterized by involuntary jerks, mostly spontaneous, localized to a part of a muscle, to a muscle

or a group of muscles, repeated, sometimes rhythmic, without segmental displacement.

They carry out a single displacement with return to the initial position. They are often localized to a segment and predominate on a part of the body, the root of a limb.

The myoclonus is favored by sensory or sensitive stimulation and by surprise. The slightest fine and precise movement gives rise to parasitic jerks that disorganize voluntary movements as well as walking, automatic gestures (coughing, yawning) and the gestures of everyday life (dressing, washing).

G. Stereotypies

Stereotypy, etymologically the "frozen character", concerns repeated, involuntary, rhythmic movements that can exist independently, likely to interfere with or intertwine with an adapted movement.

Three types are distinguished according to their location (simple movements, manipulation of one's own body or manipulation of external objects). These movements last from a few seconds to several minutes and occur several times a day.

They are mostly associated with periods of excitement and more rarely with fatigue or boredom. They do not disrupt ongoing activities. These movements are considered as stereotypies if they last for more than one month.

H. Tics

They correspond to an involuntary or semi-involuntary, brief, sudden, intermittent and stereotyped motor or vocal disorder concerning a group of muscles in functional connection and having a role in social relations. They can be transitorily controlled by the will with a rebound phenomenon and an increasing internal tension.

They rarely persist during sleep and are increased by stress and the relaxation that follows stress, decreased by concentration and distraction. They evolve in a fluctuating manner with periods of calm and resurgence in bursts.

I. Tremor

Tremor is defined as a small, regular oscillation of the body or part of the body around its equilibrium position. It is characterized by its rapidity, periodicity, amplitude and circumstances of onset. The rhythmic character of the tremor distinguishes it from most other abnormal movements.

Two entities are classically distinguished

- The resting tremor, occurring outside of any voluntary activation and

- Action tremor, which occurs during a voluntary muscle contraction.

Within action tremors, we dissociate attitude or postural tremors (during posture maintenance) from kinetic tremors (during movement).

IV. Etiologies of abnormal movements

1. Iatrogeny

Psychotropic drug-induced abnormal movements can be divided into acute and delayed movement disorders. Acute movement disorders, such as acute dystonia, akathisia, Parkinsonism, and myoclonus, begin rapidly after taking dopamine receptor blocking agents, often an antipsychotic drug.

Late movement disorders such as tardive dyskinesia and tardive dystonia occur months to years after the use of these agents 6.

A. Antipsychotics

Antipsychotic drugs have as their primary action, the dopamine D2 agonist, which is the source of the desired clinical effects but also the desirable effects, depending on the brain area affected.

Decreased dopamine levels in the nigro-striatal dopamine pathway, which is responsible for motor function, lead to the development of extrapyramidal neurological effects 7.

One of the best known extrapyramidal syndromes is the parkinsonian syndrome. Antipsychotics can also induce akathisia.

Signs of early dyskinesias may also appear very quickly after the start of treatment. Subsequently, late dyskinesias may appear 8, 9.

B. Antidepressants

They mainly induce tremors but also a parkinsonian syndrome. Tricyclic antidepressants such as amitriptyline or imipramine can induce postural tremor in the hands and aggravate the physiological tremor.

With SSRIs, postural or action tremor may be seen in up to 20% of people. 10 The onset is usually within one to two months after introduction and disappears within one month after discontinuation.

C. Thymoregulators

Tremor secondary to lithium has been the subject of several studies. Clinically, it is the most frequently observed abnormal movement, occurring in nearly 30% of those treated [10-11]. It mainly involves the hands, with a fine amplitude. It usually begins when the drug is introduced and varies in intensity and

frequency throughout the day, without any relation to the time of intake [12].

Valproic acid induces a tremor that is similar to essential tremor, which often appears within three to six months of initiation of therapy. Other anticonvulsant thymoregulatory drugs used such as gabapentin, carbamazepine and lamotrigine cause tremor.

2. Psychiatric Pathology

A. Schizophrenia

In the 19th century, from the first descriptions of schizophrenia, motor disorders such as posture and movement disorders were taken into account.

Then, from the 1950s onwards, the motor side-effects of neuroleptic treatments seem to have eclipsed these motor disorders as an integral part of the clinical picture of the disease 13.

B. Somatoform Disorders

The frequency of psychogenic abnormal movements is approximately 2-3% in a general consultation but may be as high as 30% in specialized centers (Carson *et al.*, 2000).

There are four diagnostic categories of these disorders: malingering, factitious disorder, somatic manifestations of an anxiety disorder and conversion.

The semiology of these movements is extremely varied and complex; they evolve in a paroxysmal mode: tremor, jerks, myoclonus, intermittent dystonic attacks, tics [15]. They often affect language and walking. The symptoms often occur suddenly, rapidly reaching their peak in severity and disability.

C. Catatonic syndrome

This is a psychiatric syndrome known since the beginning of the 19th century whose clinical characterization remains delicate.

It is a relatively frequent condition, generally acute in onset, characterized mainly by mutism with inertia, catalepsy with waxy flexibility and conservation of spontaneous or imposed attitudes by passive mobilization, echopraxia, bizarre, stereotyped acts, with mannerism, and even brutal impulsive behaviors of agitation and violence.

3. Neurological pathology with psychiatric expression

In many movement pathologies, there are psychiatric manifestations, thus indicating the existence of a common origin of the psychological suffering and the neurological symptoms.

The most common origin is a dysfunction of the basal ganglia which participate in the motor program but also in cognition and behavior conveying emotional and motivational information.

A. Tourette's disease

Tourette's syndrome is a neurological disorder manifested by motor and vocal or phonic tics usually beginning in childhood.

The cause of this disorder is still unknown, but the disorder appears to be hereditary in the majority of patients. In the absence of a specific biomarker, diagnosis depends on careful evaluation of the patient's symptoms and signs by an experienced clinician.

B. Huntington's Disease

Huntington's disease is a genetic, autosomal dominant neurodegenerative disorder caused by an expansion of CAG triplets in the huntingtin gene, located on the short arm of chromosome 4.

It occurs most frequently between the ages of 30 and 45, without gender or ethnic predominance. Huntington's disease classically causes intellectual deterioration, motor disorders, and severe psychiatric disorders that progress to a progressive worsening of symptoms, leading to a bedridden state and death, on average about 20 years after the onset of symptoms [17, 18].

The clinical diagnosis is therefore based on a triad composed of choreic movements, the most characteristic symptoms, cognitive impairment marked by disorganization and attention disorders, and psychiatric impairment with a depressive state, apathy, irritability and heteroaggressiveness in the foreground [19, 20].

C. Wilson's disease

This is a genetic, autosomal recessive disorder caused by a mutation in the Wilson gene, located on chromosome 13 and coding for an intracellular protein that allows the elimination of copper in the bile, so this leads to a tissue accumulation of copper mainly in the liver, brain and eye.

It is an initially hepatic disease, and then in the absence of diagnosis extra-hepatic symptoms appear, mainly neurological and psychiatric.

The three main neurological pictures, often associated with each other, are a generalized or focal dystonic syndrome with sometimes choreic movements, an intentional and attitude tremor sometimes associated with ataxia and a parkinsonian syndrome [21].

Psychiatric symptoms are present in a quarter of the patients affected, such as bipolar disorder (20%) or a major depressive state [22].

V. Course of action

The clinical evaluation of abnormal movement in psychiatry is a task that may seem difficult, but is useful in many ways. A characterization of these movements is necessary.

A first approach of this characterization can be realized at the time of the anamnesis, it is about the description of the movement, the evolution in time and the conditions.

The initial semiological analysis must be rigorous, with the aim of characterizing the abnormal movement and identifying the syndromic framework, if necessary.

The etiological investigation will lead to the elimination of an iatrogenic or lesional cause, and then somatic causes will have to be discussed.

The clinical examination of abnormal movements is ideally part of a general and complete neurological examination.

Complementary examinations are an aid to the clinical description and a basis for comparison between the different movements.

The presence of certain abnormalities on brain MRI, with and without gadolinium injection, is sometimes specific to a particular etiology or type of pathology.

A brain scan with DaTSCAN® (presynaptic ligand of the dopamine transporter) should only be performed if there is a doubt between a Parkinsonian tremor related to Parkinson's disease and a Parkinsonian tremor related to neuroleptics.

The video recording preserves the entire clinical description, allowing a semiological reanalysis afterwards, but also a comparative, evolutionary and post-therapeutic control.

The surface electromyogram is useful when the clinical examination is not univocal. It quantifies muscle activities in time and space, according to muscle groups.

At the biological level, in the first instance, a blood count, a blood ionogram, a renal check-up, an inflammatory check-up, a fasting blood sugar level, a calcemia, an albuminemia and a TSH are taken.

As a second line of treatment, carboxyhemoglobin, ceruloplasmin, cupremia, ACAN, native anti-DNA, and rheumatoid factor are measured on specialized advice.

The acquisition of certain clinical reflexes should help the psychiatrist to identify abnormal movements, to favour an adequate multidisciplinary psychiatric, neurological and possibly genetic management.

Nevertheless, some abnormal movements common in psychiatry may require a non-specialized therapeutic management or simply a medication readjustment without necessarily a specialized opinion, the main thing being to make sure of the iatrogeny in this case. In practice, we can propose the beginning of management for each of the movements mentioned earlier in the article.

A. Rigidity and akinesia

Treatment is possible with anticholinergic drugs such as tropatepin, trihexyphenidyl or biperidene. It is also possible to change molecule knowing that second generation neuroleptics present less this type of effect like risperidone, olanzapine, amisulpride. At high doses, these self-correcting mechanisms are outdated; only clozapine has a self-correcting mechanism independent of the dose used.

B. Cataplexy

First-line treatment of cataplexy is based on serotonin reuptake inhibitors, primarily fluoxetine, by a norepinephrine reuptake inhibitor, viloxazine, and by a mixed norepinephrine and serotonin reuptake inhibitor, venlafaxine.

Tricyclic antidepressants, especially clomipramine, are very active, but have anticholinergic side effects in medium to high doses. Behavioral treatment consists of avoiding the occasions that trigger cataplexy [23].

C. Akathisia

Management depends on the extent of the akathisia and the patient's psychiatric or neurologic illness. The medications causing the disorder can be managed by reducing neuroleptic doses, switching to newer neuroleptics, or even prescribing an atypical neuroleptic such as clozapine.

Effective drug treatments to relieve akathisia can also be prescribed, such as beta-blockers (propranolol) or dopaminergic depleters (tetrabenazine), which are often very effective.

D. Athetosis

The medications used are the same as for generalized dystonia. They are neuroleptics, tetrabenazine, benzodiazepines and baclofen. Lesional surgery (thalamotomy) has been used with positive results 3.

E. Ballismus

The treatment of choice is the use of neuroleptics such as haloperidol in rapidly increasing doses (49). In a second phase, the neuroleptic drug is reduced, allowing the resurgence of discrete abnormal movements, usually choreic. In the following weeks, the progressive weaning of the neuroleptics is instituted. In case the persistence of the ballism requires it, the replacement of classical neuroleptics by tetrabenazine or by atypical neuroleptics such as clozapine is undertaken.

F. Chorea

Any choreic syndrome requiring symptomatic treatment should be referred to a neurologist. Treatment, the patient should be referred to a neurologist for a first-line prescription of tetrabenazine. In the absence of efficacy or in the presence of associated behavioral disorders, as is sometimes the case in Huntington's disease, an atypical neuroleptic should be preferred.

G. Dystonia

Botulinum toxin is the treatment of choice for persistent focal dystonia. A dystonia of a limb beginning in a young adult or in a child must justify a trial of L-dopa in order not to ignore a dopa-sensitive dystonia. In case of more diffuse involvement, trihexyphenidyl may improve symptoms before discussing deep brain stimulation of the medial pallidum on a case-by-case basis and according to the underlying cause. Physiotherapy is, in the majority of cases, an indispensable complementary treatment.

H. Myoclonus

It is necessary to exclude a metabolic or iatrogenic cause. While waiting for a better identification of the generator of the myoclonus, clonazepam or piracetam can be tried in progressive doses.

I. Stereotypies

In the majority of cases, stereotypies have no psychosocial or physical impact in a patient who has had normal psychomotor development and require no treatment. In cases where stereotypies are frequent and especially if there is an associated comorbidity such as compulsive and obsessive disorders, behavioral therapy may be indicated.

J. Tics

In practice, only troublesome tics should be treated with medication. Aripiprazole is currently the first-line neuroleptic to be prescribed. For mild to moderate tics, supportive psychotherapy and cognitive-behavioral therapy may be offered.

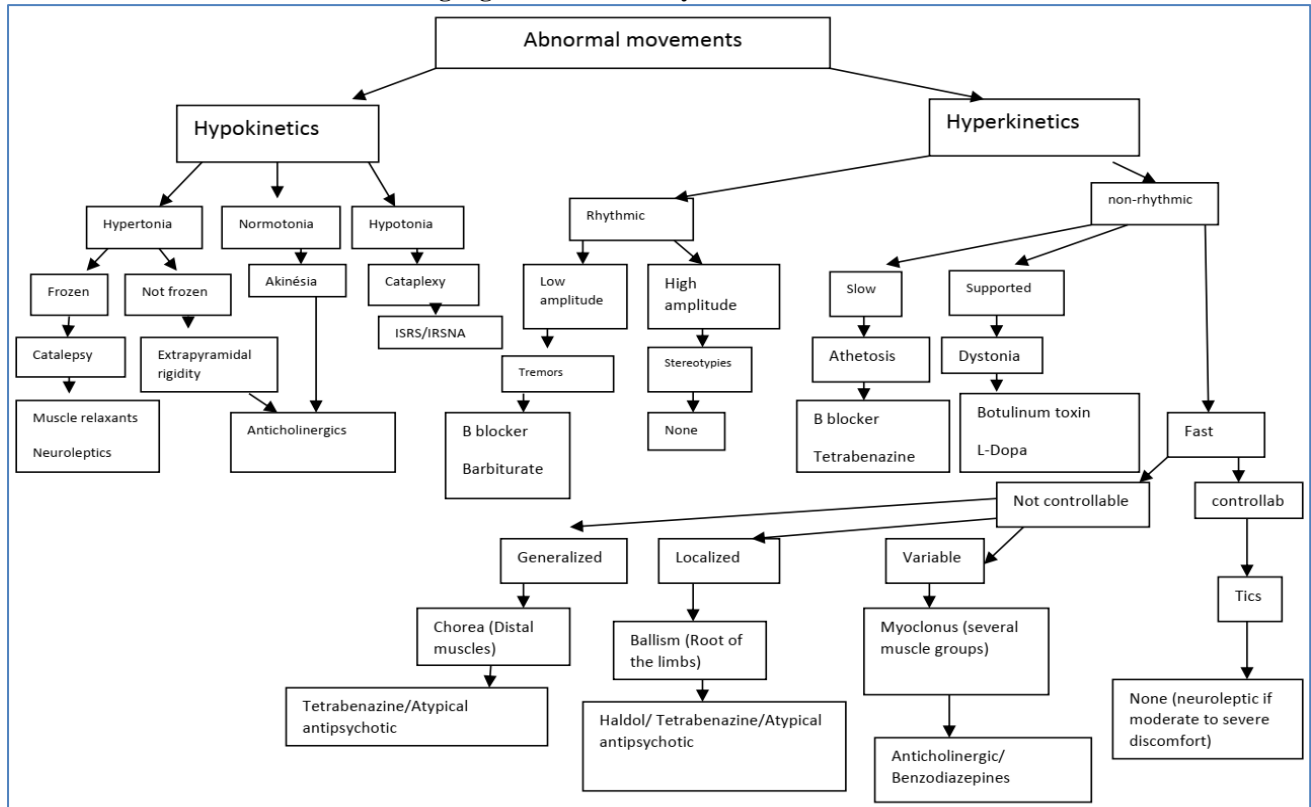
K. Tremor

If not related to a side effect, the relay is taken up by the neurologist who may be assisted by a

percutaneous polygraph recording of the tremor to

clarify its characteristics and subsequent management.

Decision-making algorithm to identify and treat abnormal movement



VI. CONCLUSION

Parkinson's and Tourette's diseases illustrate the involvement of certain brain structures, including the basal ganglia, in both motor skills and behavior.

However, psychiatrists must have the reflex to evoke them because the psychiatric picture can be isolated and the absence of specific treatment can worsen the prognosis, for example in Wilson's disease. Thus, recognition by the psychiatrist of certain disorders: tremor (Parkinson's), tics (Gille de la Tourette), dystonia (Wilson's, Huntington's) and choreic movements (Huntington's) is essential.

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