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Extrapyramidal examinations in psychiatry

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Abstract
Increased motor tone, variations in movement quantity and speed, and involuntary motor activity are examples of extrapyramidal symptoms. They consist of two categories of symptoms and associated conditions: hyperkinetic (like Parkinson's disease) and hypokinetic (like Huntington's disease). This article discusses the main extrapyramidal movement disorders, the neurology behind extrapyramidal disorders, the importance of extrapyramidal signals in the major mental illnesses, and how to elicit extrapyramidal signs.

Keywords: Extrapyramidal signs, parkinsonian signs, tremor, rigidity, motor retardation, neurologic examination

Introduction
Extrapyramidal motor performance is the most crucial aspect of a neurologic evaluation for everyday psychiatric practice out of all the components. Speech, mood, and spontaneous motor activity are just a few of the mental status exam elements that might be impacted by extrapyramidal function. Psychiatric patients frequently have extrapyramidal disorders (both iatrogenic and idiopathic), and extrapyramidal disorder patients frequently have psychiatric syndromes. As a result, it has long been understood that the basal ganglia play a crucial role in mental illnesses. Psychiatric drugs frequently have an impact on extrapyramidal signs and symptoms, so it's critical in clinical psychiatry to monitor these treatment-related side effects.
The term "extrapyramidal" describes a classic description of poor motor control, which is typically associated with dysfunction of the basal ganglia. The two main categories of extrapyramidal symptoms are hypokinetic (seen in Parkinson's disease and following brief exposure to dopamine-blocking medications) and hyperkinetic (found in Huntington's disease and following prolonged exposure to dopamine-blocking medications). Extrapyramidal symptoms are primarily observed, with the remaining ones being briefly examined directly.

Relevant Anatomy and Physiology
Extrapyramidal function refers to control of motor tone, the amount and velocity of movement, and the suppression of undesirable motor activity. Motor systems other than the pyramidal (corticospinal) tract are understandably termed *extrapyramidal*, but we should note that the term was never anatomically meaningful and has become more anomalous. Motor coordination, planning, sequencing, and inhibition, which we tend to link with the cerebellum and the frontal lobes, are not routinely considered extrapyramidal, but do involve the basal ganglia as well as cerebellum and neocortex. Also, the pyramidal tract is now known to be only one of several tracts subserving voluntary motor activity.
The forebrain basal ganglia are the brain structures most responsible for extrapyramidal control. As discussed in our review of the voluntary motor system, several parallel circuits or loops help control motor planning and control [4], decision-making, and behavioral reinforcement. These circuits involve mostly frontal cortex, cerebellum, thalamus, and limbic structures in addition to basal ganglia. They also communicate with each other, with other limbic structures to link with motivation, and with other cortical areas to link with other planning activities. Extrapyramidal control is effected by gamma motor neurons activating intrafusal muscle fibers. If we need an anatomically rational name for the systems controlling extrapyramidal motor function, it might be “basal ganglia motor system” or “intrafusal motor control system.”

Although we touched on basal ganglia anatomy in a previous "Psychiatry and Neurology" series article, we will delve a bit further here. Anatomic terms for the basal ganglia can be confusing and bear repeating. The basal ganglia consist of several functionally related nuclei. Basal ganglia circuitry is topographically and functionally organized. The basal ganglia include the caudate, putamen, nucleus accumbens, globus pallidus and subthalamic nucleus of the forebrain, and the substantia nigra and ventral tegmental area of the midbrain. The striatum (sometimes called the neostriatum) refers to the caudate and putamen. The globus pallidus (sometimes called the paleostriatum) is the third major basal ganglion (nucleus). Corpus striatum is sometimes used to refer to the caudate, putamen, and globus pallidus together. Although not included in basal ganglia, the pedunculopontine nucleus and several thalamic nuclei are highly integrated in basal ganglia motor control circuits. The ventral striatum, consisting of the olfactory tubercle and part of the nucleus accumbens, is quite distinct and part of the limbic system.

Because extrapyramidal motor control involves widely distributed circuits, it is difficult to assign roles to particular anatomic locations. However, naturally occurring and neurosurgical lesions do occur in particular locations, so we still care about the roles of these nodes in the broader circuits controlling movement. The caudate nucleus is involved in the more cognitive tasks of motor planning and goal selection. The putamen enhances sensorimotor coordination and plays a role in habit learning. The globus pallidus is involved in communication among nuclei of the basal ganglia. The subthalamic nucleus receives inputs from thalamus, pedunculopontine nucleus, and cerebral cortex, and projects to substantia nigra, pedunculopontine nucleus, and globus pallidus. The nucleus accumbens and striatum (caudate and putamen) receive most of their connections from the cortex. The striatum receives glutamatergic, dopaminergic, and serotonergic input. Striatal neurons predominantly use gamma-aminobutyric acid (GABA), but acetylcholine and others are involved.

Neurosurgical treatment of parkinsonism involves placing stimulating electrodes in one of four sites. Deep brain stimulation (DBS) of the ventral intermediate thalamic nucleus improves tremor. Stimulation of the pedunculopontine nucleus helps with gait freezing. DBS of the subthalamic nucleus and globus pallidum can be helpful with any parkinsonian signs and symptoms, and are currently the preferred sites. Electrodes are usually placed bilaterally. In Parkinson's disease, the movement disorder to which the technique has been most applied, DBS plus medication is more effective than medication alone, but the combination also increases the risk of serious adverse events.

Signs and Symptoms Movement Disorders

Parkinson's disease and related disorders

Many are misled by the terms *parkinsonian*, *parkinsonism*, and *Parkinson's disease*. Parkinson's disease refers to a specific disease, the most common form of idiopathic parkinsonism. The other two terms refer to the broader syndrome, with similar signs and symptoms but different causes. The most common other parkinsonian syndrome is drug-induced parkinsonism. Along with Parkinson's disease, idiopathic parkinsonism includes the "Parkinson-plus" or atypical Parkinson's diseases, which include multiple system atrophy, progressive supranuclear palsy, corticobasal degeneration, and Lewy body dementia. Ischemic lesions can also cause parkinsonism.

Parkinson's disease often is heralded by autonomic symptoms (orthostatic hypotension, constipation), sleep changes (REM behavior disorder), and olfactory deficits, but typically the first symptom is a rest tremor of one hand. Cardinal signs of Parkinson's disease follow the mnemonic TRAP: tremor (rest), rigidity (lead-pipe), akinesia (or bradykinesia), and postural instability. Other "parkinsonian" signs include hypophonia, micrographia, blunted affect, low spontaneous blink rate, stooped posture, short-stepped gait, motor restlessness (akathisia), and dystonia. If the presentation is typical and there is nothing in the history pointing to another possible cause, the diagnosis is very likely to be Parkinson's disease. If there is cerebellar ataxia, prominent early autonomic dysfunction (e.g., falls due to orthostatic hypotension), early dementia, pyramidal tract signs, myoclonus, supranuclear gaze palsy, and prominent apraxia, the other parkinsonian diseases should be more closely considered.

Dystonia

Idiopathic dystonia (sustained unwanted contraction of a muscle or muscle group) is easily missed and misdiagnosed. It presents with a variety of complaints, including cramping, inability to perform some task (particularly when it involves repetitive, fine, manual movements), or unsightly movements. Dystonic movements can take on a cyclic pattern and can be difficult to distinguish from tremor, and the peculiar movements (in the absence of localizing neurologic findings or abnormal test results) are often mistaken as psychogenic. The affected muscle group may be rigid, but parkinsonian signs are usually not prominent. Exposures to dopamine-blocking drugs (even metoclopramide or prochlorperazine) can precipitate dystonia.

Chorea and athetosis

The most important hyperkinetic movement disorders in psychiatry feature chorea and athetosis. These movements are frequently combined into choreoathetotic movements, in which an abrupt irregular movement (chorea) seems to launch a writhing or stretching movement (athetosis). Most prominent choreoathetotic movements are attributable to chronic exposure to dopamine-blocking drugs (tardive dyskinesia) or diphenyhydantoin (Dilantin) or to acute exposure to stimulants (sometimes called "crack dancing"). However, they can also be caused by Huntington's disease (distinguishable by family history and genetic testing), Sydenham's chorea (associated with a history of streptococcal infection), Wilson's disease (a disorder of copper metabolism, usually of adolescent onset), vascular lesions, and numerous other rare disorders.

Tic disorders

The tic disorders are another type of hyperkinetic movement disorder, dominated by motor and vocal tics. These are named Tourette's disorder or simply tic disorder, according to whether or not the patient has ever had vocal tics. These are often comorbid with obsessive-compulsive disorder; in fact, some consider tics to be a form of compulsion. They are a feature of pediatric autoimmune neuropsychiatric disorder associated with streptococcal infection (PANDAS).

They also tend to be found in attention deficit hyperactivity disorder, even without exposure to stimulants. Stimulants can precipitate tics, which may or may not remit after the stimulants are stopped.

Dementia and Movement Disorders

Any of the major dementing illnesses can include parkinsonian signs. Lewy body dementia tends to show early parkinsonian signs, which are among its diagnostic criteria. Vascular dementia may include early parkinsonian signs. Although not famous for it, extrapyramidal movement disorders can precede the emergence of frontotemporal dementia. Later in its course, Alzheimer's dementia often includes prominent parkinsonian signs.

Schizophrenia and Movement Disorders

Consistent with a long history of clinical observation, recent studies show that up to a third of patients with recent-onset, never-medicated schizophrenia have parkinsonian signs. Extrapyramidal signs in first-break schizophrenia patients previously untreated with antipsychotics predict a lower response to typical and atypical antipsychotic drugs.

Dyskinesia or "tardive-like dyskinesia" may be observed in mild form in never-medicated schizophrenia, and more prominently in older and more chronic (but still never-medicated) patients as well as some normal persons, especially the immatureand elderly. Diagnostically, such movements (particularly chorea) raise the possibilities of Huntington's disease (distinguishable by family history and genetic testing), Sydenham's chorea, PANDAS, cerebrovascular disease, and numerous other uncommon entities.

Depression and Parkinsonism

Depression and parkinsonism are statistically related and share clinical features. Depression affects nearly half of those with Parkinson's disease. Also, idiopathic major depression and parkinsonism share certain signs and symptoms, such as psychomotor retardation/ bradykinesia, blunted affect/abulia/hypomimia, sleep disturbances, and apathy. Patients with melancholicor catatonic depression in particular may have parkinsonian signs. Sometimes these patients are in an early phase of idiopathic parkinsonism, but often the parkinsonism resolves with the depressive episode.

Drug-Induced Movement Disorders

Dopamine-blocking drugs, such as the antipsychotics (both typical and atypical) and some anti-emetic drugs, are the most recognized causes of secondary extrapyramidal movement disorders. Parkinsonian side effects almost always arise within one week of reaching the necessary dose or concentration of the drug, are reversible, and can include any of the motor signs and symptoms of Parkinson's disease. When parkinsonian side effects are caused or aggravated by dopamine blocking drugs, dyskinesia rarely emerges before four months on the drug, but the effects often persist after dopamine blocking drugs have been discontinued. Tremor and postural instability are less prominent in drug-induced parkinsonism than in Parkinson's disease. Dopamine-depleting drugs, such as reserpine and tetrabenazine, can have the same side effects.

Stimulant drugs, such as cocaine and amphetamines, also can have similar effects, but via opposing mechanisms. In acute withdrawal, a stimulant user can have a parkinsonian presentation, presumably caused by dopamine depletion. While intoxicated and often persisting into withdrawal, stimulants can cause dyskinesia similar to tardive dyskinesia.

Antidepressants are known to occasionally cause both parkinsonism and dyskinesia. This has little to do with dose or duration of treatment. Duloxetine is over-represented among case reports of antidepressant-associated movement disorders, but there are no systematic comparisons. Serotonin syndrome, another side effect of some antidepressants, can include prominent dyskinetic movements, so it is important to look for other serotonin syndrome findings, such as unstable vital signs and delirium. Also it should be noted that antidepressants often induce postural or action tremors, which should be distinguished from rest tremor.

Calcium channel blockers may reduce the risk and slow the progression of Parkinson's disease, but occasionally appear to have caused (or precipitated) parkinsonism.

Many other drugs also can have parkinsonism effects, including some antihistamines, antiarrythmics, anticonvulsants, and cholinomimetics.

Examination Methods

Poverty of movement may present to simple observation as diminished gesturing, diminished arm swing while walking, or diminished facial expression (blunted affect or hypomimia, which is a reduced degree of facial expression).

Hypophonia (soft, thin speech) also shows itself in the interview. If asked to speak louder, the patient usually does so only temporarily. Of course, patients sometimes deliberately speak softly, but the distinction is almost always obvious.

Micrographia is evident in almost any writing sample. It becomes more pronounced after the first few letters, so very brief samples may fail to capture the effect. Some healthy people are habitually micrographic, but in parkinsonism the writing becomes smaller and less legible with continuation.

Posture is stooped and unstable. Stooped posture is evident while standing or sitting, with relative flexion throughout the spine. The chin may rest on the chest while seated. While standing comfortably, a light shove to the chest or back (sufficient to displace the trunk an inch or two) causes the patient to step backwards or forwards (respectively) or even to lose balance.

Gait is notable for short steps, diminished arm swing, and festination. Watch the patient walk casually, and/or ask the patient to walk five steps, turn around, and walk back to you. If the leading foot's heel strikes less than a foot-length before the trailing foot's toe, stride length is short. If the heel does not land beyond the tip of the other foot, stride length is markedly shortened. Festination is a tottering forward-leaning gait, in which fairly rapid but short steps fail to keep up with the center of gravity. To execute a 180-degree turn, several steps are required. The patient often looks unable to rotate at the waist, sometimes called "en bloc turning."

The rigidity of parkinsonism is of the lead-pipe variety. The relaxed limb (or neck), moved passively, gives a constant level of resistance throughout the range of motion, hence the term *lead pipe*. Sometimes the rigidity can only be appreciated with reinforcement, in which the patient is simultaneously engaged in some other motor task with the

other hand. One often hears talk of "cogwheel" rigidity, which refers to a ratcheting sensation produced by palpable tremor. Lead-pipe rigidity reflects parkinsonism, with or without "cog-wheeling." Lead-pipe rigidity is distinct from "clasp-knife" rigidity (initial resistance that gives way at a certain point in the range of motion). Clasp-knife rigidity, as well as velocity-dependent rigidity (less resistance when the imposed movement is slower), reflects spasticity (e.g., stroke, multiple sclerosis, cerebral palsy) rather than parkinsonism. Finally, cognitively impaired patients often seem to have difficulty relaxing as requested. Active but involuntary resistance to all movement is called gegenhalten; this tends to improve with distraction.

The tremor of parkinsonism is of the rest variety. Rest tremor is noted in a limb or other body part at rest, particularly with distraction (patients often attempt to quell the tremor). It can be intensified by anxiety. When the patient extends the hands unsupported, rest tremor ceases, but it may resume after several seconds maintaining the posture. Rest tremor is slow (about 4-6 cycles per second). When rest tremor is prominent in the second digit, so that it rubs against the palmar surface of the thumb, this is called "pill-rolling." There are several other tremors that can be confused with rest tremor, but only two other types of tremor are seen with any frequency. Postural tremor appears promptly with unsupported posture (such as hands extended), and is more rapid than rest tremor. This is the most common tremor in psychiatry (and in general) and might be attributed to essential tremor, anxiety, caffeine, theophylline, lithium, valproate, beta-adrenergic agonists, and/or withdrawl from alcohol, benzodiazepines, or barbituates. Postural tremor is often mislabeled intention tremor. Intention tremor is seen when a body part (usually a finger) approaches a target and consists of rhythmic shifting from one side to the other of the target (dysmetria).

The glabellar (Myerson) reflex is found in parkinsonism of various etiologies, as well as in diffuse degenerative brain diseases. After explanation and request to look forward and to try not to blink, tap firmly on the glabella (mid-line low forehead) six times about once every 1 to 2 seconds. A blink or partial blink is expected on the first 1 to 3 taps, but continuing to blink thereafter is abnormal. Approach in such a way that your hand is out of the patient's view (to avoid the blink response to visible "threat"). It may have high sensitivity for Parkinson's disease [50] but some studies find similar prevalence among the parkinsonian disorders [51]. It is not uncommon for patients with other parkinsonian signs to show no response at all to even the first tap. Patients with dyskinesia sometimes blink early, as if anticipating the next tap.

Blink rate is a rough indicator of dopaminergic tone. A very low rate of spontaneous blinking (<10 per minute) is found in the hypokinetic (parkinsonian) movement disorders, shortly after beginning dopamine-blocking drugs and in some other cases of psychomotor retardation. Rapid blinking (>30 blinks per minute) can be elicited by dopamine agonist drugs and is sometimes found in hyperkinetic movement disorders, including Tourette's disorder, tardive dyskinesia, and withdrawal dyskinesia. Numerous other factors, including ocular irritation, anxiety, hostility, and speaking, can also influence blink rate.

Motor restlessness (akathisia) may seem an odd companion to the other hypokinetic motor signs, but it frequently accompanies both the idiopathic parkinsonian disorders and drug-induced parkinsonism. It consists of a subjective urge to move and an objective excess of movement (usually walking). The patient may complain of anxiety, being unable to rest or relax, or may report simply enjoying walking, but will usually endorse feeling more comfortable walking than sitting. Any excess of movement, as long as the excess movement does not consist of tremor, chorea, athetosis, or tic, could be akathisia. The most convincing sign of akathisia is pacing so persistent that the patient marches in place when required to stand still.

Dystonia, the unintended, sustained contraction of a muscle group, can accompany hyperkinetic or hypokinetic syndromes. Thus, dystonia can follow a particular exposure to antipsychotic (acute dystonia), but it can also be a part of tardive dyskinesia (tardive dystonia). Writer's cramp and charley horse are common types of dystonia. In serious psychiatric and neurologic conditions, dystonia more often affects the axial muscles, causing torticollis or rotation of the lower spine. The extraocular muscles may also be involved ("oculogyric crisis"). Movement tends to elicit dystonia, and anxiety often intensifies it.

Huntingtonian signs (dyskinetic signs, abnormal involuntary movements) are those characterizing Huntington's disease, several other rare hyperkinetic movement disorders, or resulting from either acute exposure to dopamine agonist drugs or chronic exposure to dopamine-blocking drugs (tardive dyskinesia). These signs include chorea, athetosis, tic, and ballistic movements. Specific examination protocols, such as the Abnormal Involuntary Movement Scale, consist of observation with some distraction procedures. They are important to use routinely, particularly when the patient has or is at risk for these abnormalities.

Chorea is a sudden, quick, involuntary movement that can affect any skeletal muscle (including the diaphragm). It is not rhythmic like tremor and not as purposeful-looking or patterned as tic. Athetosis is a slower movement, often called writhing or undulating, that resembles deliberate stretching of a muscle group. It is most noticeable in the shoulders, fingers, and lower face.

Tic is also sudden, quick, and not rhythmic, but more patterned and purposeful in appearance. Common tics include throat clearing, vigorous blinking, sniffing, shaking the head as if to reset the hair, and vocalizing. Vocal tics, characteristic of Tourette's syndrome, more often consist of humming and meaningless syllables than the famous blurting of expletives. Tics are sometimes seen in tardive dyskinesia, but much less often than chorea

Hemiballism is sometimes seen alone or in combination with other involuntary movements. These are abrupt, large movements of the arm and/or leg, typically including extension and abduction of the limb as if throwing. Sometimes these are precipitated by similar deliberate movements (e.g., a ballistic arm movement triggered by deliberate reaching). Basal ganglia lesions, not limited to the subthalamic nucleus of tradition, cause ballistic movements, but they are also associated with hyperglycemia and human immunodeficiency infection (HIV).

Spontaneous abnormal movements, such as tremor, dystonia, chorea, tic, and athetosis, are often more evident when the patient is distracted. Structured examinations of movement (such as the Abnormal Involuntary Movement Scale) may include specific instructions for distraction, but a psychiatric interview or unobtrusive observation are at least as effective.

Movement Disorder Assessment Scales

Movement disorder assessment scales are widely available and often used in clinical as well as research settings. For hypokinetic or parkinsonian signs, these include the Unified Parkinson's Disease Rating Scale motor exam section, and the Simpson-Angus scale. For hyperkinetic or tardive-like signs, these include the Unified Huntington's Disease Rating Scale and Abnormal Involuntary Movement Scale. The Extrapyramidal Symptoms Rating Scale attempts to cover both hyperkinetic and hypokinetic signs.

These assessments can be sharpened with the use of some fairly simple instruments. Tremor measurement can be facilitated with an iPhone app. Scaling of movement velocity exploits the fact that healthy people moving between two targets have a maximal velocity proportionate to the distance between the targets, but that in parkinsonism there is no such relationship. Several aspects of parkinsonism can be assessed using a windows-based system. Writing on a digitizing pad produces sensitive data relative to subtle parkinsonism. Chorea can be quantified sensitively and accurately with instruments measuring force instability, or the ability to maintain a constant amount of motor force.

Conclusion

The extrapyramidal examination, which consists of observation and very simple tests, is important in understanding the mental status, making psychiatric diagnosis, selecting medication, and monitoring of the effects of medications.

Conflict of Interest

Not available

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