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The Phenomenology of Parkinson's Disease

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Abstract

The motor symptoms of Parkinson's disease are not limited to the cardinal symptoms of bradykinesia, rigidity, and resting tremor, but also include a variety of interrelated motor phenomena such as deficits in spatiotemporal planning and movement sequencing, scaling and timing of movements, and intermuscular coordination that can be clinically observed. While many of these phenomena overlap, a review of the full breadth of the motor phenomenon can aid in diagnosis and monitoring of disease progression.

Keywords

Parkinson's disease; phenomenology; symptoms; signs

The evaluation of a patient with a movement disorder is anchored in the phenomena observed by the clinician, and thus for the movement disorders neurologist it is the phenomenological classification (rather than neuroanatomic localization) that frames the diagnostic process. [1] In general, patients with Parkinson's disease (PD) move too little. Because it can be difficult to describe both the temporal and spatial aspects of voluntary movement in isolation, terms such as bradykinesia, akinesia, and hypokinesia are sometimes used interchangeably to describe the basic motor abnormalities seen in PD, with little regard to the differences between them. Yet a variety of related though clinically distinct motor control abnormalities occur in PD patients, and the richness of the phenomenology encompassed by these terms, as well as the subtler motor features that occur in PD, are often underappreciated and are seldom described together. This article will focus on the motor phenomenon *observed clinically* in PD, including the classic motor features of the disease and the associated lesser-cited symptoms of impaired motor control that occur (Table 1). Discussion of pathophysiology is for the most part beyond the scope of this review; however, a few key concepts are included to help to shed light on what is observed clinically. While many of the entities discussed are inextricably interrelated, an appreciation for the full

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breadth of the clinical phenomenon observed in PD can aid in diagnosis and monitoring of disease progression.

Akinesia

The term Akinesia, literally "without motion," can refer to *a decrease in automatic* movements, or a *failure or delay in the initiation* of willed movements. Even when sitting quietly or engaged in conversation, healthy people move quite frequently, unless they make a conscious attempt to suppress all movements. Changes in facial expression and small gesticulations and postural shifts are ubiquitous. However, in being so, they are usually not specifically noticed unless they are absent or markedly abnormal, as occurs in patients with PD. While evaluating the PD patient, the observant clinician will often notice akinesia in the form of a decrease in unconscious gesticulations with one or both hands (especially if the dominant hand predominantly affected), and decreased facial expression (hypomimia) and blink rate. Reduced frequency of swallowing is also a form of akinesia that can lead to sialorrhea. Decreased unconscious movements can contribute to pain and musculoskeletal problems such as frozen shoulder, [2] and nocturnal akinesia can lead to discomfort and reduced sleep quality. [3]

Akinesia can also refer to the failure or delay in the initiation of a willed movement. [4] Muscles selected but insufficiently energized to create movement at a given joint will produce recordable EMG activity (in the absence of or with a significant delay) before an observable movement. [4] Illustrative examples include the mild PD patient with a delay in the shoulder shrug on one side (a common early sign in PD), or the late stage PD patient who, in the medication "OFF" state, can exhibit an agonizingly long delay before a willed motor program is carried out. Motor blocks or freezing episodes have also been described as a form of akinesia, [5] though when occurring during an ongoing motor program or when shifting between motor programs rather that during movement initiation, additional pathological processes are likely contributing. [6]

In the research setting, akinesia usually refers to an increased reaction time, or the time from the onset of a sensory stimulus to the initiation of a motor response. [7] A delay in simple reaction time (in which a prepared motor plan is initiated upon the onset of a sensory stimulus) in patients with PD seems to be well agreed upon, [8] however studies of choice reaction times (in which the movement depends on some characteristic of the stimulus) in PD patients have had mixed findings. This may be related to differences in study design or the manner in which movement initiation was measured (e.g., onset of electromyography [EMG] activity versus onset of movement). [9] Experimental differences make parsing out exactly what components of reaction time (i.e., motor preparation and motor initiation) are abnormal in PD patients difficult based on the research to date, though the evidence is weighted toward impaired initiation of movement rather than a delay in movement preparation. [9,10] This could explain a normal choice reaction time that is dominated by movement preparation time. However, multiple other factors probably contribute as well to this form of akinesia. For example, it has been shown that patients with resting tremor have a tendency toward initiating the first agonist muscle activity of a movement concomitant with

bursts of tremor activity, [11] and recent research suggests that failure of sensory attenuation prior to movement onset might be a contributing factor to delayed movement initiation. [12]

The relationship of akinesia to bradykinesia is not well understood [13], and a delayed reaction time *per se* is not a common clinical complaint (unless freezing is included), as opposed to slow or small movements, which are often cited by patients in initial consultations.

Bradykinesia

The diagnosis of PD is based on the presence of bradykinesia and at least one of the additional cardinal features of rigidity and tremor at rest, and is the core clinical feature of parkinsonism. [14] Though the term bradykinesia is often used as an umbrella term to describe a paucity of some aspect of automatic or voluntary movements, specifically it refers to a *prolongation of the movement time*, or the time from the start to the completion of a voluntary movement. In general, this equates to a slow movement speed. Rather than akinesia, it is bradykinesia that gives rise to the general impression of slowness during movements that is noted in patients with PD. [4] The time in which a voluntary movement occurs in a patient with PD is performed is longer, and patients are slower to correct or arrest an erroneous movement, both at the proximal and distal joints. [15] Similarly, when a series of movements are performed serially, patients have a longer delay between each individual movement. [16] This can occur in both internally generated vs. externally generated (responding to sensory stimuli) movements, though it tends to be worse when movements are internally generated or cued (as described later).

The pathophysiological basis for the various manifestations of bradykinesia are not well understood, in part due to our lack of understanding of normal motor control in general. [17]

It is hypothesized that in the setting of a dopaminergic deficiency, the basal ganglia fail to both efficiently and sufficiently energize the motor cortex to reach the activation threshold required for performance of a movement of a desired size and speed. [15,17] This could account for the akinesia seen in PD patients as well, in which more time and more sensory afferent stimulation might be required to trigger a movement, resulting in an increased movement latency. Extending this hypothesis is the notion of decreased motor vigor in PD, due to impaired internally assessed calculation of the motor energetic costs and rewards associated with a movement due to the dopaminergic deficiency. [18] Recent research suggests that this motor motivational hypothesis may be an accurate representation of the energetic cost and reward assessment involved in the production of voluntary movement in normal subjects as well. [19]

Hypokinesia and the Sequence Effect

Hypokinesia refers to an inappropriately small movement amplitude. [17] Deficits in kinesthetic perception as well as the insufficient energizing of muscles (and specifically in the initial force of a movement) likely contribute to the hypokinesia seen in PD, similarly due to a higher sensitivity to motor energetic cost for internally generated movements. [20,21] Some authors have described a disconnect between bradykinesia and hypokinesia severity, [22] and others have posited that central fatigue may be a contributor as well. [23] Clinically

noted manifestations of hypokinesia include hypomimia or reduced facial expression, micrographia or small handwriting, hypophonia, dysphagia, and decreased finger-tapping size on clinical examination. [17] Like many other phenomena of PD, hypokinesia can improve with auditory or visual stimuli. [24]

The sequence effect refers to the progressive reduction in movement amplitude and speed that occurs when patients with PD perform repetitive movements. [21] It is commonly observed with finger tapping and particularly handwriting. [25] It is unique as a parkinsonian phenomenon in that it does not occur in atypical parkinsonisms, which are characterized by hypokinesia without progressive decrement. [26] The sequence effect can be seen in early stage PD and patients with severe symptoms alike, [27] and it does not appear to be improved by dopaminergic therapy. [28] It can also be helpful in diagnosing PD in patients who exhibit non-specific general reductions in activity and the speed and size of movements. As such, the presence of the sequence effect is required for most established diagnostic guidelines for PD. [29,30]

In addition to hypokinesia, many patients with PD describe weakness as a symptom of their disease. However, what they experience is more likely a complex combination of factors such as of impaired rate of force development related to bradykinesia, [31,32] co-activation of antagonist muscle activity, [33] fatigue, [34] tremor during maximal agonist contraction, [35] as well as a decrease in maximal agonist muscle contraction. [33]

Rigidity

The term rigidity refers the resistance to passive (externally imposed) movement that occurs due to increased resting muscle tone. By definition, it is a sign noted by the clinician, though patients often describe stiffness as an early symptom. When moving a joint through its range of motion in a patient with PD rigidity, the clinician may note a constant resistance that is uniform throughout the range of motion of the joint and movement trajectory. [36,37] When tremor is superimposed on rigidity, the resistance is ratchety and is called cogwheel rigidity. Rigidity becomes more pronounced during voluntary movement of the contralateral limb (the Froment maneuver), [38] which is helpful in detecting mild rigidity. Similarly, rigidity can increase during any voluntary movement (including eye movements) and when patients engage in attentional tasks. Although techniques to quantify rigidly have been developed in research settings, [39] they have not been incorporated into clinical practice.

Of the cardinal features of PD, the improvement of rigidity to levodopa is often considered one of the most reliable in determining a levodopa response. However, the requirement of a skilled clinician to detect and qualify rigidity complicates the implementation of remote or virtual clinical assessment, a difficulty that has not yet been successfully addressed in evaluations of telemedicine for PD or standardized quantitative assessment for clinical trials. Recent studies have focused on modifying the standard clinical evaluation for PD by excluding rigidity rather than seeking a proxy measure to substitute or calculate for rigidity. [40]

The etiology of rigidity in PD is complex; the clinical impression of rigidity has been shown to correlate with long latency reflex characteristics, [41] and contributions have been proposed not only from failures to relax, abnormalities in stretch reflexes and the mechanical properties of muscle, [42,43] but also from potential contributions of abnormal basal ganglia activity on spinal reflexes. [41,44] Although an examiner is necessary to appreciate rigidity directly, its effects can be seen during gait, in which a reduction in the pendular movement of the arms (arm swing) throughout the phases of locomotion in patients with PD is observed. This reduced arm swing, which can be one of the earliest and most commonly reported symptom noticed by patients with PD and their families, is more a manifestation of rigidity than bradykinesia. [45] However, as multiple studies have confirmed active EMG activity in the proximal arms during the gait cycle, [46] reduced automaticity of arm movements during walking plays a role as well.

Movement Automaticity and Dual-Tasking

Automatic movements are movements performed without directed attention, and they can even be complex movements with significant training. [4] Walking is typically automatic, and with training, activities such as typing or playing the piano can be automatic as well. During automatic movements, aspects such as speed and amplitude are selected without conscious thought. Though the ability to perform and acquire new automatic movements deteriorates with increasing age, typically older adults can perform automatic movements given enough training [47] In patients with PD, impairment in the execution of automatic movements has been recognized as a prominent deficit for decades. [48]

Almost all PD patients, including those with well-controlled and non-troublesome motor symptoms, describe impairments in automaticity and dual-tasking. In some patients, abnormalities in automatic movements can be one of the presenting symptoms of the disease (especially in patients still employed). Concurrent performance of cognitive and motor activities or multiple motor activities can be impaired, and activities such as talking while walking, listening while writing, and balancing carried objects while walking are usually described as more difficult, even in the early stages of the disease. Patients with PD have to rely (to a much greater degree) on attention-focused motor control to complete motor tasks, [49] which also contributes to the appearance of bradykinesia, as the amount of slowing seen in patients with PD when performing two movements simultaneously is greater than the sum of the reduction in speed seen with each individual movement. [13] An increase in directed attention when in the clinic can also prompt comments of “he[or she] never performs this well at home” when patients are seen for their regular follow-up visits.

Because of impaired automaticity of movements, dual-tasking can also contribute to akinesia or a delay in the onset of willed movements. A loss of automatic scaling of movement size and speed likely underlies (at least to some degree) the impairments of hypokinesia and bradykinesia seen in PD, as patients can still generate normal movement speeds and amplitudes under experimental circumstances. However, this is also complicated by abnormalities in implicit motivation due to dopaminergic deficits. [18] The dysrrhythmokinesia seen in PD patients is related as well, in that very simple repetitive movements typically can be performed automatically, unless a rhythmic movement of

another body part is performed in parallel, which in most people will degrade or alter performance of one or both movements. PD patients also specifically have a greater degree of difficulty with bi-manual movements that are anti-phase as opposed to in-phase. [50]

In the clinic, patient performance during dual-tasking usually results in poorer performance in one or both of the tasks being attempted, and anti-phase movements will sometimes revert involuntarily to in-phase movements, especially at higher frequencies, and interestingly this can be worse with external cues. [51] Patients performing repetitive movements in one hand can also demonstrate mirror movements, or involuntary movements due to homologous muscles on the opposite side of the body. [52] Similar movements (sometimes termed "motor overflow") that are matched in phase and frequency can also be seen in the jaw or at the ankle joint (toe tapping) with rapid sequential movements in the hands, and more often in patients early or in the middle of their disease course. Such movements are not specific to PD, but can be seen in both other movement disorders and other neurological disorders. [53]

Dysrrhythmokinesia

Dysrrhythmokinesia (also sometimes referred to as arrhythmokinesia or dysrhythmia) refers to an increased variability in the regularity and timing of repetitive movements. Similar to and encompassing the dysdiadochokinesia (impaired performance of repetitive sequential movements that are specifically alternating with regard to contraction and relaxation of agonist/antagonist muscle groups) that is classically described in cerebellar disorders, dysrrhythmokinesia is not specific to PD but rather can be seen in a variety of other movement disorders. Though easily observed, it is impossible to parse out the contributions of features such as bradykinesia and hypokinesia from the central timing impairment that occurs during the performance of voluntary movements in PD. That being said, explicit timing abnormalities in PD have been shown to extend beyond motor symptoms, and PD patients have impairments in both temporal sensory discrimination [54] and levodopa-responsive time estimation [55]. Clinically, rhythmic dysfunction is more noticeable to the clinician than to the patient, and its presence can be helpful in diagnosing patients with mild symptoms. Dysrrhythmokinesia is often seen earliest in the most distal movements of the fingers and feet, and especially when the movements are complex and unimanual. [56] Toe tapping is typically impaired earlier than repetitive foot stomping, and finger movements such as finger tapping tend to be the earliest impaired in the upper extremities. [4] Finger and toe tapping are helpful tasks to test in the clinic, and patients will usually have a combination of dysrrhythmokinesia and brady- and hypokinesia. As PD progresses, dysrrhythmokinesia can affect speech and gait as well, contributing to festination. Stride length variability during ambulation is another example of a form of arrythmokinesis that can contribute to falls and decreased efficiency of gait (and thus walking speed, showing the interrelated nature of timing problems with core features such as bradykinesia).

Externally vs. Internally Generated Movements and Paradoxical Kinesia

Patients with PD have long been known to have more difficulty with the execution of internally generated or cued movements compared to externally (i.e., by sensory stimuli) cued movements. [57] In research settings, a shift from internal to external cueing has been

shown to improve gait, reaction time, movement time and amplitude, and to be able to interrupt a motor block. [58–61] Difficulties in executing an internally triggered motor command in PD has been attributed to the impaired ability of PD patients to increase the excitability of the motor cortex to a state sufficient to trigger a motor command internally, possibly due to the stronger dependence of internally generated motor commands on the basal ganglia. [6] Clinically, providers often develop techniques to externalize motor cues, especially with regard to gait dysfunction and freezing of gait. Walker-mounted lasers, lines painted on the ground, or other visual targets can increase walking speed and stride length. Similarly, auditory cues such as metronomes have been shown to improve dual-tasking performance and can interrupt or prevent the freezing of gait phenomenon. [62] The beneficial effect of converting internal to external stimuli on the freezing phenomenon can be variable depending on the circumstances, and some external stimuli can worsen the symptoms of PD, such as narrow passages and horizontal barriers in the periphery. In some patients, motor performance is highly dependent upon their emotional state. [63]

Paradoxical kinesia classically refers to the brief and sudden ability of patients with PD to perform effective voluntary movements (that they could not have performed immediately prior) in the presence of a threatening or physically stressful stimulus. [64] Stories of paradoxical kinesia fitting this criterion are largely anecdotal for obvious reasons, and include the patient who is suddenly able to run when fleeing a war zone, [65] or the grandfather with severe PD running outside upon hearing an accident in search of his grandchild. [66] However the earliest descriptions [64] include descriptions of patients moving unexpectedly well without impending danger, [67] and it has been argued that the term can be applied to the use of multimodal external cues to temporarily produced surprisingly effective movement in the absence of danger or frightening stimuli. [68] A variety of non-specific possible mechanisms has been proposed to explain paradoxical kinesia in addition to increased external triggering, including increased adrenergic tone, cerebellar compensation, and activation of reserve basal ganglia function. [65,69] As discussed in the section on bradykinesia (which is the symptom most often studied and described in paradoxical kinesia), both normal subjects and patients with PD can be made to increase their maximal movement speed depending on experimental conditions. [70] Increased motivation and a sense of urgency are likely important factors in increasing “motor motivation” when stimuli are of great consequence. [18,67]

The Parkinsonian Gait and Festination

The gait of patients with PD has historically been one of the most richly described phenomena of the disease, and heavily emphasized in the original description of PD by James Parkinson in 1817. [71] In the earliest stages, patients tend to note a general decrease in their walking speed and a tendency to lag behind when walking with others. Often, a sense of dragging one leg is described. As the disease progresses, patients develop a flexed and stooped posture, with a slow shuffling quality to their gait and decreased stride length and heel strike. Difficulty getting up and down from a low sofa or in and out of a vehicle is typical. Often moderate to severe patients will find themselves collapsing into their chair upon sitting, and visuospatial dysfunction can make the process of turning to sit in a chair

from ambulation a fall risk, even when the freezing phenomenon (described below) is not present.

One aspect of the parkinsonian gait that is unique to parkinsonism is festination. Festination is a phenomenon in which the stooped posture and hypometric step size of patients with PD results in the center of gravity being located in front of the feet, resulting in an increasing step velocity. [72] The original description by James Parkinson is often cited as it conveys the phenomenon of festination as well as any subsequent descriptions. Parkinson described how the "propensity to lean forward becomes invincible, and the patient is thereby forced to step on the toes and fore part of the feet, whilst the upper part of the body is thrown so far forward as to render it difficult to avoid falling on the face…being, at the same time, irresistibly impelled to make much quicker and short steps, and thereby to adopt unwillingly a running pace. In some cases it is found necessary entirely to substitute running for walking; since otherwise the patient, on proceeding only a very few paces, would inevitably fall." [71] It is important to note that festination is less a characteristic of the parkinsonian gait and more so of PD in general, as the general phenomenon of decreasing amplitude with a compensatory increase in rate has been described in a variety of other voluntary movements, including fine dexterous movement such as piano playing [73] and axial symptoms such as speech. [74] Festination is another example of the frequent overlap that occurs in PD phenomenology, as it involves dysrrhythmokinesia as well as hypokinesia, and like many PD phenomena it commonly worsens with stress or emotional distress. [75]

Freezing of Gait

Freezing of gait (FOG) seems to be a separate entity from festination, but some studies have found an association between festination and freezing, and festination can trigger an episode of FOG. [72] In general, FOG refers to a brief and sudden episodic inability to generate effective stepping or to initiate or continue ambulation when intending to walk. [76] Clinically, freezing of gait can manifest as delayed or failed gait initiation or a halt in forward movement, often with a tremulous appearing jittering stance phase alternating from one leg to the other, and a subjective sensation of being unable to pick up the feet. When severe, a freeze can produce total akinesia. [77,78] Common triggers for FOG include narrow doorways, congested walking areas, and horizontally oriented objects in the periphery. Turning when walking or when preparing to sit down in a chair are also common triggers, as are stress, distraction, and dual-tasking. Focused attention and a shift from internal to external cueing (discussed above, such as the use of an inverted walking stick) as well as the levodopa "ON" state can sometimes overcome or reduce FOG. Both triggers and alleviating factors can vary between patients and even within the same patient at different testing times, making FOG notoriously difficult to study in the laboratory setting. [79] The freezing phenomenon can also be seen in the upper limbs or in speech, where a sudden transient break in efficient movement is also termed a motor block or upper limb freezing. [79–81] Like festination, FOG overlaps with motor automaticity, dual-tasking, difficulty with internally cued movements, [6] and hypokinesia, [79] the similarities between festination, FOG, and the decrement or sequence effect of hypokinesia have been considered in the research setting. [82]

Postural Instability

Though impairment in the protective reflexes that occur to prevent falling (i.e., postural instability) is a cardinal symptom of PD, [14] typically it occurs later in the disease course. Its presence early in the presentation of PD can suggest an atypical parkinsonism. When tested in the clinic, the examiner will typically test the recovery and degree of retropulsion that occurs after a sudden backward pull at the shoulders. Patients with mild symptoms can report frequent tripping or a requirement for focused concentration to avoid tripping on uneven surfaces despite a normal pull test. In patients with at least moderate disease, it is usually the tendency to fall backwards, or when leaning in other directions, such as when leaning to pick up an object. Patients can become aversive to this test in the clinic as postural stability increases, as it can be highly anxiety provoking. Postural stability should, however, be tested at all visits, as the development of postural impairment after perturbation can be an indicator that ambulatory assist devices are needed. As with many patient populations with impaired ambulation, patients with PD can also develop a fear of falling that can be more limiting than their actual impairment. It is more common in patients who have experienced falls previously or who also have generalized anxiety. [83] Though it can be difficult to treat, it is important to recognize fear of falling, as gait training can sometimes be beneficial in reducing gait impairment to that due to actual functional limitations.

Limb Kinetic Apraxia

The ability to perform deft finger movements requires the precise and coordinated control of the hand and forearm muscles. Timing of activation of selected agonist muscles, as well as inhibition of antagonist muscles, must occur flawlessly both within muscle groups controlling individual fingers and across groups controlling separate fingers. This must occur in a manner that correctly stabilizes joints and produces the appropriate pressures to maintain control of objects. [84] Further, motor plans must be integrated with sensory and visual feedback in real time. The inability to connect or isolate precise individual finger movements has been termed limb kinetic apraxia (LKA), and it is recognized as a disorder that straddles contemporary notions of cognitive and motor contributions to motor control. [85] Apraxia in general can be defined as impairment in the ability to perform purposeful skilled movements that is not due to abnormalities in sensation, cognition, or basic disorders of movement (such as bradykinesia or rigidity). [85] Apraxia can be generalized or task-specific, and a variety of subtypes of apraxia have been identified that describe errors in what have been considered the cognitive aspects of motor control, such as movement conceptualization, sequencing, and planned spatial or temporal components. [85,86] In LKA, there is a breakdown of previously skillful movements that become clumsy and awkward while still maintaining their gross spatial and temporal characteristics, due to impairment in isolating and/or connecting independent units of fine motor control. [87–89]

In his original essay, James Parkinson described noted that in PD, “the fingers cannot be disposed of in the proposed directions, and applied with certainty to any proposed point.” [71] Patients describe difficulty in performing activities such as buttoning clothes and tying shoelaces, and using electronic devices and smartphones are impossible for many patients even when they are well dosed on levodopa. LKA can influence many important everyday

activities such as dressing, personal hygiene tasks, eating, and cooking. [90] In the clinic, LKA is most commonly tested by rotation of a nickel. This task is correlated with the grooved pegboard test and is validated as a screening tool for impaired fine motor control. [91]

Parkinsonian tremor

Tremor is the rhythmic oscillation of a part of the body around one or more joints. As parkinsonian tremor is the focus of another article in this issue by Helmich and Dirkx, [92] it will only be discussed here briefly. The classic tremor of Parkinson's disease is a 3–6 Hz resting (initially unilateral) tremor, typically occurring initially in the distal upper extremity unilaterally. [93] Despite its well-known association with the disease, up to 25% of patients with PD do not have tremor, [94] which can sometimes lead to misdiagnosis as an atypical parkinsonism by the unexperienced clinician. Tremor direction can be wrist extension and flexion, though tremor in the pronation and supination plane or flexing or grasping movement of the fingers (sometimes described as "pill rolling") is classic. [93] PD tremor can also be present in the legs (typically in more progressed disease), and is often seen in the chin, lips, and/or jaw. In most patients, the resting tremor of PD can be disrupted by voluntary motor commands with the same limb, and activities such as squeezing a rubber ball or turning a coin between the fingers can mask tremor. Interestingly, voluntary activity in another limb as well as cognitive activity can enhance resting tremor, and it is often found when testing gait. When testing for tremor in the seated position, it can be helpful to have the patient's arms hanging at their sides, as patients sitting with their hands in their lap are often not truly at rest.

The presence of resting tremor can be highly variable with regard to presence, distribution, and severity. [95] When first noticed by patients, it can erroneously be described as an action tremor, due to its propensity to re-emerge with sustained posture. This re-emergent tremor of PD can occur after a few seconds of sustained posture and typically has a similar frequency to resting tremor. A true kinetic tremor can occur in PD as well, but is less common and of a higher frequency (5–8 Hz). Kinetic tremor (when present) may also contribute to delayed reaction times and the impression of muscle weakness in PD. [11,35] Some patients also report a sensation of inner tremor in the absence of visible oscillations around a joint (though it might be present on EMG) that is often not responsive to levodopa. [96]

Conclusion

In this review, we hope to have provided an overview of the clinical phenomenology of PD. Discussion of the pathophysiology of these phenomena was not extensive; however, the literature cited was carefully chosen to include comprehensive discussions of pathophysiology when available. Though we described the phenomena of PD separately, in reality individual phenomena rarely occur in isolation outside of a controlled research setting, and the framework of PD phenomenology that was described represents how movement is observed by the clinician rather than how it is organized in the motor system. Nonetheless, a detailed understanding of the framework of PD phenomenology can be useful in clinical practice.

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Abbreviations

PD	Parkinson's disease
EMG	electromyography
FOG	freezing of gait
LKA	limb kinetic apraxia
Hz	Hertz

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Table 1

Phenomenological features of Parkinson's disease (PD)

Classic Phenomenology of PD	
Akinesia	Decrease in unconscious movements or a failure or delay in the initiation of willed movements.
Bradykinesia	Prolongation of movement time (the time from the start to the completion of a voluntary movement).
Hypokinesia	Inappropriately reduced movement amplitude.
Rigidity	The resistance to passive (externally imposed) movement that occurs due to increased resting muscle tone.
Parkinsonian Tremor	3–6 Hz resting tremor that is typically initially unilateral and can re-emerge with sustained posture. Classic parkinsonian tremor is present in up to 75% of patients with PD but other forms of tremor can also occur.
Postural Instability	Impairment in the ability to recover and maintain balance after perturbation.
Additional PD Phenomenology	
Sequence Effect (Decrement)	Progressive reduction in movement amplitude and speed that occurs when patients with PD perform repetitive movements.
Impaired Movement Automaticity and Dual-Tasking	Impairment in the execution of learned movements when performed without directed attention, often occuring during the concurrent performance of cognitive and motor activities or multiple motor activities.
Dysrrhythmokinesia	Increased variability in the regularity and timing of repetitive movements.
Difficulty Performing Internally Cued or Generated Movements	Selective impairment in the performance of voluntary movements that are cued or generated internally, often resulting in the substitution of an external sensory cue to improve motor performance.
Paradoxical Kinesia	Brief and sudden ability of patients with PD to perform effective voluntary movements (that they could not have performed immediately prior) in the presence of a threatening or physically stressful stimulus.
Festination	Progressive increase in step velocity resulting from the center of gravity being located in front of the feet due to the stooped posture and hypometric step size of patients with PD.
Freezing of Gait	A brief and sudden episodic inability to generate effective stepping or to initiate or continue ambulation when intending to walk.
Limb Kinetic Apraxia	An impairment in the ability to connect or isolate precise individual finger movement that is not due to abnormalities in sensation, cognition, or basic disorders of movement (e.g., bradykinesia or weakness).