Editor's key points

- Parkinson disease (PD) should be considered in any patient presenting with parkinsonism, balance problems, gait changes, or nonmotor symptoms common in PD.
- Diagnosis of PD is mainly clinical, and routine use of imaging is not recommended. While motor symptoms are the core diagnostic features of PD, nonmotor symptoms are increasingly recognized as being important in the diagnosis of PD and management of patients with PD.
- Short-term levodopa challenges are not recommended for diagnosis of PD, but a long-term trial of dopaminergic treatment with clear and marked response can help confirm diagnosis.
- Family physicians should be aware of causes of parkinsonism other than PD to allow for further investigation and timely workup for other possible conditions.

Parkinson disease primer, part 1: diagnosis

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Abstract

Objective To provide family physicians an updated approach to the diagnosis of Parkinson disease (PD).

Sources of information Published guidelines on the diagnosis and management of PD were reviewed. Database searches were conducted to retrieve relevant research articles published between 2011 and 2021. Evidence levels ranged from I to III.

Main message Diagnosis of PD is predominantly clinical. Family physicians should evaluate patients for specific features of parkinsonism, then determine whether symptoms are attributable to PD. Levodopa trials can be used to help confirm the diagnosis and alleviate motor symptoms of PD. "Red flag" features and absence of response to levodopa may point to other causes of parkinsonism and prompt more urgent referral.

Conclusion Access to neurologists and specialized clinics varies, and Canadian family physicians can be important players in facilitating early and accurate diagnosis of PD. Applying an organized approach to diagnosis and considering motor and nonmotor symptoms can greatly benefit patients with PD. Part 2 in this series will review management of PD.

Parkinson disease (PD) is the fastest growing neurodegenerative condition, with prevalence predicted to double from more than 6 million globally in 2015 to more than 12 million by 2040.[1] Recognizing parkinsonism and having knowledge of the presentation, diagnosis, and management of motor and nonmotor symptoms of PD are increasingly important, particularly as access to neurologists and specialized clinics is limited in many parts of Canada.[2] Family physicians are well placed to identify symptoms, participate in diagnosis, and collaborate with specialty clinics in management of patients through the course of the disease.

Case description

Maria is a 65-year-old retired teacher. She lives in a bungalow with her husband, Doug, who works as an accountant. She presents to your office with a complaint of a rest tremor in her left hand for the past 6 months. Her past medical history includes hypertension for 10 years and type 2 diabetes for 5 years. Her medications include perindopril, amlodipine, and metformin.

On further discussion of her history, she reports having had more difficulty buttoning her shirts recently, and she complains of slowing down in general. It takes her longer to perform grooming tasks than it previously had and, when asked, she comments that she has noticed some difficulty turning over in bed. She also has noticed some shuffling in her gait but no falls. Her family history is noncontributory.

She notes she is experiencing more sleepiness and fatigue during the day, and her husband says she has yelled and moved about as if acting out dreams in her sleep for 10 years. She reports slowing of her bowels over the past year.

Sources of information

Search strategies included looking for review articles and guidelines related to PD diagnosis and management published between 2011 and 2021. A 2019 Canadian guideline, a 2010 Scottish Intercollegiate Guidelines Network guideline, and a 2017 National Institute for Health and Care Excellence guideline were used to inform evidence-based suggestions in this paper.[3-5] Evidence levels ranged from I to III.

Main message

Most family physicians are aware of the main motor symptoms and signs of PD. However, diagnosis is not always straightforward, especially early in the course of the disease. Many conditions can cause or mimic parkinsonism, and clinical features evolve over time. Sensitivity of the clinical diagnosis is high (89.2%) with poorer specificity (57.8%).[6] Access to specialist care is variable and wait lists can be long, highlighting the need for family physicians to participate in diagnosis and management.[2,7]

Diagnosis of PD remains predominantly clinical, and routine use of imaging is not recommended. Although motor symptoms remain the core features for diagnosis of PD, the importance of identifying nonmotor symptoms is highlighted in International Parkinson and Movement Disorder Society (MDS) diagnostic criteria[8] and various PD guidelines.[3-5]

Defining parkinsonism. Parkinsonism is the presence of bradykinesia and either rest tremor or rigidity, or both. Recognizing parkinsonism is the first step in evaluating patients for PD.

Bradykinesia: Bradykinesia is defined as slowness of movement and a decline in amplitude or speed (or progressive hesitations or halts) as movements are continued. A review article by Rao et al and MDS criteria suggest using finger tapping, alternating hand movements (pronation and supination, opening and closing the hand), and toe or foot tapping to assess bradykinesia.[9,10] The decline in speed or amplitude that occurs when movements are continued is seen more with PD than with other causes of parkinsonism.[8]

Rest tremor: Rest tremor is often the first symptom patients report to physicians; however, the absence of tremor does not rule out parkinsonism or PD. Rest tremor in PD has a frequency of 4 Hz to 6 Hz in the fully resting limb, commonly described as a *pill-rolling* tremor (supination and pronation). It is suppressed by voluntary movement, and it is not usually seen when holding a posture (although it may be present after sustained posture). The tremor commonly increases with mental stress (eg, doing mental arithmetic).[11] It is typically asymmetric and may involve upper or lower extremities, or both.[12] Facial and head-bobbing tremors are uncommon in PD.

A common issue is misdiagnosing essential tremor as being caused by PD. Essential tremor involves a higher frequency flexion-extension tremor with voluntary action of bilateral upper limbs, as opposed to the asymmetric pill-rolling rest tremor of PD.[11] Tremor may be present in other neurologic conditions and with vascular and drug-induced parkinsonism.

Rigidity: Rigidity in PD is sometimes called *lead-pipe rigidity* but may be better described as a velocity-independent, bidirectional increase in tone, not owing to a failure to relax (distinguishing it from spasticity and paratonia). Cogwheeling reflects tremor felt while assessing tone and is not always present in PD.[8] In those who have trouble relaxing, gently swiveling the patient's torso and observing arm movement may demonstrate limb rigidity. Subtle rigidity can be demonstrated in a limb while the patient is doing an activation maneuver, such as finger tapping or heel tapping, with a contralateral limb.[10] The pattern of rigidity provides diagnostic clues. Axial (neck and trunk) rigidity is more prominent than limb rigidity in progressive supranuclear palsy (PSP). In PSP, rigidity may contribute to a forward-flexed or extended posture, whereas in PD, a forward-flexed (stooped) posture is more typical.

Postural instability: Postural instability and falls not explained by other conditions—such as visual, vestibular, cerebellar, or proprioceptive dysfunction—are important to recognize. Many physicians use the TRAP (tremor, rigidity, akinesia, and postural instability) mnemonic, but postural instability usually occurs later in PD and is not included in the MDS criteria for PD. Prominence early in the course of illness may indicate an alternative diagnosis, such as multiple system atrophy (MSA) or PSP.[8]

Diagnosing Parkinson disease. Parkinson disease should be considered in any patient presenting with parkinsonism, balance problems, gait changes, or nonmotor symptoms of PD.[3] A 2-step approach is recommended: first, diagnosing parkinsonism, and then assessing whether the parkinsonism is attributable to PD by reviewing clinical features, evaluating for supportive features, looking for findings that make the diagnosis of PD less likely, and considering differential diagnoses.

Features supporting a diagnosis of PD: According to MDS criteria, the presence of at least 2 of the following features support the diagnosis of PD:

- clear and dramatic response to dopaminergic therapy;
- levodopa-induced dyskinesia, usually choreiform movements of the most affected side, trunk, or neck at peak dose (1 to 2 hours after a dose of levodopa)[8]; this usually emerges several years after onset of PD;
- resting tremor of a limb (asymmetric rest tremor); or
- a positive diagnostic test of either olfactory loss or cardiac sympathetic denervation on scintigraphy (not relevant in primary care).[8]

In family practice, we also find the following features helpful in diagnosing PD:

- Micrographia: Although most clinicians look for progressive micrographia, consistent micrographia may also be found (where all writing is smaller compared with before the onset of PD).[1]

- Nonmotor symptoms: These are increasingly recognized as being important in diagnosis of PD and management of patients with PD. Absence of common nonmotor symptoms after 5 years suggests an alternative diagnosis (**Table 1**).[13-24] Nonmotor symptoms can have a major impact on quality of life and function in PD; management will be discussed in part 2 of this series.
  - -Autonomic symptoms include orthostatic hypotension, gastrointestinal dysfunction (constipation, gastroparesis), temperature dysregulation, and urinary symptoms (eg, overactive bladder and nocturia). Although severe dysautonomia should not be prominent in early stages, constipation is present in 30% of patients in early stages of PD.[25]
  - -Sialorrhea (excess saliva or drooling) is seen in many forms of parkinsonism, including PD.[26] It is worth asking about and watching for this during clinical encounters. Sialorrhea is often a source of distress for patients and may be amenable to treatment.
  - -Olfactory impairment is more common in PD than in atypical syndromes and other imitators. Although most family physicians do not have ideal testing equipment for olfactory loss, paying attention to patient reports is relevant to diagnosis. A lack of hyposmia after 5 years of other symptoms would contradict a diagnosis of PD.[8]
  - -Neuropsychiatric symptoms are common in early stages of PD and increase in prevalence and severity with progressive disease. Rapid eye movement sleep behaviour disorder (acting out dreams due to lack of large muscle atonia during rapid eye movement sleep) can be a premotor symptom in up to 30% of patients in the early stages of PD and is seen in other Lewy body diseases (eg, MSA and dementia with Lewy bodies); it may predate motor symptoms by years. Depression may be seen before the onset of motor symptoms and is present in about one-quarter of patients in early stages of PD.[25] It often becomes more prominent with disease progression and can be challenging to treat.[4,27]

Alternative causes of parkinsonism: **Table 2** lists clinical features that suggest alternative causes of parkinsonism.[3,4,8,28] Although the MDS criteria distinguish between exclusion criteria and red flags, which either exclude PD or make the diagnosis of PD less likely, we have simplified the approach and language here for practical application.[3,8] Patients presenting with features in **Table 2** should be investigated further or referred early on to a specialist with expertise in movement disorders for diagnostic clarification and management.[3,4,8,28] **Table 1** provides more detail on select differential diagnoses.[13-24]

Table 1. Clinical features of select differential diagnoses of parkinsonism

CONDITION	CLINICAL FEATURES	EXPECTED COURSE	LEVODOPA RESPONSIVENESS
Medication-induced parkinsonism[13]	• Temporally related to use of antidopaminergic medications, such as typical neuroleptics (eg, haloperidol), higher-dose atypical neuroleptics (eg, risperidone >0.5 mg/d), and antiemetics (metoclopramide, prochlorperazine) • Gait disturbance less prominent • More commonly involves upper limbs • May be more symmetrical than PD	Usually improves after discontinuation of medication, but improvement may be slow (up to 1 y); persistent symptoms may reflect underlying PD	Discontinue or switch medication causing symptoms rather than treat with levodopa
Vascular parkinsonism[14]	• May have more abrupt onset and stepwise decline • Symmetrical bradykinesia and shuffling gait (may affect lower limbs more)	Depends on risk of further vascular injury	Minimal
Progressive supranuclear palsy[15-17]	• Prominent early postural instability and falls • Truncal rigidity • Extraocular movement changes, including vertical gaze palsy • "Startled" facial expression	More rapid progression than PD, with median survival 6 to 10 y[18]	Variable; up to 50% may experience some response[19]
Multiple system atrophy[20-22]	• Early and prominent autonomic failure (orthostatic hypotension, urinary dysfunction) • Cerebellar findings and inspiratory stridor may be present	More rapid progression than PD, with median survival 7 to 10 y[18]	Possible initial response to high doses[19]
DLB[23]	Dementia with at least 2 of the following for probable DLB: • Parkinsonism • Fluctuations in attention and awareness • Recurrent, well-formed visual hallucinations • Rapid eye movement sleep behaviour disorder	Rapid progression compared with Alzheimer dementia, with average survival 3 to 5 y	Variable, but may be considered after treatment with cholinesterase inhibitor and where psychosis is controlled
Normal pressure hydrocephalus[24]	• Gait disturbance with shortened step length and height ("magnetic gait") • Cognitive dysfunction, urinary urgency, or incontinence	Variable; potential improvement with CSF removal or VP shunt	None

CSF—cerebrospinal fluid, DLB—dementia with Lewy bodies, PD—Parkinson disease, VP—ventriculoperitoneal.

Table 2. Clinical features suggestive of diagnoses other than PD: ***The features listed below include but are not restricted to the exclusion criteria and red flags from the MDS diagnostic criteria for PD, simplified and explained for practical application.****

CLINICAL FEATURES[8]	RATIONALE AGAINST DIAGNOSIS OF PD
Absence of response to levodopa trial at target dosage (>600 mg/d)[4,8]	Patients with PD demonstrate clear subjective and objective improvement in motor symptoms with dopaminergic therapy
Early bilateral symmetric parkinsonism Lack of progression of motor symptoms or absence of common nonmotor symptoms (eg, constipation, orthostasis, hyposmia) of PD after 5 y	In PD, motor features are usually unilateral in onset and then progress in distribution and severity over time
Early recurrent (>1/y) falls because of impaired balance within 3 y of onset Rapid progression of gait impairment to substantial postural imbalance requiring use of a walker by 3 y and a wheelchair by 5 y ("wheelchair sign")[28]	Falls are common in older adults and often have multiple causes. The falls implicated here are those owing to postural instability. Early unexplained falls or quickly progressing mobility challenges may reflect PSP or MSA
Severe autonomic failure (eg, unexplained orthostatic hypotension, urinary retention or incontinence) in the first 5 y of disease	More likely reflects conditions such as MSA
Downward vertical gaze palsy or slowing of downward vertical saccades	More likely reflects PSP
Parkinsonism with history of stroke	Consider vascular parkinsonism unless classic PD signs present
Findings restricted to the lower limbs for more than 3 y	PD involves the upper and lower limbs. Vascular parkinsonism may be limited to the lower limbs
Parkinsonism while taking a dopamine receptor blocker (eg, typical antipsychotics, metoclopramide) or a dopamine-depleting agent	Depending on dose and time course, this may reflect drug-induced parkinsonism
Early cognitive impairment and visual hallucinations, either spontaneous or with low-dose levodopa treatment*	More suggestive of DLB
History of repeated head injury	Although PD is still possible, chronic traumatic encephalopathy should be considered
Symptoms of behavioural variant frontal temporal dementia symptoms (marked apathy, disinhibition, personality changes) or primary progressive aphasia (early impairment of speech affecting comprehension or fluency) within first 5 y	Suggests alternative pathological process such as tauopathy
Unexpected neurologic findings, such as the following: • Upper motor neuron findings (eg, unexplained weakness, spasticity, or upgoing Babinski sign) • Cerebellar abnormalities (eg, cerebellar gait or ataxia) • Early bulbar dysfunction (severe dysphonia, dysarthria, or dysphagia) • Respiratory dysfunction, including stridor and inspiratory sighs • Anterocollis (marked neck flexion) or contractures • Cortical sensory loss (eg, graphanesthesia, or the inability to recognize symbols or letters traced on the skin), limb ideomotor apraxia (inability to perform a skilled gesture with a limb upon verbal command or by imitation), or progressive aphasia • Urinary incontinence early in course without other cause	These features are not typical for PD and should prompt workup for other neurologic conditions

DLB—dementia with Lewy bodies, MDS—International Parkinsonism and Movement Disorder Society, MSA—multiple system atrophy, PD—Parkinson disease, PSP—progressive supranuclear palsy.
*DLB is an entity related to PD, with related pathophysiology. New diagnostic criteria no longer list dementia within first 5 y except for frontotemporal dementia as an exclusion criterion or red flag.[3,8] The distinction between DLB and PD warrants a more nuanced discussion, as the treatment approach to each is different. Family physicians should be alert to the fact that early cognitive dysfunction with visual hallucinations suggests DLB.

Role of imaging: Canadian guidelines do not recommend routine imaging (structural or functional) for suspected PD. Neuroimaging can be considered if clinical features suggest other neurologic entities such as normal pressure hydrocephalus or PSP (**Table 2**).[3,4,8,28]

Role of levodopa challenge in diagnosis: Short-term levodopa challenges—where patients are given single doses of levodopa combined with carbidopa, with measurement of function before and after—are not recommended. A long-term trial of dopaminergic treatment with clear and marked response can help confirm diagnosis.[3-5] Long-term challenges involve high doses of levodopa equivalent (>600 mg per day) for at least 1 month.[4,8] Older adults may not be able to tolerate these

doses owing to central nervous system or gastrointestinal side effects or owing to orthostasis.[4] Levodopa is the recommended first-line dopaminergic treatment in older individuals (>60 years) or where cognition is a concern, based on more favourable tolerability compared with other agents.[3,25]

To diagnose PD, levodopa response should be considerable, with improvement close to premorbid function.[3-5,8] It is important to document subjective and objective improvement, such as gait speed (eg, Timed Up and Go test[29]), rigidity, and bradykinesia. A reasonable starting dose of levodopa and carbidopa, or levodopa and benserazide, is 1 half-tablet of 100 mg of levodopa with 25 mg of either carbidopa or benserazide taken 3 times daily, administered 30 minutes before meals (protein reduces levodopa absorption). Levodopa should be titrated slowly in older adults, such as increasing by a half-tablet per week, to when therapeutic benefit is observed or side effects emerge.

Patients with atypical parkinsonism may have a minor response to levodopa, but the response is generally poor or short-lived and accompanied by substantial side effects, such as severe orthostatic hypotension in MSA and hallucinations and psychosis in dementia with Lewy bodies.[30]

Ongoing reassessment of signs and symptoms over the course of disease is important to ensure the correct diagnosis has been made and to provide appropriate symptom management.[5]

Conclusion

Family physicians are usually the first doctors to see patients presenting with parkinsonism. We present a practical approach of identifying parkinsonism, considering alternative diagnoses when specific clinical features are present, and diagnosing PD when supportive criteria are fulfilled. The role of long-term levodopa challenge can be considered as part of the diagnostic and treatment process, particularly when the wait for specialist clinics is long.

Part 2 of this series will review management of motor and nonmotor symptoms and will summarize the case outcome.

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All authors contributed to the literature review and interpretation and to preparing the manuscript for submission.

Competing interests
None declared

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