

PARKINSON'S DISEASE

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October 10, 2025

RECOMMENDED CITATION

mohammad looti (2025). *PARKINSON'S DISEASE*. PSYCHOLOGICAL SCALES. Retrieved from <https://scales.arabpsychology.com/?p=42683>

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Primary Disciplinary Field(s): Neurology, Neuroscience, Geriatrics

1. Core Definition and Pathophysiology

Parkinson's Disease (PD) is defined as a chronic, progressive, and highly debilitating neurodegenerative disorder of the central nervous system (CNS). It typically manifests in older adults, with onset most commonly observed between the ages of fifty and seventy. The fundamental pathology of PD involves the progressive atrophy and loss of specific neural tissue within crucial subcortical structures responsible for regulating movement. This degenerative process primarily targets the basal ganglia, the thalamus, and the reticulating activating system (RAS). These interconnected regions collectively form the essential motor control loops necessary for initiating, executing, and modulating voluntary movement. The characteristic motor symptoms of Parkinson's stem directly from the inadequate production and transmission of the neurotransmitter **dopamine**, resulting from the death of dopaminergic neurons in the substantia nigra pars compacta, a core component of the basal ganglia system.

The degradation of these specific brain areas leads to a profound imbalance in the excitatory and inhibitory signals governing motor function. Specifically, the atrophy noted in the source material--affecting the thalamus, basal ganglia, and RAS--disrupts the feedback mechanisms necessary for smooth, coordinated movement. The basal ganglia are crucial for selecting and initiating appropriate motor programs while suppressing unwanted movements. When these structures fail, the patient develops the cardinal motor features of Parkinsonism: tremor, rigidity, bradykinesia (slowness of movement), and postural instability. Understanding this neurochemical deficiency is paramount to both diagnosis and therapeutic intervention, guiding the use of treatments aimed at restoring functional dopamine levels within the motor circuits.

2. Clinical Presentation and Motor Symptoms

The clinical presentation of **Parkinson's Disease** is characterized by a specific progression of motor impairments. Initial symptoms are often subtle and unilateral, usually beginning with noticeable **rigidity** and an involuntary resting **tremor** in the muscles of one arm. This tremor, often described as a "pill-rolling" motion, is generally most pronounced when the limb is at rest and diminishes during purposeful activity. As the disease advances, these symptoms gradually spread to the leg on the same side of the body, then subsequently affect the muscles of the neck and face, and finally progress to involve the limbs on the contralateral side, leading to bilateral yet often asymmetric involvement.

Over time, the cumulative effects of rigidity and bradykinesia produce several hallmark physical

signs that dramatically impact the patient's quality of life. The face of the Parkinson's patient frequently becomes **masklike** (hypomimia), losing the ability to express emotion through facial movements due to muscle stiffness. Speech patterns are also severely affected, growing **halting and indistinct** (dysarthria), often characterized by a quiet, monotonous tone (hypophonia). A particularly distinctive and advanced motor sign is the characteristic alteration in walking known as the "festinating gait." This term, derived from an archaic word meaning "hurry," describes the patient's tendency to lean stiffly forward with a stooped posture and walk at a rapid, small-stepped pace, often appearing to be catching up with their center of gravity, significantly increasing the risk of falls.

3. Non-Motor and Psychological Manifestations

While **Parkinson's Disease** is primarily defined by its motor dysfunction, it is crucial to recognize the extensive spectrum of non-motor symptoms (NMS) that frequently precede or accompany the motor features. Remarkably, the degenerative process generally has little or no direct effect on core cognitive intelligence, yet patients frequently develop debilitating psychological and emotional symptoms. These symptoms are often secondary, resulting from the patient's profound reaction to the chronic, progressive nature of the disease and the resulting loss of autonomy and physical capability.

The psychological reactions typically observed include significant **apathy and inactivity**, leading to a marked reduction in daily physical and mental engagement. Patients often experience significant **social withdrawal**, partly due to embarrassment over their visible tremors and gait difficulties, and partly due to the generalized reduction in motivation. Furthermore, PD significantly compromises executive function, leading to difficulty in maintaining attention, an **inability to concentrate**, and a diminished capacity to maintain intellectual interest in previously engaging activities. These cognitive and affective disturbances often require focused intervention, such as psychotherapy, to manage their profound impact on daily functioning and emotional well-being.

4. Etiological Theories and Risk Factors

Despite extensive research, the precise etiology of the most common form of **Parkinson's Disease** (idiopathic PD) remains largely **unknown**. However, research points toward a complex interplay of genetic susceptibility, environmental toxins, and acquired metabolic deficiencies. Epidemiological data highlight the prevalence of the condition, with estimates suggesting approximately half a million cases within the United States alone, underscoring its significant public health burden. One identified subtype of Parkinsonism is distinctly associated with vascular issues, such as **arteriosclerosis**, which can compromise blood flow to the basal ganglia, leading to vascular parkinsonism.

Early studies, such as the work conducted by Spellman (1962), suggested indications of inherited metabolic defects in a small percentage of cases, confirming that certain genetic predispositions can increase vulnerability to the disorder. However, the majority of research points strongly toward an **acquired deficiency in brain metabolism**, suggesting that external factors play a more dominant role in the general population. A leading hypothesis posits that this metabolic deficiency may be caused by an environmental agent, possibly a **virus that attacks nerve cells**, specifically targeting those neurons leading to the motor cortex. This viral assault would trigger the cascade of neurodegeneration observed in the substantia nigra.

It is also critical to distinguish between idiopathic PD and conditions that lead to **secondary parkinsonian symptoms**, which mimic the motor features of the disease but arise from known external causes. These acquired forms include, but are not limited to, the aftermath of infectious diseases such as epidemic encephalitis, exposure to neurotoxins like **carbon monoxide poisoning**, or structural abnormalities within the brain, such as certain **brain tumors** or hydrocephalus. The identification of such secondary causes is vital because, unlike idiopathic PD, managing the underlying condition may partially alleviate the parkinsonian symptoms.

5. Therapeutic Strategies and Management

The management of **Parkinson's Disease** presents a significant challenge, as **no treatment yet devised is completely effective** in curing the degenerative process. However, modern therapeutic approaches are highly successful in managing symptoms, slowing functional decline, and significantly improving the patient's quality of life, particularly when detection occurs early in the disease course. Early detection is considered **essential** because timely pharmacological intervention can maximize benefits. The mainstay of initial treatment involves the use of **antispasmodic and muscle relaxant drugs**, which are highly effective in relieving the debilitating rigidity and persistent tremors that characterize the motor dysfunction.

Pharmacological management is often augmented by supportive therapies addressing the emotional toll of the disease. As noted, **psychotherapy** is frequently employed and often proves effective in controlling the secondary psychological reactions, such as depression, anxiety, and social withdrawal, helping patients adjust to their chronic condition and maintain functional engagement. The most important pharmacological agents are those that replace or potentiate dopamine, such as levodopa (L-DOPA), which acts as a dopamine precursor, and dopamine agonists, which directly stimulate dopamine receptors in the brain. The effectiveness of L-DOPA revolutionized PD treatment, though long-term use can lead to troublesome side effects, including motor fluctuations and dyskinesias.

In recent decades, significant advancements have been made in **surgical methods** for treating advanced PD, particularly for patients whose symptoms are poorly controlled by medication or who

suffer from severe motor fluctuations. These neurosurgical techniques have demonstrated remarkable success, arresting the progressive motor disorder in approximately 90 per cent of properly selected cases. One historical technique mentioned in early medical literature involves **cryosurgery**, where defective cells in the thalamus--a crucial relay station that switches impulses to the brain's motor centers--are precisely destroyed. This is achieved by carefully dripping a freezing solution into the affected area. Modern surgical interventions have largely shifted toward Deep Brain Stimulation (DBS), where electrodes are permanently implanted in target areas like the subthalamic nucleus or globus pallidus to deliver electrical impulses that normalize abnormal brain activity.

Further Reading

[Parkinson's disease \(Wikipedia\)](#)

[National Institute of Neurological Disorders and Stroke \(NINDS\) - Parkinson's Disease](#)

Spellman, M. (1962).