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# **Dysphagia in Parkinson's Disease**

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Additional information is available at the end of the chapter

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## **1. Introduction**

Parkinson's disease (PD) is the second most common neurodegenerative disorder among the elderly after Alzheimer's disease. It affects around 1% of the population over 65 years of age and has a prevalence of 4% or more among individuals over the age of 85 years [1]. The condition is primarily the result of a progressive, chronic loss of dopaminergic neurons in the substantia nigra and striatum and presents with or without intracytoplasmic Lewy body deposits [1-3]. A monogenic etiology is found in only around 5% of cases. In cases that do not have a monogenic etiology, the condition is known as sporadic or idiopathic PD and occurs as a result of interaction between a series of hereditary and environmental factors [3].

Bradykinesia, tremors, rigidity, and postural instability are the main motor signs of PD [4]. PD also presents with a series of non-motor manifestations, including changes in behavior, cognition, learning, and the autonomic nervous system. Dysautonomias are major complications of PD and include orthostatic (postural) hypotension (OH), constipation, anhydrosis, erectile dysfunction, sialorrhea, dysphagia, esophageal dysmotility, gastroparesis, irritable bladder symptoms, and nocturia [5-7].

Dysphagia is a problematic and sometimes dangerous feature of PD. Oropharyngeal dysphagia can have a negative impact on quality of life [8,9] and increases the risk of aspiration pneumonia, which is often a cause of death in PD [10,11]. Subjective dysphagia occurs in one third of PD patients. Objectively measured dysphagia rates are much higher, with four out of

five patients being affected. The prevalence of dysphagia in four studies was between 72% and 87%, with a pooled prevalence estimate of 82% (95% CI 77%–87%), suggesting that while the condition is common in PD, patients do not always report swallowing difficulties unless asked. This under-reporting calls for a proactive clinical approach to dysphagia, particularly in light of the serious clinical consequences of the condition [12].

The aim of this chapter is to show dysphagia as a symptom/sign and an important cause of disability in patients with PD.

## 2. Definition and classification of Parkinsonism and PD

PD is a progressive parkinsonism of undetermined cause without features suggestive of an alternative diagnosis. It responds to dopaminergic treatment and is associated with depletion of dopaminergic neurons and the presence of Lewy body inclusions in some of the remaining nerve cells [13–15].

The diagnosis of PD is based on clinical criteria as there is no definitive laboratory test to diagnose the disorder. Pathological confirmation of Lewy bodies on autopsy, a hallmark of PD, has historically been considered the standard criterion for diagnosis [13]. In clinical practice, diagnosis is usually based on the presence of a number of cardinal motor features, response to levodopa, associated symptoms, and the exclusion of other disorders [15]. When patients have a classical presentation, diagnosis of PD is relatively simple. However, differentiating PD from other forms of parkinsonism in the early stages of the disease, when signs and symptoms overlap with other syndromes, can be difficult [16]. Criteria for diagnosis of PD were developed by the UK Parkinson's Disease Society Brain Bank (Table 1) and the National Institute of Neurological Disorders and Stroke (NINDS) [7,14]. When standard clinical criteria such as the UK Parkinson's disease brain bank criteria are used, significantly more accurate clinical diagnosis of the disease is possible; however, as many as 10% of patients diagnosed with the disease in life will still need to be reclassified at post-mortem examination [17]. These criteria have been estimated to have a diagnostic specificity and sensitivity of 98.6% and 91.1%, respectively [18].

Parkinsonian disorders can be classified into four categories: primary (idiopathic) parkinsonism, secondary (acquired, symptomatic) parkinsonism, hereditary degenerative parkinsonism, and multiple system degeneration (parkinsonism plus syndromes). A number of features, including tremor, early gait abnormality (e.g., freezing), postural instability, pyramidal tract signs, and response to levodopa, can be used to distinguish PD from other parkinsonian disorders. While differences in the density of postsynaptic dopamine receptors in PD patients or patients with atypical parkinsonian disorders have been used to account for the poor response to levodopa therapy in the latter group, other explanations may also be possible [19].

UK Parkinson’s Disease Society Brain Bank
Step 1
Bradykinesia
At least one of the following criteria:
Muscular Rigidity
4–6 Hz rest tremor
Postural instability not caused by primary visual, vestibular, cerebellar, or proprioceptive dysfunction
Step 2
Exclude other causes of parkinsonism
Step 3
At least three of the following supportive (prospective) criteria:
Unilateral onset
Rest tremor
Progressive disorder
Persistent asymmetry affecting the side of onset most
Excellent response (70%–100%) to levodopa
Severe levodopa-induced chorea (dyskinesia)
Levodopa response for 5 years or more
Clinical course of 10 years or more

**Table 1.** Diagnostic criteria for Parkinson’s disease (PD) [7,14]

3. Non-motor symptoms in Parkinson’s disease

In his “Essay on the Shaking Palsy,” James Parkinson did not consider PD to be just a motor disorder, and in 1817 he described the presence of sleep disturbance, constipation, dysarthria, dysphonia, dysphagia, sialorrhea, urinary incontinence, and constant sleepiness with slight delirium [20]. What has been well established is that in PD patients, non-motor symptoms (NMS) occur in more than 90% of individuals in all stages of the disorder and include a variety of symptoms from neuropsychiatric and autonomic dysfunction to sleep disturbances and little-understood and little-reported sensory symptoms such as pain and impaired vision [21]. These NMS frequently occur throughout the course of PD and may occur concurrently with motor symptoms or precede their onset by several years [22]. In advanced PD, NMS have been recognized as important factors associated with impaired quality of life [23] and impose a considerable economic burden on patients’ families and society [24].

In contrast to the motor symptoms of PD, NMS are often under-recognized during routine clinical visits and poorly managed in clinical practice [21,25]. Indeed, they have only recently

been considered sufficiently important to warrant study by clinicians and researchers. In a prospective study of 101 PD patients, neurologists failed to identify the presence of depression, anxiety, and fatigue in over 50% of patients and the presence of sleep disturbance in 40% [26].

#### 4. Clinical features and diagnosis of dysphagia in Parkinson's disease

Oropharyngeal dysphagia in PD may limit or preclude safe oral feeding, reducing the patient's full capacity in society and resulting in social, psychological, and economic problems for the individual [27].

One of the first reports of parkinsonian dysphagia was in 1817 by James Parkinson, who described a typical case of PD with weight loss, difficulty in swallowing solids and liquids, sialorrhea, and reduced tongue movements [21].

In 1983, Longemann proposed the videofluoroscopic swallowing study (VFSS) as a means of assessing the dynamics of swallowing. Parkinsonian VFSS [27] can show specific impairments, such as those in the oral pharyngeal and esophageal phases. In the oral phase, these include orofacial tremor, difficulty forming a cohesive food bolus, prolonged swallowing time, limited tongue and mandibular excursion during mastication, and the presence of repetitive antero-posterior movements of the tongue during bolus propulsion (lingual festination). Pharyngeal phase impairments include delayed pharyngeal response with consequent stasis in the valleculae and piriform sinuses, with the risk of laryngeal penetration and aspiration, and impairment of pharyngeal muscle contraction and cricopharyngeal function [28]. Impairments in the esophageal phase of swallowing include reduced peristalsis and reduced transit time. All of these disturbances occur together with the traditional motor symptoms of PD (bradykinesia and rigidity) as a result of degeneration of the autonomic nerve system and voluntary muscle system [29, 30].

The incidence of swallowing disorders in PD varies from 50% to 100% of patients. Another characteristic reported in studies of dysphagia in PD is that it may be present without symptoms, making it difficult to identify the condition early and, consequently, to plan a more efficient therapeutic approach [27].

Interestingly, many patients who do not complain of feeding difficulties report having eliminated certain types of food that caused them swallowing difficulties, often restricting themselves to food with a purée consistency. Some patients report weight loss associated with swallowing disorders.

As a result of swallowing disturbances, parkinsonian patients may present with tracheal aspiration (entry of material into the airway), which is generally asymptomatic and known as silent aspiration. Tracheal aspiration related to swallowing is a major cause of morbidity and mortality in PD, suggesting a reduction in voluntary mechanisms that protect the upper airways [31].

## 5. Management of dysphagia in patients with Parkinson's disease

Treatment of PD has traditionally been pharmacologic and was revolutionized by the discovery that levodopa is able to penetrate the blood--brain barrier and be converted to dopamine in the central nervous system. However, the effect of pharmacologic treatment on oral communication and swallowing is still controversial.

Drug treatment appears to have little effect on speech and swallowing disturbances compared with the major effect it has on motor symptoms in the trunk and limbs. In a study on voice and swallowing, when patients were asked about the effects of medication, all reported clear improvements in general physical symptoms, but only three out of twenty-four patients reported improvements in oral communication and swallowing symptoms. This suggests that both dysarthrophonia and dysphagia are related to dysfunction of nondopaminergic neuronal pathways [27].

Although the subject of much controversy in the past, the value of speech therapy has been confirmed in several objective studies [29-31]. The effects of speech therapy on voice and swallowing were analyzed in a study, which showed that there was a 100% improvement in symptoms after therapy, particularly increased sphincteric action of the larynx [27].

Speech therapy for parkinsonian dysphagia includes exercises to increase mobility of oropharyngeal and laryngopharyngeal structures involved in mastication and swallowing, specific techniques to improve formation and propulsion of the bolus, and maneuvers that increase airway protection during swallowing. In cases of severe dysphagia, speech language pathologists work with compensatory strategies such as modifications in bolus consistency and viscosity and postural maneuvers [31].

In 1987, Lorraine Olson Raming and Carolyn Mead developed an effective treatment program for individuals with PD voice tremor. The technique is known as LSVT (Lee Silverman Voice Treatment) and was named after the first patient who received the treatment (Lee Silverman). The basic principle used in this approach involves increasing vocal effort to enable patients to speak louder. An important study reported significant improvements in swallowing in patients who received LSVT treatment. Temporal measurements such as oral and pharyngeal transport time, duration of contact between the base of the tongue and the pharynx and duration of velopharyngeal closure and laryngeal elevation were taken. There was a 51% improvement in dysphagia after LSVT [32].

Biofeedback is a technique that uses visual or auditory references and electromyography, mirrors or other tools to show physiological events to the patient. It is used to enhance learning by means of exteroceptive systems, which replace inadequate proprioceptive signals, to improve voluntary motor control, to provide more specific, faster sensory information and to facilitate motor relearning. Visual information can compensate for sensorimotor loss by allowing individuals to assimilate lost or altered information and reduce body asymmetry by reestablishing a central motor program that takes into account position and movement [32,33].

Doppler sonar has been investigated as a method for assessing swallowing [34]. The feasibility of using this method as an aid to the assessment of swallowing and the benefits it brings have

been confirmed in recent studies. The use of Doppler sonar to provide biofeedback of swallowing in PD patients not only helps them understand the swallowing process and how they can influence it, but also enables them to derive satisfaction from performing an activity over which they previously seemed to have no control.

## 6. Conclusion

Oropharyngeal dysphagia is a common symptom in PD and can develop at any time during the course of the disease. In fact, some authors have suggested that dysphagia may be one of the initial symptoms of PD.

Treatment of dysphagia in PD requires specialized rehabilitation combining treatment strategies that maximize social functioning, minimize the swallowing burden on the patient, and improve mental health [35].

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