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التوثيق الالكتروني والميكرو فيلم

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Recent Trends In Management of Phoniatic Disorders In Parkinsonism

Essay Submitted for the Partial Fulfillment of the
Master Degree in Phoniatics

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"بسم الله الرحمن الرحيم"

(سبحانك لا علم لنا إلا ما علمتنا انك

أنت العليم الحكيم)

صدق الله العظيم

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Introduction

Motor organization considers the different functions of the various levels of motor activity, which are built into a hierarchy of levels within the nervous system. Three levels that may be implicated in clinically identifiable motor speech syndromes are the lower motor neuron, the extrapyramidal, and the upper motor neuron levels. The fourth system responsible for proper production of motor speech, the cerebellum, does not initiate movement but rather serves as an error detector and error corrector of movements initiated at the other levels.

Dysarthria is that neurologic motor speech impairment which is characterized by slow, weak, imprecise, and/ or uncoordinated movements of speech musculature. Literally the term comes from the Greek dysarthron, which means "inability to utter distinctly" (*Yorkston and Beukelman, 1981*).

Parkinson's disease was first described in 1817 by James Parkinson, a British physician who published a paper on what he called "the shaking palsy". In this paper, he set forth the major symptoms of the disease that would later bear his name (*Parkinson, 1817- cited from Fahn, 1989*).

Definition:

Tanner et al. (1992) defined Parkinson's disease as a distinct clinical and neuro pathologic entity characterized clinically by bradykinesia, resting tremor, cogwheel rigidity and postural reflex impairment and pathologically by the loss of pigmented neurons, mostly in the substantia nigra, with associated characteristic eosinophilic cytoplasmic inclusions (Lewy bodies).

Parkinson's disease (PD) was also defined by *Shannak and Kish (1998)* as a slowly progressive neurodegenerative disorder characterized by a loss of nigrostriatal neurons and a reduction of striatal dopamine.

Pathology and Pathogenesis :

Parkinson's disease occurs when certain nerve cells, or neurons, in the substantia nigra die or become impaired. Normally, these neurons produce an important brain chemical known as dopamine which is a chemical messenger responsible for transmitting signals between the substantia nigra and the "next relay" station of the brain, the corpus striatum, to produce smooth, purposeful muscle activity. Loss of dopamine causes the nerve cells of the striatum to fire out of control, leaving patients unable to direct or control their movements in a normal manner. Studies have shown that Parkinson's patients have a loss of 80 percent or more of dopamine-producing cells in the substantia nigra (*Tanner and Goetz, 1989*). *Langston et al. (1996)* reported that Parkinson's disease may occur when either an external or internal toxin selectively destroys dopaminergic neurons. Environmental factors such as exposure to pesticides or a toxin in food supply is an example of the kind of external trigger that could hypothetically cause Parkinson's disease. Several pathogenetic theories are based on the fact that there are number of toxins, such as 1-methyl-4-phenyl -1,2,3,6,- tetrahydropyridi (MPTP) and neuroleptic drugs, which are known to induce Parkinsonian symptoms in human (*Fahn, 1989*). Pedigree and segregation analysis suggest autosomal dominant inheritance of a gene or genes in a subset of families with PD. There is an increasing interest concerning the role of genetic factors in the etiology of PD. Two mutations in the alpha-synuclein gene have been identified as being responsible for PD in few families (*Kuhn and Muller, 1998*).

Clinical Presentation:

* Parkinson's disease (PD) is relatively a common disease, affecting 1% of the population above the age of 65 in the UK. Males are slightly more often affected than females (*Kessler, 1972*). The classic syndrome of parkinsonism includes tremor at rest, rigidity, bradykinesia, masked face, stooped posture, and a shuffling gait. Dysarthria may be an early feature, although dysphagia usually occurs later. Significant orthostatic symptoms may predominate in some patients. Depression is a significant component in many patients and may be a feature of the early disease. The estimated frequency of dementia (which usually develops late) varies widely, but at least 15 to 20 percent of patients develop cognitive impairment.