### Chart 43-3: The Client with PD

#### NANDA, NIC, and NOC Linkages

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- Adapt the environment to aid in sleep (e.g., darken the room and decrease noises). Reducing environmental stimuli decreases external sleep disturbances.

### Using NANDA, NIC, and NOC

Chart 43–3 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with PD.

### Home Care

It is important for both the client and the family to maintain independence and self care as long as possible. To maintain function and quality of life, the following topics should be addressed.

- Realistic expectations
- Equipment suppliers
- Home environment conducive to using equipment
- Referrals to speech therapist, occupational therapist, physical therapist, dietitian

- Gait training and exercises for improving ambulation, speech, swallowing, and self-care
- Increased fluid intake of 3000 mL/day and increased fiber in every meal
- Stool softeners or laxatives as needed for bowel elimination
- Swallowing during eating and taking medications (Have suction equipment available and know the Heimlich maneuver if choking occurs.)
- Foods that can be easily swallowed (such as pureed or soft) and feed six small meals a day if possible
- Helpful resources:
  - American Parkinson’s Disease Association
  - National Parkinson Foundation, Inc.
  - Parkinson’s Disease Foundation
  - The National Institute of Neurological Disorders and Stroke

### A Client with PD

#### Walters Avneil, age 78, was diagnosed with PD at age 64. His wife died 5 years ago and he has no other family living. Mr. Avneil worked for more than 40 years as a mechanic in a large factory. He is a resident of a long-term care facility. During his last clinic visit for a review of his medications, the following assessment was made.

#### Assessment

- Elderly white male with history of PD for the past 14 years. Skin oily and damp. Tremors in both hands and the lips. Gait is slow and shuffling, with a forward leaning posture. Speech slow and slurred. Face expressionless. Has lost 10 lb since last visit 3 months ago. Has been on levodopa with carbidopa since diagnosis. States major problems are “eating problems, bowel problems, walking problems.”

#### Diagnosis

- Constipation, related to lack of exercise, decreased food intake, and effects of medications
- Impaired verbal communication, related to lip tremors, slow/slurred speech, and facial muscle involvement of PD
- Imbalanced nutrition: Less than body requirements, related to difficulty swallowing and chewing
- Impaired physical mobility, related to rigidity and bradykinesia

#### Expected Outcomes

- Have a soft stool at least every other day.
- Practice exercises provided by speech therapist twice a day.
Huntington’s disease, also called chorea, is a progressive, degenerative, inherited neurologic disease characterized by increasing dementia and chorea (jerky, rapid, involuntary movements). It is a single-gene autosomal-dominant inherited disease that causes localized death of neurons of the basal ganglia (Porth, 2002). The exact cause is unknown, but postmortem studies have demonstrated a decrease in gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter in the basal ganglia. There is also a decrease in acetylcholine levels, suggesting that the manifestations are the result of an imbalance in dopamine and acetylcholine. Although carriers can be identified, there is no cure for the disease. Huntington’s disease causes progressive chorea, speech problems, and dementia.

Because the client is usually asymptomatic until age 30 to 40, he or she may already have passed the gene to the next generation. The psychologic impact is devastating to clients and their families. The family not only experiences guilt from passing the disease from one generation to the next, but also is faced with the overwhelming long-term care needs of those affected. It is common for several family members to be afflicted with the disease.

**PATHOPHYSIOLOGY**

Huntington’s disease causes destruction of cells in the caudate nucleus and putamen areas of the basal ganglia. Other areas of the brain, such as the frontal lobes, may selectively atrophy. Several neurotransmitters and their receptors are decreased, including GABA and acetylcholine. The neurotransmitter dopamine is not affected in Huntington’s disease, but the decrease in acetylcholine results in a relative excess of dopamine in the basal ganglia. Whereas in Parkinson’s disease a deficit of dopamine causes slow movement or lack of movement, in Huntington’s disease the opposite occurs: There is a relative excess of dopamine, causing excessive, uncontrolled movement.

**MANIFESTATIONS**

Manifestations primarily involve abnormal movement and progressive dementia (see the box on page 1425). The progression and sequence of manifestations varies somewhat; however, initially the psychologic manifestations are more debilitating than the choreiform movements.

Early signs of personality change include severe depression, memory loss with decreased ability to concentrate, emotional lability, and impulsiveness. The client experiences frequent mood swings ranging from uncontrollable periods of anger to apathy. Eventually, signs of dementia, including disorientation, confusion, and lack of sense of time, become evident and interfere with self-care.

Motor symptoms usually parallel personality and mood changes. The motor symptoms worsen with environmental stimuli and emotional stress but are absent when the client is sleeping. Initially, movement problems are described as “fidgeting” or restlessness, followed by progressive worsening of