



Parkinson's Disease Self-Directed Learning Package For Nursing Staff

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Learning Objectives

On completion of this package the participant will have a basic understanding of:

- Parkinson's disease (PD), its aetiology and incidence.
- The pathophysiology of Parkinson's disease.
- The Clinical Phenomenology & Symptomatology including:
 - i. Motor Symptoms
 - ii. Non-Motor Symptoms
 - iii. Neuropsychiatric Symptoms
- Commonly used medications in the management of Parkinson's disease
- The use of a Parkinson's diary in the management of a patient with PD
- The role of the multidisciplinary team in the management of a patient with PD
- Device Assisted Therapies in the management of PD
- Nursing Considerations when caring for a patient with PD



Glossary & Abbreviations

Akinesia – slowness and difficulty of initiation of movement.

Anosmia – loss of sense of smell

Bradykinesia - slowness or poverty of movement

Dyskinesia – involuntary, unnatural movements, which can affect any part of the body. Generally a side effect of PD medications. Similar movements can occur in other diseases e.g. Huntington's disease.

Dystonia – abnormal cramping postures, more often of the limbs which can be painful. Dystonia is most often, but not always, experienced when the patient is under-medicated with PD medications, for instance during the night or early morning.

Tremor – regular rhythmic oscillation of a part of the body, jaw, neck, mouth, trunk, limbs and /or extremities.

'OFF' – A Parkinsonian state when a PWP is under-medicated and manifesting Parkinsonian symptoms.

'ON' – A Parkinsonian state when a PWP is optimally medicated and Parkinsonian symptoms are well controlled or diminished.

PD – Parkinson's disease

PWP – Person with Parkinson's disease

RIGIDITY - Stiffness, increased resistance to movement. Generally detected in limbs and neck and associated with OFF state. Sometimes described as 'cog wheeling' resistance to movement.

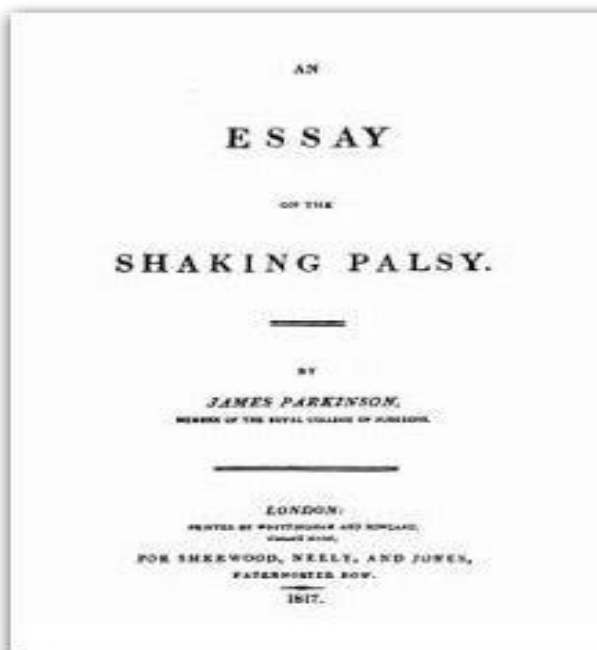
FREEZING OF GAIT- brief episodes of an inability to step. Can occur during initiation of first step, during walking or on turning and with the feet seeming to be stuck to the floor. This can result in extremely short steps being taken.

Introduction

Parkinson's disease (PD) is the second most common neurodegenerative disorder after Alzheimer's Disease and is the most common disorder in the neurology sub-specialty of 'Movement Disorders'. In Australia, Parkinson's disease affects approximately 1% of the population over 55 years and around 60,000 Australians with the mean age of onset is around 60 years. Males are slightly more affected with ratio of around 5:4 (Deloitte 2015). It is a multi-system disease that is slowly progressive with no cure (Hayes M et al. 2010 p.144). The aim of treatment is to maximise the quality of life for the person with Parkinson's (PWP) at each stage of disease progression.

History

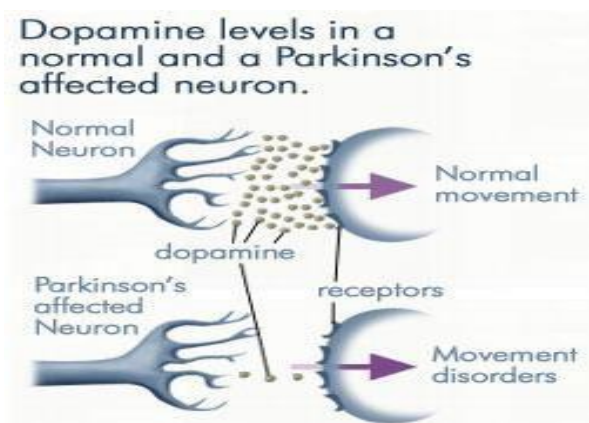
PD was first formally described by Dr James Parkinson in 1817 in 'An essay on the shaking palsy (*paralysis agitans*)'. James Parkinson described the cardinal signs of rigidity, bradykinesia and resting tremor that he had observed in 6 people. He was also able to identify some non-motor features of PD such as constipation and sleep disturbance. But it was not until 1861 and 1862 that a French neurologist, Dr Jean-Martin Charcot, renamed *paralysis agitans* (shaking palsy) to Parkinson's disease in recognition of Dr James Parkinson's contribution. Despite the age of this publication, the content of this document is still relevant to this day.



Pathophysiology

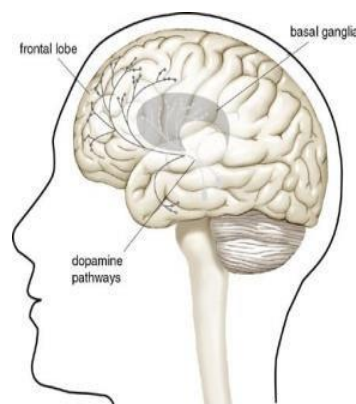
The traditional understanding of the pathophysiology of PD begins with the destruction of dopamine producing neurons in the substantia nigra located in the midbrain. These structures are part of the basal ganglia which is responsible for initiation and controlling voluntary movements. The reduced production and action of dopamine lead to reduced neuro-transmission and stimulation to the motor cortex causing motor dysfunction (McCance & Heuther, 2006 pp 538). However, more contemporary thinking that is widely accepted is known as the Braaks Hypothesis. The Braaks hypothesis proposes that there are 6 stages of PD.

Diagram 1. Comparison of Dopamine levels in normal Neuron and a PD affected neuron.



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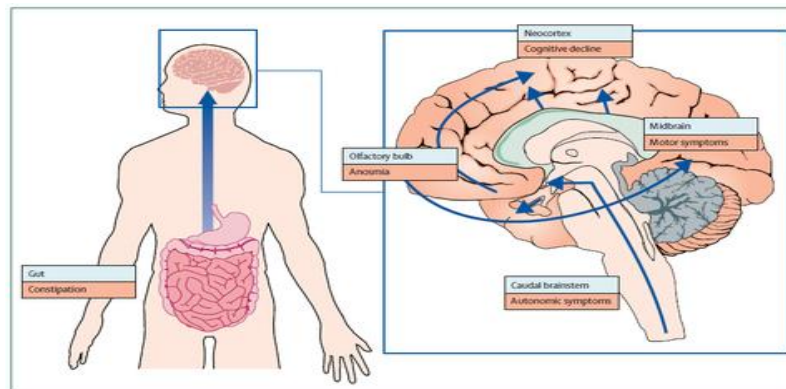
Diagram 2. Location of the Basal Ganglia



University of Fribourg 2010, 'dopamine'

Stages 1 and 2 are the premotor or prodromal phase of PD which suggests the pathophysiology of PD begins in the gut causing gastrointestinal symptoms commonly manifesting as long standing constipation years before the diagnosis of PD. The progression of the pathophysiology into the medulla causes autonomic dysfunctions such as temperature imbalance, postural hypotension, urinary and erectile dysfunction. Further progression to the pons causes the sleep disturbances such as REM sleep behaviour disorders, insomnia and day time somnolence (Braak et.al 2003, p 208)

Diagram 3. Braak's Hypothesis – Lewy Pathology

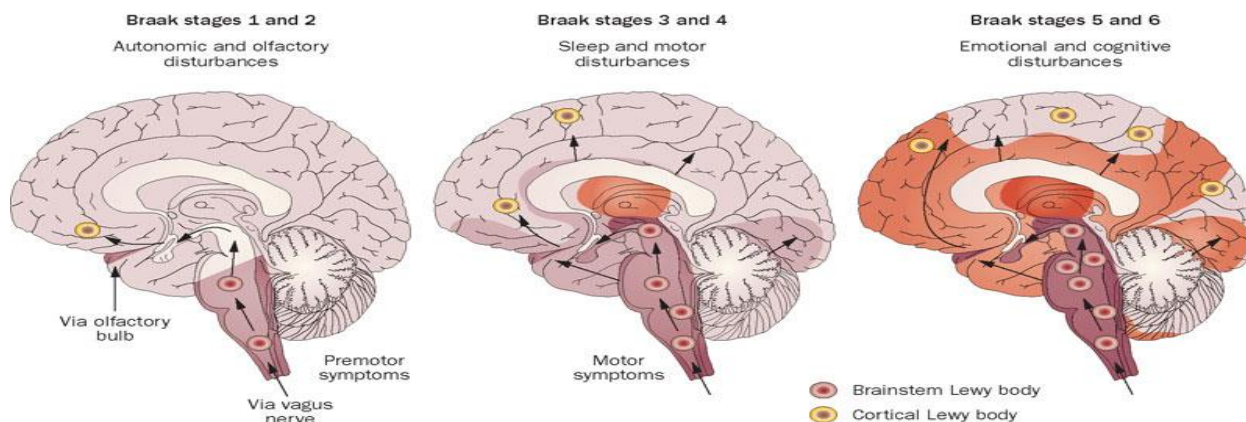


Source: Elodi A et.al. 2010, p1130

Stages 3 and 4 are classed as the motor or clinical phase of PD when the midbrain is affected causing the manifestations of the motor symptoms such as tremor, rigidity, akinesia and postural imbalance which allows for the clinical diagnosis of PD to be made (Braak et. al. 2003, p209). By the time of mild symptom manifestation and diagnosis it is accepted that 60-80% of dopamine producing cells are already lost.

Stages 5 and 6 are caused by further progression of the pathophysiology into the forebrain and the rest of the cortex which leads to the cognitive impairments such as dementia, hallucinations and psychosis and can exacerbate pre-existing anxiety and depression (Braak et. al. 2003, p209).

Diagram 4. Stages of Braak's Hypothesis



Source: (Doty R, 2012 p330)

Pre-Motor / Prodromal Phase	Motor / Clinical Phase	Cognitive Phase
<p>Gut → Constipation</p> <p>Medulla (Lower Brainstem) → Autonomic Dysfunction (e.g. postural hypotension, urinary and erectile dysfunction, temp. imbalance)</p> <p>Pons → REM Sleep Behaviour Disorder (RBD), insomnia, daytime somnolence</p> <p>Olfactory Bulb – Anosmia</p>	<p>Manifestation of motor symptoms of PD</p> <p>Clinical Diagnosis of PD: Tremor Rigidity Akinesia Posture and gait disturbance Other motor symptoms of PD</p>	<p>Cognitive Impairments</p> <p>Dementia</p> <p>Hallucinations</p> <p>Psychosis</p> <p>Depression & Anxiety</p>

Clinical Phenomenology & Symptomatology

Signs and symptoms of PD are explained below but it is important to be aware that these vary greatly from one person to another. Disability and the perception of disability can also vary from one person to another.

Parkinson's disease is commonly described as a multi-system disease and although a large component are motor symptoms of PD, the non-motor symptoms can be just as debilitating and dramatically impact a person's quality of life. The features of PD can be considered in three main domains:

- i. Motor Symptoms
- ii. Non-Motor Symptoms
- iii. Neuropsychiatric Symptoms

Motor Symptoms

Classic PD motor symptoms are often remembered as the acronym T.R.A.P:

- Tremor
- Rigidity
- Akinesia
- Postural Instability

1. Tremor

Tremor is a rhythmic, regular and oscillating motion of the hand and the tremor is often referred to as 'pill-rolling' tremor that does not interfere with activity. It is usually seen as an early sign and is most often the first sign in PD. It is described as a resting tremor, which subsides with movement and is abolished during sleep. Tremor is seen most commonly in an upper limb but can occur in any part of the body (e.g. Jaw, neck, foot). (Deuschl et.al. 1998, p.3). Around 60-70% of people with PD have a resting tremor. (Donaldson et.al, 2012, p 243) The tremor, however, can progress over time to the other upper limb or to the lower limbs, face and chin and might then be seen in a PWP in action posture or 'ON' state. Tremor does sometimes contribute to the person's disability and may also be a source of embarrassment. Tremor may also be caused by other disorders e.g. essential tremor, dystonic tremor.

2. Rigidity

Muscles become rigid which results in immobility and, with many people, muscular or joint pain. The rigidity in PD is often described as 'cog-wheeling'. Rigidity of musculature is seen more easily in the neck and wrists on testing.

3. Akinesia & Bradykinesia

Bradykinesia describes the slowness or poverty of voluntary movement that affects the muscles of the trunk, limbs, face, speech, swallowing or mastication. Akinesia refers to the difficulty in initiating movement and both of these symptoms are common in people with PD and can be one of the most frustrating symptoms in PD. (MDS n.d)

4. Postural Instability / Impaired Postural Reflexes

PD patients will commonly have a stooped posture and reduced or diminished arm swing particularly on the more affected side. Impaired postural reflexes in PD results as difficulty in regaining balance once balance has been disrupted e.g. Foot caught on rug. It causes difficulty with regaining/maintaining balance and therefore falls are more likely. A PWP is best assessed by a physiotherapist for the most appropriate walking aid as some aids are considered dangerous for a PWP.

5. Shuffling Gait

In gait, both step height and stride length are also often reduced. Festination, where the person takes small ever-increasingly faster steps without control, retropulsion, the same phenomenon in reverse, and propulsion, the inability to stop and is (often described by PWP as a sensation of being pushed from behind) contribute to falls in PD particularly where postural reflexes and balance are impaired.

6. Freezing of Gait

'Freezing' or motor block can also occur where the person is suddenly unable to move, and it may take an external cue to be able to move again. Freezing of gait may occur on initiation of walking, on turning and especially at doorways and narrow corridors but can also be seen in other motor sequences. (Nutt et.al 2011 p734)

The patient can be encouraged to learn tricks to combat freezing by pretending to walk upstairs, marching, stepping over lines, or an object, humming a tune etc. These are called cues and PD patients can do most things when prompted with a cue. Most recently, laser pointers and laser lines attached to walking aids have been used with some success. It is a matter of finding the method which best suits the individual patient.

Although difficult to arise from a chair, the PWP may suddenly propel forward after getting up. When lying in bed they may experience difficulty in turning. Cueing can also be used to assist a patient to get in or out of bed or turn in bed, without physical assistance, even when they appear completely immobile.



Nursing Considerations

Safety

There are many aspects of safety to be considered. The PWP who has a stooped posture with the lack of ability to correct posture, short shuffling steps, freezing of gait and/or impaired postural reflexes has an increased risk of falling on turning or tripping over obstacles such as a rug or carpet in the home. Falling backwards is also fairly common as a result of impaired postural reflexes.

The need for supervision should be assessed in ambulation, transferring, dressing and hygiene. Independence is fostered within the confines of safety and due to the **changeable nature of this condition it must be constantly reassessed.**

All assessments must consider the state of the patient i.e. ON/OFF/ DYSKINETIC

Mobility

As discussed above, gait is impaired in almost all PWP. A Parkinsonian gait is described as slow, shuffling with reduced step height with or without freezing of gait. The patient does not swing their arms normally and they are often stooped and may be leaning to one side. Holding objects in their hands or arms, or doing two things at once i.e. talking, can distract PWP from concentrating on task of walking and should be discouraged. If they require a walking aid, this should be organised by a physiotherapist as some walking aids may increase the risk of falls to the PD patient. Medication may improve gait disturbances if the PWP's gait difficulties are co-related to their 'OFF' state.

Nursing a patient with PD can be very challenging. The patient that can pass you on his way to the newsagents having just had a quick shower independently can be the same patient who cannot mobilise enough to get to the toilet 2 hours later and is found incontinent and distressed sitting rigidly in their chair.

A PWP may experience difficulty initiating movement overnight and this may lead to difficulties in turning in bed. Regular overnight pressure area care may be required as a result.

7. Dystonia

Dystonia is a common symptom in Parkinson's disease in which muscles contract involuntarily, which can force certain parts of body into abnormal movements or postures and may be with or without pain. Pain in PD is under-reported and is reasonably common. Dystonic pain is usually relieved when the PWP reach their 'ON' state. (Derrey et.al 2010 p 1264)

8. Speech & Swallowing Difficulties

In PWP, volume and projection of speech tends to be softer and the speech can be monotonous. Some people talk quickly or in a stammering fashion, while others find their speech slurred and difficult for others to understand. Dysphagia or difficulty in swallowing can impact on a PWP's normal fluid and diet intake. A speech pathologist assessment is required in such circumstances to prevent complications such as aspiration pneumonia. A pathological swallow is not common and mostly seen in patients with advanced disease. A PWP may benefit from a speech pathology referral to assess dietary problems because of reduced swallowing and rigidity of swallowing musculature. Consideration should be given to the possibility of fluctuating dysphagia as a result of 'OFF' period fluctuations as a PWP may have a safe and effective swallow during their 'ON' states but then be unsafe during 'OFF' states.

Speech pathology can also assist with voice projection as a reduction in volume and clarity, with stuttering and perseveration on sounds are commonly found, especially in latter stages. Because of a lack of facial expression and difficulty in speech, communication can be difficult and the patient can present in the same way as people with cognitive impairment. This can be frustrating for the patient. A speech pathology program known as the Lee Silverman Voice Training (LSVT LOUD) developed in 1987 can improve vocal loudness, intonation, and voice quality for individuals with PD. (LSVT Global n.d)



Nursing Considerations

Communication / Swallowing

Be patient when communicating with the person and give them time adequate time to provide answers. Aids, such as a communication board, may be helpful.

Always ensure a PWP receives their Parkinson's medications on time. Patients should be sat upright to eat meals and preferably not in bed. Encouraging and educating patients to eat during their 'ON' periods allows them to feed themselves with greater independence and also avoid any 'OFF' period swallowing difficulties. Assistance when opening items is often required. Further monitoring the PWP's ability to swallow and their oral intake via a food chart is an important nursing consideration to prevent malnutrition and weight loss during their stay in the hospital/facility. This will also provide appropriate records for dieticians to make adjustments to the PWP's oral intake and to ensure appropriate nutrition is provided. There is no specific diet for PD but soft nutritious meals high in fibre and complex carbohydrates are best for those with advanced disease. Encourage the patient to practice strategies suggested by the speech pathologist.

9. Reduced Facial Expression & Reduced Blink Rate

Mask-like face (hypomimia) sometimes referred to as 'poker face', it is characterised by reduced eye blinking and facial expressions. PWP may also be slow to respond to conversations/questions again making communication challenging. These can lead to problems with communication as emotions are more difficult to express, it is therefore important to listen to the content of the conversation as the lack of facial movement does not allow normal facial gestures. This can add to difficulty in engagement and make the person look as if they are staring or not interested in the conversation. Lack of blinking can also cause dry and watery eyes.



Nursing Considerations

Give PWP time to respond to questions/ conversations and be mindful that lack of facial expression does not mean patient is disinterested or refusing to engage in conversation.

Eye care – advocate for frequent eye care is attended and consider eye refresher drops.

10. MOTOR FLUCTUATIONS ('ON' / 'OFF' Phenomenon & Dyskinesia)

A PWP can fluctuate dramatically through different phases of medication cycle. They may have vastly different motor and cognitive abilities that they experience at different stage of their medication cycles. In the 'OFF' period or pre-medicated state, the person may manifest typical motor symptoms such as tremors, bradykinesia, rigidity and gait disturbance. (Tsui D, 2014 p35)

In the 'ON' period, the medicated state, the same person may be functioning close to the level of a normal person with minimal interruptions from their Parkinson's symptoms.

Dyskinesia is a side effect of PD medications and not a symptom of PD. It occurs when dopamine plasma levels are too high causing a state called 'peak-dose' dyskinesia which is characterised by chorea-like movements. These movements are involuntary, chorea-like or writhing movements which can affect any part of the body. However, dyskinesia can also present in different clinical forms at different points of a PWP's medication cycle. (Hayes M, et al. 2010. p 147)

Diagram 5. Relationship between 'OFF', 'ON' and Dyskinesia

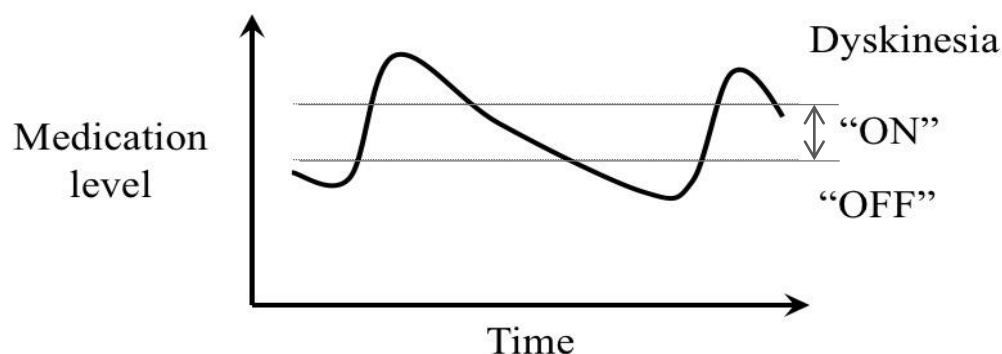


Diagram used with permission of Dr Neil Mahant (neurologist)



Nursing Considerations

Do not interpret fluctuations or inability to perform tasks as being related to a lack of cooperation of the PWP.

Coordinate timing of nursing care / ADL's, therapy sessions, meals, showering, transferring and ambulation when the PD medications take effect and the PWP enters into their 'ON' state. It requires less nursing time and intervention to assist a PWP to the shower in their 'ON' state than it is to assist a PWP in their 'OFF' state when they may require 2 person transfer to commode and maximal assistance in showering and dressing.

Non-Motor Symptoms

1. Bowel & Bladder Dysfunction

Constipation is common and is due to many factors as well as the disease process. Reduction of dopamine neurons in the gut, slowed gastric motility, inadequate roughage and fluid in the diet and reduced exercise can all contribute to constipation (Donaldson et.al, 2012 p 296). A PWP requires strict bowel monitoring and following of regular, daily bowel regime. Constipation may affect absorption of PD medications which can result in failed doses, worsening 'OFF' periods and increased length of stay of hospital admissions.

Urinary urgency and frequency of micturition and nocturia are probably the most reported symptoms, but incontinence and retention of urine can also occur. A PWP may also commonly experience urinary urge incontinence because of reduced mobility and inability to reach the toilet in time. (Donaldson et.al, 2012 p 296) Urodynamic studies are beneficial in highlighting if an underlying problem is present.



Nursing Considerations

Elimination

Many patients with PD have problems with constipation and require extra fibre and natural bowel softeners. Fluids should be encouraged and exercise is beneficial as the gut loses its normal motility.

A PWP may need medications to assist normal bladder function and some will require the insertion of a suprapubic catheter. In some cases the patient is incontinent because they are unable to get to the toilet in time and so trying to improve their motor function may result in better bladder function and independence.

Nocturnal incontinence can occur due to the urgency and or difficulties in mobility. Ensure regular attendance to toileting in form of offers of assistance and/or pad checks/changes overnight to prevent patient discomfort as well as reduce the risk of pressure area injuries and skin breakdown whilst in hospital. Consider creating toilet routines in home environment.

Be mindful of level of assistance and changing needs in relation to PWP 'OFF' and 'ON' period function. A PWP who experiences urgency may try to rush to bathroom which can increase their risk of falling. Prompt attendance to requests for assistance to bathroom can reduce the risk of falls.

Stool chart to ensure regular, quality bowel motions should be included to monitor, prevent patient discomfort and ensure early intervention to prevent constipation.

2. Orthostatic Hypotension

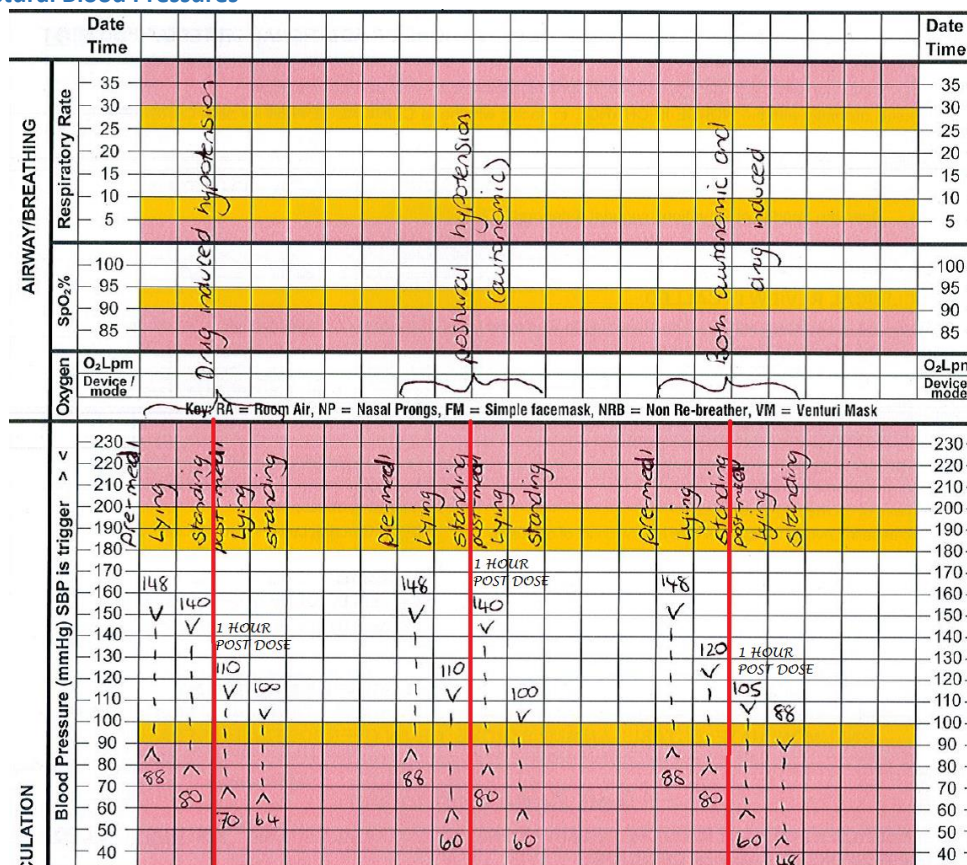
Orthostatic hypotension is defined as a systolic blood pressure drop of 20mmHg or a diastolic drop of 10mmHg within 3 minutes of standing. (Schoffer K, et.al, 2007 pp 1543) The symptoms can include light-headedness, dizziness, visual disturbance and/or syncope secondary to cerebral hypoperfusion which increases the risk of falls and other injuries by causing dizziness or fainting. Orthostatic hypotension is a common problem amongst elderly patients but the impact on morbidity and mortality in patients with Parkinson's disease is even more severe affecting around 20% of this population (Mukai S, et.al. 2002 p 253).

The two main contributing factors include the autonomic dysfunction caused by the Parkinson's disease itself and the use of dopaminergic medications used to treat Parkinsonism which may exacerbate or induce orthostatic hypotension due to its effect on vasodilation (Mukai S, et.al. 2002 p 260). People who have been treated for hypertension before their diagnosis of PD may find they may not require their anti-hypertensives after starting Parkinson's medications.

There are several types of drugs that are used to treat orthostatic hypotension such as the commonly used Domperidone. The other main medications used in the treatment of orthostatic hypotension in Parkinson's disease include Midodrine which is an arterial and venous vasoconstrictor, Fludrocortisone which works by expanding the extravascular body fluid volume (Schoffer K L, et al. 2007 p1543) and pyridostigmine which causes a generalised cholinergic response including increased tone of skeletal and intestinal musculature. (Mims online n.d)

Diagnosis of orthostatic hypotension is confirmed by strict and accurate measurement of lying and standing blood pressures for 24-48 hrs. Lying and standing BP should be taken immediately prior to the administration of the PD medications (namely Levodopa type medications e.g. Sinemet®, Madopar® and Stalevo®) and 1 hour post dose, for each dose PD over 24-48 hours. Patient should be in supine position for 5 minutes prior to taking lying BP and standing for 2-3 minutes prior to taking standing BP.

Diagram 6. Postural Blood Pressures



Example 1	Example 2	Example 3
Drug Induced Hypotension	Postural hypotension secondary to autonomic dysfunction	Combination of drug induced and autonomic dysfunction



Nursing Considerations

Accurate and consistent collection of lying and standing BP's pre and post dose provides valuable information to make adjustments to treatment if required. PWP with suspected / untreated orthostatic hypotension are at an increased risk of falls. Need for supervision of these at risk patients when they ambulate needs to be considered and assessed. Encourage PWPs to increase fluid intake to avoid dehydration. Consider alerting treating medical staff to any concerns or changes.

3. Temperature Imbalance

The person with PD may feel hot when others are cold or the reverse and excess sweating is a common complaint. (Hayes et.al 2010 p 148)

This phenomenon is not always helped by levodopa and may be worsened both during episodes of dyskinesia and /or OFF periods.



Nursing Considerations

Ensure appropriate comfort measures are taken during these times - excessive sweating patients are at increasing risk of skin breakdown especially if appropriate pressure area care is not attended. Comfort measures can be offered such as wet cloth, extra blankets etc.

4. Sleep Disorders

A PWP sleep may be interrupted by motor and/or non-motor symptoms including painful dystonia and/or frequent nocturia, however, primary sleep disorders may also occur. Rapid-Eye-Movement (REM) sleep behaviour disorder is caused when paralysis that normally occurs during REM sleep is incomplete or absent, allowing the person to 'act out' his or her dreams usually characterised by violent limb and trunk movements. It is commonly recognised as a marker of early PD. (Goldman J.G, et.al 2014 p 435). Restless legs syndrome is also frequently found in PWP and often responds to dopaminergic therapy. (Ondo, W.G. et.al 2002 p 421).

Other sleep disorders in PD include daytime sleepiness (somnolence) and insomnia which may be disease and/or treatment related. (Goldman J.G, et.al 2014 p 439)



Nursing Considerations

REM SBD can cause injury and impacts to PWP and partners who share a bed.

Educate re sleep hygiene: limiting daytime naps, avoid alcohol and caffeine drinks late in evening.

5. Sexual Difficulties

This arises from the autonomic dysfunction in the disease process, the restrictions in mobility, the changes in family dynamics and self-image of the person with PD. It is a much ignored problem possibly due to the reticence of the PWP and their partners to talk about this sensitive subject or the fact that there are often more immediate issues. Referral to a sexual health clinic may be beneficial, and /or discuss with GP.



Nursing Considerations

Inappropriate sexual behaviour in PWP can occur as a form of Impulse Control Disorder and is usually related to dopamine agonist treatment or higher doses of levodopa. It can be very upsetting for both PWP and family members and present clinical challenges to nursing staff when caring for such patients.

Documentation and reporting of such behaviours is crucial to ensure appropriate management measures can be provided to support these patients. For example, ensuring what form the behaviour is (inappropriate touching, grabbing, verbally sexual comments), in what context, how frequent the behaviour takes place, and whether the behaviour is troublesome at all are important factors to document and report to the medical team, Community Nurses may consider joint visits.

Nursing interventions for such behaviours include identifying the triggers of such behaviour – for example, male patient may be triggered by female staff members or female patients - interventions such as ensuring same sex nursing staff when possible or during personal care and placing the patient in a same gender room may assist in the management of such behaviours.

Other interventions include distraction strategies such as giving the patient something to hold such as stress balls to occupy the hands during routine care such as taking vital signs (Joller P, et.al 2013 p 257)

Neuropsychiatric Symptoms

1. Bradyphrenia

Slowness in thought processing may lead to delay in replying to questions and can be compounded by word-finding difficulties. Many people with PD also report a lack of concentration which adversely affects conversation. (Donaldson et.al, 2012 p 261). There may be noticeable differences in the same person when the medications are working or when they have entered into their 'OFF' state.

It may take a lot of time for a person with PD to gather their thoughts, concentrate on the topic of conversation and to process their thoughts. This can be very frustrating for the person as their speech may also be affected. Hence the person with PD requires more time for effective communication.



Nursing Considerations

Allow PWP time to formulate thoughts, be patient and encouraging to assist in minimising frustration.

Be mindful of potential cognitive changes between 'ON' and 'OFF' periods.

2. Depression and Anxiety

These are common symptoms. Many studies have shown depression in PD is much higher than in comparable chronic conditions (estimated 40-60%). The reason for this is not completely understood but neurotransmission imbalances, as well as reactive response to the limitations imposed by the condition have to be considered. Anxiety is also fairly common, although it may be restricted to the 'OFF' state and in that case it generally resolves with levodopa. (Stein, M et.al; 1990 p219). Keep in mind that anxiety and unstable moods may impact on the person's absorption of their Parkinson's medications as delayed gastric emptying can occur with anxiety. (Hayes M. et al, 2010 p 148)

3. Hallucinations, Psychosis and Dementia

These are seen in a large percentage of people with advanced PD and more common with elderly onset. It may also be exacerbated by some Parkinson's medications, particularly in the use of dopamine agonists and anticholinergics. A reduction in dosages and frequency of PD medications may be considered. Caution with antipsychotic medications which can markedly worsen motor function. Clozapine and quetiapine are the preferred medications for use with PWP. (Hayes M et.al 2010 p 148)



Nursing Considerations

Reorientation and reassurance of patients experiencing hallucination can assist in reducing these symptoms. Safety of patient, other patients and staff must be considered and appropriate measures taken. Consider referral to appropriate support services, for both PWP and carer. These changes in behaviour can be extremely stressful on carers.

Parkinson's Medications

Medications in Parkinson's disease are used to provide symptomatic relief and work best at the beginning of PD. As time passes the drugs may be less reliable in relieving motor symptoms. There are many reasons why this occurs such as delayed gastric emptying, anxiety and continued reduction in the person's own dopamine production. Side effects, in particular dyskinesia, often worsen. To this point no agent has been shown to slow progression of the disease. The range of medications, with varying formulations and preparations is confusing and are common causes of medication errors. There are six different classes of medications currently in use. Please note the following information is only a quick guide and does not replace full drug information in Australian Medicine's Handbook.

1. Levodopa based Medications

This remains the Gold Standard treatment for PD. Levodopa is a precursor to dopamine which is combined with either carbidopa or benserazide. Carbidopa and Benserazide prevents the conversion of levodopa to dopamine in the peripheries by inhibiting the enzyme (DOPA Decarboxylase or DDC) that synthesizes levodopa to dopamine before it reaches the blood brain barrier. Side effects are often dose dependent and include nausea and vomiting (particularly on commencement of treatment), drowsiness, mood changes, alteration in libido, hallucinations, postural hypotension, dyskinesia and motor fluctuations but generally these drugs are well tolerated over time. [Australian Medicines Handbook (AMH)]

Sinemet® / Kinson® (Levodopa/Carbidopa) Tablets strengths available are:

Regular Release Tablets:

- Sinemet 100/25 tablets
- Kinson 100/25 tablets
- Sinemet 250/25 tablets

Controlled Release Tablets:

- Sinemet CR 200/50 tablets (Controlled Release)

Stalevo® (Levodopa/Carbidopa/Entacapone)

- Stalevo 50/12.5/200
- Stalevo 75/18.75/200
- Stalevo 100/25/200
- Stalevo 125/31.25/200
- Stalevo 150/37.5/200
- Stalevo 200/50/200

Madopar® (Levodopa/Benserazide) Tablets strengths available are:

Regular Release Tablets/Capsules:

- Madopar 100/25 scored pink tablets
- Madopar 200/50 scored pink tablets
- Madopar 50/12.5 blue/grey capsule
- Madopar 100/25 blue/pink capsule
- Madopar 200/50 blue/brown capsule

Rapid Release Tablets:

- Madopar Rapid 50/12.5 dispersible, scored, white tablet
- Madopar Rapid 100/25 dispersible, scored, white tablet

Controlled Release Capsule:

- Madopar HBS 100/25 blue/green capsule

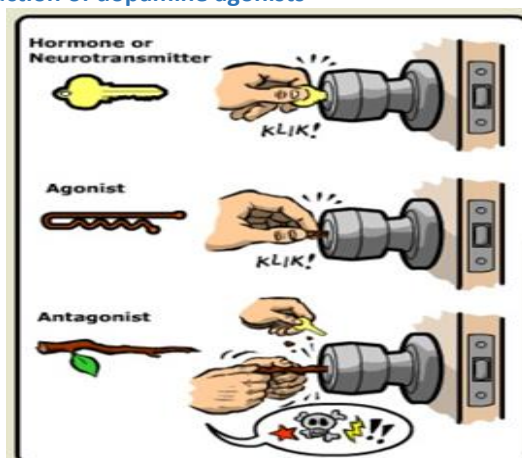
Duodopa® (Levodopa/ Carbidopa Intestinal Gel)

- 20mg levodopa/5mg carbidopa per mL (100mL cassette = 2000mg)
Specialised mode of delivery of Levodopa via PEG-J tube into the small bowel.

2. Dopamine Agonists

Agonists act on the dopamine receptor sites by mimicking the action of levodopa but are not a direct replacement for dopamine. (ed. Brooks 2000) In combination with levodopa they can decrease levodopa dosage and improve motor fluctuations. (Hayes M, et.al 2010 p 145)

Diagram 7. The Mechanism of the action of dopamine agonists



Side effects are similar to levodopa although dopamine agonists are possibly more likely to exacerbate or cause hallucinations or confusion (particularly in the older person). The ergot derived drugs may cause cardiac valve disease, pulmonary or retroperitoneal fibrosis, and as a result Cabergoline and Bromocriptine are not currently widely used in Australia for Parkinson's disease. (AMH, 2017)

Dopamine Agonists have also been associated with a serious side effect known as “Impulse Control Disorder” (ICD) (AMH, 2017) which may manifest as compulsive gambling, hyper-sexuality, over-eating, compulsive spending and obsessive compulsions in a percentage of susceptible patients.

- Apomorphine subcutaneous injection/infusion. Available 20mg/2mL, 50mg/ 5mL, PFS 50mg/10 ml and vials 100mg/20ml
- Sifrol® (Pramipexole) is a short acting non-ergot derived dopamine agonist. Tablet strengths available are 0.125mg, 0.25mg and 1mg. Extended Release strengths are 0.375, 0.75mg, 1.5mg, 2.25mg 3mg, 3.75mg and 4.5mg.
- Rotigotine (Neupro®) transdermal patch is a nonergolinic dopamine agonist. Available in 2mg, 4mg , 6mg and 8 mg patches

3. COMT inhibitors

COMT inhibitors inhibit an enzyme called catechol-o-methyl transferase (COMT) which metabolises levodopa. They need to be administered at the same time as a dose of levodopa and dopa-decarboxylase inhibitor. This enables higher and more sustained levodopa plasma by 30%. COMT inhibitors may be found in combination drugs such as Stalevo®. Side effects include dizziness, dyskinesias, gastrointestinal upset, urine discolouration and they may worsen confusion. (AMH, 2017)

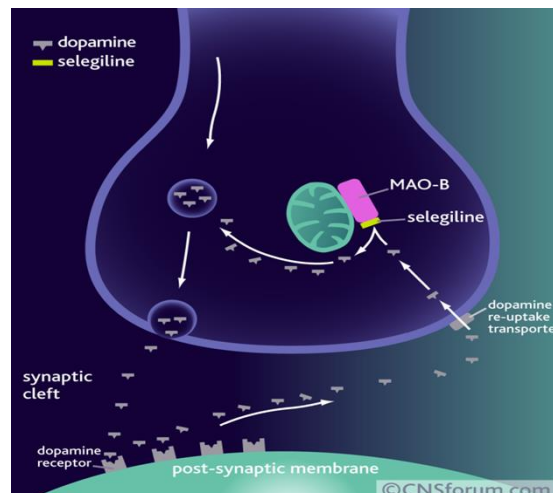
- **Comtan® (entacapone)** 200mg tablets.
- **Stalevo® (Levodopa/Carbidopa/Entacapone)**
 - Stalevo 50/12.5/200
 - Stalevo 75/18.75/200
 - Stalevo 100/25/200
 - Stalevo 125/31.25/200
 - Stalevo 150/37.5/200
 - Stalevo 200/50/200
- Tasmar (Tolcapone) 100mg tablets are not marketed in Australia due to reports of hepatotoxicity. Availability is possible via the Special Access Scheme.

4. MAO-TYPE B Inhibitor

This drug is a selective inhibitor of monoamine oxidase type B, which is one of the enzymes that catabolises dopamine in the brain and blocks dopamine reuptake. MAO-B's mildly enhance dopaminergic effects. Side Effects include sleep disturbances, postural hypotension, headache, nausea, vomiting and confusion. Potential drug interactions with pethidine and selective serotonin reuptake inhibitors (SSRI's). (AMH, 2017)

- Eldepryl, Selgene (selegiline) 5mg tablets.
- Azilect (Rasagiline) 1mg tablets

Diagram 8. The Mechanism of the action of MAO-B inhibitors



5. Anti-Cholinergics

The two main neurotransmitters in the basal ganglia are dopamine and acetylcholine. It is hypothesised that a lack of dopamine alters the balance in neurotransmission in the basal ganglia and so anticholinergics are prescribed to decrease the amount of acetylcholine and in turn attempt to return the balance. Anti-cholinergics are used mainly in tremor-dominant patients. They should not be stopped abruptly. Avoid these agents in the elderly where possible. They are contraindicated in the confused patient as they may increase their confusion. Common side-effects including blurred vision, memory difficulties, dry mouth, constipation, urinary retention, aggravation of glaucoma and psychiatric adverse effects mean that these drugs are not well tolerated. (AMH, 2017)

- Artane (benhexol) 2mg and 5mg tablets.
- Benztrop (benztropine) 2mg tablets.
- Akineton (biperidin) 2mg tablets.

6. Amantadine

This drug possibly acts as a glutamate antagonist as well as some anti-cholinergic activity. It is mostly used for treatment of severe dyskinesias and has been described to be effective in treating freezing of gait. Side Effects include insomnia, confusion, postural hypotension, dizziness and blurred vision. It is usually advised that Amantadine is not to be administered later than the afternoon as it may cause sleep disturbance and confusion. (AMH, 2017)

- Symmetrel® (amantidine) 100mg capsules

Important Medication Issues

1. Withdrawal & Nil by Mouth Status

Abrupt withdrawal or reduction of Parkinsonian medications may cause serious withdrawal symptoms similar to neuroleptic malignant syndrome. Dopaminergic drugs should **NOT** be ceased abruptly as this can lead to a condition similar to akinetic crisis, which is life-threatening (characterised by severe rigidity, hyperpyrexia, and increased PD signs). Nil by mouth (NBM) status should be reviewed urgently. The consideration of a nasogastric tube insertion to administer PD medications may be necessary if no other means of administration are available. When patients are scheduled for surgery please ensure that PD medications are charted and to be given with sips of water or contact the anaesthetist for further instructions. Levodopa may need to be reduced in the post-operative state as confusion in the elderly with PD is common and can be exacerbated with high levels of levodopa. (AMH, 2017)

2. Prescribing of Medications

Unlike other medications, it is recommended that PD medications should be prescribed using the brand name, the dosage and formulation of the tablet/capsules, the number of tablets or capsules and time of administration of medication to prevent any misinterpretations leading to medication errors. It is also easy to neglect checking of the second component of the dopaminergic agents, the DOPA decarboxylase inhibitor (i.e. Carbidopa and Benserazide) and prescribing the brand name can help reduce this issue.

For example, writing Sinemet 100/25 (x2½) tabs is clear and reduces the risk of medication errors. If the above was prescribed as Sinemet 250mg, it can be interpreted wrongly as Sinemet CR 200/50, Sinemet 250/25 and Sinemet 100/25 (x2) tablets.

Diagram 9. Example of Prescription of PD Medications

Date	Medication (Print Generic Name)		Tick if Slow Release	
16-3-16	Stalevo 150/37.5/200		<input checked="" type="checkbox"/>	0700
Route	Dose	Frequency & NOW Enter Times		1000
p.o.	1	q 3h		1300
Indication	Pharmacy			1600
Parkinson's Disease				1900
Prescriber Signature	Print Your Name		Contact	
[Signature]	NAME			
Date	Medication (Print Generic Name)		Tick if Slow Release	
16-3-16	Sinemet CR 200/50		<input checked="" type="checkbox"/>	
Route	Dose	Frequency & NOW Enter Times		
po	1	nocte		
Indication	Pharmacy			
Parkinson's Disease				
Prescriber Signature	Print Your Name		Contact	
[Signature]	NAME			2200
Date	Medication (Print Generic Name)		Tick if Slow Release	

3. Timing of Medications

Timing of PD medications is one of the most important nursing considerations. Even a 15 minute delay in dosing may make a significant difference in symptom control and could mean the patient is sitting rigid and in pain for the length of time it takes for the medication to work. Remember levodopa has a short duration of action.

GIVE THE PARKINSON'S MEDICATIONS AT THE EXACT TIME ORDERED. QID does not necessarily mean the times of the medications are 0600, 1200, 1800 and 2400hrs. The timing of the dose is individualised according to the patient's PD symptomatology. It is important to ask each patient what times they take their PD medications when planning their individualised schedule.

4. Interactions of Meals with Levodopa Absorption

It is best for most patients on levodopa medications (Kinson®, Madopar®, Sinemet®, Stalevo®) that they are given half an hour prior to meals as ingestion of food and, to a lesser extent, protein intake in the meal can interfere with absorption of levodopa and cause fluctuations in levodopa concentrations, although this is not the case with all patients. (Baruzzi, A. et al 1987 p 534)

Levodopa is absorbed in the small intestine and not in the stomach. (Baruzzi, A. et al 1987 p 532) When Levodopa medications are taken with or after meals, absorption of medication is delayed due to the entero-gastric reflex. This causes the pyloric sphincter to tighten and hence delay absorption into the small intestine until gastric emptying occurs, which can take from 30 minutes to 2 hours. (Marieb EN. 1992 p789).

Content of meals may also effect rate of absorption of Levodopa medications, although to a lesser extent than gastric emptying. A high protein meal creates competition between protein molecules and Levodopa. 'competition between the products of protein digestion and Levodopa for the carrier system across the gut wall might also influence drug absorption' (Baruzzi et.al 1987 p534)

5. Drugs to Avoid

Commonly used medications (see list) may cause severe Parkinson symptoms or an acute dystonic reaction in a PWP. For example, Maxolon® has dopamine antagonist activity while Stemetil® has an anti-dopamine action. A medical review to add these medications to the 'allergies & adverse drug reactions' section of the medication chart is suggested. Domperidone and/ or Ondansetron is generally the alternative anti-emetic that is used with a PWP.

Medications Contraindicated in PD	Class of Drug
Metoclopramide (Maxolon®)	Antiemetic/ antinausea
Prochlorperazine (Stemetil®)	Antiemetic/ antinausea
Haloperidol (Serenace®)	Antipsychotic
Promethazine (Phenergan®)	Antihistamine

6. Parkinson's Diary

Assessment of the effects of Parkinson's medications is imperative, particularly where new medications are being trialled or where the regimen has been altered. An hourly awake diary of 'OFF/ON' status can help to highlight where medication needs to be changed (See Diagram 10).

Similarly documenting dyskinesia can show where the dosage may need to be reduced or better spaced. There may also be a comment section to report any unusual phenomena.

Diagram 10. Example of a completed hourly Parkinson's Diary plotting the 'OFF' and 'ON' states of the PWP throughout their waking day.

Date: __/__/__	06	07	08	09	10	11	12	13	14	15	16	17	18	19	20	21	22
Dyskinesia +++																	
Dyskinesia ++																	
Dyskinesia +											X	X					
"ON"			X	X		X				X			X		X	X	
Off +		X			X	X		X	X					X			X
Off ++	X																
Off +++																	
Comments																	

7. Multidisciplinary Involvement

People with PD may require input from members of the multidisciplinary team at some point in the progression of their PD.

Referral to the following members of multidisciplinary team may be beneficial:

- speech pathologists for speech and swallowing difficulties
- physiotherapists to assist with mobility and falls prevention and cueing techniques to manage 'freezing' episodes
- dietician to provide guidance and advice on caloric intake and protein interaction with levodopa
- social workers to assist with linking into service provision
- psychologists to provide counselling to both PWP and carer throughout disease process and changes
- Occupational therapists to provide assessments for home equipment and aids
- Parkinson's Nurse
- Pharmacist

Device Assisted Therapies in Parkinson's disease

Currently there are three Device Assisted Therapies (DAT) in Parkinson's disease. These therapies include Apomorphine, Duodopa® and Deep Brain Stimulation (DBS). Apomorphine and Duodopa® are both infusion therapies and DBS is a neurosurgical intervention. They are based on the concept of continuous stimulation to minimise pulsatile, sudden or unexpected fluctuations. (Hayes M et al, 2010 p 147)

Apomorphine

Apomorphine is a subcutaneous injection for the treatment of Parkinson's disease. Apomorphine is a dopamine agonist that works to stimulate the dopamine receptors in the substantia nigra, more specifically the D1 and D2 receptors. It can be given as an intermittent rescue injection using a heparin/insulin syringe or via a continuous subcutaneous infusion. Although it is derived from the morphine molecule it has no narcotic, analgesic or addictive properties. The dilution and infusion rates on the pump are carefully individualised to each patient.

Duodopa® - Levodopa/Carbidopa Intestinal)

Duodopa® is an intestinal gel of Levodopa/Carbidopa (20mg/5mg/ml) enclosed in a 100 ml cassette for the treatment of Parkinson's disease. It is administered, via a PEG-J tube, directly into the duodenum or jejunum where the absorption of the medication is optimal. The tubing includes an outer tube (the PEG tube) with an inner tube (the PEJ tube) threaded through it. The connection of the tubing has two ports. The shorter/side port is the PEG, or gastric, port and the longer/front port is the PEJ, or intestinal port, where Duodopa® is administered. Please ensure Duodopa® is always being administered through the intestinal port. The infusion rates on the pump are also carefully individualised to each patient. (Tsui D. 2014 p 36)

Deep Brain Stimulation

Deep brain stimulation is a neurosurgical option in the treatment of Parkinson's disease. The treatment involves stereotactic insertion of electrodes into target sites in the brain, usually the subthalamic nucleus (STN) or globus pallidus internus (GPi), and connecting the electrodes to an implanted pacemaker to control the abnormal firing of neurons by delivering steady and continuous electrical impulses to the target sites. (Hayes M, et.al 2010 p 147)

Patients who have undergone insertion of DBS electrodes will have pacemaker implanted in right subclavicular region (or occasionally the abdomen) and electrodes subcutaneously from pacemaker, over collarbone, up the neck and behind the ear. Some patients with devices implanted may not be able to have MRI's.



TAKE AWAY GOLD STANDARDS OF NURSING CARE FOR EACH PWP

- Ensure contraindicated medications are completed in ALLERGIES and ADVERSE DRUG REACTION box on medication charts.
- Medications are correct
- Medications are given ON TIME EVERY TIME
- ALL assessments completed in ON and OFF state
- Bowel chart completed and constipation management initiated if required.
- PATIENCE with PWP



Questions

1. Which group of neurological diseases is PD is classified under?
2. What is the cause and pathophysiology of Parkinson's disease (in your own words)?
3. What is dopamine and what is it responsible for?
4. Parkinson's disease is a natural aging process.
TRUE or FALSE
5. Resting tremor in PD occurs when the patient is asleep.
TRUE or FALSE
6. What are the 3 domains of Parkinson's symptoms, describe and give 2 examples of each one?
7. Why is Levodopa combined with carbidopa?
8. Name 3 drugs that are to be avoided in Parkinson's disease?
9. Why is timing of Parkinson's medications strict and crucial?
10. Give 2 reasons why a speech pathology review would be beneficial to the Parkinson patient?
11. What is 'ON / OFF' Phenomenon?
12. Describe relationship between ON/OFF and dyskinesia.
13. Explain how ON/OFF and Dyskinesia can affect your nursing care.
14. What nursing care considerations are required during the 'OFF' period?
15. During a night shift, what nursing care needs to be attended to throughout the night?
16. What are the three Device Assisted Therapies for PD and briefly explain each of them.
17. What is the aim behind the three device assisted treatments in Parkinson's disease?



- 18. What is meant by the term “freezing” in PD and how can it be overcome?**
- 19. PWP are at increased risk of falls – what can be included within nursing care to manage such risks?**
- 20. What is the suggested anti-emetic to use in patients with PD?**
- 21. Scenario: You have assume care of a PWP who is booked for surgery the next morning and has been ordered nil by mouth from midnight – Should the patients medications be given and please provide rationale?**
- 22. Why are Parkinson’s medication prescribed using the brand name – please give an example of how you are likely to see the medication written on the medication chart?**



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