

Health Care for Older People

Holistic Approach

PARKINSONS DISEASE

Sri Lanka Association of Geriatric Medicine

2019

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An attempt to develop and promote multidisciplinary mutual coordination and collaboration among the team involved in care of older patients at various levels in the health and social service sector.

“Teamwork divides the task and multiplies the success.”

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Parkinson's disease: an Overview

Prof. Saman Gunatilake

Parkinson's disease (PD) is a progressive neurological disorder that results from loss of dopaminergic neurones in the substantia nigra. The cause of neuronal damage remains largely unknown, but believed to be associated with both genetic and environmental factors. PD is characterized by motor and non-motor symptoms. The main motor features are rigidity, tremor, and hypokinesia. Non-motor symptoms include: neuropsychiatric conditions; autonomic disturbances, sleep disorders and sensory symptoms. Quality of life (QoL) for patients with PD is severely affected by both motor and non-motor symptoms. Input from a multidisciplinary team improves outcomes for patients and carers.

Epidemiology

About 5.2 million people suffer from PD worldwide. It is commoner in Europe and North America than in Africa; this could reflect a difference in life expectancy since PD is mainly, a disease of elderly. Parkinsonism is an umbrella term for several neurodegenerative diseases. A person has a 2.5–3 times higher risk of developing PD if a first-degree relative has the disease. However, familial PD is rare (<5%). onset after the age of 50 years is less likely to be genetically influenced. The average age of onset is 65 years. Young-onset PD, i.e. onset under the age of 40 years, accounts for about 5–10% of all cases.

Causes

The cause of PD remains largely unknown. Age is not the only factor contributing to development of PD. A combination of an inherited susceptibility and exposure to environmental risk factors could cause PD and this warrants further research.

Genetics

There is little doubt that genetic factors contribute to development of PD, but their significance is yet to be established. Advances in genetic research have enabled identification of 12 genes associated with PD (PARK1 to PARK11, and NR4A2). Each gene mutation expresses different clinical features, with some overlap.

Environment

The evidence of an association between environmental risk factors and development of PD is weak and the literature should be interpreted with caution. Suggested environmental factors include exposure to pesticides (eg, MPTP), herbicides or heavy metals (manganese, copper). Conversely, cigarette smoking is negatively associated with development of PD. Other such proposed protective factors are coffee consumption, drinking alcohol and physical activity.

Pathology

There are three pathological hallmarks of PD: the presence of Lewy bodies (LBs); neuronal death in pars compacta of substantia nigra; and the loss of pigmented neurons (neuromelanin) in pigmented brainstem nuclei. LBs are protein aggregates of abnormal synuclein and other neurofilaments within the neuronal cytoplasm. The exact role of LBs in pathophysiology of PD has yet to be established but believed to be pathogenic in PD, leading to neuronal cell death. Nevertheless, the presence of LBs can be asymptomatic and not associated with PD. LBs are also found in other neurodegenerative diseases (eg, Lewy body dementia, Alzheimer's disease, multisystem atrophy). Number of LBs increases with age, correlating with increasing incidence of PD among elderly. Degeneration of dopaminergic neurones in substantia nigra pars compacta leads to profound depletion of dopamine within the brain. Compensatory mechanisms are so effective that the clinical symptoms of PD only develop when 80% of dopaminergic neurones have degenerated.

Symptoms

The clinical features of PD are classified into two groups — motor and non-motor symptoms.

The classic motor features are:

Resting tremor — fine rhythmic movement (4–6 Hz) and often one of the first signs of PD; initially seen in one upper limb and typically involving both later.

- Bradykinesia — slowness of movement, usually in the upper body.
- Hypokinesia — poverty of movement, including impassive facial expression and loss of arm swing when walking and progressing to general difficulty with fine movements.
- Rigidity — feeling of resistance to passive movement, which begins unilaterally and progresses bilaterally in advanced disease; the term “lead pipe” is used to describe continuous resistance, whereas “cogwheel rigidity

refers to more jerky rigidity caused by superimposed tremor.

Other motor symptoms that develop in later stages of PD include motor freezing and postural and gait instability. Postural disturbances include stooped posture, stumbling, which frequently lead to falls. Gait becomes slow with shuffling and festination (involuntary quickening). Sudden stopping or the inability to initiate movements is referred to as “freezing”. This is different from “Switching off”, which is a motor complication of PD drug therapy where the treatment effect wears off abruptly before the next dose is due.

A greater awareness of non-motor features of PD is now seen. Widespread use of the PD “non-motor symptoms scale” questionnaire has shown they often have an equal or greater impact on quality of life than motor complications. Patients may not be aware that non-motor symptoms are related to PD. Although these are often difficult to control, it is important that they are addressed and treated to minimize their impact.

Non-motor manifestations include:

- Neuropsychiatric symptoms — dementia (24– 31%), depression (30%), anxiety, visual hallucinations
- Autonomic — constipation, urinary incontinence, hyperhidrosis, sialorrhoea, postural hypotension
- Sleep disorders — rapid eye movement sleep behaviour disorder, restless leg syndrome, vivid dreams, narcolepsy
- Sensory symptoms — pain (dystonic and nondystonic), paraesthesia

Diagnosis

Parkinsonian syndrome, characterised by bradykinesia and at least one of the following:

- Muscular rigidity
- Resting tremor (4–6Hz)
- Postural instability unrelated to primary visual, cerebellar, vestibular or proprioceptive dysfunction

Accurate diagnosis of PD relies on clinical examination and a thorough review of history. No definitive laboratory or imaging tests are available to confirm the diagnosis.

There is a high error rate in the diagnosis of PD (24–35% of diagnosed cases are false positives) because of the lack of definitive laboratory or imaging tests to

confirm diagnosis. Evidence suggests that the error rate is 47% in primary care compared with 6–8% in tertiary care and, therefore, if PD is suspected patients should be referred (preferably untreated) to a movement disorder clinic for diagnosis. The UK Parkinson's Disease Society's "brain bank criteria" can be used to aid diagnosis and ongoing patient review.

Supportive criteria for PD

≥3 of the following are required for a definite diagnosis:

- Unilateral onset
- Resting tremor
- Progressive disorder
- Persistent asymmetry affecting the side of onset most
- Excellent response to levodopa
- Severe levodopa-induced chorea
- Levodopa response for over five years
- Clinical course of over 10 years

Differential diagnosis

Some neurodegenerative and Parkinsonian conditions (eg, multisystem atrophy, progressive supranuclear palsy, Wilson's disease and cortico-basal degeneration) can present with symptoms similar to PD and can complicate diagnostic accuracy. Evaluation of age of onset, family history, symmetry, eye movements, tremor, presence of dementia, onset and nature of falls, levodopa response, cardiovascular autonomic failure and bladder disturbance are helpful in differentiating these.

Essential tremor (ET) is a symmetrical, task-related tremor of 8–10 Hz occurring mainly in upper limbs. To differentiate between PD and ET patients are often asked to complete a writing test. This usually reveals progressively bigger text written by ET patients compared with PD patients for whom the writing becomes smaller.

Drug-induced Parkinsonism is the second commonest cause of Parkinsonian symptoms. (Idiopathic is the first). Drugs commonly implicated are antipsychotics (typical antipsychotics more often than atypical) and anti-emetics, due to their dopamine-blocking properties. Also associated are amiodarone, calcium channel blockers, lithium and anti-epileptics (e.g. valproate and phenytoin). Drug-induced Parkinsonism is under diagnosed and can lead to permanent symptoms in 15% of cases even after withdrawal of the causative medicine; it

is not clear whether these cases reflect an unmasking of underlying idiopathic PD. Tremor is less prominent in them and the onset is usually bilateral and symmetrical.

Prognosis

Rates of disease progression vary considerably among PD patients. PD in itself is not fatal — the cause of death for most people with PD is a secondary co-morbidity (e.g. pneumonia). This has led to discrepancies in the mortality rates reported in epidemiological studies. Overall, most studies conclude that PD does reduce life expectancy, with reported mortality hazard ratios varying from 1.5 to 2.16. Some 25–40% of patients with PD will develop dementia, which is thought to contribute heavily to the reduced life expectancy. Mortality rates associated with PD have remained largely stable. A decrease in mortality was noted in the late 1970s and early 1980s and this was followed by an increase back to previous levels (explained by the introduction of levodopa, which is believed to delay death by about five years).

Motor symptoms of Parkinson's disease and the management

Dr Ajini Arasalingam

Introduction

The core motor features of Parkinson's disease (PD) include asymmetry, bradykinesia, resting tremor and rigidity. Whilst anticholinergics were the main treatment for Parkinson's disease in the past, over the last few decades focus changed to ameliorating the dopaminergic deficit, bringing levodopa as the main modality in the management of motor symptoms of PD. Many other options are emerging giving the clinician a wide variety of pharmacological options to choose from. However, the trick of the trade depends on individualizing therapy. Further disease modifying drugs or neuroprotective drugs do not exist at present, whilst the future for them is promising. Recent drugs tried as disease modifying which failed in PD trials include co-enzyme Q10, creatine, pramipexole and fetal transplants.

When should treatment be started?

Therapies that can slow or halt the disease, if available, should be started immediately, however none exist currently. Symptomatic therapy is less likely to be effective for mild symptoms, but as stated in the outset this has to be individualized as even mild reduction in symptoms may be beneficial to certain groups of patients (e.g. those involved in fine work). In other cases, the physician may delay the introduction of treatment until the patient has significant functional disability.

Early PD management

Levodopa has proven to be more effective than MAO-B inhibitors and dopamine agonist (DAs) in symptomatic control, particularly motor symptoms (2017 update of the NICE guidelines). The long-term quality of life gains (including dyskinesias) associated with initial levodopa therapy over DAs and MOA-B inhibitors as an initial therapeutic agent had been studied in the PD – MED UK trial showed that risk versus benefits favour initial treatment with levodopa further reinforcing the NICE guideline recommendation – “offer levodopa to people in the early stages of PD whose motor symptoms impact on their quality of life.”

As the disease progresses, the dose of levodopa is increased gradually. The upper limit depends on the development of dyskinesia which is dose dependent. The

initial dose of levodopa for a patient with average body weight will be 100mg thrice daily (with meals) which is then gradually increased to 100mg five times per day with the additional doses in the mid-morning and mid-afternoon. The maximum dose is 600mg per day (8-9mg/kg body weight) with a double dose with breakfast or the maximal dose tolerated by the patient. Doses above the recommended may lead to dyskinesias and wearing off which will require adjuvant therapy.

Adjuvant therapy

Adjuvant therapy includes DAs, MAOB inhibitor or catechol-O-amine methyl transferase (COMT) inhibitor. A systematic review of placebo controlled trials with DAs, MAO inhibitor or COMT inhibitor suggested that DAs and tolcapone were more effective than entacapone and MAO inhibitors in reducing “off time” and levodopa dose without an excess of side effects. The PD MED LATER trial showed that there was no benefit in quality of life in using a DA compared to a MAOB inhibitor or COMT inhibitor. More evidence on what adjuvant therapies should be added and when is expected from the PD MED LATER study, however older patients seem to be less tolerant to add on therapy, probably due to onset of cognitive impairment. There is lack of evidence comparing the efficacy and choice of a DA and MOAB inhibitor. Further there is no evidence for efficacy and safety of selegiline, Rasagiline or the newer safinamide. Selegiline is commenced first as it is cheaper.

Non-ergot DAs (pramipexole, ropinirole, rotigotine) are preferred over the ergot derivatives due to the poor side effect profile of the ergot derivatives. Entacapone is a weak adjuvant therapy and tolcapone is avoided due to hepatotoxicity.

Rational polypharmacy

With disease progression, adjuvant agents will be added one on top of the other. Thus in addition to levodopa many patients with PD will be on an MAOB inhibitor, a DA and a COMT inhibitor leading to rational polypharmacy.

Advanced PD with severe motor complications

NICE guidelines recommend the use of amantadine as an anti-dyskinesia drug based on a Cochrane review of three randomized controlled trials. Apomorphine (DA) is not effective orally due to extensive first pass metabolism. Patients with intermittent severe ‘off periods’ benefit from bolus rescue injections, while those with many ‘off periods’ benefit from subcutaneous infusions.

Continuous infusion of levodopa directly into the jejunum (Duodopa©) reduces off time and improves motor function, activities of daily living and quality of life.

Surgical options include bilateral sub-thalamic stimulation surgery to switch off the nucleus. NICE guidelines recommend STN stimulation for patients with motor complication refractory to best medical treatment, who are medically fit with no clinically significant active mental health problems such as dementia or depression.

Therapeutic options in the geriatric population

Data indicates that dopamine agonist (non-ergot derivatives) are effective in the treatment of motor symptoms in early PD. Data also suggests anti-depressive effects with dopamine agonist improve quality of life.

Complications of drug treatment/ disease per se:

On and off state: After a few years of levodopa the motor response to levodopa fluctuates, reported as the effects not lasting up to the next dose (wearing off). If the wearing off was predictable the doses frequency is increased (reducing the time between two successive doses). Some patients may also experience non-motor symptoms in addition to motor symptoms. Higher risk of developing fluctuations is associated with younger age of disease on set, longer use of levodopa and higher individual doses of levodopa. These motor symptoms are described as on and off states.

Reemergence of PD symptoms, before the next dose of medication is called predictable wearing off, while random and sudden return of PD symptoms not related to the timing of levodopa is unpredictable off state. Wearing off can be managed with dopaminergic agonist (ropinirole or pramipexole (both immediate and controlled release forms), rotigotine patch and subcutaneous apomorphine) COMT inhibitors (entacapone), MAOB inhibitor (selegiline, rasagiline) and levodopa preparations (carbidopa/levodopa controlled and extended release).

Rarely a transient worsening of symptoms at the beginning of the dose or end of dose often presenting as an increase in tremor is known as beginning of dose worsening and end of dose rebound.

Dystonia: Dystonia is painful affliction of the distal leg, foot and toes with

abnormal posturing. When occurring during the off phase is termed off period dystonia. These were managed with dopamine agonist, COMT inhibitors, MAOB inhibitors and levodopa preparations. Focal off period painful dystonia could be treated with botulinum toxin.

Peak dose dyskinesias: These are mixed chorea and dystonia of neck and limbs, increased with mental and physical activity. Usually occurs in the on state. Amantadine is the treatment of choice. Diphasic dyskinesia affects the leg and present as high amplitude dystonia, with stereotypic kicking and a funny gait, also occurring in the 'on' state. This can be managed by dopamine agonist.

Freezing: Off freezing is a transient difficulty in initiating or continuing a movement, while turning or due to sudden stress or anxiety. Freezing in the on period is rare.

References

1. PD MED Collaborative Group, Gray R, Ives N, et al. Long term effectiveness of dopamine agonists and monoamine oxidase B inhibitors compared with levodopa as initial treatment for Parkinson's disease (PD MED): a large, open label, pragmatic randomized trial. *Lancet* 2014; 384(9949):1196-205.
2. National Institute for Health and Care Excellence. Parkinson's disease in adults: diagnosis and management. London 2017.
3. National Collaboration Centre for Chronic Conditions. National Institute for Health and Clinical Excellence (NICE) Guidelines – Parkinson's disease: diagnosis and management in primary and secondary care. London: Royal College of Physicians, 2006.
4. Clarke C, Patel S, Ives N, et al. A large randomized trial assessing the quality of life in patients with later PD. Results from PD MED LATER. *Parkinsonism & Related Disorders* 2012;18 (Suppl 2): S33.
5. Stowe R, Ives N, Clarke CE, et al. Meta-analysis of the comparative efficacy and safety of adjuvant treatment to levodopa in later Parkinson's disease. *Movement Disorder* 2011; 26 (4):587-98.

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Non motor manifestations of Parkinson's disease

Dr Chandana Kanakarathne

Though the Parkinson disease (PD) is defined by motor features patients are also affected with many non motor symptoms (NMS). These features may be present at very early stages of the disease or more commonly, towards the advanced stages, compromising the quality of life (QoL) significantly. Certain features, known as pre-motor features, may occur years before the appearance of motor features. To achieve the best QoL in PD, motor as well as NMS need appropriate attention. Most NMS which are described in the article show a poor response to dopaminergic therapy and other pathways, including the serotonergic and noradrenergic pathways are implicated in pathogenesis.

1. Impaired olfaction

Includes impairment of odour detection, identification and discrimination

Common (in 90%)

No successful interventions

2. Gastro-intestinal symptoms

A) Drooling

Treatment

Non-Pharmacological: speech therapy

Pharmacological: only if non-pharmacological treatment is not available.

glycopyrronium bromide (anticholinergic) 1mg - 2mg TDS

Other topical anticholinergics (1% atropine) sublingually and (Hyoscine) – if risk of cognitive impairment is minimal

Botulinum toxin A – injection of salivary glands (If above treatments are not effective or contraindicated)

B) Nausea and vomiting

Common

Treatment

Best choice is Domperidone. Avoid Metoclopramide.

C) Dysphagia

It occurs in both oral and pharyngeal phases in PD. May manifest as a cough when ingest food.

Assessment: Speech therapist's input along with video fluoroscopy and upper gastrointestinal endoscopy, endoscopic evaluation of swallowing and oesophageal manometry are used.

Interventions

1. Exercises to promote tongue strengthening, tongue control and voice exercises
2. Alteration of the consistency of food and drink
3. Advice on the organisation of the bolus of food within the mouth and frequency of swallowing
4. Lee Silverman method

D) Delayed Gastric emptying

Common in PD occurring at all stages and results in many upper gastrointestinal symptoms and poor absorption of PD drugs.

Treatment

Dopramidone – may be useful but no studies to support its use

Macrolides – may increase gastric emptying, short term

E) Constipation

Affects at least 50%, due to loss of central and colonic dopaminergic neurons

Presentations

Sigmoid volvulus, Ano-rectal dysfunction, colonic dysmotility with high faecal loading

Treatment

1. Improve mobility and exercises
2. Increase fibre intake in diet (>15 g/d)
3. At least 1.5L of fluid intake a day
4. Macrogols may be more effective than lactulose
5. Dopaminergic medication
6. Defaecation training, puborectalis botox, sacral nerve stimulation and abdominal massage

3. Genito-urinary symptoms

A) Urinary dysfunction

Nocturnal frequency is the commonest symptom, Day time urgency and frequency develops later. This is usually due to Detrusor hyper-reflexia.

Management

1. Exclude UTIs
2. Urodynamic studies

3. Overactive bladder syndrome- Antimuscarinics (oxybutinin, propiverine, solifenacin and tolterodine). A drug that does not cross the blood brain barrier (tolterodine and trospium) is better than oxybutynin.
4. Mirabegron, a 3-adrenoceptor agonist, for detrusor over-activity with no anticholinergic activity and cognitive effects, and hence may be useful in PD but no convincing evidence available.
5. Detrusor botulinum toxin injections may be used.
6. Retention may need catheterisation.

B) Sexual dysfunction

Reduced sexual drive is reported. Also, hyper-sexuality can occur as an ICD.

Erectile dysfunction may occur in 60-70% of male patients with PD.

Treatment: sildenafil

4. Sleep disturbances

Nearly all patients develop nocturnal NMS and this can happen early in the disease. A thorough sleep history is mandatory.

Pathogenesis:

Degeneration of central sleep regulation centres in the brainstem and thalamocortical pathways is an important cause. Motor symptoms, anxiety, depression, and dopaminergic treatment, increased urinary frequency at night also may contribute.

Several tools have been validated as bedside tools.

Epworth sleep scale (ESS)

Parkinson's disease Sleep scale (PDSS)

Scales for outcomes in PD - sleep' (SCOPA-sleep)

Polysomnography and multiple sleep latency tests are the gold standard diagnostic evaluations.

Types of sleep disturbances

A) Insomnia

Difficulty in falling asleep or maintaining sleep as disturbed by nocturnal akinesia, 'wearing-off' overnight with re-emergence of motor symptoms, pain and stiffness, nocturia, RLS and obstructive sleep apnoea (OSA).

Management

To improve 'wearing off' at night, optimise levodopa therapy or add a long acting levodopa or DA at night. If available, rotigotine patches or apomorphine infusions may be used. Melatonin may be used to improve sleep wake cycle.

B) Excessive Day time Sleepiness – (EDS)

Common and may affect up to 50%

Leads to poor concentration and memory

Tendency for road traffic accidents

Some develop 'Sudden onset of sleep' similar to narcolepsy

Pathogenesis: Multi-factorial (disease itself, medication)

It's associated with cognitive impairment in later stages

Management

1. Improve quality of nocturnal sleep
2. Treat other causes of EDS such as depression, OSA and review medication (Ex: sedatives)
3. Drug treatment: Modafinil – 200-400mg/day especially for 'sudden onset of sleep'
4. Avoid driving. Caution on swimming, climbing ladders etc.

C) Nocturnal akinesia (inability to turnover)

Treatment

1. Oral levodopa or DAs at bed time
2. Rotigotine patch –if above oral agents are ineffective

D) Restless leg syndrome

Unpleasant or uncomfortable sensations in the legs and an irresistible urge to move them.

Treatment

Pramipexole (125 ug) or ropinirole (250ug) or Rotigotine patches (starting dose of 1mg) at bed time with dose titration.

E) Periodic Limb Movements of Sleep (PLMS)

Involuntary movement of the limbs during sleep

Treatment– Levodopa at night

F) REM Behavioural Disorder (RBD)

RBD is a parasomnia, here patients physically enact their dreams in the forms of vocalisations and abnormal movements. It may predate motor symptoms by up to several years in 40% of the patients.

Treatment

1. Clonazepam : 0.25-0.5mg, maximum 1mg – at bed time
2. Melatonin : 3-6mg at bed time

5. Autonomic dysfunction

These are common (50%) and affects many systems.

A) Orthostatic Hypotension

Common presentations are dizziness, falls, syncope and visual disturbances. It can cause discomfort and heaviness over the neck and shoulders known as coat-hanger pain. Prevalence – 50%

Assessment

- Check for postural blood pressure drop routinely
- 24 hour blood pressure monitoring

Management

1. Consider adjusting existing medications
2. Avoid sudden head up – on rising from bed stay seated for a few minutes before standing up
3. Avoid large meals and alcohol
4. Ensure adequate fluid and salt intake
5. Avoid excessive heat
6. Elevate bed head by 20-30 degrees
7. Consider elastic stockings

Drug Treatment

1. Midodrine – first line – May cause supine hypertension and warrants regular check-ups and periodic 24h BP monitoring. If it happens consider night time GTN patch or a short acting vasodilator antihypertensive
2. Fludrocortisone – if above not available/contraindicated – maximum 400 µg
3. Domperidone
4. L-DOPS (Doxidopa) - a pro-drug of noradrenaline and adrenaline. May be better tolerated
5. Pyridostigmine for mild to moderate orthostatic hypotension

B) Excessive sweating

Common but no satisfactory treatment is available.

C) Bladder/ sexual dysfunctions – see section for genito-urinary conditions

6. Menal health issues

A) Apathy

Prevalence 30%

This may be attributable to disturbances in basal ganglia and fronto-cortical connections, cognitive impairment and simply due to Fatigue

Potentially helpful drugs: DAs, Amantadine, modafanil, methylphenidate and amphetamines

B) Anxiety

Prevalence – 50%

Include panic disorder, generalized anxiety and phobias.

Anxiety and panic attacks may occur with 'off periods', hence helped by adjusting dopaminergic treatment.

Anxiety and mania – may occur with excess of levodopa ingestion (dopa dysregulation syndrome – see below)

Treatment

1. Selective serotonin reuptake inhibitors (SSRI)
2. Benzodiazepines for short term management of significant symptoms

C) Depression

It may occur in up to 10-70% of patients. Severe depression may occur in 17%.

Diagnosis:

A structured interview involving relatives and carers is the gold standard for diagnosis. Scales may be used but none are specially designed for PD. (Beck Depression Inventory, the Hospital Anxiety and Depression Scale, Geriatric Depression Scale, Hamilton Depression Rating Scale (HAM-D)).

Treatment:

1. SSRI with usual dose titration (caution with those using MOA-inhibitors (selegiline)) due to rare but dramatic complication of serotonin syndrome
2. Mirtazapine
3. TCADs (trazodone)
4. Serotonin and noradrenaline reuptake inhibitor (SNRI) venlafaxine
5. Selective noradrenaline reuptake inhibitor - reboxetine
6. Pramipexole
7. Electroconvulsive therapy in refractory cases

D) Psychosis

This Include visual hallucinations, illusions (sense of presence), delusions and

paranoia.

Affects 60% and common in older onset PD with cognitive impairment and RBDs. Florid persistent psychosis in later disease is strongly correlated with increased mortality and for nursing home placement. Psychosis can be associated with DAs and less commonly, with levodopa.

Assessment

General medical assessment for causes

Medication review: may need to gradually reduce the dose of dopaminergic treatment. (MAO-B inhibitors, DAs, COMT inhibitors)

Tail off and stop anticholinergics and Amantadine

Treatment

1. Delusions and hallucinations - If well tolerated by the patient, families and carers there is no need to intervene.
2. Quetiapine in low dose – used but evidence not strong
3. Low-dose Clozapine has best evidence but may have side effects.
4. Avoid – Haloperidol, olanzapine due to the risk of worsening Parkinson features
5. Rivastigmine is used with some effectiveness but evidence for its use is not strong

E) Cognitive impairment and Dementia

Prevalence – 30%

Specific deficits include impaired executive function, reduced verbal fluency and visuospatial abilities.

Cognitive assessment can be done with Montreal Cognitive Assessment (MOCA).

Management

1. Review medication
2. Gradual and slow withdrawal of DAs, anticholinergics, Amantadine and selegiline
3. cholinesterase inhibitors for mild to moderate dementia – Most effective are Rivastigmine and Memantine

7. Impulse Control Disorders (ICDs)

ICDs can develop in any PD patient who is on any dopaminergic therapy (but mainly DAs), at any stage in the disease course. There are many ways it is manifested.

Ex: Punding, Pathological gambling, Hyper-sexuality, Compulsory buying, Binge eating

Prevalence – 14%

Factors increasing the risk of ICDs

- DA therapy
- A history of previous impulsive behaviours
- A history of alcohol consumption and/or smoking

It is preferable, when initiating DA therapy, educating the patient and the family about ICDs.

Treatment

Withdrawal of DAs and replacing with levodopa therapy

8. Dopamine Dysregulation Syndrome (DDS)

A disorder that can happen when patients escalate their treatment with levodopa beyond that is needed for motor control.

Management:

1. Attempt to reduce therapy - may lead to dysphoria, pain and anxiety
2. Atypical antipsychotic drugs and psychological input may be necessary
3. May try DAs

9. Miscellaneous

A) Fatigue

It's common and carries a negative impact on QoL. Fatigue may occur independently or as a result of other NMS such as depression.

B) Pain

Common (up to 40%)

Various pain syndromes described

Pain due to dystonia and dyskinesia : commonest

Pain related to motor fluctuations

Coat hanger pain in neck and shoulders due to postural hypotension

Secondary causes such as musculoskeletal pain - osteoporosis, arthritic changes and contractures

Oral (burning mouth syndrome) and genital pain rarely occur

C) Thermoregulatory dysfunction

No effective treatment recommended

10. Pre-motor NMS

They may be useful as screening tools for PD in future

Main features

1. Impaired olfaction
2. RBD
3. Episodic major depression
4. Constipation
5. Excessive daytime somnolence
6. Fatigue
7. Abnormal colour vision/visual perception
8. Erectile dysfunction
9. Pain (often unilateral) Pain often evident on side first affected at motor PD diagnosis
10. Cognitive impairment

Deep Brain Stimulation of Sub-Thalamic Nucleus (STN-DBS) on NMS

Improvements in sleep architecture, urodynamics, constipation and non motor fluctuations (sensory, dysautonomia, cognition) noted. Effect on neuro-behavioural symptoms has shown both improvements and deleterious effects. Modest deterioration of cognition may occur.

Whenever you assess someone with PD always make effort to pay attention to NMS. Remember to use the multi-disciplinary team effectively in a coordinated manner. Without optimising NMS it's not possible to achieve the best quality of life to the PD patient.

References

1. Wishart S, Macphee GJA. Evaluation and management of the non-motor Features of Parkinson's disease. *Therapeutic Advances in Chronic Dis* (2011);2(2):69-85.
2. Parkinson disease NICE Guidelines 2017.
3. Noyce AJ et al. The prediagnostic phase of Parkinson's disease. *Journal of Neurology Neurosurgery Psychiatry* 2016;87:871–878.
4. Titova N, Chaudhuri KR. Non-motor Parkinson disease: new concepts and personalised management. *The Medical journal of Australia*, (2018) 208(9), 404-409).

Following is a validated NMS questionnaire to be filled by the patient. This will help you to identify the NMS in a patient

PD NMS QUESTIONNAIRE

Name: Date: Age:

Centre ID: Male Female

NON-MOVEMENT PROBLEMS IN PARKINSON'S

The movement symptoms of Parkinson's are well known. However, other problems can sometimes occur as part of the condition or its treatment. It is important that the doctor knows about these, particularly if they are troublesome for you.

A range of problems is listed below. Please tick the box 'Yes' if you have experienced it **during the past month**. The doctor or nurse may ask you some questions to help decide. If you have **not** experienced the problem in the past month tick the 'No' box. You should answer 'No' even if you have had the problem in the past but not in the past month.

Have you experienced any of the following in the last month?

- | | Yes | No | | Yes | No |
|---|--------------------------|--------------------------|--|--------------------------|--------------------------|
| 1. Dribbling of saliva during the daytime | <input type="checkbox"/> | <input type="checkbox"/> | 16. Feeling sad, 'low' or 'blue' | <input type="checkbox"/> | <input type="checkbox"/> |
| 2. Loss or change in your ability to taste or smell | <input type="checkbox"/> | <input type="checkbox"/> | 17. Feeling anxious, frightened or panicky | <input type="checkbox"/> | <input type="checkbox"/> |
| 3. Difficulty swallowing food or drink or problems with choking | <input type="checkbox"/> | <input type="checkbox"/> | 18. Feeling less interested in sex or more interested in sex | <input type="checkbox"/> | <input type="checkbox"/> |
| 4. Vomiting or feelings of sickness (nausea) | <input type="checkbox"/> | <input type="checkbox"/> | 19. Finding it difficult to have sex when you try | <input type="checkbox"/> | <input type="checkbox"/> |
| 5. Constipation (less than 3 bowel movements a week) or having to strain to pass a stool (faeces) | <input type="checkbox"/> | <input type="checkbox"/> | 20. Feeling light headed, dizzy or weak standing from sitting or lying | <input type="checkbox"/> | <input type="checkbox"/> |
| 6. Bowel (fecal) incontinence | <input type="checkbox"/> | <input type="checkbox"/> | 21. Falling | <input type="checkbox"/> | <input type="checkbox"/> |
| 7. Feeling that your bowel emptying is incomplete after having been to the toilet | <input type="checkbox"/> | <input type="checkbox"/> | 22. Finding it difficult to stay awake during activities such as working, driving or eating | <input type="checkbox"/> | <input type="checkbox"/> |
| 8. A sense of urgency to pass urine makes you rush to the toilet | <input type="checkbox"/> | <input type="checkbox"/> | 23. Difficulty getting to sleep at night or staying asleep at night | <input type="checkbox"/> | <input type="checkbox"/> |
| 9. Getting up regularly at night to pass urine | <input type="checkbox"/> | <input type="checkbox"/> | 24. Intense, vivid dreams or frightening dreams | <input type="checkbox"/> | <input type="checkbox"/> |
| 10. Unexplained pains (not due to known conditions such as arthritis) | <input type="checkbox"/> | <input type="checkbox"/> | 25. Talking or moving about in your sleep as if you are 'acting' out a dream | <input type="checkbox"/> | <input type="checkbox"/> |
| 11. Unexplained change in weight (not due to change in diet) | <input type="checkbox"/> | <input type="checkbox"/> | 26. Unpleasant sensations in your legs at night or while resting, and a feeling that you need to move | <input type="checkbox"/> | <input type="checkbox"/> |
| 12. Problems remembering things that have happened recently or forgetting to do things | <input type="checkbox"/> | <input type="checkbox"/> | 27. Swelling of your legs | <input type="checkbox"/> | <input type="checkbox"/> |
| 13. Loss of interest in what is happening around you or doing things | <input type="checkbox"/> | <input type="checkbox"/> | 28. Excessive sweating | <input type="checkbox"/> | <input type="checkbox"/> |
| 14. Seeing or hearing things that you know or are told are not there | <input type="checkbox"/> | <input type="checkbox"/> | 29. Double vision | <input type="checkbox"/> | <input type="checkbox"/> |
| 15. Difficulty concentrating or staying focussed | <input type="checkbox"/> | <input type="checkbox"/> | 30. Believing things are happening to you that other people say are not true | <input type="checkbox"/> | <input type="checkbox"/> |

All the information you supply through this form will be treated with confidence and will only be used for the purpose for which it has been collected. Information supplied will be used for monitoring purposes. Your personal data will be processed and held in accordance with the Data Protection Act 1998.

Developed and validated by the International PD Non Motor Group
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Falls, Parkinsonism and Frail elder

Dr Ramila Wickramatunga Varendran

Epidemiology

Falls are a common presenting factor in those who have both frailty and Parkinsonism. We may be putting the cart before the horse when focusing on falls as it is likely that underlying frailty is the primary contributor. It is concerning that a recent prevalence study showed that the prevalence of frailty is higher in lower to middle income countries, along with pace of ageing. This has significant health policy implications with a need to focus on proven interventions that can reduce both morbidity and mortality in this group.

The aetiology of falls is multi-factorial. Underlying visual impairment, cognitive limitations, depression, poor mobility associated with neuro-muscular problems or rheumatological issues, cardio-respiratory limitation and environmental barriers can all have a contribution. For those with Parkinson's disease rigidity, akinesia, tremor, postural hypotension, depression and dementia increase the risk of falls.

The underlying features associated with frailty include sarcopaenia, osteoporosis and increased cardiovascular morbidity which all play a significant part in poorer outcomes for those with falls. It is encouraging that a recent study of elderly Sri Lankans showed that they are aware of the multiple components that result in higher risk of falls. This suggests that there is scope to widen this knowledge base to improve outcomes.

In addition to underlying medical factors, medications also play a role with both falls and Parkinsonism. The exact epidemiology of this phenomenon is poorly identified in studies of lower to middle income countries. Reducing polypharmacy would be useful for this group.

Interventions

Despite a paucity of models of management of falls in lower- and middle-income countries, there are investigations and interventions that have been reviewed in high income countries. Though these should be modified to reflect resource utilization, it is important to accept that they need to be multipronged and individualized.

It is important to understand that though there is significant overlap of factors that predispose to falls, they are a complex phenomenon resulting from multi-morbidity. Investigation of these individuals requires a comprehensive wide-angle approach with a focus on modifiable factors with realistic achievable goals that are pursued over a protracted time. This explains why it is important to consider having multidisciplinary teams which focus on client related goals rather than single disease entities.

Ideally these multidisciplinary teams should be able to share information to enable adequate care provision without overlap. Having a joint multidisciplinary clinic with a common database of information that is shared can improve efficiency. These interventions should involve prevention of frailty with nutrition and exercise in addition to a focus of fall reduction. Patients with Parkinson's disease have displayed lower levels of Vitamin D; this may be another area that is targeted with nutrition.

There are significant environmental factors that can contribute to the prevention of frailty like better nutrition and modification of the environment with rails, flat pathways and areas that enable regular exercise.

Discussion

Though a medical model requires the diagnosis of underlying factors to work on a management plan, this complex problem of falls with a background frailty does not always yield a single disease entity. Most of the effective interventions included exercise, nutrition and environmental modification in addition to medication review. Although the ideal intervention needs to be individualized it may be useful from a population health view to consider general system factors as these may have higher health economic impact. In middle income countries having environmental factors that can be accessed freely may have a bigger advantage. Safe public walking areas, with open exercise equipment and subsidized nutritional supplement could be considered.

The high levels of literacy combined with recent data showing relatively high levels of knowledge around falls related pathophysiology in Sri Lanka is encouraging. This article suggested that media including television, radio and newspapers have a significant impact of increasing the knowledge of community based older adults. The use of "Google" rather than libraries with curated information is an example of outside the square technology that has functional benefit. Providing further information via these channels on

methods to prevent frailty can be an area that improves overall outcomes for the population. General screening of falls risks as well as increased awareness of preventative methods can be discussed in this manner.

Detecting older individuals who are at high risk when they have interactions with any aspect of the health care system is important particularly in resource limited settings. The combination of self-administered questionnaire with the timed up and go test has shown high efficacy as a screening tool. The limited amount of time required for the timed up and go test makes it more attractive and could be used in community health settings. Although this is ideally done with a physiotherapist it can be considered in an inter-disciplinary situation with other allied health members who work with a physiotherapist.

Despite the practice of multi-factorial interventions to reduce falls and more importantly outcomes associated with falls, there is a paucity of evidence showing significant benefit. This may partly relate to the timing in that after a person falls it may be relatively late in the disease course to intervene. The main area that showed some evidence of benefit was fracture prevention.

The combination of frailty falls, and Parkinsonism significantly increases the risk of fractures with devastating outcomes. Patients with Parkinson's disease may have higher rates of both osteoporosis and low Vitamin D. This may be an area that is targeted with nutrition and treatment. Nutrition is a contributor to both falls and frailty. Dietary patterns that include higher protein intake, Vitamin D supplementation, anti-oxidants and long chain fatty acids show promise in the reduction of sarcopaenia. Sarcopaenia is the component of frailty that directly results in falls with reduced muscle mass translating to functional deficits. One of the studies included in the recent Cochrane review suggested benefit of a dietary intervention in the acute setting; with better outcomes with reduced fractures in the intervention group.

Bleeding and fractures are complications of falls that result in high morbidity and mortality. Encouragement of appropriate anticoagulation is advocated, using oral anticoagulation for those with stroke risk, unless they have multiple falls with head strike.

While addressing Parkinsonism requires medical assessment, the role of the pharmacist in the community setting is significant. This is particularly important where medications may be accessed without a prescription. In addition to the focus on reducing Polypharmacy, treatment of osteoporosis along with the appropriate use of anticoagulation is very important.

The other area to explore is preventative community strategies prior to people reaching the older years. We may be seeing poorer outcomes as we are targeting the population after the proverbial horse has bolted. Something to consider in populations with large proportions reaching the geriatric bracket is multidisciplinary efforts including medical, dietary, physiotherapy and nursing screening at an earlier age.

Although falls, parkinsonism and frailty are complex in aetiology, addressing risk factors in the community particularly at early stages may reduce the poorer outcomes faced by this group. This provides food for thought and research in middle income settings with consideration for cost effectiveness.

References

1. Siriwardhana DD, Hardoon S, Rait G et al Prevalence of frailty and pre frailty among community dwelling older adults in low income and middle-income countries: a systematic review and meta-analysis. *British Medical J Open* 2018;8: e018195. doi:10.1136/bmjopen-2017-018195
2. Karadsheh MS, Weaver M, Rodriguez K et al. Mortality and revision surgery are increased in patients with Parkinson's disease and fractures of the femoral neck; *Clinical Orthopaedics and related research* (2015) 474: 3272-3279 doi: 10.1007/s11999-015-4262-5
3. Gamage N, Rathnayeke N, Alwis G. Knowledge and Perception of Falls among Community Dwelling Elderly: A Study from Southern Sri Lanka. *Current Gerontology and Geriatrics Research*. (2018), Article ID 7653469, 8 pages doi.org/10.1155/2018/7653469
4. Sleeman I, Aspray T, Lawson R et al; The role of Vitamin D in disease progression in early Parkinson's disease; *Journal of Parkinson's. Di* 2017; 7: 669-675
5. World Health Organisation. World report on ageing and health. 2015. Geneva, Switzerland.

Psychiatrist's role in managing Parkinsonism

Dr Malsha Gunathilake and Prof. Varuni de Silva

Parkinson's disease (PD) is a neurodegenerative disorder characterized by classic motor symptoms of bradykinesia, resting tremor and rigidity. However, PD also includes many non motor symptoms. Prominent among them are cognitive impairment, depression, psychosis, impulsivity, sleep disorders, autonomic failure and pain syndromes. Patients with PD have a higher risk of developing psychiatric complications. This can result in poorer overall prognosis, more disability and poor quality of life. Hence, a psychiatrist has a significant role as a member of the multidisciplinary team, in managing patients with PD and improving outcomes. In this article, different psychiatric conditions associated with PD and their management will be further discussed.

Depression is probably the commonest psychiatric co morbidity in patients with PD. However the most challenging co-morbidity is psychotic symptoms as these even complicate the management of PD. Dementia due to Parkinson's disease may have to be differentiated from Lewy Body dementia (LBD). Some of these challenges in diagnosing and managing are discussed below.

Depression is one of the commonest psychiatric disorders reported in patients with PD. A study from Sri Lanka reported a prevalence of depression of 37.5% among them. Therefore, it is important that patients with PD are screened for depression. Advanced PD, anxiety, cognitive impairment, psychosis, long duration of illness, younger age of onset, features of atypical parkinsonism, female gender and requiring higher doses of levodopa are identified as risk factors for depression in PD. SSRI(Selective Serotonin Inhibitors)are considered first line treatment. SNRI (Serotonin and Norepinephrine Inhibitors) such as venlafaxine and TCA (Tricyclic Antidepressants) are also effective. However, SNRI are associated with worsening of motor symptoms and TCAs are poorly tolerated due to their anticholinergic side effects.Evidence also supports the use of ECT (Electro Convulsive Therapy) for severe depression in patients with PD. ECT has dual benefit in this group as it improves both depressive and motor symptoms in PD.CBT(Cognitive Behavioural Therapy) has a significant benefit in mild to moderate depression especially in patients with PD who do not have executive dysfunction. There is some evidence for the beneficial effect of deep brain stimulation (DBS) especially focused on the sub-thalamic nucleus (STN)

and rTMS (Repetitive trans-cranial magnetic stimulation) in management of depression in patients with PD. However studies conclude that these effects are short lasting. DBS and rTMS are not yet available in Sri Lanka.

Studies report that the prevalence of psychotic symptoms in PD ranges from 15.8% to 75% Visual hallucinations being the commonest. In patients with visual hallucinations it is important to look for the presence of delirium first. If delirium is excluded, it is important to evaluate the temporal relationship between onset of psychosis with anti-parkinsonian medications. Visual hallucinations and features of Parkinsonism could also be present in LBD. A careful history and examination is required to differentiate between these conditions. Patients with PD can also develop auditory hallucinations and delusions. If psychotic symptoms are due to anti-Parkinson medication, dosages should be adjusted. In situations where multiple drugs are used this should be done in the order of anticholinergics, selegiline, amantadine, dopamine receptor agonists, catechol-O-methyltransferase inhibitors and lastly levodopa.

If adjustment of anti-Parkinson medication is inadequate to control psychotic symptoms it may be necessary to start treatment with antipsychotics. However, antipsychotics can make symptoms of PD worse. Therefore, close collaboration between the psychiatrist and the neurologist is necessary in order to negotiate two aspects. Atypical antipsychotics; clozapine, olanzapine, and quetiapine are recommended in the management of psychotic symptoms. Usually quetiapine as the first choice as it has the lowest risk of worsening of motor symptoms in PD. Small dose of clozapine may be considered when other antipsychotics are not effective. ECT can also be used in resistant cases.

Cognitive deficits of different degrees varying from mild cognitive impairment to severe dementia are common in PD. The differentiation between PD dementia and LBD are based on the onset of symptoms. If patients with established PD develop dementia one year or more later the diagnosis favours PD dementia. If patients develop features of LBD such as visual hallucinations, cognitive decline and fluctuating levels of consciousness followed by Parkinson's features, the diagnosis is LBD. PD dementia may respond to cholinesterase inhibitors such as donepezil and rivastigmine. However in patients with psychotic features antipsychotics should be used cautiously as they can worsen Parkinson's features and precipitate autonomic instability in LBD.

Because of the high risk of depression, dementia and psychotic symptoms, patients diagnosed with PD should undergo brief cognitive testing, screening for depression, Rapid Eye Movement (REM) sleep behaviour disorder and psychotic symptoms.

The prevalence of dementia in PD varies from 31.3% to 75%. Risk factors for dementia are old age, severity of motor symptoms and presence of visual hallucinations. Patients with dementia of PD have higher dysfunction in the domains of attention, executive and visuospatial functioning with lesser involvement of memory and language. Rivastigmine and Donepezil are found to be effective in dementia in PD.

Excessive day time sleepiness, insomnia, restless leg syndrome (RLS) and REM sleep behavior disorder (RBD) are the commonest sleep disorders in PD. First step in management is proper diagnosis which involves history from the patient and the bed partner followed by polysomnography if necessary. Then look for causes including sleep hygiene, medications, and comorbid conditions. Depending on the type of sleep disorder, sleep hygiene is used as the first line treatment option. Clonazepam is recommended for of RBD. Melatonin has been evaluated for insomnia. There is some evidence for the efficacy of long acting dopamine agonist like cabergoline in RLS.

Patient with PD are at risk of developing anxiety disorders. They may be seen in 19.8% to 67% of patients with PD. The commonest include panic disorders, social phobia and generalized anxiety disorders. Female gender, early onset PD, presence of depressive symptoms, severe PD, postural instability, gait dysfunction, motor fluctuations are risk factors for development of anxiety disorders. As there are no definitive studies on use of anxiolytic medications in patients with PD, usually the recommendations are based on the studies of elderly without PD. There are some specific issues related to the management of anxiety in patients with PD as some anti-parkinsonian medications are associated with development or variation in anxiety symptoms. In such cases it is important to adjust doses or replace those medications. SSRIs are considered first line treatment. Benzodiazepines are generally not recommended as they increase the risk of falls, and may worsen cognitive impairment and Parkinson features. Relaxation and CBT are also useful.

Impulse control disorders (ICDs) such as pathological gambling and hypersexuality have been reported to be higher in patients with PD those receiving dopaminergic agents. Reduction or discontinuation of dopaminergic

agents will lead to the resolution of symptoms.

Suicidal behavior is also reported to be higher in patients with PD compared to general population. Risk is higher among those with depression, anxiety, hopelessness and history of ICDs.

Apart from above major psychiatric conditions, apathy and anhedonia are also commonly reported in patients with PD without depression leading to significant functional impairment.

Psychiatrists also have to be mindful about drug induced Parkinsonism caused by treatment with antipsychotics. Careful selection of antipsychotics especially in patients who develop extra pyramidal symptoms is necessary to prevent morbidity and improve compliance.

Because the risk of psychiatric illness is high in PD the psychiatrist has an important role in identification and treating them adequately to better the outcome. A collaborative management plan where the patient is jointly managed by the neurologist and the psychiatrist is necessary when psychiatric comorbidities are present.

References

1. Grover S, Somaiya M, Kumar S, Avasthi A. Psychiatric aspect of Parkinson's disease. *Journal of Neurosciences in Rural Practice* .2015;6 ;1.
2. Taylor D, Paton C, Kapur S. Parkinson's disease. *The Maudsley prescribing guidelines in psychiatry*. 12th edition. Willey Blackwell. London. UK;529.
3. Faber R, Trimble MR. Electroconvulsive therapy in Parkinson's disease and other movement disorders. *Mov Disord* 1991; 6:293-303.
4. Dobkin RD, Rubino JT, Allen LA et al. Predictors of treatment response to cognitive-behavioral therapy for depression in Parkinson's disease. *J Consult Clin Psychol* 2012;80:694-9.
5. Couto MI, Monteiro A, Oliveira A et al. Depression and anxiety following deep brain stimulation in Parkinson's disease: Systematic review and meta-analysis. *Acta Med Port* 2014;27:372-82. 87.
6. Epstein CM, Evatt ML, Funk A et al. An open study of repetitive transcranial magnetic stimulation in treatment-resistant depression with Parkinson's disease. *Clin Neurophysiol* 2007; 118:2189-94.

Pharmacology in management of Parkinson disease

Prof. Nirmala Wijekoon

What to achieve with drug therapy in Parkinson disease (PD)?

Parkinson disease has no cure. There is no drug proven to be effective in slowing or reversing the natural course of the disease. Thus the goal of drug therapy is to control signs and symptoms and keep the patient functioning as long as possible while minimizing adverse effects. However with time, patients become disabled despite drug therapy due to the disease process as well as due to adverse effects of drugs. Each patient's therapy needs to be chosen according to his/her disease stage and main symptoms.

What drugs are recommended for treatment of motor symptoms of PD?

First line treatment of early PD include levodopa coupled with carbidopa, non-ergot derived dopamine agonists and monoamine oxidase B (MAO-B) inhibitors. Levodopa gives the greatest benefit, especially in patients who are elderly or have cognitive decline. Adjuvant therapy for later PD with motor complications of levodopa includes dopamine agonists (DAs), MAO-B inhibitors and catechol-O-methyl transferase (COMT) inhibitors. There is no evidence of improvement of motor symptoms with amantadine. Amantadine is considered in advanced disease if dyskinesias are not controlled by existing therapy.

The comparative efficacy and risk of adverse effects of these drugs are shown in Table 1.

Table 1: Drugs recommended for treatment of motor symptoms of PD

First line treatment of early PD				
	Levodopa	Dopamine agonists (DAs) Pramipexol Ropinirole Rotigotine	MAO-B inhibitors Rasagiline Selegiline	

Improvement of motor symptoms of PD	More	Less	Less	
Improvement of activities of daily living	More	Less	Less	
Motor complications	More	Less	Less	
Other adverse effects <i>hallucinations</i> <i>ICD</i> <i>excessive sleepiness</i>	Less	More	Less	
Adjuvant therapy for later PD with motor complications				
	Dopamine agonists Pramipexol Ropinirole Rotigotine Apomorphine	COMT inhibitors Tolcapone Entacapone	MAO-B inhibitors Rasagiline Selegiline Safinamide	Amantadine
Improvement of motor symptoms of PD	Improve	Improve	Improve	No evidence
Improvement of activities of daily living Improve	Improve	Improve	Improve	No evidence
Improvement in off-time	More	Less	Less	No studies reporting these outcomes

Other adverse effects <i>hallucinations</i> <i>ICD</i> <i>excessive</i> <i>sleepiness</i>	More	Less	Less	No studies reporting these outcomes
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Levodopa

Current levodopa preparations include levodopa/carbidopa immediate-release (IR) tablets, levodopa/carbidopa controlled-release (CR) tablets and levodopa/carbidopa orally disintegrating tablets. The orally disintegrating tablet is bioequivalent to oral IR tablets. It dissolves on tongue but not absorbed in mouth until it travels in saliva to absorption sites in the proximal small bowel. Levodopa/carbidopa/entacapone combination is useful in advanced PD with motor fluctuations.

Dopamine Agonists (DAs)

Non-ergot derived DAs include pramipexol, ropinirole, rotigotine and apomorphine. Rotigotine is administered as a transdermal patch which provides more continuous dopaminergic stimulation than oral medication. It is particularly useful in swallowing difficulties. Subcutaneous injection of apomorphine is effective for temporary relief (“rescue”) of off-periods of akinesia in patients with advanced disease. Ergot-derived DAs (pergolide, bromocriptine and cabergoline) are not first line treatments.

MAO-B inhibitors

Selegiline is an adjunctive therapy for patients with a declining or fluctuating response to levodopa. It has only a minor effect on motor symptoms as monotherapy. Rasagiline is more potent and effective as monotherapy for early PD and in later disease as adjunctive therapy. The newest MAO-B inhibitor, safinamide is indicated for reducing response fluctuations in levodopa therapy.

COMT inhibitors

Tolcapone and Entacapone are not used as monotherapy in early disease. They are indicated in patients receiving levodopa who have developed response fluctuations.

Anticholinergic drugs

Trihexyphenidyl hydrochloride (benzhexol) improve tremors but has little

effect on bradykinesia. They shouldn't be given in dyskinesia and/or motor fluctuations.

What drugs are currently registered in Sri Lanka?

These include levodopa/carbidopa(IR)tablets, levodopa/carbidopa/ Entacapone combination tablets, pramipexole, ropinirole, bromocriptine, entacapone and benzhexol.

Adverse Effects of dopaminergic drugs

Table 2: Adverse effects of dopaminergic drugs

Peripheral AEs	anorexia, nausea, vomiting cardiac arrhythmias orthostatic hypotension
Central AEs	confusion, psychosis ICDs (eg. pathological gambling, hypersexuality, binge eating, compulsive shopping) depression, anxiety, agitation, euphoria nightmares, insomnia, somnolence, narcolepsy
Motor complications	dyskinesias end of dose akinesia on-off phenomenon
Miscellaneous	Levodopa Mydriasis and precipitation of acute glaucoma precipitation of gout abnormalities of smell or taste Ergot-derived DAs Painless digital vasospasm pulmonary, retroperitoneal, and pericardial fibrotic reactions cardiac valvulopathy MAO-B inhibitors Precipitation of hypertension

Nausea and vomiting

All dopaminergic drugs can cause nausea/vomiting and particularly, apomorphine. However tolerance develops rapidly. Taking doses with food can help. Metoclopramide, prochlorperazine and other centrally acting dopamine-blocking anti-emetics should be avoided as they may worsen PD. A short course of Domperidone (peripherally acting D2R antagonist) is an option for intractable nausea and vomiting.

Impulse control disorders (ICDs)

ICDs can develop with any dopaminergic therapy at any stage in the disease course. They are generally under-reported and often unrecognized by health care professionals. The risk is highest with DAs. Other risk factors include an impulsive personality, a history of substance abuse and a family history of ICDs.

Motor complications

Motor complications are more likely with longer duration of treatment and higher levodopa doses (≥ 600 mg/d). Therefore symptomatic treatment of mild Parkinsonism is best avoided until there is some disability or significant social impairment. Compared to levodopa, motor complications are less frequent and are limited to dyskinesias with DAs and MAO-B inhibitors.

Dyskinesias

Dyskinesias are reported in up to 80% of patients receiving levodopa for more than 10 years. Commonest manifestation is choreoathetosis of face and distal extremities. It is a dose-related adverse effect but there is individual variation in the dose required. Amantadine and clozapine may help to improve troublesome dyskinesias.

End of dose akinesia (wearing off effect)

Wearing off of drug effect occurs towards the end of a dosage interval resulting in akinesia. This is related to timing of levodopa dosing thus predictable. Management options include smaller, more frequent dosing, using levodopa CR preparations and using DA, COMT inhibitor or MAO-B inhibitor as adjuvant therapy. High protein meals may interfere with levodopa absorption therefore giving drugs 1 hour before meals can be helpful.

On-off phenomenon

On-off phenomenon is unpredictable switching between off-periods (akinesia) and on-periods (improved mobility) often with marked dyskinesia.

Management of such patients is challenging. For those with severe off-periods, subcutaneous apomorphine may provide temporary benefit but at the expense of an increase in dyskinesias.

What are the adverse effects of anti-cholinergic drugs?

They are poorly tolerated by elderly or cognitively impaired and may cause confusion, hallucinations, blurred vision, constipation, urine retention and dry mouth causing swallowing difficulties. Acute suppurative parotitis reported as a complication of dryness of mouth.

Precautions needed in drug therapy for motor symptoms

Anti-parkinsonian drug therapy should never be stopped abruptly as this carries a risk of neuroleptic malignant syndrome and rhabdomyolysis. This also causes acute akinesia. The practice of temporarily withdrawing people from their anti-parkinsonian drugs ('drug holidays') to reduce motor complications should not be practised and they are also of little help in the management of on-off phenomenon.

Treatment with dopaminergic drugs is associated with development of ICDs. It is important to make patient and carers aware of these. Medication withdrawal or dose reduction is needed until symptoms resolve.

All anti-parkinsonian drugs are known to cause psychotic symptoms (delusions and hallucinations). They can deteriorate existing psychiatric illnesses or precipitate relapses. In those with active and/or severe psychotic illnesses dopaminergic drugs should be avoided.

Excessive daytime sleepiness and sudden onset of sleep can occur with levodopa and DAs. Patients should be warned of risks and caution with driving or operating machinery.

Hypotensive spells can occur with DAs particularly during first few days of treatment and levodopa can cause postural hypotension. This increases the falls risk associated with old age and PD itself and educating regarding falls prevention is important.

Levodopa and DAs should be used cautiously in IHDs and arrhythmias as there is a small but increased risk of arrhythmias. Recent myocardial infarction is a contraindication. Levodopa is contraindicated in angle-closure glaucoma.

Levodopa is a precursor of melanin and has potential to activate malignant melanoma and should be used with particular care in patients with a history of melanoma or suspicious undiagnosed skin lesions.

Before starting treatment with DAs which are ergot derivatives (bromocriptine, pergolide, cabergoline) cardiac valvulopathy should be excluded with

echocardiography. There is a risk of pulmonary and retroperitoneal fibrosis and patients should be monitored for dyspnoea, persistent cough, chest pain, cardiac failure, abdominal pain and renal failure.

Harmful drug interactions should be avoided. MAO-B inhibitors should not be taken with other MAOIs and should be used with caution in patients receiving TCADs, SSRIs or tramadol because of the risk of serotonin syndrome. Levodopa should not be used concomitantly or within 2 weeks of use of MAO-A inhibitors.

When anti-cholinergic drugs are considered in elderly, particular attention needs to be paid to adverse effects like urine retention, constipation and swallowing difficulties.

Place of drug therapy in management of non-motor symptoms

Orthostatic hypotension

The patient's existing medications need to be reviewed for anti-hypertensives, diuretics, dopaminergic drugs, anti-cholinergics and antidepressants. Midodrine, an alpha agonist is indicated for management if a correctable cause is not found. If midodrine is contraindicated, not tolerated or ineffective, Fludrocortisone is an option.

Daytime sleepiness

Modafinil, a centrally acting sympathomimetic can be considered, once a detailed history has excluded reversible pharmacological and physical causes.

Sleep disturbances

Clonazepam or melatonin is considered if other possible causes are corrected.

Psychotic symptoms

If these symptoms are troublesome, quetiapine can be tried. Reducing the dosage of dopaminergic drugs is also useful. Clozapine is another option but close monitoring is needed. Quetiapine and clozapine doses needed for people with PD are lower than the doses required in other indications. First (chlorpromazine, haloperidol) and second-generation (olanzapine, risperidone) antipsychotic drugs should be avoided because they can aggravate Parkinsonism.

Parkinson's disease dementia

Cholinesterase inhibitors (donepezil and rivastigmine) are indicated for mild or moderate PD dementia. As the benefits of these drugs are modest, an initial trial of 2 to 3 months is indicated. If no benefit is seen or if there are serious adverse effects, drug therapy should be stopped. When stopping, the dose is slowly tapered to avoid sudden cognitive and behavioural deterioration.

Drooling

Drug therapy is considered only when non-pharmacological management is ineffective. Options include glycopyrronium bromide (muscarinic anticholinergic drug) and botulinum toxin type A injections into salivary glands.

Neuro-protective therapy

Neuro-protective therapy aims to slow, halt or reverse disease progression by limiting the loss of dopaminergic neurons in basal ganglia. Although no therapy has been proven to be neuro-protective and approved for clinical use, several agents are being investigated. These include rasagiline, safinamide and pramipexol. Pre-clinical studies suggest selegiline may reduce disease progression, but clinical trials have yielded ambiguous results. Studies have not found vitamin E and co-enzyme Q10 to be effective.

References

1. National Institute for Health and Care Excellence. Parkinson's disease in adults: diagnosis and management: NICE; 2017 [cited 2019 January 09]. <https://www.nice.org.uk/guidance/ng71>
2. Sellbach A, Silburn P. Management of Parkinson's disease. Australian Prescriber 2012;35:183–8
3. eTG complete. Parkinson disease. Melbourne (VIC): Therapeutic Guidelines Ltd; 2017. [cited 2019 January 09]. https://tgldcdp.tg.org.au/viewTopic?topicfile=parkinson-disease#MPS_d1e587
4. Katzung BG. Basic and clinical pharmacology. 14th ed. New York: McGraw-Hill Education; 2018
5. National Medicines Regulatory Authority, Sri Lanka. Registered products. NMRA ;2019 January 17 [cited 2019 January 17]. http://nmra.gov.lk/index.php?option=com_product&Itemid=127&lang=en

Nurses' role in the management of Parkinson's disease

Ms Kalpani Abhayasinghe

“I am trying to smile but the rigid muscles often make it difficult. I am not being rude. I am not being miserable. I have Parkinson's.”(Terry Kavanagh: A patient with PD)

Parkinson's disease (PD) causes significant disabilities limiting quality of life (QoL) of the affected people and their caregivers. More than 40 percent of people with PD will experience severe disability accompanied by poor locomotion, and communication problems (Tosin et al. 2016). Appropriate management of the condition and early interventions are important as it can alleviate some of the most distressing features.

Progress of PD can be described in four stages from a practical, management perspective: i.e. diagnosis, maintenance, complex and palliative. In the early stages of PD pharmacological treatment can be very effective in managing symptoms. However as the disease progresses into complex and palliative stages, the individual may experience more difficulties and multi-disciplinary care approach is essential. This includes rehabilitation, physiotherapy, language therapy and advanced nursing care.

Nurses' role in the care of people with PD

The nurse, as a member of the MDT, has an important role in caring for a person with PD. There are three goals to achieve when providing such care. (1) Delay disease progression, (2) Relieve symptoms and (3) Preserve functional capacity of the patient. Nursing interventions include symptom management using pharmacological and non-pharmacological methods, take measures to avoid complications, communicate with care team, patient and family, raise concerns for improving QoL, and planning for palliative and advanced nursing care. Nurses direct the plan of care to meet the individual needs of the patients, with an understanding of their physical, cognitive and behavioural potentials and encourage adaptations to the limitations imposed by the disease.

Nursing care during early stages of PD

When a person with Parkinson's is in hospital for treatments, it is the nurse who has the most direct contact with the patient. This means they are able to observe and monitor patient's functional level, record his responses to

pharmacological and non-pharmacological treatments throughout the day and assist with implementing strategies to improve them. They will have more opportunity to observe gait, balance, risk of falls, posture and movements which is essential for setting goals and treatment programmes by the MDT and more importantly convincing and getting the patient and family on board. They can educate the patient and the family about the benefits of daily low intensity exercises.

Nurses also need to display patience and empathy with patients and the family members. Patients may require a long time to complete activities and also their level of function may change from one day to the next. It is important to attend to patient's physical care needs as well as emotional, psychological and social aspects. Family members need to know the condition and may need psychological support to cope with the situation. Educating and raising awareness regarding the disease and management of the condition at early stages may increase their support in long term care.

Motor symptoms of PD can be managed by pharmacological treatments such as Levodopa and Dopamine agonists. However, before starting the treatments nurses should discuss the life style of the individual, adverse effects and risk of having medication as some medication can involve excessive sleepiness, psychotic symptoms such as delusions and hallucinations and impulse control disorders. Patients experience the symptoms are largely in restraint, while they are 'on' but when they are "off", they are not, making it more difficult for them to comprehend and move about. This is called "on/off nature" of the condition, that might change from "on" to "off" like a light switch, therefore it is extremely important they receive their medication on time, every time. Anti-parkinsonian medicines should not be withdrawn abruptly because of the risk of neuroleptic malignant syndrome (NICE, 2019).

Nutrition of PD patients is another concern. It is vital to make sure that patients have access to a varied and balanced diet. Advise people with PD to take vitamin D supplements and avoid a reduction in their total daily protein consumption. People with PD should not take over the counter nutritional supplements without medical advice (NICE, 2019). Patients with swallowing difficulties might need NG or PEG feeding.

Nursing care during rehabilitation and later stages

Nurses can attend the advanced care needs of the patients such as eating,

drinking, washing, walking, incontinence or constipation. Careful assessment, documentation and monitoring of the disease progress, communicating with the MDT, providing psychological support for patient and family are major concerns. It is necessary to educate the patient and family members regarding available support services, for example, personal care, equipment and practical support, financial support and advice, care at home and respite care. People with PD should have a comprehensive care plan agreed between the person, their family members and the MDT.

It is important to understand pain and physical needs of the patient as PD affects communication. Communication difficulties can be overcome by understanding patient's behaviour, gestures and body language and respond by speaking slowly and clearly so the patient can understand. Maintaining eye contact is important when communicating with a person with PD. Both verbal and written communication should be encouraged throughout the course of the illness while making sure it does not intimidate the patient.

Interventions for morbidity relief (ex: pain management), safety, prevention of complications (falls, bed sores) and maintaining psychological health of the patient and the family members are also important at complex and palliative stages. As the disease progresses 70% of patients will get some cognitive decline and this may lead to a diagnosis of dementia. Potential deterioration in ability to perform ADLs may require additional support at home and increase caregiver burden.

Nurses need to set mutual, realistic goals with the patient and family to promote learning and adapting to physical, cognitive or behavioural changes. Autonomy and QoL of the patient matters and mutual understanding between nurses and family members are very important.

In complex and palliative stages patients with PD suffer from swallowing difficulties, tend to aspirate leading to pneumonia. It is also important to note that increased disability makes patients more vulnerable to infections. Loss of balance can cause falls that result in serious injuries. People with PD mostly die due to these causes. Pain management is also crucial. Advanced nursing interventions and careful planning of care is necessary at these stages with involvement of the patient, family and health and social care professionals. Palliative care for the patient should be holistic, that facilitates a person centered approach to support the patient and family members' personal,

social, psychological and spiritual needs. The palliative care plans should include the preferences of patients for how their condition should be managed as it progresses, and about their wishes. Providing emotional support to the family members in bereavement is also an important role for nurses who work in palliative care settings.

Parkinson's disease currently has no cure. Long-term care plan for patients with PD is essential to monitor progression of the disease. Management of PD not only includes patient care but also assistance toward their caregivers. However, as with many chronic diseases, people with PD can live for many years and in later stages they may need institutionalized care that is beyond the ability of the informal caregivers. Informal caregivers need to be empowered and supported in order to minimize the caregiver burden. Promoting community and home based care services are important aspects in this regard.

Countries like the UK offer people with PD an accessible point of contact with specialist services usually provided by a Parkinson's disease nurse specialist. PD nurse specialists often visit families, provide care, health education and emotional support to patient and the family members. It is essential and timely we initiate community and home based care services with PD specialized nurses in Sri Lanka.

Reference

1. Goroll AH, Mulley Jr AG. Primary care medicine: Office evaluation and management of the adult patient. (2009) 6th ed. Philadelphia: Lippincott Williams &Wilkins; p1223-1225.
2. Greenwood RJ, Barnes MP, McMillan TM, Ward C. Handbook of Neurological Rehabilitation(2003) 2nd ed, Routledge, New York.
3. NICE Guidelines (2019), Parkinson's disease in adults: <https://www.nice.org.uk/guidance/ng71/resources/parkinsons-disease-in-adults-pdf-1837629189061>
4. Shin JY, Hendrix CC. Management of patients with Parkinson disease. The Nurse Practitioner; (2013)38(10): 34–43.
5. Tosin MHS, Campos DM, Andrade LT et al. Nursing interventions for rehabilitation in Parkinson's disease: cross mapping of terms. Revista Latino-Americana de Enfermagem; (2016)24:e2728.

How to utilize physiotherapy and other complementary therapies in Parkinson's disease?

Ms Nadeesha Kalyani

Parkinson's disease (PD) is a complex neurodegenerative disorder, and by 2030, the number of people living with PD is expected to double because of the increasing aging population. Parkinson's disease primarily results from the deficiency of the neurotransmitter dopamine and the loss of function of dopaminergic cells in basal ganglia, and is manifested by a broad spectrum of motor and non-motor symptoms.

The traditional approach of managing PD has been centred around medication, especially levodopa which was considered the "gold standard" and many more other medications have followed. Deep brain stimulation is a more advanced treatment option used. To date these remain the first line treatments however, they provide symptom attenuation only. A full recovery is highly unlikely even in patients who receive best-practice medical care (i.e. surgery and medication). Despite advances in pharmacological therapy and surgical procedures, the disabling motor and non-motor symptoms in PD persist, leaving many opportunities for inclusion of rehabilitation therapies as an adjuvant to standard medical care. Consequently, there is a growing body of evidence supporting physiotherapy, especially in the form of exercises as a viable supplementary intervention in treating PD. A review by Margaret et al. categorised these supplementary interventions as resistance training, aerobic training, balance, gait or cued training and complementary exercises (i.e. dance) (Mak, Wong-Yu, Shen, & Chung, 2017). In PD, it has been found that exercise stimulates dopamine synthesis in the remaining dopaminergic cells and hence reduces motor as well as non-motor symptoms. It is argued that exercise improves overall well-being among individuals with neurodegenerative diseases by improving physical and cognitive functions. Furthermore, evidence suggests that people with higher levels of routine physical activity are at lower risk for developing PD.

Resistance training

Resistance Training has been found to improve muscle strength in people with PD. Muscle weakness may be a primary symptom of PD which can contribute to postural instability and gait difficulties, and has also been identified as a secondary cause for bradykinesia. Resistance training in the treatment of

PD aims at improving the above issues. These exercises are mainly focused at strengthening of the knee extensors and flexors, leg muscles, plantar flexors, trunk flexors, extensors and rotators (Roeder, Costello, Smith, Stewart, & Kerr, 2015).

Aerobic training

Aerobic exercise may reduce the detrimental effects of neuromuscular slowing in PD, by improving the subject's ability to initiate and perform appropriate movement patterns. Aerobic training involves the use of large muscle groups and a training intensity of 60–75% maximum heart rate or 40–50% heart rate reserve. Stationary bicycle training, walking training with treadmill, walking with music are some of the aerobic training. Past studies have demonstrated that aerobic training improved cardiopulmonary fitness, walking performance (walking speed, stride length and gait stability) and quality of life in PD.

Gait training

Gait training is focused on improving gait parameters such as speed and/or stride length which are impaired in PD. These are being practised as over ground walking as well as with a treadmill and more advanced gait training with a robotic gait trainer. The belt speed of the treadmill or robotic gait trainer is progressively increased and the training is done with or without body-weight support. These interventions have been found to be effective in improving gait speed, stride/step length and the ratio of single to double leg stance time, which facilitated to normalise the shuffling gait pattern.

Balance training

Exercises are prescribed to challenge various PD-specific components of impaired balance. These trainings include self-destabilization of the center-of-body mass (performing voluntary motor actions in static or dynamic conditions e.g., transferring body weight onto the tips of the toes and onto the heels), externally induced destabilization of the center-of-body mass (maintain balance while standing on foam support base) and coordination between leg and arm movements during walking. Balance training positively influences postural stability, improve the level of confidence perceived during daily life balance activities, and reduce the frequency of falls in patients with PD.

Cued training

Cueing is defined as using external temporal or spatial stimuli to facilitate movement (gait) initiation and continuation. These cues are categorised as visual cues (lines or markers on the ground and treadmill), rhythmic auditory

cues (metronome beats or music at a pre-set frequency) and somatosensory cues (tactile sensation given to a body part). Visual and auditory cues are found to have an immediate and powerful effect on gait performance by minimizing freezing and festination of gait in PD. Patients are instructed to pay attention to the cues and to step on the line or markers or to step in time with an auditory or somatosensory cue. Particularly, the visual cues are provided in the form of lines at regular intervals on the floor, and these may draw attention to the stepping process and enhance the optical flow to improve gait. When visual cues when coupled together with auditory cues, such as the instructor's commands, the patients can take large steps with a high cadence (steps per minute), leading to a fast speed comparable to that of normal.

Dance

While the traditional exercise programs may assist and benefit patients with PD, long-term adherence to these modalities has proven to be challenging. A systematic review assessing exercise adherence in PD revealed that reduced enthusiasm was a common reason for reduced participation. Therefore, researchers are seeking novel, community-based interventions that facilitate uptake and enjoyment. As a result, dance has become an emerging management option. It is the therapeutic use of movement to support the intellectual, emotional, and motor functions of the body (Earhart, 2009). There are numerous dance styles such as tango, ballet, ballroom and improvisation, as well as models designed especially for PD, such as DfPD® methodology currently being used for managing the symptoms of PD. Dance acts as a form of exercise as well as possesses numerous cueing strategies among which the rhythmic music used in dance acts as a strong auditory cue to facilitate movement. Compared with traditional gait or balance training or other rehabilitative interventions, dance is a safe, fun alternative way to achieve functional changes and improvements in mobility, gait, balance and QoL in PD. While understanding the independent effects of each supplementary intervention in the context of improving the symptoms of the disease, the current focus is on the holistic approach in managing PD by using a combination of exercise modalities, complementary therapies along with medical management that is most likely to be optimal for patients with PD. Hence it is essential to find out how the different management options act together to provide the best possible outcome for the patients. Improving the awareness of the clinicians and therapists to explain and direct the patients for the most effective combination of management options is vital.

References

1. Earhar, G.M. Dance as therapy for individuals with Parkinson disease. *European journal of physical and rehabilitation medicine*,2009; 45(2), 231.
2. Mak MK, Wong-Yu IS, Shen X et al. Long-term effects of exercise and physical therapy in people with Parkinson disease. *Nature Reviews Neurology*,2017 13(11), 689.
3. Roeder L, Costello JT, Smith SS et al. Effects of resistance training on measures of muscular strength in people with Parkinson's disease: a systematic review and meta-analysis. *PLoS One*,2015 10(7), e0132135.

Life and home adaptations for patients with Parkinson's disease: Occupational Therapy perspective

Mr Nandana Welage

With advancement of medical treatment, symptoms of Parkinson's disease (PD) can now be controlled for a longer period. However, PD remains a progressive disease that gradually limits activities of daily living (ADLs), income, social and leisure activities. Many patients thus, eventually refrain from participating in social activities and become isolated.

Occupational therapy in general, focuses on improving or maintaining the level of meaningful activities and providing necessary therapeutic support to increase social participation. In PD, Occupational Therapist (OT) enables the person to participate in ADLs by working with them to improve their ability to engage in tasks they want to, need to, or are expected to do. In many cases, this may require modifying an occupation (task) or the environment to better support engagement.

Frames of reference

Several frames of reference (approaches) are used by OT to design treatment protocols. Commonly used frames of references for patients with PD are explained below:

- Learning frame of reference: an approach to educate the patient about the management of their condition. This has two approaches; (a) cognitive approach which provides advice on time management and energy conservation, and (b) behavioral approach which provides anxiety management techniques for both the patient and caregivers to cope with everyday challenges.
- Compensatory frame of reference: used when progressive nature of the disease lead to reduced function and requires compensation. This also has two approaches; (a) adaptive approach which provides behavior modification and rearrangement of physical environment, and (b) rehabilitative approach which provides alternative support in care assistance or use of appropriate assistive devices and equipment.
- Development frame of reference: an approach to assess sequential gross and fine motor skills individualized and guided by OT.
- Biomechanical frame of reference: an approach based on improving physical abilities such as joint range of motion, strength, and endurance

when necessary. This approach is helpful in mobility training.

Assessment and intervention

During assessment, OT gathers information on ADLs and concerns of the caregivers. Based on the findings, meaningful goals are set, appropriate frames of references are identified and often “Hoehn and Yahr Staging of PD” are used when planning interventions.

Hoehn and Yahr Staging of PD

Stage One:	Symptoms mild
	Usually presents with tremor of one limb
Stage Two:	Symptoms bilateral
	Posture/gait affected
Stage Three:	Significant slowing of movements
	Early impairment of equilibrium while walking or standing
Stage Four:	Rigidity/bradykinesia
	Tremor may be less than earlier stages
Stage Five:	Cachectic stage
	Requires constant nursing care

For the purpose of clarity occupational therapy intervention is described below in two phases.

Initial phase

This phase includes first three stages of Hoehn and Yahr Staging of PD. OT generally aims to maintain functions and daily lifestyle as much as possible. Therapeutic activities are designed based on biomechanical, developmental and learning frames of references. Following are an elaboration of such.

Restorative activities

Individual and group activities are provided to restore or improve gross motor abilities such as limb mobility, trunk rotation, postural stability and upper limb function. OT also trains fine motor activities to improve finger dexterity and hand function. For example, if writing is impaired, patient is trained to master patterns of writing first, and gradually progressed to actual writing.

Personal care

OT advises to select (a) the type of garments comfortable and easy to take off

and put on while preserving self-esteem and dignity, and (b) the right footwear for comfort, support, and easy to wear.

Grooming

Patient is trained to brush hair, shave and apply makeup and to trim finger and toe nails. These activities encourage physical movements, coordination, dexterity, self-esteem and enable to retain control over the appearance. Advice regarding a suitable hairstyle, types of razors and suitable techniques of grooming activities are also provided.

Eating and drinking

In PD, the ability of eating and drinking is usually affected due to rigidity, bradykinesia and tremor. These symptoms slow down pace of eating, drinking and swallowing. Therefore, OT supports in maintaining correct posture and movements and advices on frequency of meals, using hands and cutleries.

Washing and bathing

Safe positions and arrangement of items to be kept in the washing and bathing area are advised. If measures are not safe, alternatives and some adaptive equipment are introduced.

Work

OT encourages patients to continue employment for as long as possible. Regular engagement of work is beneficial for maintaining physical and mental health. However, continuing work depends on tasks involved in that particular employment. If needed, energy conservation methods and workplace modifications are introduced.

Leisure

Patients are encouraged to maintain existing leisure activities and hobbies. Enjoyable activities enhance both physical and emotional wellbeing.

Later phase

This phase includes the last two stages of Hoehn and Yahr Staging of PD. The general aim of OT is to assist the patient and family/caregiver to modify and adapt lifestyle to meet the needs of the patient. Treatment is designed based on compensatory and learning frames of references. Following are some elaborations.

Remedial activities

Remedial activities are more closely related to skills of daily living. Activities are designed based on the interest and importance within the lifestyle of the person concerned. The objective of remedial activities is to preserve remaining movements, strength, and coordination.

Mobility

As the disease progresses, the patient becomes less mobile in and around home. OT ensures maintenance of existing movements and mobility to control symptoms such as rigidity and helps to structure the day so that activities which need more mobility are scheduled at the time when drugs display maximum effect. OT also rearranges home environment for patient to be safe and comfortable. At the very last stage, patient may be confined to wheelchair. OT will then assess and select the most suitable type of wheelchair and train caregiver to handle and maintain it. Patient may also need constant assistance to transfer from wheelchair to bed/toilet, in and out of car etc. OT trains caregivers on how to prevent back injuries while handling patients. Caregivers are also trained to use assistive devices such as walkers, wheelchairs, and hoists.

Home care and safety

As the mobility decreases, the tendency to fall will increase. Therefore, environmental adaptations such as railings, non-slip mats, walking aids are also introduced. Advice regarding lighting, floor coverings, and introduction of assistive devices for self-care are provided. As the condition deteriorates, patient will need constant support. An alarm call system may become useful.

Leisure

Patients are encouraged to pursue their interests and leisure activities as far as possible. If patient is unable to continue, OT helps to select alternative, creative leisure activities which stimulate and enhance happiness. Family members are encouraged to maintain their own leisure activities as they too should maintain optimum physical and mental wellbeing.

Conclusion

Evidence suggests that meeting an OT at early stages of PD is beneficial. When a patient is referred, OT conducts an in-depth assessment to identify functional problems and demonstrates ways to practice difficult tasks more easily or advice on using strategies, techniques, gadgets, or equipment and new technologies. OT may also suggest some useful changes at work or home environment to promote and prolong health and wellbeing of the patients and their families.

References

1. Dixon L, Duncan DC, Johnson P et al. Occupational therapy for patients with Parkinson's disease. 2007 Cochrane Database of Systematic Reviews (3).
2. Foster ER, Bedekar M, Tickle-Degnen L. Systematic review of the effectiveness of occupational therapy-related interventions for people with Parkinson's disease. *American Journal of Occupational Therapy*, 2014; 68(1), 39-49.
3. Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. *Neurology*, 1967;17: 427-442.
4. Parkinson's UK. (2015). Occupational therapy and Parkinson's. <https://www.parkinsons.org.uk/information-and-support/occupational-therapy>
5. Turner A, Foster M, Johnson SE. Occupational therapy and physical dysfunction-principals, skills and practice. (4th ed.) 1996. New York: Churchill Livingstone.

Speech and Language therapy in Parkinson's disease

Ms Saumya Rathnayake

Parkinson's disease (PD) is a chronic and progressive neurological condition, which could give rise to many difficulties including speech, language, and cognition and swallowing problems. Understanding of the pathophysiology of these impairments has evolved over time. In this article one of the commonest frameworks in disability, "International Classification of Function, Health and Disability" (ICF) (WHO, 2010) model will be explained in terms of how it's used in Speech and Language therapy for people with PD using two case study examples.

The ICF model explains the dynamic interaction between the domains of body structure and function, activity and participation (figure 01) and how those domains are affected by health conditions and environmental factors.

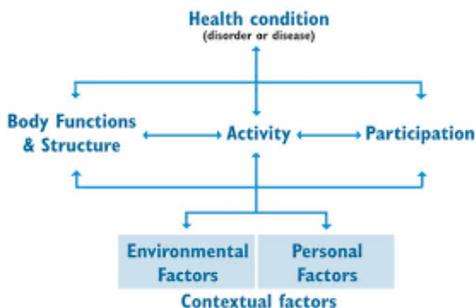


Figure 1: ICF model

Based on ICF model (Figure 1) the health condition in PD could be identified as dysfunction of the basal ganglia, caused by degeneration of dopamine-producing cells in the substantia-nigra (Keus et al, 2014). "Activity limitations" and "restrictions in participation" specific to communication and swallowing were described as impairments in the cognitive and mental functions, voice, speech and language functions, impairments in neuromusculoskeletal and movement-related functions, digestive functions and sensory functions (Keus et al 2014).

Activity limitations and restrictions in participation include deficits in communication, acquiring new knowledge, emotional support, ability to cope with stress, relationships, family dynamics, self-care including eating

and drinking. Environmental factors are specified in to limitations in five main areas as Products and technology (e.g. assistive devices, access to homes and public buildings and or financial assets), natural environment and human-made changes to environment (e.g. population density, climate) support and relationships (e.g. family ,friends ,health professionals) Attitudes towards PD(social attitudes and norms individual attitudes and beliefs)and lastly availability of services, systems and policies (social support, health services and education). This is a domain that could easily be changed and improved if effective multi-disciplinary management was implemented rather than changing the health condition. Finally, personal factors such as motivation, education level, age and gender could also contribute to PD disability level.

Determining the treatment for PD will depend on careful evaluation and focus on all above areas as PD is a multifactorial disease, where medication or treatment of one entity alone will not be helpful in the long-term management. A study done by a group of health professionals who developed a MDT method called “Parkinson’s Net” among 699 PD patients and health professionals such as nurses, Speech therapists, Physiotherapists, occupational therapists and Neurologists, showed interesting results in treatment outcome . Parkinson’s Net was developed based on a similar principle to the ICF model aiming to increase communication and networking among PD patients and Health professionals. Results of the study observed that the quality of care increased while satisfaction of patient and health professional were also increased. The importance a holistic approach in speech therapy is always emphasized and the following examples will show how to identify aims and treatment areas based on ICF modal in speech therapy.

Case Study 1

Mr Jamal is 62 years and diagnosed with PD two years ago. Currently, he receives therapy for his speech and language difficulties. According to the ICF model, based on Body Functions and Structures domain, he has got hypokinetic dysarthria with rigidity of articulators, tremor of lingual movements, poor respiratory support for sentence level expressions and decreased pitch variation with moderately affected speech intelligibility. Apart from this, word recalling difficulties with perseveration at times were present. Many patients with PD will experience symptoms similar to Mr Jamal, where issues in voice quality, articulation, volume, pitch, prosody and intelligibility of speech are common.

Activity participation limitations that were reported were as follows: Increased

social isolation, frequent communication breakdown due to poor speech intelligibility and word recalling issues, difficulty in speaking over the phone or in a noisy environment, and having difficulty in using voice after physical exercises. This led to restricted participation in social events such as community gatherings and family occasions. He tends to spend more time alone and is dependent on others for activities such as banking, shopping.

Treatment of Mr Jamal was focused on improving his communication skills while reducing restricted participation. Listed are few of the approaches that was incorporated into Mr Jamal's therapy.

1. Principles form Lee Silverman approach (based on improving the voice quality by intentionally using the voice in higher volume with clarity), Ramio et al. 1994
2. Articulatory kinematic approaches(looking at specific articulators and its range, speech and accuracy of movements, Wong et al., 2012)
3. Subsystems approach (identifying which subsystem to prioritize for speech production, Ramio et al, 1994)

Although Mr Jamal does not require any alternative and augmentative communication (AAC) support, many patients with severe PD with poor intelligibility of speech skills might require supported AAC systems. There are high techno (such as computer-based applications) and low techno (picture board, writing boards) based AAC systems (Ramio et al,1994, Pinto et al, 2004)

Apart from above approaches, the theoretical model discussed by Morris & lansek (1990) has been met with positive experiences in many Parkinson's centres. This model consists of the following five basic assumptions and these principles can be used with all PD patients.

In Parkinson's disease,

1. Normal movements are possible and what required is suitable activation.
2. Any complex movement needs to be broken down into smaller components prior to execution of the movement.
3. Each little segment of movement should be preferment at a conscious level.
4. Use an external clue to initiate and maintain movements.
5. Avoid simultaneous motor or cognitive tasks.

Communication partner training, Group therapy and specific community participation oriented goals could also be used to improve the function of social

and community participation. Family members and friends will be invited for speech and language therapy sessions.

Case Study 2

Mrs Seetha, a 54 years old housewife, was diagnosed with PD and she was referred to speech therapy. During initial assessment, it was observed that Mrs Seetha has dysphagia and is at risk of developing pneumonia. Neither she nor her family was aware of the consequences of dysphagia. However, her daughter reported Mrs Seetha had lost 12 kg of weight during last three months.

During the assessment of dysphagia, therapist conducted a detailed clinical assessment as well as requested Fibro Endoscopic Evaluation of Swallow (FEES) which is a common instrumental dysphagia assessment used in speech therapy. Based on results of these assessments Mrs Seetha, had difficulties in the oral level of dysphagia, as she chews food too long (hypokinesia) and/or kept food in her mouth without swallowing it (due to akinesia). She also had poor sensory awareness and had delayed swallow trigger. These symptoms are commonly seen in many patients with PD (Deane et al,2001).

The oral level difficulties can be treated with motor regionalization principles, where the patient was explained and taught to perform the process in conscious steps by using specific cues ((Hartelius and Svensson, 1994). Seetha also had aspiration for thin liquids and she was explained how to modify food with different swallow manoeuvres. She was given a specific meal plan to continue. Although there was no requirement for alternative feeding methods for her if a patient found to have aspiration for all food consistencies and/or if he or she is unable to eat and drink safely with usual techniques, such patients will be recommended to continue with alternative feeding methods. Apart from swallowing food, many PD patients will have issues in managing saliva and various medication and therapeutic methods could be used to manage that issue (Manor et al, 2007).

In conclusion, Speech and Language therapy in PD is managed at both individuals well as in participation level. The ultimate goal in treatment is to minimize barriers in active participation while increasing the opportunities to communicate within the environment, which leads to increase the QoL of the individual.

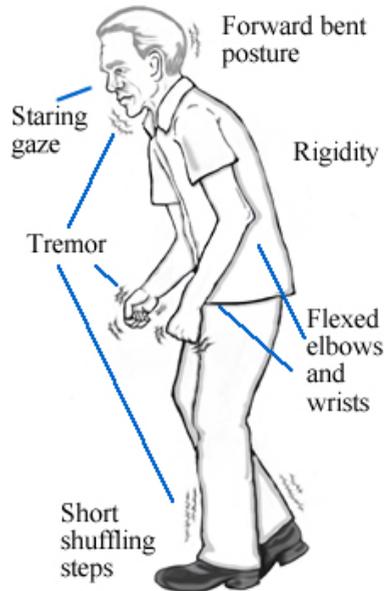
References

1. Deane KH, Whurr R, Playford ED et al. Speech and language therapy for dysarthria in Parkinson's disease, *Cochrane Database Syst Rev*,2001;(2):8-12.
2. Hartelius L, Svensson P. Speech and swallowing symptoms associated with Parkinson's disease and multiple sclerosis: A survey. *Folia Phoniatr Logop*;1994;46(1):9-17
3. Manor Y, Giladi N, Cohen A et al. Validation of a swallowing disturbance questionnaire for detecting dysphagia in patients with Parkinson's disease,*Mov Disord*, 2007;(13):1917-21.
4. Morrison EB, Rigrodsky S, Mysak ED. Parkinson's disease: speech disorder and released infantile oroneuromotor activity. *Journal of Speech, Language and Hearing Research*.1990. 13(3):655.
5. Munneke M, Nijkrake MJ, Keusetal SH, "Efficacy of community-based physiotherapy networks for patients with Parkinson's disease: a cluster-randomized trial," *The Lancet Neurology*, 2010(9). 46–54.

Living with Parkinson's disease

Dr J.B. Peiris

The first challenge in living with Parkinson's disease (PD) is coping with comments and inquisitive queries of friends and relations. Comments usually include the symptoms you are trying to hide or ignore. Comments may be direct and embarrassing like 'why aren't you smiling' or 'why are you drooling', 'why is your hand shaking', 'why are you bending and shuffling'. Next you start feeling sorry for yourself with thoughts like 'why me', 'is this the end', 'will it be painful', 'will I be able to walk, talk and eat,' will I have to suffer', 'how long will I last', will I be shaking all the time etc. These are difficult questions to answer but the patient has to be told that all is not lost and it is likely to be better than what he has seen in others in the past. It is imperative for the physician to lighten his anticipated fears about the illness. For a start the patient can be told that it is not as bad as a cancer and that there is medication to alleviate most of the symptoms for many years, and that is really due to insufficiency of a neurotransmitter which can be replaced with oral medications.



Having PD, you could still have many years of near normal life. A good example, is Pope John Paul II, once dubbed God's Athlete for his physical presence, who was able to perform his duties for a decade after the diagnosis was made. So did Sir Roger Bannister, the London neurologist famed for being the first man to cross the 4 minute barrier for the mile and Muhammed Ali (Cassius Clay) who inadvertently brought it on himself by subjecting himself to repeated head trauma in the boxing ring. Unfortunately, most lay people identify PD with the most severe form of the 'shaking palsy' they have seen.

The severity and interference with daily activities varies widely from person to person. There is much you can do to proactively affect the course of your PD and live a full, happy and healthy life. PD can be considered a deficiency

disorder more common with advancing age. Parkinson's affects everyone differently, and there are treatment options for the main symptoms of poverty and slowness of movement (bradykinesia) tremor, rigidity and postural instability, manifesting as unsteadiness and falls. Parkinson's subjects are able to execute voluntary movement, especially suddenly as demonstrated by their ability to catch a ball thrown to them.

Treatment options

The breakthrough in treatment was the production of the neurotransmitter laevodopa, (the deficiency of which in the extrapyramidal system is responsible for the symptoms) now sold in combination with carbidopa or benserazide, to reduce unwanted effects. It is important for the patient to be told that the cerebral cortex is relatively unaffected. The reader is encouraged to watch the film 'awakening', available on you tube, where patients who were frozen and staring vacantly into space, started dancing after treatment with L. dopa. Stereotactic brain surgery was performed to alleviate tremor but has now been replaced by deep brain stimulation.

Physiotherapy and rehabilitation

The basis is to attempt and do what you are unable to do. Basically the difficulties encountered

- Difficulty in initiating – initiating walking can be assisted by commencing lifting foot off the ground.
- Dragging of feet can be prevented by lifting the feet off the ground and taking longer strides
- Slowness of walking can be 'prevented' by attempting to walk faster

Overall, trying to imitate the 'march' of Hitler's army helps- long strides, high leg lift, and a free arm swing.

Medications and other treatments

Parkinson's is more than just a movement disorder, and the non-motor symptoms can often be more debilitating than the tremors or dyskinesia. Stress increases the symptoms of PD. However, symptoms come and go at varying intensities, so what may be causing you to have a bad day today may disappear tomorrow.

Drug treatment

Drug medication is best commenced by a neurologist. Main drug used is a preparation of levo-dopa. Generic preparations are cheaper and effective.

There are a few newer drugs which are indicated in special instances like intolerance, side effects or lack of response. The treating physician will decide on when to commence drug treatment, commencing with a small dose and increasing gradually as necessary. You are allowed to adjust the dose to suit your needs – the amount you need at the time you need, bearing in mind unwanted effects. The drug can be taken before, with or after meals depending on tolerability.

Non pharmacological treatment

Generally speaking, you may not need psychiatric or psychological assistance but you will benefit from the input of a well meaning spouse, friend or relative, to have a positive frame of mind to cope with changing or even progressive symptoms.

Practical tips for day to day activities

Standing – erect posture

In assuming erect posture you have to correct all the abnormalities shown in the figure, particularly the bent posture by

- Straightening head and neck
- Holding shoulders back
- Straightening spine and arms

When assuming erect posture from lying or seated, do it slowly with help, if needed. Prop yourself with arms on chair or bed, bring your buttocks to edge, lean forward and push up with hands to standing position. Stand firmly and steady yourself, before attempting to walk.

Walking

Adopt the exact opposite of the current walk –viz: longer, quick, high steps instead of the short, slow, shuffle. Both hands must be free when walking. Loose items on the floor like rugs and wires can be dangerous.

Talking

Learn to talk slowly and louder. It may help to record your speech and plan how you can speak slowly and loud.

Writing

Writing becomes smaller with time. Correct this by writing big letters in script form, letter by letter. If helpful, use a copy book.

Sleeping

Do not sleep bent up, which will accentuate bent posture when standing.

Eating/ Swallowing

Chew your food well and eat slowly. Avoid talking while eating, as you may choke.

Making the food mushy or semi solid would help if swallowing is difficult.

Washing and Shaving

May be easier while seated and support with elbows

Bathing

Showering is better than trying to get in and out of bath tubs. Here too consider doing it seated.

Driving

In the early phase, even for several years, your movements and reflexes may be unaffected and you may be able to drive, but it would be prudent to avoid driving without help.

Toileting and Grooming

Propping the elbows on a table while seated may help brushing teeth and combing hair.

Getting in and out of bed

Bend the knees up, turn the head in the direction of getting up, reach across the body with other arm, swing the legs over and sit up.

Conclusions

After months or years you will understand the disorder and the limitations better and learn to cope with it and live actively with it for many years. Obey the instructions of your physician and therapist. Have a positive attitude following examples of those coping well with the disease.

Parkinson's patients and care givers

(Persons who give all round help and protection and who know something about all aspects of Parkinson's disease)

Prof. Arjuna Aluwihare

There are many matters - emotional support, physical help, entertainment, cleaning, financial help (with banks etc) and/or support- all with variables; this note has a mixture of personal knowledge and information from others! It is vital to state at the outset, that the patient must have the freedom to do whatever they want whenever they want and not feel controlled or policed- subject to safety, the stage of the disease and important timings.

The care giver may be a spouse or a child who can hug and kiss or an employee who may or may not reside in the same premises day and/or night or both. They can give encouragement and support, talk politely without 'strange' and patronizing accents like talking to someone incapable of understanding (patronizing and 'scolding' tones do not help- any carer anger has to be with the disease not the patient!) and carer can avoid giving third party messages by sign or voice language- all of which are sensitively picked up by the patient who feels something is being done 'behind their back' and may get angry and difficult -needing lengthy communication, affection and encouragement until this hostile feeling is reversed and trust reestablished. The relative has the advantage of using hugs and such like to help.

The caregiver is physically and emotionally helpful to the patient, she helps with communication, feeding, visits to toilet (without risk of loss of dignity of the patient- such as can be caused if a toilet door is not shut!), washing and bathing, sorting of dressing, help with packing and planning for any trips, bedmaking and keeping the bedroom clean, etc. I reiterate, the type of care giver will vary depending on the family situation and the extent of the illness. As the illness gets worse more fulltime/professional support may be needed. A lot of common sense and self control is needed by the carer, and the ability to be happy to be helping is vital. The PD patient also in their better moments must be encouraged to appreciate the work of the care giver and help by doing things alone (safely!) and support caregiver breaks.

Positive activities help. Even just being or doing things together- games, jigsaws, listening to music, watching TV or visits to friends'/ relatives' (or inviting them

home) help. Seeing children or grandchildren is always a help, or even talking to them by telephone, skype or visual 'Whatsapp calls'.

Caregiver respite for short periods or longer so that he/she can do other work, go out etc, and give the patient respite from caregiver is valuable. A semi-isolated rest area for the carer with access to toilet, television and refreshment, can help the caregiver and patient 'rest' from each other and relaxation. Even corners of a garden can help. The caregivers may also find a caregiver type group-to help avoid feeling 'isolated and being uniquely stuck in a corner. In such groups or with others she needs to be cautious to avoid talking negatively about 'her' patient- this is a breach of confidence and trust- the disease one can complain about! Being a carer is stressful but this is much reduced by being aware of the satisfaction of doing it and if some free time is found to do other work, and when one remembers the satisfaction it gives the patient and shown by the patient.

All this helps the persons involved to be patient and relaxed when possible. The patient reacts favorably to patience and vice versa. The caregiver must take a day at a time and also both patient and carer can make the most of each day without 'hangovers'. One way this be shown is by a reminder to the patient how much she can do and anyway to do whatever is possible and view this as an achievement.

The mobility, ability to do other activity (eg sewing, cooking), motility and tremors, sleep disturbance with dreams, drowsiness, vary in a day in PD and one can watch them change rather rapidly as time goes on. For a long time many things are possible- but done more slowly. Even if the patient cannot physically do something they can often remember how things can and should be done and advise others, encouraging this is useful (they spot errors on other people's part). Much can be achieved by encouraging what is possible and asking, not ordering, and suggesting different ways of doing things (walking with high steps or even sideway, or with some articulated rhythm) An example of this is when the 'shuffler' has to climb stairs, they are slow but free of shuffle as a different part of the brain helps. There are episodes of rapid foot movement- especially in evening and if tired- and the carer must be there and walking stick MUST prevent a fall. If a fall occurs it is worth checking limb and joint movement before rushing to pick the patient up to make sure no bone might be injured, which would be excruciatingly painful.

Eating/feeding varies depending on tiredness, attractiveness of food, hunger and the stage of disease. If tired or if the patient has had a dream or woken too early or too fast there can be episodes of irrational behaviour and apparent hostility to the carer. Anything dangerous (like pouring boiling water) has to be prevented and forbidden but as far as possible with words of affection and gentle directions and sometimes physically with reasons given to the patient- hugs where possible- rather than scolding.

Urine control varies as in anybody but timing is important. Bowel functions with variable constipation depending on diet, fluid intake, exercise levels, and timing. Level of consciousness is usually good but mixed with periods of hallucinations (usually visual sometimes auditory) or variable interpretation of talk, memory and clarity of what is said. These hallucinations or imaginary people do not reduce their ability to detect patronizing or insulting behaviors or comments by others.

The Parkinson's drugs (especially dopamine type combinations) have side effects like hallucinations and dyskinesic movements which may need other medications. Medication may also include supplements and blood pressure or cholesterol pills. Timing of these is important and the patient or carer may have to use a diary to keep the timings and details of tablets written down and an alarm. A dosette box can help. Delaying a drug can aggravate stiffness, tremors, gait and feeding ability. Imagination is needed to facilitate the tablet use.

Doctors' visits and feedback to doctor- should be done as instructed and if the spouse or grown up children cannot go with patient, the carer should do so that he can communicate back and forth. This is as or more important than the matter of prescription renewal! A holistic view of the patient is vital. The doctor communication needs to be multidimensional about the medications, symptoms and signs, hallucinations, dementia like symptoms, what makes the patient better or worse and the carer has to ensure this!

The timing of meals and resting times is also very important for enjoyment and avoiding confusion. The meals and timing need to be supervised, regularized and help may be needed with feeding orally (or in more advanced patients by tubes). It is very important that the food has enough protein and roughage including green vegetable and fruits and adequate

but not excessive calories. Fluid intake must be adequate (tea and coffee if too much lead to fluid loss) or there will be dehydration- constipation being part of this. Water may have to be warm for drinking and mouth washing. These patients tend to collect phlegm or food particles between the voice box and upper end of the swallowing passage and mouth washes or throat gargles with warm salt water may be necessary after meals and other times to help clear phlegm. Another trick to help phlegm (and also tablets) is to eat a small piece of banana or papaw as the phlegm gets swallowed with this. It is also relevant to mention that a small bowl into which the patient can spit should be available, with some tissues.

Exercise- this is almost certainly part of daily routine and will involve carer reminders and help. A physiotherapist may visit from time to time and can advise on physical help and exercises. Also walking or cycling machines may be used. Walking the patient, moving around in the house and garden as much as and for as long as possible is a vital part of the carer's role. The use of various walking routines is useful such as a reminder to take slow high deliberate steps or even sideways if the patient is stiff or shuffling, or pretending to walk on stairs. Stairs should have a stable handrail. It is wise to use a walking stick as difficulties increase, for support, especially at times- often evenings when unpredictably rapid shuffling movements take place involuntarily, and at times when this is known to happen the carer should be with the patient. The patient MUST always have a hand free to help grab a support if need be and a walking stick in the other hand. Carrying things around must be discouraged as the patient may try to protect what is carried rather than looking for urgent support- and then fall onto the floor- risking a fracture or a bruise.

The quantity of rest and number of rest periods and their timing will vary as the condition progresses in order to optimize function in terms of quantity and enjoyment. The nights' sleep should be as continuous as possible depending on bladder function, the use of 'staydry' underwear or diapers, the night carer availability and -if essential- medication timing.

Towards the end more complexities arise- carrying may be needed. Feeding may need nasal tubes or peg gastrostomies. Urine control may proceed to night pads, bedpans and later may be to catheters, bowel issues may end with using aperients, soluble enemas or even digital disimpaction.

An important reminder restated- on caregivers relationship with spouse, family and patient. Whoever the care giver is she has to remember not to overreact to what the patient may say or do as apparently irrational, confusing or hostile actions are usually part of the illness (one needs to remember that patient does not enjoy the illness and being fed up with it may produce various reactions). A good caregiver is a godsend. Love, patience and encouragement are keys to good care for both patient and family- rebounding on caregiver(s)!

