Physician Guide

Non-motor symptoms of Parkinson's Disease

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Disclaimer

The goal of this booklet is to provide a practical simple overview of non-motor manifestations and their treatment. It is not intended to be a comprehensive guide, and some potential treatment options for some manifestations are not discussed. Neither does each treatment apply to each patient – this guide cannot be used as a substitute for clinical judgement. Finally, the field of non-motor problems of PD is rapidly changing – much of this advice may change – information can be considered current to approximately September, 2012.

Acknowledgement

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Introduction

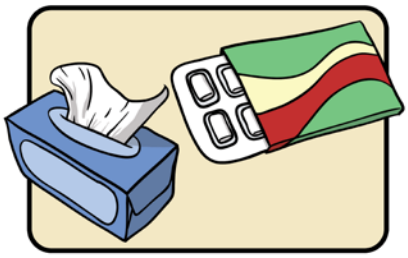
Parkinson's – Not just a motor disease

Parkinson's disease (PD) is classically considered as a motor disease, with tremor, rigidity, bradykinesia and gait problems as the classic motor features. However, non-motor manifestations (NMM) of PD have become increasingly recognized – they can often be more disabling than the motor symptoms. Non-motor manifestations of PD result from neuronal degeneration in widespread areas of the brainstem. Unfortunately, NMM are often under-recognized, and therefore, undertreated. The goal of this booklet is to provide a guide to recognizing and managing these NMM, so that quality of life of your patient with PD can improve.

Most NMM fall into one of three major categories: autonomic dysfunction, cognitive and psychiatric symptoms, and sleep disorders. They can occur throughout PD; some of them, such as olfactory dysfunction, constipation, depression, and rapid eye movement sleep behaviour disorder (RBD) can precede the motor symptoms of PD. Others, especially cognitive symptoms such as hallucinations and dementia, tend to occur late in PD.

How to screen for Non-Motor Problems

A clinical tool that was developed to screen for NMM in PD is the NMS-Quest. It consists of 30 yes-no questions each aimed at identifying a different non-motor symptom. This booklet addresses each of the NMM in the NMS-Quest. They are presented in order of their appearance on the questionnaire. A copy of the NMS-Quest is supplied at the back of this booklet. The NMS-Quest is a tool designed to be used in the office setting – for example, in our clinic, patients fill out the NMS-Quest in the waiting room before their visit, so that it can be reviewed during the visit. In our clinical experience, this saves both time (since we do not have to ask about symptoms that are not present) and helps us pick up important treatable manifestations of the disease. You are encouraged to copy the questionnaire and use it for patient care.



Drooling

What is the prevalence? Approximately 10% of patients

What do patients experience?

When mild, drooling usually occurs during the night or at mealtimes, but as it progresses it can occur any time during the day. Other than the social disability involved, serious complications can include choking on saliva and aspiration pneumonia.

Why does it happen?

Drooling is mainly due to impaired mouth movements and swallowing rather than saliva overproduction. It is therefore primarily a manifestation of the akinesia of PD.

Possible Treatments:

Non-pharmacological:

Advise the patient that eating chewing gum or a hard candy often decreases drooling by triggering increased spontaneous swallowing movements.

Pharmacological:

1. Anticholinergics:

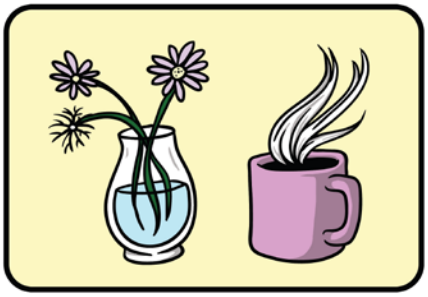
a) Atropine: Try 1-2 drops once per day of 1% atropine (anticholinergic) solution under the tongue to decrease saliva production.

b) Glycopyrrolate: 2-3 doses (total 1-2mg/day).

Potential side-effects are similar to the use of any anticholinergic agent: mouth dryness, blurry vision, constipation, urinary retention, and worsening of hallucinations and memory problems. However, these side effects are uncommon at the doses mentioned above. Anticholinergics should be used with special caution in patients with dementia.

2. Botulinum Toxin Injections: Botulinum toxin inhibits the cholinergic parasympathetic and the postganglionic sympathetic activity of the salivary glands, thus reducing saliva production. The parotid and/or submandibular glands can be targeted. The parotid gland is easiest to localize, although caution must be given to avoiding the facial nerve. Often, we start with injections to just the parotid gland (typically 30-50 Units of botulinum toxin A each gland), adding the submandibular gland (often with ultrasound guidance) in resistant cases.

3. Dopamine therapy: Since drooling is due to impaired mouth movements, it may improve with dopaminergic therapy.



Olfactory & Taste Dysfunction

What is the prevalence? Olfactory loss affects up to 90% of patients

What do patients experience?

Olfactory dysfunction (hyposmia or anosmia) and sometimes taste alterations occur in PD. Since these changes occur gradually, it is difficult for the patient to recognize them. Hyposmia often precedes the motor symptoms of PD, and therefore, it may be useful in the future as a screening tool to identify those at risk of PD. These symptoms are not serious, but in some cases they may cause decreased appetite.

Why does it happen?

Olfactory dysfunction occurs due to the degeneration of the anterior olfactory nucleus and olfactory bulb, one of the first brain areas to degenerate in PD. Furthermore, sniffing, which enhances olfaction, is impaired in patients with PD. Olfactory dysfunction may also be caused by smoking, rhinitis, head trauma, and other neurodegenerative conditions.

Fast Facts:

- Olfactory loss can predict PD
- Other "parkinsonisms" (MSA, PSP, etc.) may have intact olfaction, a feature sometimes useful in differential diagnosis

Screening:

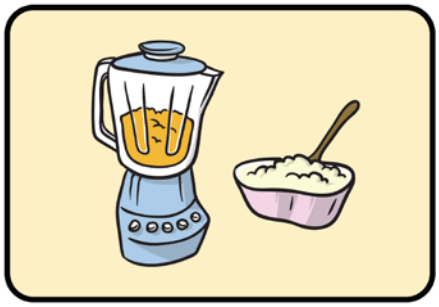
There are two principal options for testing olfaction.

1. UPSIT (The University of Pennsylvania Smell Identification Test). This test consists of 40 'scratch and sniff' pads (or 12 in a brief version) which release odours when scratched with a pencil; patients choose the correct odour from four available options. Some odours may be foreign to some patients, and this may affect their test scores.

2. Sniffin Sticks which are felt-tip pens infused with odours and are used to detect the patient's olfactory threshold.

Possible Treatments:

No treatments are currently available for olfactory or taste dysfunction in PD.



Choking and Swallowing Difficulties

What is the prevalence? Approximately 50% of patients

What do patients experience?

Although mild swallowing problems can be experienced early in the disease, severe dysphagia usually occurs only in advanced PD. The patient may have trouble swallowing food, liquids, or pills. Complications include malnutrition, aspiration pneumonia, and choking.

Why does it happen?

Swallowing difficulties are due mainly to dysfunction in the oral and pharyngeal phases. Oral-pharyngeal dysphagia may be associated with poor activation of tongue and cheek muscles, cricopharyngeal dysfunction, and incomplete relaxation and in coordination of the upper-esophageal sphincter. Most swallowing difficulties in PD are due to impaired bolus transport across the pharynx.

Possible Treatments:

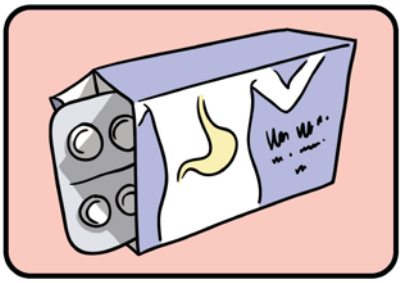
Non-pharmacological:

1. Advise the patient to avoid hard, dry foods that are difficult to swallow.
2. Ensure that the patient follows correct eating habits: proper posture while eating, smaller portions, clear mouth before speaking.
3. In some cases, increasing the patient's antiparkinson drug dosage may improve mouth movements and swallowing.

Special interventions:

Occupational/speech therapy. In most cases, patients with swallowing problems should be referred to an occupational/speech therapist for assessment and therapy. Barium swallow examinations are commonly performed to localize the deficit (oral, pharyngeal, esophageal), and to rule out alternative causes. Consider the referral to a gastroenterologist if the patient is more symptomatic.

A gastric feeding tube may be required in severe cases.



Nausea and Vomiting

What is the prevalence? Approximately 20% of patients

What do patients experience?

Patients can experience a sensation of abdominal bloating even without medications, or can present with nausea and vomiting when they start a new antiparkinson drug.

Why does it happen?

Nausea and vomiting can occur as a primary feature of disease, but are commonly a side-effect of dopaminergic and other antiparkinson medications. Patients often experience nausea and vomiting when they begin a new drug. A sensation of bloating is common even without medications and may occur due to decreased stomach movements – this is related to degeneration of autonomic neurons in the peripheral nervous system (Meissner's plexus) and brainstem.

Possible Treatments:

If due to a new medication, symptoms often disappear with time. Therefore, if symptoms are mild, additional medications may not be required.

Pharmacological:

Dopamine receptor antagonists: Domperidone. A dopamine antagonist that blocks dopamine's peripheral effects, domperidone does not cross the blood-brain barrier (BBB) and therefore does not interfere with dopamine's effect on the brain. This is very effective in the case of levodopa-induced nausea – typical dose is 10 mg with (or half hour before) each levodopa dose. Because of potential cardiac arrhythmia at high dose, maximal domperidone dose is 10 mg TID.

Change in dopamine therapy:

Taking dopaminergic medications with meals may reduce nausea, but this also results in decreased absorption of levodopa, potentially worsening motor symptoms.

> ***Fast Facts:***
> • Whereas domperidone can help nausea considerably, do not use dopamine antagonists that cross the blood brain barrier (e.g. Prochloroperazine/Stemetil, Metoclopramide).
> *They will worsen PD motor symptoms!*

Constipation

What is the prevalence? Approximately 75% of patients

What do patients experience?

Constipation is defined as fewer than 3 bowel movements per week or straining to pass stools. Constipation often precedes motor symptoms. Aside from the distress it adds to daily life, complications include megacolon, pseudo-obstruction, volvulus, and bowel perforation. Therefore, severe constipation should not be neglected.

Why does it happen?

Constipation is a symptom of dysautonomia, and is mainly due to reduced colonic motility and occasionally ano-rectal dysfunction. Peripheral and brainstem autonomic nuclei degeneration causes increased intestinal transit time and constipation. Parasympathetic cholinergic denervation can lead to sphincter dyssinergia, in which the co-ordinated relaxation of the anal sphincter is impaired, leading to inability to defecate normally.

Fast Facts:
- Constipation may predict PD
- One of the commonest NMM in PD

Possible Treatments:

Non-pharmacological:

Exercise & Diet is the first line of treatment for constipation.

1. Adequate hydration
2. Dietary fiber (bran, prunes, etc.)
3. Physical exercise

Pharmacological:

1. Bulking agents (Psyllium, Metamucil) and stool softeners (Colace 100 mg BID). In most cases, bulking agents are not sufficient, and stimulants/osmotics are needed.

2. Stimulant laxatives & osmotic agents:

a) Senokot: is a natural laxative available in tea or pill form.

b) Isosmotic macrogol solution/polyethylene glycol 3350 (1-3 standard doses/d) or **Lax-a-day** (17g/day)

c) Lactulose 30-60 cc BID

d) Lubiprostone 24 ug QD to BID

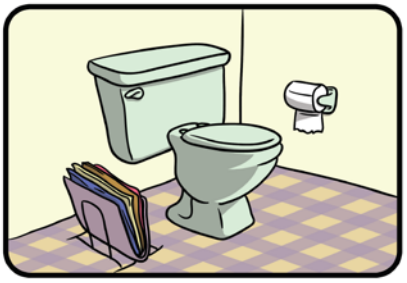
Note: Classic laxatives may lead to an atonic colon and cause electrolyte imbalance (hypokalemia). Although they are generally not recommended for extended periods of time, many patients nevertheless require daily laxatives.

3. Cholinomimetics: Pyridostigmine Bromide (30-60 mg TID-QID). Consider as the option to treat constipation associated with PD when patients also suffer from orthostatic hypotension, because it can treat both symptoms.

4. Suppositories (e.g. Glycerine) and **enemas** may be required in resistant cases.

5. Reduce or discontinue drugs with anticholinergics activity.

6. Add Domperidone.



Fecal Incontinence

What is the prevalence? Uncommon (less than 10%)

What do patients experience?

Fecal incontinence is the involuntary loss of stool. It is a relatively rare non-motor manifestation, and it is usually seen with urinary incontinence.

Why does it happen?

Fecal incontinence is most likely related to dysautonomia. Functional incontinence--inability to get to the bathroom in time because of motor/gait impairment--is relatively common in late disease.

Possible Treatments:

Pharmacological:

Fecal incontinence can be improved by antiparkinson medications (particularly if functional incontinence is prominent) therefore incontinence may be a signal that dopaminergic medications should be increased.

Non-pharmacological:

Discuss the use of incontinent products with the patient (e.g., adult diapers, pads, guards).

Bladder Dysfunction

What is the prevalence? Over 50% of patients experience some form of bladder dysfunction

What do patients experience?

A diverse range of bladder symptoms are experienced by PD patients. The commonest are related to detrusor hyperreflexia, including nocturia, urinary urge, urinary frequency, and incontinence. Urinary retention/detrusor hyporeflexia is less common. Mild bladder symptoms are common in early PD - incontinence occurs in more advanced PD. In some cases, it is difficult to diagnose the nature of the impairment (or combination of impairments) on history alone – if this is the case, consider urodynamic studies.

Why does it happen?

Bladder dysfunction is due to degeneration of autonomic bladder neurons, motor areas, and higher control areas. Furthermore, degeneration of the substantia nigra (SN), which inhibits urination, also leads to bladder dysfunction.

Fast Facts:

• Many men with bladder dysfunction are misdiagnosed as having prostatic hypertrophy. Any male patient must therefore be aware of the possibility that urinary symptoms are due to PD, to avoid unnecessary prostate surgery.

Possible Treatments:

Non-pharmacological:

General measures for treating urinary urgency and incontinence include avoiding coffee and limiting water ingestion before bedtime, etc.

Pharmacological:

A. Detrusor hyperreflexia (urgency, frequency):

1. Dopamine therapy: Levodopa generally improves detrusor hyperreflexia and urgency

2. Anticholinergics: The first line of treatment for an overactive bladder.

- **Oxybutynin** (5mg 3-4 times/day or 1 patch twice/week) – may have more central anticholinergic effects than the others in this class

- **Tolterodine** (2mg -3 times/day).
- **Solifenacin** (**Vesicare**) (5-10mg/day)
- **Darifenacin** (7.5-15mg/day)
- **Trospium Chloride/Trosec** (20-40mg/day)

Note: Anticholinergic medications such as these can worsen constipation, impair memory, and cause hallucinations, therefore be cautious when prescribing to patients with dementia.

3. Alternative treatments:

- **Botox injection (serotype A)** injection in detrusor muscle may reduce bladder overactivity.

B. Nocturia:

Desmopressin nasal spray (10-40mcg/night nasal spray) - decreases urine production. It can also be used to treat orthostatic hypotension. Caution should be given to avoiding excessive fluid intake when taking desmopressin. Drinking too much water causes decreased sodium in the blood (rare) and electrolyte imbalance.

C. Hyporeflexia (urinary retention):

Bethanechol Chloride (25-75mg/d)

Pain

What is the prevalence? Approximately 33-66% of PD patients experience pain directly related to PD

What do patients experience?

Pain in PD presents as stiffness, cramps, spasms, or muscle pain, usually occurring in the calves, neck, or back. Both primary PD pain and secondary pain exist in PD. Primary pain often occurs during off periods (i.e. when antiparkinson medications 'wear off' in patients who fluctuate). Pain can also be associated with dyskinesias and early morning dystonia. PD also can decrease pain thresholds, so that other secondary pain syndromes worsen in the presence of PD.

Why does it happen?

Decreased pain thresholds in PD can be due to the degeneration of dopamine-dependent centers that regulate pain inhibition. Norepinephrine degeneration in the locus coeruleus is also associated with pain in PD. Cramping, dystonia, and muscle rigidity due to the primary manifestations of PD can also be painful.

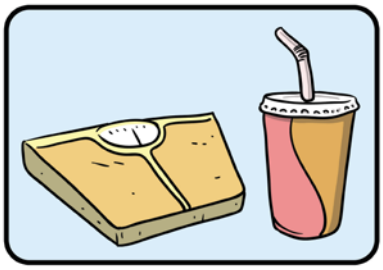
Possible Treatments:

Non-pharmacological:

Stretching, massage, a warm bath, and over-the-counter pain medications may help.

Pharmacological:

Adjust antiparkinson medication: Increasing dopaminergic therapy may help both primary and secondary pain in PD. If the pain is occurring during off periods, reducing fluctuations may be helpful. Therefore, pain may be a signal that dopaminergic medications should be adjusted.
Dystonic pain mostly responds to PD drugs or Botulinum Toxin.
Agents to treat orthostatic hypotension (for example, "coat-hanger pain") may be useful if the pain is linked to autonomic failure.
Antineuropathic treatment (Gabapentin, Pregabalin, Lamotrigine and tricyclic antidepressants) may be useful.
Tricyclic antidepressants or selective serotonin and noradrenaline reuptake inhibitors can be useful especially if pain is linked to depression.



Weight Loss & Weight Gain

Why does it happen?

Weight loss occurs in many neurodegenerative diseases, including PD. This weight loss is often independent of energy intake. Normally, the brain integrates information on nutrient stores and energy expenditure, and then alters energy intake. In neurodegenerative diseases, this process may be affected. In PD, weight loss is related mainly to a decrease in fat tissue. Factors such as dyskinesia, swallowing dysfunction, altered smell and taste, nausea and vomiting, or other medication side effects can contribute to weight loss.

Weight gain is much less common than weight loss. Weight gain may occur due to binge eating (an impulse control disorder), which is often a side-effect of dopaminergic agonists. Atypical antipsychotics such as quetiapine and clozapine are also associated with weight gain.

Possible Treatments:

Weight Loss:

Early Disease: Dopaminergic therapy adjustment

Late Disease: Oral nutritional supplements (e.g. Ensure, Boost) can often help. In rare cases, percutaneous endoscopic gastronomy may be required.

Weight Gain: If due to binge eating from dopamine agonists, reduction in dose may be helpful (see impulse control disorders section).

Cognitive Dysfunction and Dementia

What is the prevalence?

Up to 70 % of patients with PD will eventually develop cognitive impairment (either mild cognitive impairment or dementia)

What do patients experience?

Patients with early disease demonstrate subtle changes on neuropsychiatric tests of mental flexibility and executive function, but these changes are usually asymptomatic.
Parkinson's Disease Dementia (PDD) usually occurs in patients in later stages who are above 65 years of age. Predominant symptoms include bradyphrenia (slow thought process), impaired memory (due to retrieval more than encoding problems), impaired attention, visuoperceptual/ visuospatial dysfunction, and dysexecutive syndrome (poor planning, rigidity, etc.).

Why does it happen?

Lewy Body degeneration of cortical structures is the major underlying cause of PDD, but Alzheimer-like changes and vascular lesions may contribute in many cases. Probable risk factors for PDD include age (>65), hallucinations and delusions, family history of dementia, depression, advanced disease, and REM sleep behavior disorder.

Diagnosis:

Commonly-used screening tests for cognitive dysfunction include:

1. **Mini Mental Status Examination (MMSE):** a test score of 25/30 or less indicates with functional impairment is considered within the demented range in PD. Note that it is notably insensitive to the cognitive abnormalities of PD.
2. **The Montreal Cognitive Assessment (MoCA)** tests more completely the visuospatial and executive dysfunctions that occur in PD. Scores <21 may indicate dementia, and <26 indicate mild cognitive impairment.
3. **Other potential instruments include the Mattis Dementia Rating Scale (which takes 30 minutes to administer), the ACE-R, the ADAS-COG, CAMCOG, SCOPA-COG, etc.**

Ask questions if the patient can:
- Manage finances?
- Use equipment such as telephones and remote controls?
- Act appropriately in social situations?
- Take their medication independently?

Cognitive impairment is only considered dementia if it impacts the person's ability to carry out daily activities.

Possible Treatments:

Non-pharmacological:

1. Remain cognitively active.
2. Regular exercise: PD risk in humans is probably reduced by midlife exercise. No studies have addressed whether exercise influences dementia risk in PD, however PD patients who exercise vigorously may improve cognitive scores.
3. Healthy diet.
4. Control vascular risks: Blood pressure, Diabetes, Cholesterol levels.
5. Control other reversible factors: sudden onset cognitive impairment can be a sign of a superimposed acute medical process (sepsis, etc.).

Pharmacological:

Medication review: ensure there are no medications that cause cognitive impairment, such as anticholinergic medications (including tricyclic antidepressants), benzodiazepines, etc.

Cholinesterase Inhibitors: Rivastigmine **(Exelon)** (1.5-6mg BID) and Donepezil **(Aricept)** (5-10mg QD) have had randomized trial evidence of benefit in PD. Side-effects of cholinesterase inhibitors include nausea, vomiting, and diarrhoea (often these are less of a problem in PD patients who already suffer from constipation). Tremor can occasionally worsen with cholinesterase inhibitors, but this is rarely a practical problem.

Dopamine therapy is limited in treating PDD, but it may improve subtle cognitive deficits seen in early PD.

Pitfalls:

• If the dementia occurs at the same time or within a year of motor symptoms, the diagnosis is formally defined as **Dementia with Lewy Bodies**, rather than PD dementia (although these are very similar diseases and classification may change).
• Anticholinergics, tricyclic antidepressants and sedatives can worsen cognitive function and should be used with caution in PD patients.

Hallucinations

What is the prevalence? Up to 40 % of patients have visual hallucinations

What do patients experience?

Hallucinations usually occur in later stages of PD. Auditory (whispers, music), gustatory, olfactory, and tactile hallucinations are uncommon in PD, while visual hallucinations are more common. These often occur in low-light settings or when one's conscious state is altered (i.e. sleep/wake transitions). Initially, illusions (misinterpretations of visual objects) are common – for example, a spot on a wall becomes a bug. As they progress, they become less dependent on the environment, more vivid, and often threatening. Initially, the patient usually realizes that the hallucinations are not real, but eventually, they may not be able to distinguish between reality and the imaginary.

Why does it happen?

Degeneration of the visual and perceptual areas in the cortex is associated with hallucinations in PD. Hallucinations are often side effects of antiparkinson medications, but in advanced disease they can occur without dopaminergic medications. Other risk factors for hallucinations include cognitive impairment, advanced age, long disease duration, and depression.

Fast Facts:

• Abrupt onset of hallucinations may be a sign of an acute illness, such as infection, dehydration, or toxicity!

Possible Treatments:

1. **Look for triggers:** Infection, urinary retention/colonic obstruction, metabolic causes, etc.
2. If the hallucinations are not caused by an underlying medical condition, the following step-wise approach can be useful:

    a) **Taper or discontinue sedative medications:** Sedating antidepressants, benzodiazepines, and other medications that cloud the consciousness should be discontinued.

b) Taper antiparkinson drugs: Drugs with the highest risk of inducing confusion and psychosis should be reduced first. Taper antiparkinson drugs in roughly the following order:

i. **Anticholinergics**
ii. **Amantadine**
iii. **Dopamine agonists**
iv. **MAO-B inhibitors**
v. **Levodopa** (generally provides the best motor benefit for the least amount of hallucinations)

c) Cholinesterase inhibitors: **Rivastigmine (Exelon)** and **Donepezil (Aricept)** improve cognitive function, behavioural symptoms, and psychiatric symptoms. If there is coincident cognitive impairment, this may be the first-line treatment for hallucinations or psychosis. Side-effects include nausea, vomiting, diarrhea, and upset stomach.

d) Atypical antipsychotics: Clozapine (Clozaril) and **Quetiapine (Seroquel)** can be useful agents for psychosis. These are the most 'atypical' of the atypical neuroleptics.

i. **Clozapine** (12.5-25mg hs): The only atypical antipsychotic that does not worsen motor symptoms. Clozapine may, in fact, treat some symptoms of PD such as tremor or dyskinesia.

Side-effects: Sedation, dizziness, drooling, postural instability, orthostatic hypotension, weight gain, and **leucopenia (0.38%)/ agranulocytosis** (fatal, but rare). Therefore, a regular neutrophil count is necessary.

ii. **Quetiapine** (12.5-50mg hs): The effect on psychosis is less well-established, but may help in many cases. Side effects are similar to Clozapine, but leucopenia does not occur, saving the patient the need for regular blood tests.

Pitfalls:

• Do NOT use Olanzapine, Risperidone, or any 'typical' neuroleptics. They worsen motor symptoms and have many more adverse effects.
• Consider cholinesterase inhibitors if borderline dementia is present.
• Antipsychotics have been associated with increased all-cause mortality in large trials of dementia. The mechanism of this is unclear.

Depression

What is the prevalence? Approximately 20-50 % of patients

What do patients experience?

Depression precedes motor symptoms in approximately 30% of cases. PD depression typically differs from depression in the general population – there are less expressed feelings of sadness, little tearfulness or guilt, and a low suicide rate. On the other hand, there is prominent anxiety, anhedonia, and apathy. There are difficulties in diagnosing mild depression in PD because clinical features overlap with motor features of PD; hence clinicians should have a low threshold for diagnosing depression in PD.

Fast Facts:

- Depression can precede PD and is often found early in the disease.

Why does it happen?

Although there can be situational depression due to manifestations of disease, depression in PD is likely a primary disease manifestation. It may be due to complex dysfunction of numerous structures including noradrenergic, serotoninergic, and dopaminergic regions of the brainstem. Furthermore, mood swings and depression can occur with wearing off or during off periods.

Screening tools:

- **Beck Depression Inventory (BDI)** (cut off score 13/14)
- **Hamilton Depression Rating Scale (HDRS)**

Possible Treatments:

Pharmacological:

1. Especially if motor control is suboptimal, consider **dopamine agonists** such as **Ropinirole** or **Pramipexole**. There is evidence that dopamine agonists act as mild antidepressants.

2. **Tricyclic antidepressants: Nortriptyline (25-75 mg hs)** has been tested in trials using PD patients, and in some cases may be more effective than other agents. However, caution must be used in patients at risk for hallucinations, and cognitive impairment.

3. **Selective serotonin reuptake inhibitor (SSRI): Citalopram (20 mg/day), Venlafaxine XR** (up to a maximum dosage of **225 mg/day**) and **Paroxetine** (up to a maximum dosage of **40 mg/day**). These are the SSRI's medication that have been demonstrated as effective in randomized controlled trials of antidepressants in PD.

4. Other newer antidepressants such as Reboxetine, Mirtazapine, and Nefazodone may be effective but have not been studied.

Non- pharmacological:

Cognitive Behavioural Therapy (CBT): Although not studied in PD, this has been consistently shown as effective for depression in the general population, and may therefore be useful in PD.

Observation:

There is a theoretical interaction between all antidepressants and MAO-B inhibitors. This may not be clinically manifest – data on Rasagiline is lacking, but Selegiline has been used for over 20 years in combination with TCA's and SSRI's, with only very rare sporadic reports of serotonin syndrome. Nonetheless, this is a common cause of pharmacist 'flags', so it is important to warn patients about this.

Pitfalls:

- Treatment for PD-related depression may differ than in the general population.
- Dopamine agonists have antidepressant properties.

Anxiety

Fast Facts:
- Anxiety can also be a preclinical PD risk factor.

What is the prevalence? Reported in 30-40% of patients, frequently co-occurs with depression

What do patients experience?

Symptoms can include panic attacks, phobias, or generalized anxiety disorder. Patients often find they ruminate excessively on upcoming events. Anxiety may precede diagnosis of PD. In some patients, anxiety can be related to drug-induced motor fluctuations. When patients with PD have depression, anxiety is commonly a prominent component.

Why does it happen?

The cause of anxiety is unclear. Brainstem areas involved in mood and anxiety regulation degenerate in PD. The presence of anxiety during off periods suggests that dopaminergic denervation may be an important cause.

Fast Facts:
- Anxiolytic agents may be contraindicated in PD and others may worsen symptoms of the illness.

Possible Treatments:

Pharmacological:

The optimal pharmacological treatment for anxiety in patients with PD has not been established.
See depression section for details of antidepressant treatments.

If the anxiety is occurring during off periods, adjust dopaminergic medications to decrease off periods and motor fluctuations.

Pitfalls:

Benzodiazepine medications are often used for anxiety, but in PD, they can be associated with falls, ataxia, and cognitive dysfunction.

Apathy

What is the prevalence? Reported in 12-16 % of patients

What do patients experience?

Apathy is the lack of motivation, interest and emotion. The patient expresses indifference and has no goals for the future. Patients often note that they no longer enjoy elective activities. Caregivers often complain that the patient no longer participates in social or physical activity.

Why does it happen?

Degeneration of 'goal-directed' areas (frontal subcortical areas) or reward centers (dopamine projections between the ventral tegmental area and nucleus accumbens) may cause apathy. Both dopaminergic and cholinergic neuron degeneration are associated with apathy.

Fast Facts:

- Apathy is a distinctive symptom of PD independent of depression, somnolence, and fatigue.
- Apathy is characterized by isolated lack of motivation and initiative.

Possible treatments:

In general, apathy is difficult to treat, and measures are unlikely to be successful.

Non-pharmacological:

Education of patients and caregivers regarding apathy as symptom of PD different from depression or laziness, can be helpful to improve patient and caregiver distress.

Pharmacological:

Dopamine therapy: Occasionally, increasing dopaminergic therapy, particularly use of dopamine agonists, helps apathy.
Methylphenidate can possibly be useful, but utility may be limited due to side effects.

If the apathy is occurring during off periods, administer drugs to decrease off periods and motor fluctuations.

Sexual Dysfunction

What is the prevalence? Approximately 50% of patients

What do patients experience?

Sexual dysfunction (SD) in PD includes erectile dysfunction (ED), difficulty reaching orgasm, decreased libido, and decreased genital sensitivity. On the other hand, patients can occasionally have increased sex drive (hypersexuality), usually related to dopamine agonists. Sexual dysfunction is reported more commonly by men than women, perhaps because it is more easily identified in men.

Why does it happen?

Erectile dysfunction occurs as part of autonomic degeneration, with parasympathetic and sympathetic denervation. Sexual dysfunction can also be due to motor dysfunction, medications, or mood disorders. Testosterone deficiency can be implicated in some cases.

Aberrant sexual behaviour and drive including hypersexuality are impulse control disorders, which in susceptible patients is linked to dopaminergic drug treatment (see Impulse control disorders section).

Possible Treatments:

Pharmacological:

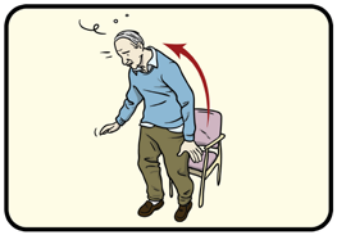
First line therapies for erectile dysfunction involve the phosphodiesterase inhibitors. These include:

1. **Sildenafil Citrate (Viagra)** 50mg-100mg before intercourse
2. **Vardenafil (Levitra)** 10mg before intercourse
3. **Tadalafil (Cialis)** 20mg before intercourse

Most second line therapies are administered by urologists and are outside the scope of this booklet.

Hormone Replacement therapy can help some women with certain sexual dysfunction. However, risk/benefit ratio must be carefully determined.

Hypersexuality: See impulse control disorders section.



Orthostatic Hypotension

What is the prevalence? Seen in 30-58% of patients

What does the patient experience?

Orthostatic hypotension (OH) is formally defined as a drop in systolic blood pressure by ≥ 20 mmhg or diastolic pressure ≥10 mmhg from supine to standing. Due to cerebral auto-regulation, many patients with OH are asymptomatic. Symptoms can include light headedness, fatigue, headache, shoulder-ache (coat-hanger pain) and cognitive slowing after standing up or occasionally after large meals. Falls and blackouts may occur if the OH is severe.

Why does it happen?

OH in PD occurs due to the failure of the baroreceptor reflex (both its cardiovagal and sympathetic branches) and to cardiac sympathetic denervation. Dopamine therapy may also cause OH.

> ***Fast Facts:***
> • With early and severe orthostatic hypotension, consider Multiple System Atrophy as an alternate diagnosis.

Possible Treatments:

Non-pharmacological:

1. Encourage water intake
2. Increase salt intake
3. Avoid big meals
4. Head of bed elevated at night
5. Compressive stockings

In practise, these measures can be complex in the context of motor disability, and are often poorly tolerated by PD patients.

Other recommendations which can be useful:

1. Advise the patient to stand up slowly and carefully.
2. Leg exercises before standing may prevent OH by bringing pooled blood back into circulation.
3. Dehydration should be avoided.

Pharmacological:

1. Re-assess antihypertensives: Because of orthostatic hypotension, the typical **24 hour** BP in PD 10 mm lower than in age-matched controls.

In general, medications for orthostasis have not been thoroughly studied in PD.
Options include:

2. Domperidone (10 mg TID): A peripheral D2 receptor antagonist blocks OH effects of dopaminergic therapy. It does not cross the BBB, and therefore, does not affect dopamine levels in the brain. It may be useful even in the absence of dopaminergic therapy.

3. Physostigmine (Mestinon) 30-60mg QID can help OH, and has the added benefit of treating constipation. Increased drooling and exacerbation of urinary dysfunction can occur.

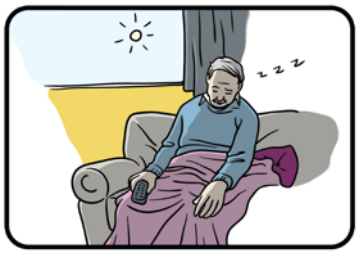
4. Midodrine (2.5-10mg TID): An alpha-adrenergic agent and a vasopressor. Like options 5 and 6, it can cause supine hypertension. Other side-effects include piloerection, scalp pruritus, paresthesia, urinary retention or urgency.

5. Fludrocortisone (0.1-0.3mg/day): A mineralocorticoid that treats OH by increasing renal sodium absorption and plasma volume.

6. Desmopressin (10-40ug nasal spray or 100-400ug orally at bedtime): Desmopressin treats OH as well as nocturia. It increases plasma volume by acting on the renal tubule V2 receptors.

Pitfalls:

• Midodrine and Fludrocortisone may cause hypertension in the supine position. In patients on dopaminergic therapy, Domperidone may be the first-line option.



Excessive Daytime Sleepiness

What is the prevalence? Approximately 50% of patients

What do patients experience?

Patients with EDS may fall asleep while driving, during a conversation, or in public places such as theatres. If the EDS is severe, the patient may experience sudden sleep episodes called 'sleep attacks'. Naps are often unrecognized by patients, so caregiver input is essential.

Why does it happen?

Degeneration of regulators of the sleep-wake cycle, particularly the reticular activating system and circadian rhythm generators, are likely responsible for much of EDS in PD. However, levodopa, anticholinergics, amantadine and especially dopamine agonists, can cause drowsiness. Although poor sleep at night can result in EDS, on average patients with EDS sleep longer and 'better' than those without, reflecting an overall increased sleep drive. Although sleep apnea is not clearly increased in PD, it is nonetheless a potentially reversible cause of sleepiness; however the connection between apnea and EDS in PD is unclear. The presence of comorbid medical and psychiatric disorders like depression and anxiety should be considered.

Possible Treatments:

Non-pharmacological:

1. A stimulating daytime environment and exposure to intense light during the day may be of use.
2. Regular mild exercise.

Pharmacological:

1. Review the entire medication list. EDS can be caused by antiparkinson drugs. Decreasing such medications (especially dopamine agonists, anticholinergics, amantadine) may help. Note that Selegiline is metabolized to amphetamine products, so theoretically can help EDS (although it can also be associated with sleepiness, like any other dopaminergic agent).

2. Caffeine (100 -200 mg BID)**:** Caffeine is commonly used for EDS in the general population. PD patients are often non-coffee drinkers, so addition of caffeine in tablet or beverage could potentially help EDS. According to some short-term studies, caffeine may also improve some motor and non-motor aspects of PD.

3. Modafinil (100-400mg/day): The only proven agent for EDS in PD. Modafinil is a psychostimulant with unknown mechanism of action. Side-effects include headache and nausea.

4. Atomoxetine (40-80 mg)**:** This selective norepinephrine reuptake inhibitor (SNRI) may have alerting effects in PD based on possible enhancement of locus ceruleus (LC) activity and, also, enhance executive function in patients with PD without dementia, according to recent preliminary open-label study.

5. Other stimulants (e.g. **Methylphenidate**), may be useful, but have not been well studied in PD.

Insomnia

What is the prevalence? Some insomnia affects up 60-80% of patients

What do patients experience?

There are two major types of insomnias:

Sleep-onset insomnia: trouble falling asleep

Sleep-maintenance insomnia: trouble staying asleep and waking too early

In general, PD patients tend to be more troubled by sleep-maintenance insomnia. Many patients notice that they have become increasingly 'early to bed, early to rise'.

Why does it happen?

Insomnia can be due to many causes. Motor symptoms such as bradykinesia, tremor, dyskinesia, restless legs syndrome frequently interfere with sleep. All antiparkinson medications can cause insomnia, particularly evening Selegiline, which has amphetamine metabolites. Neuropsychiatric symptoms such as hallucinations and delusions often disrupt sleep. Nocturia is common in PD. Finally, degeneration of sleep-promoting and circadian regions of the brain is an important cause of insomnia.

Fast Facts:

- Insomnia is the most common non-motor complaint.
- Sleep maintenance insomnia much more common in PD

Possible Treatments:

Non-pharmacological:

1. Sleep hygiene measures: These include having regular sleep hours, avoiding excess time in bed and daytime naps, having regular get-up time, using the bed for sleep only, scheduling time to relax before bedtime, being physically active during the day, ensuring adequate sun exposure, making the bedroom quiet, dark, and comfortable, minimizing stimulants during the evening, and avoiding large evening meals. Sleep hygiene may be especially useful when used with other strategies.

2. Cognitive behavioural therapy (CBT): Although not studied in PD, CBT is a proven and highly effective treatment of primary insomnia. Goals include alteration of patient's dysfunctional beliefs and misconceptions about sleep and insomnia. CBT also helps to reformulate anxiety-provoking thoughts regarding sleep. Different approaches can be used to improve sleep including sleep hygiene training, relaxation training, stimulus control, and sleep restriction.

Pharmacological:

1. Assess if medications are a cause. If Selegiline is being taken in the afternoon or evening, move it to a.m. and noon. Sedative drugs that cause drowsiness during the day should be avoided.

2. Nonbenzodiazepine cyclopyrrolones: Selectively bind GABA receptors, inducing a hypnotic effect. They decrease latency to sleep initiation, increase duration of sleep, and reduce episodes of awakening. Examples include **Zopiclone** (7.5 mg hs) and **Eszopiclone** (2-3 mg hs).

3. Histaminergics: Doxepin (5-10 mg hs) is classified as a tricyclic antidepressant, it has selective histaminergic antagonistic action at low doses. In pilot studies it has been particularly effective in sleep maintenance insomnia in PD.

4. Melatonin: in pilot studies, it has improved perception of sleep quality but effect on total sleep time is small.

5. Dopaminergic medications: If motor manifestations such as tremor or pain secondary to rigidity are disrupting sleep, addition of dopaminergic therapy may be useful. In some patients, dopaminergic agents (especially dopamine agonists) can promote sleep. Options include controlled release levodopa at bedtime, levodopa upon awakening in the early morning, or addition of long acting agents to the daily regimen.

6. Sedating antidepressants: Trazodone, low dose **Desipramine**, have sedating properties. These may be especially useful if there is associated depression, but watch for anticholinergic effects.

7. Benzodiazepines are short-term options only, can be useful in some, but caution is needed in patients with cognitive impairment. Habituation must be avoided.

REM Sleep Behaviour Disorder (RBD)

What is the prevalence? Found in 50% of patients with PD

What do patients experience?

Rapid Eye Movement Sleep Behaviour Disorder (RBD) is characterized by the absence of muscle atonia during REM sleep. Patients act out their dreams, resulting in talking, limb jerking or screaming during their sleep. Patients may fall out of bed, injure themselves, or hurt their bed partner. Patients often note that dreams have become more violent. In many cases RBD precedes motor symptoms and can be a marker of other problems, especially cognition.

Why does it happen?

Degeneration of lower brainstem nuclei is probably involved in RBD, particularly in the peri-locus ceruleus area.

Fast Facts:

- RBD has considerable potential as a marker for PD.
- Up to 65% with idiopathic RBD develop PD or Lewy Body Dementia in 10 years.

Misdiagnosis of RBD is common. Sleep apnea, nocturnal seizures, and sleep walking (non-REM parasomnias) can mimic RBD. If doubt exists, diagnosis is **made by sleep study (Polysomnogram).**

Clinical features of RBD:

- If patient reports dream enactment behaviour ask: if this corresponds to dream content (i.e. “Acting out your dreams”).
- Falls out of the bed are common; however, sleep walking is atypical.
- Should return quickly to normal alertness when woken during episodes.

Possible Treatments:

Non-pharmacological:

Warn the patient about injury and consider bed-safety measures (e.g. moving sharp objects away from the bed, using bed rails, placing pillows or mattresses at the side of their bed, sleeping apart from spouse, etc.).

Pharmacological:

1. **Remove triggers:** antidepressants, including SSRI's and tricyclics, can trigger or worsen RBD. Wellbutrin is not associated with RBD.
2. **Clonazepam** (0.25-2 mg at bedtime). The first described treatment of RBD, it helps up to 90%. As with any benzodiazepine, caution needs to be used in patients with cognitive impairment, excessive daytime somnolence and falls.
3. **Melatonin** (3mg-12mg at bedtime): Melatonin works by directly restoring REM atonia. It is recommended as first line therapy for patients at risk of cognitive dysfunction or excessive daytime somnolence.

If clonazepam and melatonin alone or in combination therapy fail, additional drugs may occasionally help, including dopaminergic therapy, donepezil, etc.

Pitfalls:

- Antidepressants trigger or worsen RBD.

Restless Leg Syndrome (RLS)

What is the prevalence? Approximately 30% of patients

What do patients experience?

The patient feels discomfort in their legs and feels the need to constantly move their legs. RLS usually occurs when sitting or lying down, and it is usually worse during the evening and night. RLS can be associated with periodic limb movements of sleep, which are involuntary limb movements that occur every 20-60 seconds while sleeping. It is unclear whether the prevalence of RLS is the same in the general population as it is in PD, but it is often more severe in PD. It is important (and difficult) to distinguish RLS from other leg symptoms that are common in PD such as pain due to off periods, cramps, and dyskinesia.

Essential diagnostic criteria of RLS: An urge to move the legs that:

1. Is usually accompanied or caused by uncomfortable and unpleasant sensation in the legs
2. Begins or worsens during periods of rest or inactivity such as lying or sitting
3. Is partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues
4. Is worse in the evening or night than during the day

Why does it happen?

RLS is often hereditary and can be associated with depleted iron stores. It occurs in about 10% of the general population in Europe and North America. Although dopaminergic therapy is the mainstay of treatment of RLS, long-term use results in augmentation, the tendency for symptoms to worsen in severity and occur earlier during the day. By virtue of their need for long-term dopaminergic therapy, PD patients are at risk of augmentation.

Possible Treatments:

Pharmacological:

1. Iron: Check ferritin levels, if low consider iron supplementation (e.g. FeSO4 300 mg QD).

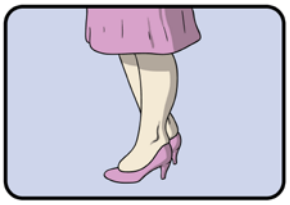
2. Dopamine therapy: Levodopa (Sinemet), Pramipexole (Mirapex), Ropinirole (Requip) may improve RLS in PD if they are taken at night. Often, changes in dopaminergic therapy are not practical because of other issues at night-time.

3. Gabapentin (100-300 mg QD-BID) can reduce RLS and may be a good option when dopaminergic therapy is already maximized. The main side effect is somnolence. Topiramate, Carbamazepine and Valproate may also possibly be efficacious.

4. Opioids: All opioids can also help RLS and can be used in moderate-severe cases.

Pitfalls:

• Early studies suggest that Domperidone occasionally can worsen RLS, perhaps via changes in melatonin secretion.



Leg Swelling

What do patients experience?

Leg swelling is common in PD. It usually occurs in the lower leg, and it is usually not severe.

Why does it happen?

Leg swelling is a common side effect of antiparkinson medications, particularly dopamine agonists.

Possible Treatments:

1. If the leg swelling is not severe and it is not bothersome to the patient, treatment may not be necessary.
2. Advise the patient to wear **compression stockings** and to sit with their legs raised.
3. Reduction of dopaminergic therapy may help if symptoms are severe.

Pitfalls:
• Rule out other causes of leg swelling: Congestive heart failure, renal failure, electrolyte imbalance, etc.
• Leg swelling related to PD has a close relation to starting new medications.

Excessive Sweating

What is the prevalence? Approximately 30-50% of patients

What do patients experience?

PD patients sometimes experience excessive sweating. Usually, hyperhidrosis manifests in the axilla, the palms, the soles of the feet, and the face, but in PD it often also involves the whole body – "drenching sweats."

Why does it happen?

Hyperhidrosis usually occurs during off periods (muscle stiffness) or during on periods with dyskinesias. Dysautonomia is associated with both hypohydrosis and hyperhydrosis.

Possible Treatments:

1. Dopaminergic medication adjustment: Changing the timing of medication to reduce off periods may improve excessive sweating.
2. Botox Injections to the axilla, palms, and soles can occasionally be used; however, they are rarely required in PD patients**.**

Diplopia and Visual Abnormalities

What do patients experience?

Visual abnormalities in PD include diplopia, impaired colour and contrast discrimination, and visuospatial defects. Impaired colour and contrast discrimination may precede motor symptoms and patients can be unaware of these deficits. Impaired contrast sensitivity can impair driving, particularly at night. Diplopia is less common and usually occurs while reading.

Fast Facts:

• Visual changes (color vision loss) may have potential as a predictor in PD

Why does it happen?

Impaired colour and contrast discrimination may be due to retinal dysfunction (degeneration of dopaminergic neurons in the retina) and/or to dysfunction of visual cortex.

Diplopia is often due to convergence insufficiency; essentially, the eyes 'get tired' from holding a prolonged convergence position. Major extra ocular movement abnormalities may be a sign of another degenerative process, especially progressive supranuclear palsy.

Defects in visuospatial function are associated with cognitive dysfunction in PD. They can be associated with hallucinations (see sections on cognitive dysfunction and hallucinations).

Possible treatment:

Increasing dopaminergic medications can improve convergence insufficiency.

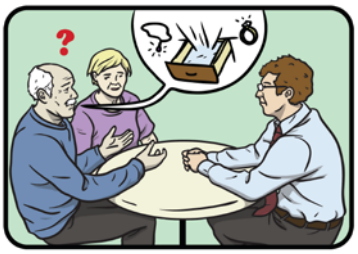
Discuss with patient that vision problems in PD have important implication in driving abilities.

Driving in PD

• Lots of reasons to have problems:
- Visual abnormalities
- Motor speed
- Attentional deficits, reaction time deficits
- Dementia
- Excessive somnolence - sleep attacks

• Most patients with mild disease are safe drivers, and many PD patients stop spontaneously

• When in doubt, most provincial/state agencies have on-road testing available



Delusions

What is the prevalence? Approximately 5% of non-demented and 15% of demented patients with PD.

What do patients experience?

Delusions are false beliefs that are not based on fact. They frequently are paranoid in nature, with feelings of persecution, jealousy, spousal infidelity and fears of impoverishment. Visual hallucinations can trigger delusions – a common delusion in PD is squatters living in attics, sheds, the garage, etc. which is directly related to vivid hallucinations of people.

Why does it happen?

Delusions are less common than hallucinations, although the two symptoms often coexist, and occur in patients with cognitive impairment, and therefore are part of advanced stage of PD. Like hallucinations, delusions, can be side effects of antiparkinson medications or can be induced by concurrent infection.

Possible Treatments:

Delusions can be treated with the same medications used to treat hallucinations in PD.

Fast facts:

• Delusions are uncommon, but almost always require treatment!!

Impulse Control Disorders

What is the prevalence? Approximately 10-15% of patients

What do patients experience?

Impulse control disorders (ICD) are characterized by failure to resist an impulse, drive or temptation to perform an act that is harmful to the person or to others. These include pathological gambling, hypersexuality, binge eating (eating abnormally large amounts of food or lack of control), excessive shopping, and punding (repetition of useless tasks). Unrecognized impulse control disorders can have devastating effects, resulting in bankruptcy and deterioration of close relationships.

Dopamine Dysregulation Syndrome (DDS) is another form of ICD, which is experienced like an 'addiction' to levodopa. Patients with DDS experience severe anxiety and irritation during off periods, and will take excessive medication to prevent any sign of an off period even without their physician's approval.

Why does it happen?

ICDs are a side-effect of dopaminergic medications. ICDs are caused principally by D3 receptor overstimulation. Patients on dopamine agonists such as Pramipexole (Mirapex) and Ropinirole (Requip) have the highest likelihood of developing ICD. Overall, approximately 15% of patients on dopamine agonists can experience ICD. ICD can also occur with high doses of levodopa, and with deep brain stimulation of the subthalamic nucleus (DBS of STN).

DDS is more common with levodopa than other dopaminergic medications.

Clinical red flags for the development of DDS:
- Young-onset PD (<45 years of age)
- Previous history of alcohol or illicit drug abuse
- Impulsive sensation-seeking personality traits, previous "risk-taking" activities

Possible Treatments:

Non-pharmacological:

Vigilance for this problem is essential. Patients are often not aware of the connection to dopamine agonists, so will not mention them. Patients taking dopamine agonists and their caregivers must be screened for ICD at each visit.

Pharmacological:

Reduce or stop dopamine agonist. This is the first step in treating ICD. This almost always resolves the symptoms, although not always completely. Often patients are able to tolerate lower doses of agonists without behavioural problems, but continual surveillance is needed.

Antidepressants. If patients require dopamine agonists for their therapy or if symptoms persist, use of SSRI/SNRI antidepressants may help.

Some Useful References

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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
1. Dribbling of saliva during the daytime	☐	☐
2. Loss or change in your ability to taste or smell	☐	☐
3. Difficulty swallowing food or drink or problems with choking	☐	☐
4. Vomiting or feelings of sickness (nausea)	☐	☐
5. Constipation (less than 3 bowel movements a week) or having to strain to pass a stool (faeces)	☐	☐
6. Bowel (fecal) incontinence	☐	☐
7. Feeling that your bowel emptying is incomplete after having been to the toilet	☐	☐
8. A sense of urgency to pass urine makes you rush to the toilet	☐	☐
9. Getting up regularly at night to pass urine	☐	☐
10. Unexplained pains (not due to known conditions such as arthritis)	☐	☐
11. Unexplained change in weight (not due to change in diet)	☐	☐
12. Problems remembering things that have happened recently or forgetting to do things	☐	☐
13. Loss of interest in what is happening around you or doing things	☐	☐
14. Seeing or hearing things that you know or are told are not there	☐	☐
15. Difficulty concentrating or staying focussed	☐	☐
16. Feeling sad, 'low' or 'blue'	☐	☐
17. Feeling anxious, frightened or panicky	☐	☐
18. Feeling less interested in sex or more interested in sex	☐	☐
19. Finding it difficult to have sex when you try	☐	☐
20. Feeling light headed, dizzy or weak standing from sitting or lying	☐	☐
21. Falling	☐	☐
22. Finding it difficult to stay awake during activities such as working, driving or eating	☐	☐
23. Difficulty getting to sleep at night or staying asleep at night	☐	☐
24. Intense, vivid dreams or frightening dreams	☐	☐
25. Talking or moving about in your sleep as if you are 'acting' out a dream	☐	☐
26. Unpleasant sensations in your legs at night or while resting, and a feeling that you need to move	☐	☐
27. Swelling of your legs	☐	☐
28. Excessive sweating	☐	☐
29. Double vision	☐	☐
30. Believing things are happening to you that other people say are not true	☐	☐

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
For information contact: susanne.tluk@uhl.nhs.uk or alison.forbes@uhl.nhs.uk

PD NMS Questionnaire

Nom: ..... Date: ..... Âge: .....

Numéro d'identification du centre : Homme ☐ Femme ☐

Les problèmes non-moteurs reliés au Parkinson

Les symptômes du Parkinson reliés mouvement sont très bien connus. Par contre, d'autres problèmes peuvent parfois apparaître en raison de cette maladie ou de son traitement. Il est important que le médecin soit au courant de ces problèmes, particulièrement s'ils sont gênant pour vous.

Plusieurs problèmes sont énumérés ci-dessous. S'il-vous-plaît, cochez la boîte « Oui » si vous avez eu ce problème **au cours du dernier mois**. Le médecin ou l'infirmière pourraient vous poser quelques questions afin de vous aider à choisir une réponse. Si vous n'avez pas eu ce problème au cours du dernier mois, cochez la boîte « Non ». Vous devriez répondre « Non » même si vous avez eu ce problème dans le passé, mais pas au cours du dernier mois.

Avez-vous eu un des problèmes suivants au cours du dernier mois?

	Oui	Non
1. Salive qui dégoutte durant la journée	☐	☐
2. Perte ou changement dans votre habilité à goûter ou à sentir	☐	☐
3. Difficulté à avaler de la nourriture ou à boire, ou problème d'étouffement	☐	☐
4. Vomissement ou sensation de malaise (nausée)	☐	☐
5. Constipation (moins de trois selles par semaine) ou avoir besoin de fournir un effort soutenu afin d'évacuer une selle	☐	☐
6. Incontinence intestinale (fécale)	☐	☐
7. Sensation que votre intestin n'est pas complètement vidé après avoir été à la toilette	☐	☐
8. Sensation d'urgence pour uriner qui vous fait courir pour aller à la toilette	☐	☐
9. Se lever régulièrement la nuit pour uriner	☐	☐
10. Douleur inexpliquée (non causée par une maladie connue telle l'arthrite)	☐	☐
11. Changement inexpliqué de votre poids (non causé par un changement de diète)	☐	☐
12. Problème à se rappeler des choses qui se sont produites récemment ou oubli de choses à faire	☐	☐
13. Perte d'intérêt pour ce qui se passe autour de vous ou pour les choses à faire	☐	☐
14. Voir ou entendre des choses que vous savez ou qui vous a été dit qui n'étaient pas là	☐	☐
15. Difficulté à se concentrer ou à rester « focussé »	☐	☐
16. Sensation de tristesse, d'avoir les « blues » ou une perte d'énergie	☐	☐
17. Sensation d'anxiété, apeuré, paniqué	☐	☐
18. Avoir moins d'intérêt pour la sexualité ou plus d'intérêt pour la sexualité	☐	☐
19. Avoir de la difficulté à avoir des relations sexuelles quand vous essayez	☐	☐
20. Se sentir étourdi ou faible lorsque vous êtes debout après avoir été assis ou couché	☐	☐
21. Chute	☐	☐
22. Trouver cela difficile de rester réveillé en faisant des activités telles que travailler, conduire ou manger	☐	☐
23. Difficulté à s'endormir la nuit ou resté endormi durant la nuit	☐	☐
24. Rêves intenses, d'apparence réelle ou rêves épeurants	☐	☐
25. Parler ou bouger durant votre sommeil comme si vous « actez » vos rêves	☐	☐
26. Sensation désagréable dans vos jambes le soir ou lorsque vous vous reposez et une sensation que vous devez bouger	☐	☐
27. Enflure de vos jambes	☐	☐
28. Transpiration excessive	☐	☐
29. Vision double	☐	☐
30. Croire que des choses vous arrivent alors que d'autres personnes disent que ce n'est pas vrai	☐	☐

Toutes les informations que vous donnerez via ce questionnaire seront gardées confidentielles et seront seulement utilisées dans le but pour lequel vous avez complété ce questionnaire.