PHARMACY MAGAZINE CPD MODULE
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module 241

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Welcome to the two hundred and forty first module in the *Pharmacy Magazine* Continuing Professional Development Programme, which looks at supporting patients with Parkinson's disease.

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GOAL

To enable pharmacists to provide proactive medicines optimisation services for people with Parkinson's disease (PD) and their carers.

training excellence NPA National Pharmacy Association Quality Seal 2015 in association with CIG

OBJECTIVES

After studying this module you should be able to:

- Recognise early signs and symptoms associated with possible PD and signpost appropriately
- Identify medicines which may be harmful in PD
- Plan and conduct a MUR for a person taking levodopa or a dopamine agonist.

First in professional development pharmacy magazine

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CPD

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This module is suitable for use by community pharmacists as part of their continuing professional development. After reading this module in the magazine or online, complete the scenarios and post-test at **www.pharmacymagazine.co.uk** and include in your personal learning log. CPD is one aspect of professional development and can be considered alongside other activities for inclusion in your **RPS Faculty portfolio.**

Supporting patients with Parkinson's disease

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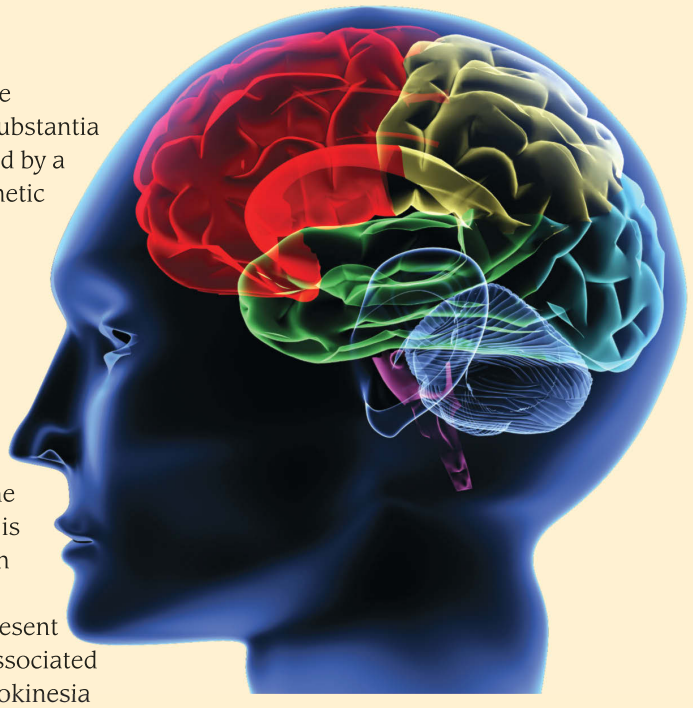
Introduction



Parkinson's disease (PD) is a neurodegenerative disease of the dopaminergic neurones in the substantia nigra and is thought to be caused by a complex interaction between genetic and environmental risk factors.

The current NICE Clinical Guideline 35 (CG35), which is due to be updated in 2017, defines PD as:

"A progressive neuro-degenerative condition resulting from the death of the dopamine-containing cells of the substantia nigra. The diagnosis is primarily a clinical one based on the history and examination.

"People with PD classically present with the symptoms and signs associated with parkinsonism, namely hypokinesia (i.e. poverty of movement), bradykinesia (i.e. slowness of movement), rigidity and rest tremor."

PD potentially affects all muscle groups to an extent where it limits daily activities and also encompasses autonomic dysfunction (constipation, erectile and urinary dysfunction, hypotension and dizziness), neuropsychiatric conditions (depression, dementia, hallucinations and

psychosis), pain, lethargy/fatigue, and has a major impact on quality of life for the individuals and their carers and families.

Increasingly recognised is the presence of prodromal symptoms occurring up to 20 years prior to diagnosis. The commonest early presenting symptoms in the 10 years before PD are constipation, fatigue, dizziness, hypotension, erectile and urinary dysfunctions. Later onset symptoms (occurring four to five years before diagnosis) are tremor, balance and rigidity.

Depression occurred in 10 per cent of all people diagnosed with PD two years prior to a diagnosis being made, compared to only 4 per cent in people without a PD diagnosis.

Prevalence

Parkinson's disease is the fourth commonest neurological condition in the UK behind stroke, all forms of dementia and epilepsy. In terms of progressive neurodegenerative diseases it is second only to Alzheimer's disease.

In the UK PD affects between six to 11 people in every 6,000 (the average size of a general practice list) and, on average, there will be five to eight people on PD medication in every community pharmacy. There is a family history of PD in 20-30 per cent of cases but only 5 per cent of these are attributed to single gene defects. Having a genetic risk present does not mean PD is inevitable.

Parkinson's is rare below the age of 50 years – such cases are known as juvenile PD and account for only 5 per cent of all PD cases – but affects 1 per cent of the population over 65 years, rising to 2 per cent in people over 80 years. Of note, 50-80 per cent of people with PD may develop Parkinson's disease dementia (PDD), although this seems more related to increasing age than PD itself.

Neuropathological changes

During the disease process there is progressive loss of dopaminergic cells from the substantia nigra pars compacta in the brain stem, projecting into the striatum (caudate nucleus and putamen-basal ganglia). This cell loss results in decreased dopamine (as well as serotonin and GABA) in the striatum. PD is not caused by an imbalance of acetylcholine and dopamine as once thought; its underlying mechanism is more complex than that.

Table 1: Learned voluntary actions

Examples of learned voluntary actions are:

Walking	Eating	Cooking
Washing	Talking	Climbing stairs
Household chores	Dressing	Using public transport
Bathing	Shopping	Going to the toilet
Answering the phone	Gardening	Cleaning teeth
Driving	Working	Playing sport
Dancing	Having sex	Writing
Enjoying hobbies	Turning over in bed	Socialising
Rising from a chair	Getting out of bed	Fine movement activities

The basal ganglia controls muscle tone and provides smooth muscle voluntary movements. PD symptoms present as impairment in carrying out learned voluntary actions (see Table 1) when 80 per cent of dopaminergic cells are lost.

Every person with PD has a different set of signs and symptoms (see Table 2), so care and treatment needs to be individualised.

Other signs associated with PD may be present, but these are not diagnostic in their own right. They include:

- Hypomimia (reduced facial expression)
- Micrographia (very small hand writing that slopes off the page)
- Impaired blinking
- Reduction in speech volume
- Autonomic abnormalities
- Difficulty swallowing/excess saliva and/or drooling
- Sleep dysfunction
- Neuropsychiatric disturbances
- Gait abnormalities.

Table 2: Associated symptoms of PD

Common problems would include:

Dribbling/drooling	Anxiety
Swallowing	Tremor
Incontinence	Insomnia
Constipation	Behaviour
Pain (muscle spasm)	Confusion
Freezing	Nightmares
Depression	Memory
Slowness	Hallucinations

People may also complain of pain, tiredness, depression and constipation.

Medication-induced parkinsonism

Any medication that blocks the action of dopamine can cause parkinsonian-like symptoms (tremor, rigidity, bradykinesia, dystonia), which account for 7 per cent of people presenting with suspected PD (see Table 3). These medication-induced effects respond poorly to levodopa therapy. Dependent on the agent, slow withdrawal is recommended, with 60 per cent of patients recovering in two months, but others may take up to two years.

A number of medicines can cause tremor and these should not be prescribed for people with PD unless agreed by a specialist neurologist, and then used at the lowest possible dose for the shortest period of time.

Non-pharmacological treatments and support services

These are extremely important for people with PD at any stage of their disease. Treatments (whether pharmacological or non-pharmacological) should be tailored to individual need. For example, physiotherapy aids retention of muscle strength and mobility; occupational therapy supports maintenance of daily activities (washing, dressing, tips for un-freezing); speech and language therapy enables communication; patients may need help with swallowing (eating and drinking); and social services are necessary for access to disability and financial support.

Staging in Parkinson's disease

There are four stages to PD:

- Establishing the diagnosis
- Early or maintenance PD
- Complex or later PD
- Palliation.

Treatments do not cure or halt progression, but they do improve quality of life.

Early PD

In early PD the aim is to preserve dopaminergic function for as long as possible by using dopamine supplementation. Levodopa preparations, non-ergot derived dopamine agonists and MAOI-B inhibitors are all first-line options

Table 3: Medicines associated with parkinsonism

Medication class	Example
Anti-emetic	Metoclopramide, prochlorperazine, cinnarizine
Antipsychotics	First generation: dose dependent – avoid haloperidol Second generation: dose dependent effects – avoid. Clozapine least association with movement abnormalities
Antihypertensives	Calcium channel blockers
Causality unproven	NSAIDs, captopril, amiodarone, phenytoin, valproate, lithium, oral contraceptives, SSRIs

in early PD. The choice is dependent on the individual circumstances (e.g. age, preference, tolerability), and after the short and long-term benefits and drawbacks of each class of medicine have been explained to the patient.

Historically, levodopa with a decarboxylase inhibitor (DCI) has been the gold standard of treatment, but tolerance to levodopa occurs over time. Tolerance is estimated at 10 per cent year-on-year, making it inappropriate to use first-line in younger patients as it will be ineffective within 10 years.

Anticholinergic agents, amantadine and beta-blockers are not recommended in early PD due to lack of evidence or limited efficacy. (See Table 4 for treatment options.)

Complex or later PD

After five to 10 years of taking a levodopa preparation, between 50-70 per cent of people experience ON or OFF episodes.

An ON episode is when a person with PD can perform activities of daily living as normal for them; OFF is when they completely freeze and voluntary movement is difficult or impossible. This can be extremely frightening and may cause a great degree of anxiety and concern for the person with PD and his/her family. These fluctuations in symptom control may not necessarily relate to the timing of medicines administration.

Eighty-five per cent people taking levodopa experience 'wearing off' of drug efficacy, with 37 per cent experiencing a sudden ON/OFF and 34 per cent a delayed ON response to their usual treatment.

Parkinson's disease can be hugely debilitating and have a major impact on quality of life

In later PD an adjuvant agent is required to reduce complications and improve quality of life. There is no single adjuvant agent of choice so selection is based on patient preference after the short and long-term benefits and drawbacks of each class of medicine have been explained to them and what they had been originally prescribed is taken into account.

If a person has been on a levodopa preparation first-line, then either a COMT inhibitor or a non-ergot derived dopamine agonist could be introduced. In the latter case, the dose of levodopa must be reduced to prevent dyskinesia and neuropsychiatric effects, such as hallucination and psychosis. Conversely, if a person has been on a non-ergot derived dopamine agonist first-line, then either a levodopa preparation or a COMT inhibitor could be added. (See Table 5 for treatment options.)

Counselling point

If someone is experiencing an episode of freezing, there are ways in which movement can be re-started. This will depend on patient experience and choice.

Often verbalising the action a person wants to do (e.g. lift the foot forward, take a step and walk through the door) may help them to do this. Singing, whistling a tune, counting or stepping over a carer's foot or holding a stick and tapping the foot they want to move may also help.

Never pull or push someone as they could fall and injure themselves. Remember: this situation can be very frightening for an individual and they may need lots of gentle, calm reassurance.

Reflection exercise 1

One of your patients with early Parkinson's disease has had a minor accident while driving. His carer is very concerned. What advice would you give and what are your responsibilities in terms of protecting other road users by informing the DVLA? (See dvla.gov.uk/medical.aspx for further information.)

Palliative stage

This stage is characterised by a lack of efficacy of medication, increasing side-effects and movement disorders. Neuropsychiatric symptoms and/or dementia may be present with problems in swallowing, eating, communication or completing activities of daily living.

The aims at this stage are to reduce unnecessary medication, and address freezing episodes, postural instability, falls, mood disturbance and dementia (rivastigmine is licensed for Parkinson's disease dementia), while facilitating communication and individualised patient care and carer support.

You can complete this module online at **pharmacymagazine.co.uk** and record your learning outcomes and the impact on your practice in your **personal learning log**

Pharmacological agents:
Levodopa + decarboxylase inhibitor

To treat the symptoms of PD the amount of dopamine available in neuronal synapses needs to be increased. Exogenous dopamine is rapidly broken down in the peripheral circulation by decarboxylase and catechol-O-methyl transferase (COMT) enzymes. To ensure the maximum amount is able to cross the blood-brain barrier, levodopa (LD) is generally combined with a decarboxylase inhibitor (DCI) (carbidopa in Sinemet [co-careldopa] or benserazide in Madopar [co-beneldopa].

These agents are as effective as each other, but one may be preferable to an individual. It is imperative that they are always taken as prescribed, as even small errors in dose or timing may affect the well-being of the patient for some days. If in doubt, pharmacists should always ask the person for confirmation that they are dispensing their usual medication.

If a dose is missed in early PD, little effect may be seen on movement, but in later PD dopamine storage is impaired so the effects will be greater.

In early PD levodopa preparations are taken with or after food to minimise nausea and vomiting (domperidone is ineffective), but in later PD doses are taken *before* food so absorption is not impaired by the presence of proteins.

People with PD are encouraged to leave protein-rich meals to later in the day after they have taken the majority of their levodopa doses in order to improve its absorption as well as the more predictable effects on PD symptoms at any stage.

Immediate release preparations

Levodopa immediate release preparations generally have a short half-life so require three to four times daily dosing to a maximum of five doses in 24 hours. Only immediate release preparations should be prescribed in early PD.

Levodopa preparations are started at 50mg once daily, increasing every three to four days until a dose regimen of 50mg three times daily is reached. This can then be titrated (when appropriate) to 100mg three times daily. This slow titration is to avoid side-effects, especially nausea, postural hypotension and neuropsychiatric effects.

Pharmacy teams should be able to anticipate side-effects of levodopa and highlight these to individual patients. Interactions with other medicines should also be predicted and avoided where possible.

Controlled release preparations

In the past controlled release preparations were often used first-line in the hope of delaying tolerance to levodopa, but this is no longer considered appropriate and is strongly advised against. Controlled release preparations should only be initiated in complex PD by a specialist, in order to simplify medication regimens. The bioavailability of dopamine from controlled release preparations is less than that from immediate release products (60-70 per cent compared to 90-100 per cent), and this necessitates careful levodopa dose increases to compensate.

Bioavailability of controlled release products is increased when administered with food, but decreased if antacids have been taken.

The nomenclature of these preparations can be confusing and care should always be taken by pharmacists and pharmacy technicians when dispensing them to ensure the correct product has been selected.

Reflection exercise 2

How many people with Parkinson's disease are currently on your PMR? When was the last time you invited them for a medicines use review?

Table 4: Pharmacological options in early PD

Agent	First choice option?	Side-effects	Special cautions
Levodopa & DCI	Yes – good degree of symptom control. Immediate release ONLY	Increased motor complications and other adverse effects	Keep dose as low as possible to reduce development of motor complications. Maximum amount 800mg daily in maximum of five daily doses
Dopamine agonist (DA)	Yes – moderate degree of symptom control	Reduced motor complications. Increased other adverse effects, especially with ergot derivatives. Also associated with impulsive control disorder and narcolepsy	Titrate to clinically effective dose. If side-effects prevent this, another agonist should be tried or a medicine from another class. If an ergot-derived agonist, monitor renal function and ESR, and take chest x-ray before and annually
MAOI-B inhibitor	Yes – limited degree of symptom control	Reduced motor complications. Increased other adverse effects	Slows breakdown of DA in striatum, boosting levels & prolongs LD half-life. Apoptosis form of programmed cell death thought to be important in several neuro-degenerative disorders including PD. Blocks conversion MPTP to MPP+. Wafer selegiline (Zelapar 1.25mg = 10mg oral). Not metabolised by buccal to l-amphetamine by 90 per cent. On tongue before breakfast – no drinking/eating for five minutes. Contraindication: any antidepressant (washout if necessary)
Anticholinergic	No – lack of evidence and limited efficacy	Cause neuropsychiatric side-effects, dry mouth, blurred vision, urinary retention, exacerbation of glaucoma and constipation. NOT for use in elderly	
Amantadine	No – lack of evidence	Ankle oedema, confusion, livedo reticularis, hallucinations	Last dose at 2pm
Beta-blockers	No – lack of evidence	Symptomatic treatment of postural tremor in PD only	Blood pressure/pulse measurement

Non-ergot derived oral dopamine agonists

Dopamine agonists were originally developed as a levodopa-sparing agents in early PD but the reality of delaying the need for levodopa preparations in the long-term has not been realised.

The original ergot-derived dopamine agonists (bromocriptine, cabergoline, pergolide) are associated with potentially severe fibrotic adverse effects requiring increased monitoring, and are no longer first-line options in this class. The recommended non-ergot derived dopamine agonists are now ropinirole, pramipexole and rotigotine.

Dopamine agonists bind directly to post-synaptic receptors to give a more reliable and sustained biological response and therefore, theoretically, should be more effective in early PD. They are not stored by degenerating dopaminergic neurones and so reduce dopaminergic turnover and resultant free radical production at the synapse. They also have a longer half-life, resulting in less frequent dosing schedules.

The dopamine receptor sub-type specificity varies between agents, so if there is little gain with one agent, another should be tried.

Apomorphine is a parenteral dopamine agonist given by continuous subcutaneous infusion or intermittent subcutaneous 'rescue' injection for OFF or freezing episodes. It is powerfully emetogenic and requires at least 48 hours' pre-treatment with domperidone. As determining the dose requires stopping PD treatment overnight, apomorphine is only started in specialist centres. There is evidence for eight years of continuous usage, but it is also associated with hypotension and development of skin nodules.

Adverse effects include nausea, dyskinesia, orthostatic hypotension, somnolence (sudden sleep attacks with ropinirole and pramipexole), dizziness and psychiatric disturbances including impulse control disorders such as pathological gambling, hypersexuality and binge eating. (It should be remembered that dopamine is involved in the reward pathway of addiction.)

Monoamine oxidase B (MAOI-B) inhibitors

For some years it was thought that selegiline, due to its MAOI-B activity, had a neuro-protectant role and an anti-apoptotic effect (preventing cell death), hence its use as first-line treatment in early PD to try and prevent the need for early levodopa therapy. However current NICE and SIGN guidance is very clear that MAOI-B inhibitors should not be used as neuro-protectant therapy for people with PD except if part of a clinical trial.

The place of MAOI-B inhibitors (selegiline and rasagiline) in PD is as a first-line treatment option (less effective than levodopa or dopamine agonists) or as an adjunct to therapy

Reflection exercise 3

One of your patients with Parkinson's disease has routine surgery booked for a knee replacement. She is very concerned about how she can ensure she takes her medications at the times she needs them and not at the usual ward medication round times. What information, support and assurance can you offer her?

Table 5: Pharmacological options in later PD

Agent	First choice option?	Effects	Monitoring	Special cautions*
Dopamine agonist	Yes – moderate degree of symptom control	Reduced motor complications. Increased other adverse effects	Titrate to clinically effective dose. If side-effects prevent this, try another agonist or a medicine from another class	Ergot agonist derivatives: annual monitoring of renal function, ESR and chest x-ray
COMT inhibitor	Yes – moderate degree of symptom control	May reduce motor fluctuations	COMT inhibitors should be taken 20 minutes prior to levodopa for best effect, but adherence is difficult. A triple combination of levodopa, carbidopa and entacapone should be offered	Tolcapone should only be used after entacapone has failed or has intolerable side-effects. Liver function tests required every two weeks in the first year, then every four weeks for six months and every eight weeks thereafter. (Risk of fulminant liver failure)
MAOI-B inhibitor	Yes – moderate degree of symptom control	Reduced motor complications (dyskinesias). Increased other adverse effects		
Amantadine	Non-significant result	Reduced motor complications		
Apomorphine	No – limited degree of symptom control	Intermittent injections may reduce OFF time in people with severe motor complications. Continuous subcutaneous infusion may reduce OFF time and dyskinesias in people with severe motor complications. Increased other adverse effects		Initiation restricted to expert units with facilities for monitoring
Modified-release preparation of levodopa+DCI	No – can be used but not first option	Can reduce motor fluctuations	Ensure timing intervals. Be aware of signs of excess dopamine when adding or changing therapy	

* Never withdraw antiparkinsonian medication abruptly as this can lead to akinesia or neuromalignant syndrome, both potentially fatal.

to reduce motor fluctuations in complex or later PD. Selegiline is metabolised to methamphetamine and amphetamine, producing euphoria as an adverse effect. It also causes psychiatric disturbance and increased risk of all-cause mortality. With insomnia another adverse effect, oral dosing is prescribed before 2pm. Licensed buccal dosage forms by-pass this metabolic route and subsequently the associated adverse effects.

Catechol-O-methyltransferase (COMT) inhibitors

To understand the place of catechol-O-methyltransferase (COMT) inhibitors, it is important to understand the metabolism of levodopa. In the absence of a decarboxylase inhibitor (DCI), 70 per cent of levodopa undergoes decarboxyation and 10 per cent by COMT. In the presence of a DCI, an increased proportion is metabolised by the COMT route. By adding in a COMT-inhibitor, the central and peripheral availability of levodopa is increased, delaying its elimination and prolonging the duration of action.

There are two licensed COMT inhibitors – entacapone and tolcapone – although tolcapone is not a first-line choice due to its association with precipitating life-threatening hepatotoxicity. Entacapone is therefore the preferred agent and is given at the same time as a dose of levodopa with a decarboxylase inhibitor to a maximum of 2g daily. It can colour urine reddish-brown and has a number of side-effects including cardiac, neuropsychiatric and, rarely, hepatic side-effects.

Important considerations with PD medication

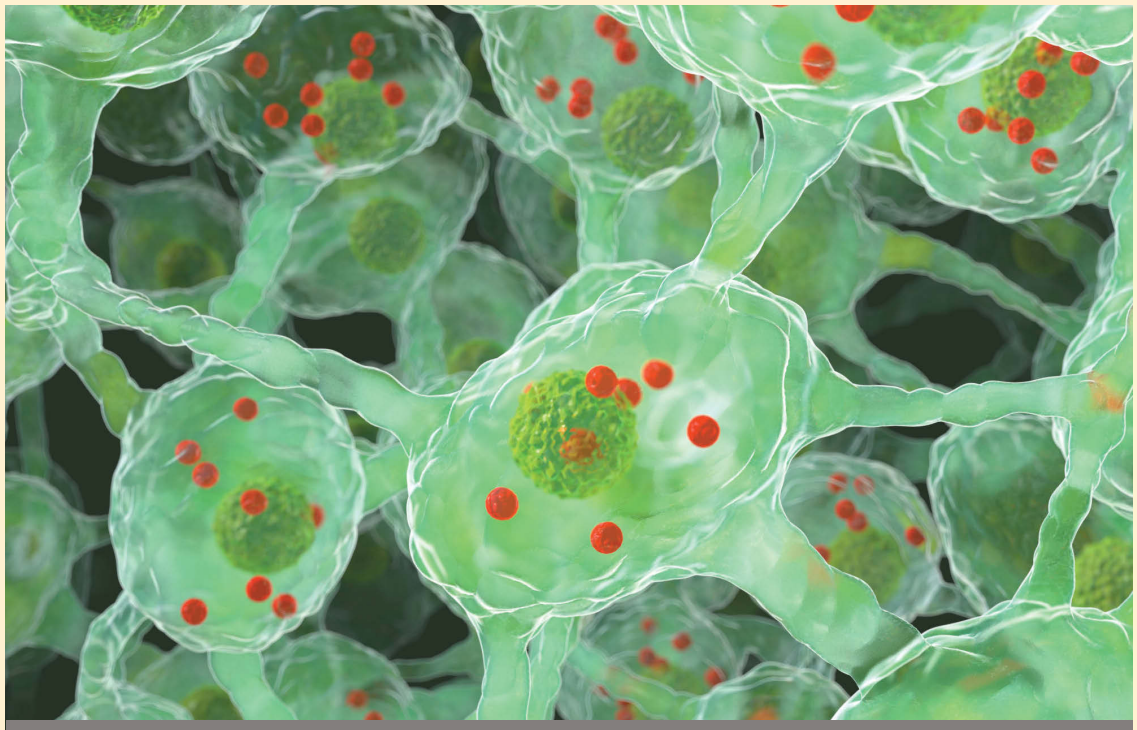
PD is a fluctuating, debilitating disease and the efficacy of pharmacological treatments also fluctuates, as does the response to treatment. It is important to remember that trapped within an uncooperative body is a cognitively intact person.

The clinical features of PD profoundly affect an individual's ability to communicate and can prejudice how people communicate with them because the altered body language and facial expressions of people with PD can seem threatening to others.

With 55 per cent of communication via body language, 38 per cent dependent on tone and volume, and only 7 per cent on the words spoken, it is important to adapt your communication skills to help the person communicate effectively with you. Due to reduced speech volume, conversations may be better in a quiet consultation room rather than at the medicines counter.

It is extremely important to remember that antiparkinsonian medicine should **never** be withdrawn abruptly or allowed to fail suddenly due to lack of absorption (e.g. in gastroenteritis or abdominal surgery) because of the risk of acute akinesia (total loss or impairment of the power of voluntary movement for the person, akin to paralysis) or neuroleptic malignant syndrome occurring.

People with PD who are hospitalised, awaiting surgical procedures or admitted to care homes, should have their medication given to them at the times appropriate to them and not be constrained by the organisation's routine. Self-medication should be a preferred option.



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Computer artwork of neurons (green) affected by Lewy bodies (orange) in the brain of someone with PD

Medicine doses or timing should be adjusted by/or only after discussion with a specialist. This may be a consultant neurologist, elderly care physician, Parkinson's nurse or other suitably trained healthcare professional.

Parkinson's UK has published guidance for both community and hospital pharmacists on the most appropriate use of PD medicines. It is important to be aware of the key recommendations. (See **parkinsons.org.uk/professionals/resources/key-information-community-pharmacists-booklet**.)

Information gathering

Parkinson's UK would like community pharmacists to ask patients the five questions below, rate the answers and then provide the patient and their specialist with a copy of the outcome. You could do this during a MUR.

1. When was your last hospital appointment with a Parkinson's specialist?

Within the last six months 0
Within the last 12 months.....5
More than 12 months 15
Have never seen a specialist for the management of my Parkinson's 20

Reflection exercise 4

Could you signpost a person newly diagnosed with Parkinson's disease to local support groups for people with the condition and their families? Visit the Parkinson's UK website (parkinsons.org.uk) and NHS Choices (nhs.uk/conditions/Parkinsons-disease/Pages/Introduction.aspx). Use these to create your local list. Patient access to carer support and social services may need referral by a GP.

2. How would you rate the control of your Parkinson's symptoms at the moment?
Well controlled 0
Somewhat controlled 5
Poorly controlled 15
Not controlled 20

3. Do you remember to take your medication every time?
Yes 0
Misses the occasional dose (one a week) 5
Misses regular doses (one a day) 15
Not compliant – I do not have a clear understanding of my medication regimen 20

4. Do you ever take additional Parkinson's medication over and above what your specialist has advised?
No, never 0
Infrequently 5
Most of the time 15
All of the time 20

5. Are you experiencing any new motor symptoms (e.g. freezing, increased tremor or gait problems) since your last hospital appointment?
No 0
Not sure 5
Occasionally 15
Yes 20

Total score from assessment questions = outcomes of total score

0-15 Satisfactory review. No action necessary
15-30 Direct to local Parkinson's nurse
30+ Intervention required. Refer to GP for onward referral to Parkinson's specialist

Autonomic symptoms in PD

Symptoms	NICE guidance & comments
Incontinence	Highly anticholinergic agents may impair cognitive function and worsen constipation and dry mouth
Erectile dysfunction	Affects intimate relationships
Dysphagia (impaired swallowing)	This may affect eating and lead to weight loss and impaired verbal communication skills
Weight loss	Refer for nutritional advice
Orthostatic hypotension	Can be exacerbated by medication treatment and lead to falls
Excessive sweating	Embarrassing for the individual
Sialorrhoea	Embarrassing for the individual
Constipation	Ensure a stimulant laxative and/or softener is prescribed as appropriate

50+ Urgent intervention required. Patient to see GP as soon as possible for referral to Parkinson's specialist.

A medicines use review could also cover the following areas:

- Handling of medication packaging
- Loss of smell: may affect appetite
- Dry mouth: may impair eating/communication
- Visual disturbances: affect mobility and increase risk of falls
- Falls: any fall should be investigated and the person referred appropriately
- Sleep/mood disturbance: refer for treatment
- Daytime hypersomnolence: refer for review
- Memory/dementia: refer for specialist advice
- Pain: this is from muscle spasms in dystonias and dyskinesias, refer for treatment; physiotherapy may also help.

Other common issues in PD include mood and autonomic symptoms. The autonomic nervous system works to control the unconscious or automatic functions of heart rate, digestion, breathing rate, perspiration, urination and sexual arousal. These issues may emerge during a MUR discussion. If they do, the patient should be signposted and/or referred for specialist review.

As swallowing difficulties increase over time and the adverse effects of PD medication become less tolerable, there may be a need to change the formulation of a medication to one that is more suitable for the individual.

Some people may need different formulations of the same agent, e.g. a dispersible levodopa preparation with a standard release preparation taken in the morning to aid mobility and/or a controlled release preparation at night to prevent OFF periods.

The manufacturer's guidance in the SPC should always be used for conversion rates when changing between formulations to take into account differing rates of absorption. Generic drugs are available but absorption rates may differ, so people should be titrated and maintained on the same brand.

Cautions, contraindications and common side-effects should always be checked to ensure patient safety.

Further reading and useful resources

- Key information for community pharmacists: parkinsons.org.uk/sites/default/files/publications/download/english/b0148_ keyinformationforcommunitypharmacists.pdf
- NICE Clinical guideline 35, 2006: nice.org.uk/guidance/cg35/evidence/cg35-parkinsons-disease-full-guideline2
- NICE Pathways for Parkinson's disease: pathways.nice.org.uk/pathways/parkinsons-disease
- Kearney D, Dunsmure L. Parkinson's disease management. *Clinical Pharmacist* 2011 (3) 368-373
- Blochberger A, Jones S. Parkinson's disease clinical features and diagnosis. *Clinical Pharmacist* Vol 3 December 2011 p361-366
- Ruxton K, Woodman RJ, Mangoni AA. Drugs with anticholinergic effects and cognitive impairment, falls and all-cause mortality in older adults: A systematic review and meta-analysis. *British Journal of Clinical Pharmacology* Volume 80, Issue 2, pages 209-220, August 2015. DOI: 10.1111/bcp.12617
- Nannor N. Understanding essential tremor. *Pharmacy professional.* March 2010. P6-41
- NICE Clinical Knowledge Summaries for PD: cks.nice.org.uk/parkinsons-disease
- SIGN 113. Diagnosis and pharmacological management of Parkinson's disease. A national clinical guideline. January 2010. sign.ac.uk
- bestpractice.bmj.com/best-practice/monograph/147.html
- Hypersalivation and oral glycopyrronium bromide: nice.org.uk/advice/esuom15/chapter/key-points-from-the-evidence

Reflection exercise 5

Now that you have completed this module, invite two patients with PD for a MUR and use some of the tips you have learnt here. If the patient cannot come to see you at the pharmacy, consider requesting permission to do a MUR by telephone.

This module is also online at pharmacymagazine.co.uk

PARKINSON'S DISEASE

assessment questions

1. What diagnostic test can be used to identify PD?
a. A CT scan
b. A MRI scan
c. Presence of a genetic missense
d. None. Diagnosis comprises the history of symptom onset, exclusion of other causes and baseline imaging

2. Which is NOT a first choice option in early PD?
a. Dopamine agonist
b. Levodopa and DCI
c. Beta-blocker
d. MAOI-B inhibitor

3. Which of the following is NOT recommended as a non-ergot derived dopamine agonist?
a. Ropinirole
b. Pramipexole
c. Cabergoline
d. Rotigotine

4. In relation to PD, which of the following is FALSE?
a. 20-30 per cent of all cases have a family history
b. In 20-30 per cent of all cases there is a familial history and corresponding genetic cause
c. Five per cent of all cases of PD are due to a genetic cause
d. Five per cent of all cases of PD are classed as juvenile parkinson's disease

5. Which statement regarding dopamine agonists is FALSE? They are:
a. Associated with risk-taking behaviours
b. Associated with binge eating
c. Theoretically more effective in early PD because they bind directly to post-synaptic receptors
d. Theoretically more effective in late stages of PD because they bind directly to post-synaptic receptors

6. As a class of medicines, which statement is TRUE about the two levodopa + decarboxylase preparations?
a. They work in the same way
b. If one does not prove clinically effective, neither will the other
c. They must never be used in combination
d. If severe side-effects occur with one agent, it is always worth trying the second agent

7. Which statement about selegiline is TRUE?
a. It can be used for its neuro-protectant effect in early PD
b. Oral selegiline can cause euphoria and insomnia
c. The wafer formulation can cause euphoria and insomnia
d. Oral dosing is always prescribed before 11am

8. Which pharmacological option in later PD should be taken 20 minutes prior to levodopa for best effect?
a. Amantadine
b. Apomorphine
c. COMT inhibitors
d. MAOI-B inhibitors

You may need to consult other information sources to answer the questions.

CPD record

Pharmacy Magazine CPD November 2015

Use this form to record your learning and action points from this module on **Supporting patients with Parkinson's disease** or record on your personal learning log at pharmacymagazine.co.uk. You must be registered on the site to do this. Any training, learning or development activities that you undertake for CPD can also be recorded as evidence as part of your RPS Faculty practice-based portfolio when preparing for Faculty membership. So start your RPS Faculty journey today by accessing the portfolio and tools at www.rpharms.com/Faculty.

Activity completed. (Describe what you did to increase your learning. Be specific)
(ACT)

Date: Time taken to complete activity:

What did I learn that was new in terms of developing my skills, knowledge and behaviours? Have my learning objectives been met?*
(EVALUATE)

How have I put this into practice? (Give an example of how you applied your learning). Why did it benefit my practice? (How did your learning affect outcomes?)
(EVALUATE)

Do I need to learn anything else in this area? (List your learning action points. How do you intend to meet these action points?)
(REFLECT & PLAN)

You can also record in your personal learning log at pharmacymagazine.co.uk

*** If as a result of completing your evaluation you have identified another new learning objective, start a new cycle. This will enable you to start at Reflect and then go on to Plan, Act and Evaluate. This form can be photocopied to avoid having to cut this page out of the module. You can also complete the module at www.pharmacymagazine.co.uk and record on your personal learning log**