Parkinson’s disease and services

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• Weston General Hospital
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Weston General Hospital, Weston.
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Consultant Referrals

• Neurologists specialising in PD/movement disorders - NBT
  Dr P Heywood
  secretary 0117 414 6689
  &
  Dr A Whone
  secretary 0117 414 6690

  Dept Neurology
  Southmead hospital
  Fax 0117 414 9475.

  Dr E Coulthard [PDD/LBD]
  secretary 0117 414 6691

  For urgent queries call on call neurologist via NBT switchboard
  0117 950 5050.

• Physicians with a special interest in elderly care & Parkinson’s NBT
  Dr J Richards / Dr R Barber
  secretary 0117 414 6444
  Admin is Southmead Hospital for Cossham PD clinic.

  BRI/SBCH
  Dr Tobin / Dr Allain
  Dept. of Medicine for the Elderly. BRI

  N Somerset
  Dr Kumar, Weston General Hospital or
  Specialist Older Peoples Team, Clevedon

  Neuropsychiatrists: Dr Seddon, Dr Mohan, Dr Herrod, Rosa Burden Centre, Southmead Hospital
Voluntary sector: Parkinson’s UK

- National Helpline 0808 800 0303.
- Website [www.parkinsons.org.uk](http://www.parkinsons.org.uk)
  - Have a wide variety of information resources, factsheets on every aspect of PD, including medications etc.
  - Have online training modules for GPs

- **Bristol branch of Parkinson’s UK** = peer support for patients & spouses, meet on the first Saturday of the month in Bristol. Also
  - a small group meet in Thornbury,
  - also coffee mornings, Tai Chi, Nordic walking, Irish Dancing & other events at various venues at various times across the year.
Parkinson’s UK Community Support

• Bristol, S Glos and BANES

  Kevin Carter
  Community Information & Support worker.
  Tel 0844 225 3697, Email kcarter@parkinsons.org.uk

• N Somerset

  Sarah Barter
  Community Information & Support Worker
  Tel 0344 225 3698
  Email sbarter@parkinsons.org.uk
Joint statutory and voluntary sector working

Parkinson’s DROP IN

• Run by Parkinson’s Nurse Specialist and Parkinson’s UK Community Support Worker
• On the first Thursday of the Month
• At various venues around the city [rolling venue schedule each month] for people in South Gloucestershire and Bristol e.g. two venues in S Glos and two in Bristol.
• Informal questions/advice/support over coffee and biscuits
Specialist Support

PSP Association
(supporting people with Progressive Supranuclear Palsy & Cortico Basal Degeneration (CBD))

PSP House
167 Watling Street West
Towcester
NN12 6BX

Tel 01327 322410
Email psp@pspeur.org
www.pspeur.org

Helpline for South West Region:
Jane Stein
Email Jane.Stein@pspassociation.org.uk

Multiple System Atrophy Trust

Southbank House, Black Prince Road, London, SE1 7SJ

Tel 0207 940 4666
Email office@msatrust.org.uk
Jill.lyons@msatrust.org.uk
www.msatrust.org.uk

Remember also
• Alzheimer’s Society Website have lots of useful information on behaviour in dementia etc. for families &
• The Carers Centre. 0117 965 2200
Parkinson’s nurse specialist role

Aim is to

• Improve quality of life & clinical outcomes. Reduce hospital admissions & GP attendances

• NICE PD Guidelines: “Specialist nursing care is a key development in improving care to people with PD and has quantifiable benefits in terms of reducing admissions and outpatient attendances”

Service =

• Telephone support, information & advice

• Outpatient clinics at Downend Clinic, Yate Westgate Centre, Thornbury Hospital OPD, [?Southmead].

• Home visits / Nursing home visits

• In-reach, ward reviews [NBT]

• Drop In

• Education and Training
Parkinson’s nurse specialist role

- **Provision of information, support and advice** to:
  - the patient
  - their partner/family
  - health and social care staff working with someone with Parkinson’s

- **Assessment**: symptoms [motor & non-motor]

- **Review of medications** & prescribing under *Independent Nurse Prescribing Guideline*. Helping to optimise medications & minimise side-effects to enhance QoL.

- **Referring on to MDT members** (Health, Social Services, third sector). Working in partnership to help establish an individualised package of care to help promote QoL (NICE). Equipment needs etc.

- **Spouse/Carer support** & ensuring they are aware of other support services, sign posting

- Acting as a **resource for patients and professionals**: sign posting to other services.

- **Informal counselling**

- **Education & Training**: raising awareness of PD
NICE Guidelines: Parkinson’s

• Refer untreated to a specialist Consultant in Parkinson’s

• Review diagnosis regularly

• NICE Guidelines: MDT early interventions, promoting healthy lifestyle, exercise, particularly physiotherapy, Occupational Therapy, Speech and Language Therapy

• Refer to specialist PD nurse

• “Regular review will allow appropriate management of non-motor symptoms such as depression, psychosis, dementia, sleep disturbance……”

• “Drug management can be complex and may need frequent adjustments….”

• “Improving communication with patients and families will help empower patients to participate in decision making and promote self-management”.

• Palliative care should be considered during all phases (give opportunity to discuss end of life issues)
PD NICE Guidelines

• Physiotherapy should be available, with particular consideration to
  – Gait re-education, improvement of balance
  – Enhancement of aerobic capacity
  – Improvement of movement initiation
  – Improvement of functional independence [inc. ADLs]

• Occupational Therapy should be available, with particular consideration to
  – Maintenance of work/employment and family roles, and leisure activities
  – Improvement and maintenance of transfers and mobility
  – Improvement of personal self-care activities such as eating, drinking, washing and dressing
  – Environmental issues to improve safety and motor function
  – Cognitive assessment and appropriate intervention
PD NICE Guidelines

• Speech and Language Therapy, with particular consideration of:
  – To improve vocal loudness, pitch range
  – Teaching strategies to optimise intelligibility
  – Ensuring effective means of communications are maintained, including use of assistive technologies
  – Review/management to support safety and efficiency of swallowing to minimise the risk of aspiration
  – Lee Silverman Voice Treatment

People with PD should have regular access to [which can be provided by a Pd Nurse Specialist]:

• Clinical monitoring & medication adjustment as required
• A continuing point of contact for support
• A reliable source of information about clinical and social matters of concern to people with PD, their spouses/family and sources of available support
• 100% of PwPD should have access to a Parkinson’s specialist nurse
Palliative care in PD

- NICE
  
  “Palliative care requirement should be considered throughout all phases of the disease, not limited to end of life stage.”

  “People with PD and their carers should be given the opportunity to discuss end-of-life issues with appropriate healthcare professionals”. Complete Advanced Care Planning Forms: share outcomes with other professionals/MDT

  - PD is a very heterogenous condition and patient specific, knowing when the time for EoL care has come can be difficult.

  - Early referral to palliative care services for their expertise, respite care/Day Care, counselling, home care services

  - For their view on symptom management [a fresh set of eyes]
Case 1

- Mrs S.T. a 63yr old widow, attends your sugery with shaking in her hands; worse on right.
- She has a PT job 4hrs a day in a canteen, has been spilling drinks etc. Worse in right hand.
- Has become anxious about retaining her job.
- Needs the money and enjoys the social aspect of her job.
- Recently a colleague also mentioned she seemed slower.

- Medication history: Aspirin, Simvastatin, Atenolol, Metoclopramide.

- Father had tremor.

What are the key issues for this lady?
Case 1

Key issues

- Wants/needs to maintain work, but tremor affecting work
- Slowing up
- Family history relevant or not?

- Elements to check
  - Is the tremor unilateral? Is it limited to hand (head)?
  - Gait: is it normal, or shuffling, arm swing, poor posture
  - Drug history: Metoclopramide – relevant?

- What are the possible differential diagnoses?
Case 1: Possible differential diagnosis

– Drug induced Parkinsonism (as Metoclopramide is a DA receptor antagonist)

– Essential Tremor [ET]

– iPD

What might your plan be?
Case 1

Plan:
• Stop Metoclopramide to see if symptoms improve
• Should you do bloods for thyroid etc.? Consider ET.....

N.B. ET doesn’t exclude PD (a significant % go on to develop PD)

• If symptoms subside when Metoclopramide is removed no further action
• If symptoms persist.......refer untreated to secondary care specialist in PD.
• In cases of diagnostic uncertainty specialist will refer for a DAT scan.
• If scan positive...........To treat or not to treat?
Case 1: Treatment options

Are symptoms interfering with functional ability/ ADLs/ QoL?

Yes - start PD medication

• Given her age what are your treatment options?

• What are your reasons?
Case 1

- Dopamine agonist & gradually titrate
  - Requip XL
  - Mirapexin/Pramipexole PR
  - Rotigotine [transdermal patch]

- Selegiline/Rasagiline [MAO-B inhibitor]

- But if need a ‘quick fix’ to do job effectively consider levodopa - Sinemet or Madopar 62.5mg tds for two weeks increasing to 125mg tds.

- Keep dose as low as possible and add in other classes when an increase is required.
Case 2

- Mrs BW, 62 yr, otherwise well
- 3yr history Parkinson’s.
- Now presents with discomfort in bed at night, includes cramp. Also taking longer to “switch on” in the morning i.e. feels more stiff and movements are slower at this time.

- Current treatment:
  - Madopar 125mg QDS
  - Zelapar 1.25mg OD [buccal melt form of Selegeline]

- What are the key issues for Mrs BW

- What would your treatment options be?
Case 2

Issues
• Lack of sleep, discomfort in bed. Cramp at night.
• Takes time to get going in the morning as ‘wearing off’ over night so first dose of morning ‘less effective’.

Treatment options for night time:
• Could add in a controlled release/modified release preparation of levodopa to be taken at bed time (e.g. Half Sinemet CR 25/100mg/Madopar 25/100mg CR/Caramet CR 25/100mg. Later increase CR to 50/200mg if required.

Treatment option for early morning:
• Could add a dispersible Madopar tablet in the morning as a “kick start”, 62.5mg (12.5/50mg) increase to 125mg i(25/100mg) if required; the dispersible form is quicker acting so can help reduce likelihood of a delayed “on” in the morning.

or
• Could add in a long acting/prolonged release dopamine agonist.
  Dopamine agonist options: Rotigotine/Neupro 24hr skin patch, Pramipexole PR once a day, or Requip XL
Case 3

• Mr AJ, A 69yr old male, lives alone, presents with a 3 year history of decline in function at home.
  – Slowing down, tasks of the day take longer.
  – Takes longer to get moving after sitting for some time

He has made an appointment to see you as he feels he is less independent and not getting out and about as he has previously.

• What are the key issues for this gentleman?
• What is the likely diagnosis?
Case 3:

• Key issues:
  – Normal activities of living are harder including washing and dressing. Things are an effort.
  – Keeping up with housework is likely to be harder
  – An effort to get out to see his friends so doing less, so risk of social isolation.

• What is the likely diagnosis?

• What is your management plan?
Case 3:

- **Possible diagnosis:**
  - Parkinson’s
  - Depression
  - OA
  - Check not SOB on exertion

- **Management plan:**
  - Physical examination/history and exclude other causes
  - Refer to a specialist untreated [NICE], who starts meds
  - Refer to a physio [NICE]
  - Once seen by specialist GP helps to titrate dose up.
  - Specialist or GP refers to Parkinson’s nurse specialist [NICE]
Case 4

- Mr P, a 70yr old man with IHD & PD. Lives with wife. Second floor flat, no lift. History:
  - Diagnosed with PD for 6 yrs [initial presentation fatigue, shuffling gait, stooped posture, rest tremor and micrographia].
  - Treatment now = Sinemet CR 50/200mg QDS and Ropinirole 6mg tds. Until recent months good symptom control, no recent PD specialist follow-up but now….
  - Drooling of saliva
  - Some coughing when eating
  - General decline in mobility, occasional falls
  - Low mood, daytime tiredness, early morning waking, worries about wife who has had recent diagnosis of OA.
  - Wife worried about his worsening memory, also has accused her of putting something in his food and has started to see a cat in the garden when he looks out, which she doesn’t see.
  - These symptoms fluctuate, sometimes mild confusion, other times lucid.

- What are the key points for a management plan?
Case 4: Issues

- **Drooling**: it may be hard to use cognitive strategies due to memory impairment so consider meds but may worsen cognition/memory. SLT can help.

- **Reduced swallow** (coughs on eating, common in PD): provide basic advice & refer to S&LT.

- **Possible anxiety & depression**: use a screening tool, talking therapies/meds. Together with anxiety, depression is a very common non-motor symptom of PD. May require Old Age Psychiatry referral if persists.

- **Hallucinations and possible paranoia** [or loss of sense of taste – common in PD]. Possible drug induced psychosis, or early signs of dementia. **Refer back to secondary care for review +/- memory assessment.**

- OT equipment needs in flat. Is housing appropriate in light of progressive mobility difficulty. Refer to OT, physio. Falls assessment.

- Refer to Parkinson’s nurse to review & for support; can adjust meds.

- **Increasing carer strain**: trigger referral for **Carers Assessment** for respite +/- care package, and a **needs assessment** for the patient
Parkinson’s disease

- Degeneration of the substantia nigra and nigrostriatal pathway causes depletion of dopamine in the basal ganglia, resulting in problems with initiation and control of voluntary movements: planning, regulating, sequencing, timing, and monitoring of movement.

- The mesolimbic dopaminergic pathway is also implicated in pathogenesis so alterations in motivated behaviour, emotional behaviour may exist.

- Other brain regions involved hence other neurotransmitters so dopamine is not the whole picture [research]
Classification of PD

- Early onset [long duration, high doses of medication, early motor fluctuations]. May have gene mutation.

- Tremor dominant [less motor fluctuations, less morbidity]

- Akinetic, rigid [increased incidence of depression & dementia, more rapid progression]

- Postural instability & gait disorder with falls [later onset]
Differential Diagnosis

Diagnosis of idiopathic Parkinson’s disease can be difficult, especially in early disease, 20% of PMs are found to have an alternative diagnosis

- **Idiopathic PD**
- **Essential tremor (bilateral hand, head, rarely rest tremor, often familial)**
  NB a significant % ET also develop PD; also nearly 25% PD found to be ET
- **Parkinson’s plus syndromes** (MSA – Multiple System Atrophy, PSP – progressive supranuclear palsy, CBD – corticobasal degeneration, LBD – Lewy body dementia), which have a more rapid progression with atypical features e.g. early falls, early autonomic dysfunction, gaze palsy, early dementia etc.
- **Parkinsonism** due to secondary causes:
  - Drug induced Parkinsonism – onset usually symmetrical, due to dopamine antagonists e.g. antipsychotics/neuroleptics [haloperidol]; anti-emetics: [Stemetil, Metoclopramide]
  - Vascular Parkinson’s (often lower body Parkinsonism, less responsive to Pd meds)
  - Normal Pressure Hydrocephalus, very rare
  - Trauma (repeated head trauma/boxing), very rare
  - Toxins (carbon monoxide, manganese, MPTP, etc. – target basal ganglia), extremely rare
  - Metabolic (Wilson’s disease: a dysfunction of copper metabolism (in the young)) very rare
Cause of Idiopathic Parkinson’s disease

- Unknown: likely to be multifactorial (an interaction between environment and genetics)

Risk factors include:

- Age, gender (oestrogen partly protective), Male>female (1.5: 1)

- Environmental factor may trigger: e.g. toxins such as pesticides, herbicides, industrial chemicals, heavy metals, farming communities.

- Genetic susceptibility (normal genetic variation which influences risk)

- Genetic mutations identified in approx. 5-10% of cases; autosomal dominant or recessive. Usually young onset PD (20s or 30s). Often have different phenotype (dystonia common & early dyskinesia).

Genes which encode the proteins alpha-synuclein and Parkin, implicated in PD, over 12 different mutations identified.
Endogenous causes of iPD

- Endogenous toxin(s) resulting in cell death
  - Oxidative stress (OS) medicates cell death in PD (free radicals/ROS are toxic to the cell so disrupt cellular function).
  - PM evidence of reduced AO defence mechanisms in PD (i.e. free radical scavengers such as glutathione, co-enzyme Q10 etc. reduced). Also the brain is more vulnerable to OS, specifically the S-Nigra
  - Mitochondrial dysfunction a key event leading to cell death as cellular function is disrupted due to low levels of ATP
  - Role of Iron? High in S-Nigra, Fe may inhibit complex 1 activity in mitochondria
  - Protein misfolding, aggregation, accumulation, so disrupt cellular functioning (packaged into Lewy bodies - ? To limit damage ?)
  - The normal system to clear proteins is disrupted hence accumulation.
  - Excitotoxicity: role of glutamate on receptors in substantia nigra
  - Synaptic dysfunction
Diagnosing iPD

No specific test in life so diagnosis (probable/possible) is made by:

- **Clinical history** *(unilateral onset, diagnostic criteria)*

- **Clinical examination**: may show cardinal signs; consider “red flags”: atypical features (diagnosis may be more complex in the elderly as 30% may have tremor, 50% rigidity, 90% slowness as part of natural ageing process)

- **Blood tests** to exclude other causes for symptomatology (thyroid, vitamin B12 etc.)

- **Brain scan (MRI / CT)** to exclude tumour, bleed, hydrocephalus, small vessel disease etc. [MRI not usually necessary if symptoms are typical]

**DAT scan**: aids diagnosis where there is uncertainly, so more able to commence treatment with confidence. Use where symptoms may be atypical or mild; helps differentiate between ET and tremor dominant PD
  - Radio labelled ligand binds to dopamine transporters (DAT) on the nigrostriatal presynaptic membrane – so used to assess integrity of terminals (extent of degeneration)
  - In PD there is a loss of DATs so reduced uptake of the ligand during scan (initial loss is in the putamen)

- **Diagnosis confirmed by post mortem examination** (Lewy bodies in CNS)
DAT Imaging
Diagnostic Criteria

Bradykinesia (slowness of initiation of voluntary movement with progressive reduction in speed and amplitude of repetitive actions) and at least one of the following:

• Muscular rigidity
• Rest tremor [4-6Hz] - 30% will not have tremor
• Postural instability [not caused by primary visual, vestibular, cerebellar or proprioceptive dysfunction]

Supportive prospective positive criteria for PD: three or more required for ‘definite’ diagnosis:

– unilateral onset; rest tremor; progressive disorder; persistent asymmetry; excellent response to levodopa; severe levodopa-induced chorea; clinical course for > 10 yrs
Epidemiology

• Approx. 1:500 of the population develop PD. Prevalence increases with age:
  
  • 1 in 100 over 65
  
  • 1 in 50 over 80
  
  • Average age of onset - 62

NB
• 1 in 7 of those diagnosed are under 50
• 1 in 20 are under the age of 40
Motor Symptoms

- **Mobility:**
  - *Initiation difficulties,* slow shuffling gait often with stiffness associated, (difficulty getting out of chair/bed etc.) – **so early physio, OT referral**
  - Lack of dexterity/fine finger movements (clumsiness) – **OT may help**
  - Postural instability:
    - Posture: stoop/forwards flexion, reduced arm swing
    - Poor balance particularly on *turning/changing direction*……often results in…..
  - **Falls:** a major cause for admission; 90% become fallers (early falls = red flag MSA, PSP). Contributing factors include: postural hypotension, visual disturbance, infection, environmental factors (poor lighting, rugs etc), age. Fractures are a major cause of morbidity and mortality. **So ref: Falls Assessment**

- **Tremor** – often at rest but also in action so may cause functional impairment *[OT]*

- **Festination** (short rapid steps, body propelled forward, difficulty stopping so often result in falls) *[Physio]*

- **Dystonia** (& cramp), often very painful

- **Restlessness:** akathesia

- **Freezing**
  - A motor block / an unpredictable gait failure. Temporary inability to move, especially when changing direction/turning
  - Cueing strategies may help (internal: targeted attention, mental rehearsal, external: strips, metronome), freezing often not helped by meds.
  - Freezing is not an “off” period, rather a shorter lived temporary “sticking “ of gait.
Falls in PD

• Inevitable due to balance disturbance
• Meds do not particularly help reduce falls
• Promote exercise to maintain strength
• **NICE:** early referral to physio & OT
• Consider cognition; lack of insight to risk
• Consider any postural hypotension
• Are other meds contributing
Non-Motor Symptoms of Parkinson’s disease

Non-Motor Symptoms (NMS) represent the greatest management challenge as do not generally respond to dopaminergic therapies as origin base is an alternative neurotransmitter or a mix of neurotransmitters.

NMS have a major impact on QoL
Autonomic symptoms

- Constipation
- Erectile dysfunction
- Urinary problems
- Postural hypotension
- Sialorhoea [drooling of saliva]
- Heat intolerance/excessive sweating
Constipation

- Can precede motor symptoms [? a prodrome]
- Will worsen PD symptoms, poor absorption of medication. May cause confusion in the elderly.
- What is their fluid input? Japanese study = lack of thirst, but also intake may be low due to fear of urgency/incontinence. Possible connection with apathy/lack of motivation.

Management:

- Try dietary changes, increase fluids, try linseeds, herbal remedies, encourage exercise. Med review – are other meds exacerbating the problems e.g. trihexyphenidyl etc.
- Laxatives
  - Osmotic laxatives: Movicol
  - Stimulant [Docusate Sodium, Senna] Try to avoid long term use
- Referral to Continence nurse specialist
Bladder disturbance/overactive bladder

Urinary problems include detrusor instability, hence frequency, urgency, nocturia, incontinence. Big impact on QoL, restricts lifestyle [where is the next toilet?]. Poor mobility will compound these symptoms.

• Severity of urinary symptoms has been shown to be related to neuro disability rather than the duration of the disease or age.

• Nocturia – significant for some, impact on sleep. Likely to need help physically getting out to use toilet/commode/bottle so impacts on spouse’s sleep. May increase falls risk.

• Urgency & functional incontinence so patients often avoid fluids, exacerbating the problem and compounding constipation.
Management of bladder dysfunction

• Give fluid/dietary advice: avoid caffeine, fizzy drinks, alcohol. Ensure drinking a sufficient quantity.

• Try pelvic floor exercises/bladder re-training.

• Refer to continence nurse specialist to establish if residual, advise on aids to help, may need convene, particularly for night.

• Try antimuscarinic. Solifenacin and Trospium are drugs of choice as less central side effects [don’t’ X BBB to same extent as Oxybutynin etc]. Mirabegron.

• In males there may be a double impact from benign prostatic hyperplasia: enlargement / obstruction.

• Consider referral to neuro-urologist
Erectile dysfunction & female dysfunction

- Reduced arousal, reduced interest, hard to reach orgasm/ejaculate, may result in stress & depression. Do other meds for co-morbidities contribute?

- Consider referral to erectile dysfunction (urology) nurse specialist.

- Consider Phosphodiesterase type-5 [PDE 5] inhibitors such as Sildenafil (Viagra), Tadalafil (Cialis), start with small dose, then increase until the effective dose is found.

- Female dysfunction: lubrication, stimulation etc. [menopause]

- ALSO consider effect of bradykinesia and rigidity on sexual ability/performance. Might seem too ‘staged’ as have to pick good times.

Postural Hypotension

- Often occurs after several years of PD, and is more common in the elderly [so possibly multifactorial]. Occasionally seen in early disease (particularly in MSA).

- Defined as fall in B/P of at least 20mmHg systolic or 10mmHg diastolic within 3 mins of the standing position.

- Check B/P immediately then 1 min after standing, then if differ re-check at 2 min intervals until returned to baseline or 5 min have elapsed.

- Review anti-hypertensives. Check fluid history, increase if low & add salt to diet, compression stockings. Raising head of bed 30°.

- Fludrocortisone [with salt and fluid] if resting B/P acceptable, titrate dose accordingly. Check b/p regularly.

- Rarely Midodrine, should be accompanied by regular B/P monitoring.
Heat intolerance/excessive sweating

- Often affects face/head and neck
- Related to the disease itself rather than being a side effect of medication i.e. a primary autonomic abnormality, which sometimes is improved by levodopa.
- PM studies of PwPD have shown hypothalamic lesions.
- Studies have suggested low concentration of DA in hypothalamus might cause profuse sweating.
- Off symptom sweating should be distinguished from the sweating that can occur during dyskinesia [this isn’t usually as profound as the off period “drenching sweats”]
- Sweating is usually seen in fluctuating patients rather than those without motor fluctuations.
Sialorhoea: drooling of saliva

• Impaired/infrequent swallow rather than hypersecretion. Can affect up to 78% PwPD

• Results in pooling of saliva and increased risk of aspiration pneumonia. Pooling often results in drooling, often compounded by a flexed head posture

• Is socially embarrassing and can lead to isolation, so may worsen an existing depression
Treatments for drooling of saliva

- Information sheets, cognitive strategies / cueing (swallow reminder), positioning
- Refer to SLT for strategies to help
- Gum, sugar free sweets [bring swallowing to the conscious], sage capsules, red grape juice

**Anticholinergics:**
- Hyosine/Scopolamine patch [1mg/72hrs]
- Atropine (eye drops) sublingually (0.5mg 1%, one drop sublingual BD)
- Atrovent spray in mouth (not much evidence for) ?

- NB: side effects especially in the elderly (confusion, blurred vision, constipation, urinary retention) often limit their use

- Amitriptyline ?
- Botulinum toxin injections
Non-Motor Symptoms

• **Communication**
  - non-verbal e.g. reduced facial expression; micrographia
  - verbal e.g. monotone, hypophonia, dysarthria, slowness of thought processing (bradyphrenia) hence a delay in producing a response, so allow time, don’t rely on spouse. Consider advanced care planning early if speech difficulty.

• **Swallowing difficulty** – dysphagia, delay in triggering swallow. May result in weight loss. Patients advised on altered textures/soft diet, dietary supplements. Risk of aspiration pneumonia, dehydration, malnutrition may follow.
  In advanced disease PEG may be appropriate: know your patient’s wishes, weigh up benefit.

Referral to Speech and Language Therapist for speech & swallow assessment as well as for help with saliva control.
Non-Motor Symptoms

• **Sleep disturbance (40-98%)**
  - difficulty turning in bed (due to rigidity), discomfort. Pain
  - Cramp. Early morning dystonia
  - Anxiety and depression [panic attacks]
  - RLS
  - insomnia [includes frequent waking]
  - vivid dreams, nightmares
  - PLMS
  - REM behaviour disorder [15-60% v 0.5% gen pop. 20% REMBD developed PD within 5yrs]
  - hallucinations
  - Nocturia

  - **Hypersomnolence in daytime** [can be of sudden onset, e.g. fall asleep when talking/eating. Increases with disease duration, and cognitive decline, & meds]

Promote sleep hygiene, treat any anxiety & depression, consider RLS treatments, night sedation

Some patients may require sleep studies

• A significant impact on QoL
Non Motor Symptoms

- **Behavioural change**
  - Mood change. Frustration, loss of self confidence, social embarrassment (due to tremor, dyskinesia)
  - Personality change: rigid/ lack flexibility of thoughts, routine driven
  - **Apathy**

- **Depression & Anxiety [& panic attacks].**
  Depression may precede motor symptoms [so is it a risk factor or a prodromal feature?]
  - Both are a major influence on QoL
    - Are associated with increased risk of disability
    - Often seen in younger onset PD & elderly [bimodal pattern]
    - 2 x impact of motor problems
    - Anxiety hard to treat

- Try relaxation / self help strategies, other primary care interventions / counselling, CMHT. Optimise PD meds.

- Citalopram. Mirtazapine [as noradrenergic loss also] (15mg for anxiety).
Non-Motor Symptoms

- **Fatigue** (one of the most disabling symptoms)
  - Is separate from excessive daytime sleepiness
  - Increases with disease progression
  - Rating scales exist
  - Optimise dopaminergic meds
  - Has a significant impact on QoL

- **Visual impairment**
  - blurred &/or double vision
  - impaired visuospatial function, ↓ spatial orientation & awareness results in an increased falls risk. Refer to optician to optimise vision.

- **Sensory disturbance & Pain**
  smell (early anosmia/hyposmia 68% at diagnosis) & taste perception

  **Pain**: 60-87% prevalence:
  More pain when “off” including dystonic pain.
  - RLS nocturnal pain (three DA agonists now marketed for RLS)
MCI and Dementia

- Cognitive and memory problems
  - Many patients experience MCI [even in early stages]
  - Dementia is common (around 30% +)
    ↓ concentration & attention, word finding difficulties, ↓ problem solving, ↓ planning, ↓ concept formation, visuo-spatial impairment, altered sleep:wake cycle etc.
  - Recognising a change – subtle initially, spouse may do all the taking, spouse may unintentionally take over.
  - Try AchE inhibitor such as Rivastigmine at an early stage as may stabilise cognitive and functional abilities. [ECG first as contra-indicated in bradycardia].
  - A diagnosis can be helpful for the family, can open up routes for support.
  - Remember practical issues such as LPA, a Will, advanced care planning etc. AD website. Carer support.
Treatment for PD

- A Non-pharmacological approach to the treatment of PD is often just as important as pharmacological options and should run along side medicine management.

- NICE Guidelines: MDT early interventions, promoting healthy lifestyle, exercise (known to improve outcomes, possibly increases BDNF in CNS). With disease progression/increasing disability re-referral to MDT over regular intervals is key to effective management.
Voluntary Organisations
Psychological Interventions
Carer Support
Complimentary Therapies
Psychological Interventions
Voluntary Organisations
GP & Specialist
Social Services
PD Nurse Specialist
Community Nursing
Physiotherapy
Occupational Therapy
Speech & Language
Dietician
Continence Advisor
PARKINSON'S
Medications for PD: PD NICE Guidelines

- Patients should be referred to a specialist untreated.
- Review diagnosis regularly.
- Medication should be delayed until the degree of motor disability interferes with ADLs/QoL.

As no cure aim is to alleviate symptoms by correcting dopamine deficiency:

- **Pharmacological.** No standard treatment, rather treatments should be directed to the patient’s symptoms taking into account situational factors, age, other concurrent medical diagnoses.

  No evidence of neuroprotection.

No need to rush into prescribing. Is there functional impairment? Is there an urgent need to prescribe (young at work)? Consider long term effects. There is no universal first choice of medication [see NICE PD Guidelines].
Pharmacological Treatment

• Dopamine replacement: Levodopa (L-dopa) (a precursor to dopamine)
  
  - Sinemet/Co-careldopa
  - Madopar/Co-beneldopa
  
  - Various preparations exist: standard, controlled release, dispersible
  - Levodopa does not restore “normal function” within the basal ganglia (mechanisms of storage and release differ from normal physiology)
  - Short half life so regular dosing required over the day (TDS, QDS → ↑)

• Short term side effects:
  - nausea (co-prescribe with Domperidone)
  - postural hypotension (may need Fludrocortisone at some point)

• Long term side effects:
  - motor complications: dyskinesia, motor fluctuations, so delay use where able

NB: Sinemet/Madopar have the potential for competition with dietary amino acids resulting in delayed onset of action or dose failures (individual variation in gut transit time affects absorption; gut transit time can be delayed in PD) so aim to give L-dopa at least 30-45 mins before food

NB: A small % of PwPD will be unresponsive to L-dopa

• Duodopa (new gel based L-dopa: intra-intestinal (PEJ) delivery via pump over waking day/24hrs)
Dopamine Agonists

- **Pramipexole** (Mirapexin) PR, prolonged release once a day prep’ or immediate release TDS.
- **Ropinirole** (Requip XL) 24hr prep or immediate release TDS.
- **Rotigotine** (Neupro) a 24hr transdermal patch OD
- **Apomorphine** (subcutaneous bolus injection, via a APO-go PEN or continuous infusion via specific Apo-Go pump)

- Mimic effect of dopamine (act by directly stimulating post synaptic dopamine receptors)
- Longer duration of action than L-dopa, so less motor fluctuations
- Used as monotherapy or an adjunct
- Gradual titration to minimise side effects so takes time to achieve optimum effect
- No competition with dietary amino acids so less problem with variable absorption

Side effects (more prominent in the elderly) include:
- **nausea** (usually co-prescribe Domperidone), give at meal times
- **postural hypotension/dizziness/syncope** – may result in falls, faints
- **peripheral oedema** so may need to reduce dose
- **daytime sleepiness** (somnolence) can be a sudden onset of sleep (inform risk driving & operating machinery), reduce dose if significant
- Neuropsychiatric: psychosis: **hallucinations**, confusion, **paranoia**, delusions (reduce dose)
- **Impulse control disorders / compulsive behaviours** including: hypersexuality, gambling, excessive shopping, over eating (due to dysfunction in limbic /DA reward systems in the brain), reduce and possibly stop.
Pharmacological Treatment

• **Anticholinergics: treatment of tremor** (aim to restore DA:Ach balance)
  – Caution in elderly due to potential side effects
    • **Trihexyphenidyl**
      – Side effects: dry mouth, **confusion**, constipation, retention, blurred vision

• **Enzyme inhibitors**: prevent breakdown of dopamine/l-dopa peripherally +/- or centrally
  (COMT = catechol-O-methyl transferase, MAO-B = monoamine oxidase B breakdown l-dopa/dopamine so enzyme inhibitors minimise this metabolism)
  – **Selegeline** a centrally acting MAO-B inhibitor, given OD or BD, take a.m. as can disturb sleep
  – **Zelapar** a buccal melt form of Selegeline, OD, a.m.
  – **Rasagiline/Azilect** a MAOB Inhibitor, OD, a.m.
  – **Entacapone** a peripheral COMT inhibitor, taken with each dose of levodopa, **not** separately, it enhances efficacy of levodopa
  – **Tolcapone** a centrally & peripherally acting COMT inhibitor, TDS (**need 2wkly LFTs**)  
  – **Stalevo** a combination tablet Sinemet and Entacapone (may improve adherence)

• **Others**
  – **Amantadine** (glutamate antagonist) **used in the treatment of dyskinesia** – exact mechanism of action unknown, ? via NMDA receptors. Start once a day, BD (TDS occasionally). May cause confusion and hallucinations. Take no later than 3p.m. as can disturb sleep
NICE guidelines 2006: Options for treatment

- Nice 2006: refer untreated to specialist. Treatment choice is individual.

Early Disease
- Young or those with little co-morbidity - a dopamine agonist (Pramipexole, Ropinirole, Rotigotine). Generally less suitable in elderly due to side effect profile. Also sl less effective so if still working may need levodopa as treats symptoms more effectively, but delay use of l-dopa in young where able.

- The elderly: levodopa = Sinemet or Madopar
  - Various preparations: regular, controlled release, dispersible.
  - Keep dose as low as possible to reduce risk of motor complications.

- Early monotherapy: MAO-B inhibitor (Rasagiline/Azilect. Selegeline/Zelapar). Less effective in symptom control but may ‘buy time’

- Anticholinergics: Trihexyphenidyl [risk of cognitive impairment but can be very useful to treat early stage tremor dominant PD.]
<table>
<thead>
<tr>
<th>Initial therapy for early PD</th>
<th>First-choice option</th>
<th>Symptom control</th>
<th>Risk of side effects</th>
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<td>Motor complications</td>
<td>Other adverse events</td>
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<tr>
<td>Levodopa</td>
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<td>Dopamine agonists</td>
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<td>MAOB inhibitors</td>
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<tr>
<td>Anticholinergics</td>
<td>✗</td>
<td>Lack of evidence</td>
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<tr>
<td>Beta-blockers</td>
<td>✗</td>
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<tr>
<td>Amantadine</td>
<td>✗</td>
<td>Lack of evidence</td>
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</table>
NICE guidelines: Later disease

The majority of patients will need levodopa at some stage (Sinemet or Madopar).

• Use an adjunct to reduce motor fluctuations: no one class has been found to be superior to another [PD NICE]
  – Dopamine agonist
  – MAO-B inhibitor (Rasagiline/Azilect. Selegeline/Zelapar].
  – COMT inhibitors (Entacapone / Stalevo)

• For dyskinesia: Amantadine

• Apomorphine subcut’ pump for waking day for motor complications including dyskinesia  [cost]

• Duodopa intestinal gel for waking day administration via PEJ  [cost]
<table>
<thead>
<tr>
<th>Adjuvant therapy for late PD</th>
<th>First-choice option</th>
<th>Symptom control</th>
<th>Risk of side effects</th>
<th>Other adverse events</th>
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<tr>
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<td>COMT inhibitor</td>
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<td>MAOB inhibitors</td>
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<td>Amantadine</td>
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<td>Apomorphine</td>
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<td>Duodopa</td>
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Summary of common side effects

• **Nausea.** Co-prescribe with Domperidone if it occurs [10mg tds with or just before the PD meds]

• **Peripheral oedema.** Elevate legs, compression stockings, reduce the dopamine agonist or may need to stop it

• **Postural hypotension.** Monitor fluids, check sitting and standing b/p, add fluids & salt to diet if low, compression stockings, [raise head of bed], Fludrocortisone [Midodrine].

• **Daytime sleeping.** Excessive, sudden onset. Reduce meds if possible and if a bother; warn about not driving etc.

• **Hallucinations and paranoia.** Exclude UTI, chest infection, constipation, a recent fall/head injury etc. Reduce meds where able [stepwise reduction in the various classes, some more of a culprit than others: seek advice]. Try Rivastigmine [check ECG 1st] or Quetiapine. Ref to Old Age Psychiatry or CMHT. Red flag for LBD.

• **Compulsive behaviour.** Gambling, over spending, sex, porn on-line, over eating. Reduce meds, GamCare, Gamblers Anonymous etc.

• **Motor Complications:** dyskinesia, wearing off, dose failures, On-Off phenomenon.
Potential problems with pharmacological approaches

After several years of treatment the margin between benefit of medication and unwanted effects becomes progressively narrower. Over 50% of pts develop motor complications after 3-5 yrs (some much sooner):

- **Dyskinesia**: involuntary abnormal chorea like movements

- Motor Fluctuations
  - ‘Wearing off’ of medication, i.e. therapeutic response to L-dopa shortens over time resulting in a return of symptoms at the end of each dose.
  - **Dose failure** &/or significant delay in onset of action of a dose (due to poor absorption)

Timing of medication can therefore be crucial for patients with PD.

- **“On-Off” effect** characterised by very sudden changes in mobility from being “on” (mobile) to being “off” (immobile) within minutes. Can occur randomly several times over the day, can be unrelated to medication timings. Off periods can be associated with non-motor symptoms e.g. anxiety & panic, shortness of breath, depression. (On periods can be associated with a euphoric mood and hyperactivity)
Age differences regarding likelihood of developing Motor Fluctuations

- Pre 40yrs – 100%
- 40-59yrs – 50%
- 60-69yrs – 25%
- Over 70yrs 16%

Motor fluctuations are thought to be related to absolute levodopa dose, so keep low.

Taken from: Vlarr, A et al 2011. Practical Neurology, 11, 145-152,
Strategies to help reduce motor complications

- Seek advice from PNS [email] or specialist for following possibilities

- Delay introduction of L-dopa / keep levels as low as possible

- Prescribe adjuvant drugs

- **Wearing off** – try adding other classes of meds to enhance therapeutic control e.g. COMT inhibitor, a DA agonist, a MAO-B inhibitor. Increase frequency +/- dose. Try addition of CR preparation in daytime.

- **Delayed “on” or dose failure**: review timings of meds, ensure not given with food, manipulate protein load to evening. Try addition of an agonist, try a “kick start” with Madopar dispersible

- **Dyskinesia**: reduce meds (particularly L-dopa) if possible; could add COMT inhibitor or agonist if lack of efficacy on lower dose if clinical context allows. Try Amantadine: titrate up, give no later than 3p.m.

- **On-Off syndrome**: try adjunct e.g. agonist or MAO-B inhibitor or COMT inhibitor. Try Apomorphine. Try Duodopa
Progression of Parkinson's disease

- **Early phase**
  - "the good years"
  - Ability to store dopamine
  - Threshold for morning dose only

- **Wearing-off**
  - Short off-periods

- **Wearing-off with dyskinesias**
  - Predictable fluctuations with peak-dose dyskinesias
  - Defined therapeutic window

- **On-off fluctuations**
  - Unpredictable fluctuations
  - Very narrow therapeutic window

- **L-Dopa Blood level**

- **Time (years)**
  - ~0-4
  - ~4-7
  - ~7-10
  - ~>10

- **Therapeutic Window**

Colors:
- Green = Normal mobility
- Blue = Parkinsonism
- Red = Dyskinesia
Further considerations with medications

- Avoid neuroleptics due to risk of Neuroleptic Malignant Syndrome, as they block DA receptors in CNS (DA antagonists) e.g. Haloperidol
- Avoid other dopamine antagonists such as Metoclopramide & Stemetil
- Try to avoid benzodiazepines (but may be needed for anxiety)
- Review anti-hypertensives: do they need reducing in the light of postural hypotension?
- Age (biological vs chronological in prescribing decisions)
- Cognitive impairment predisposes hallucinosis (so go easy on meds)
- Involve patient in prescribing decisions (adherence involves joint decision making/a shared agreement, some wish to delay treatment)
- PD meds should not be withdrawn abruptly due to risk of NMS
- See SPC if in doubt, or ring for advice
- V. rare behavioural problem = Dopa dysregulation syndrome = early increases/self-escalation in meds’, resistance to reduce (hoarding), > male, punding, gambling, criminal behaviour, hypersexuality
Some General Considerations

• **Individuality of symptoms:** people vary in how PD affects them (wide phenotypic range).

• Symptoms may vary day to day or hour to hour (good and bad days).

• Drug tolerance varies between people so individual management is key: there is no “one size fits all” in medication management.

• Symptoms and the way the medication works will be influenced by mood/emotional state (anxiety/depression) & by diet/absorption (erratic or a delay in gastric emptying will influence medication absorption)

• Other concurrent illnesses / infections (UTI, chest etc.), constipation, may worsen PD symptoms so require prompt treatment.

• Adherence issues. Regimens can be complex: is the patient able to manage their own meds (compliance aids: dossett box).
Anticipatory Care Management of an Acute Deterioration in Parkinson’s Symptoms

Consider these 4 Steps

1. Is infection present?
   NB: PwP do not always display signs of infections therefore consider carrying out the tests/observations below;
   1. Urinalysis if positive send to microbiology
   2. Blood tests for infection, delirium, falls inc: FBC, U&E’s, CRP, TFT’s, B12 folate, LFT, calcium, random glucose.
   3. Examine chest - risk of aspiration refer to SLT for swallow assessment
   4. Any wounds, infected rashes
   *Falls Check lying & Standing BP after 3 minutes to rule out postural hypotension
   Complete a Multifactorial Falls Assessment

2. Is the PWP constipated
   Have bowels opened within the last 2/3 days (or consistent with patient’s normal bowel habit

3. Has the patient been prescribed any new medication within the last month?

4. Is the PWP taking their medication as prescribed?
   Check the dose, time, formulation is correct. Any missed doses. Any PD medication side effects, or that of other medications they are taking

Positive infection screen
   Once treated patient should return to their normal level of functioning

If PWP has symptomatic postural hypotension
   1. Check PwP is drinking adequate fluids
   2. Check & review other medications e.g. anti-hypertensive’s
   3. Consider compression hosiery if not contra-indicated to other conditions e.g. vascular/arterial
   4. Refer to Specialist Nurse to discuss Parkinson’s medication regime to see if requires adjustment
   5. Refer to Specialist Consultant for discussion as to whether anti-hypotensive medication indicated and appropriate for the patient

Devised by Kay Baggle, Kathryn Prout, Parkinson’s Nurse Specialists
Adapted from Barnes, L PDNS, Peninsula Community Health
Increase in PwP Social care Needs/Carer Stress
Consider referral to Care Direct 01179222700
Carers Centre 01179652200

Refer to Parkinson’s Nurse Specialist
Kay Baggle PNS South, Inner city & East Bristol 01179190289
Kathryn Prout PNS North & West Bristol, S. Glos 01174141593

Consider simplifying the drug regimen if possible in conjunction with the Parkinson’s Nurse and refer to the Community Pharmacist/GP dispensary for assessment if necessary

Yes

Consider discontinuing the medication or use an alternative & symptoms should improve after two weeks

No

Yes

Yes

Yes

No

Yes

No
How to Manage A Deterioration in Your Symptoms if you have Parkinson’s

Consider these 4 steps

1. You may have an infection?
   Do you have urinary symptoms?
   Obtain a urine pot & take a urine sample to your surgery for testing.
   Your GP may also want to examine your chest if you are coughing or if your breathing is problematic, any skin rashes if present &/or take bloods for infection/falls, measure your lying and standing blood pressure
   - Yes

2. Are you constipated?
   Has it been more than 2/3 days since you last opened your bowels
   - Yes
     Constipation present
     Increase your decaffeinated fluid intake to 1.5 litres per day
     Increase your fibre intake
     If no improvement discuss with your GP whether you need to start a laxative or if currently taking, change to one which is going to be more effective
   - No

3. Have you bought or been prescribed any new medications within the last month?
   - Yes
     Is it a medication to avoid in Parkinson’s?
     - Yes
       Is it one of the medications listed overleaf?
     - No

4. Are you taking your Parkinson’s medication as prescribed and on time?
   Have you missed or delayed any doses?
   - Yes
     If YOU ARE CLEARLY VERY ILL SEEK MEDICAL ADVICE
   - No
     Require Support for possible increased social care needs/carer stress
     Consider contacting Care Direct-01179222700
     Carers Centre-01179652200
     South Gloucester Social Services-01454868007
     Telephone your:
     1. Community Pharmacist
     2. GP
     3. Parkinson’s Nurse Specialist

   - Requiring Support for possible increased social care needs/carer stress
   - Devised by Kay Bagley, Kathryn Prout, Parkinson’s Nurse Specialists
   - Adapted from Barnes, L PDNS, Peninsula Parkinson’s Nurse Specialists

   NB: it could take a couple of weeks for symptoms to settle
   If you have ongoing concerns with your bladder you may wish to contact your local Continence Advisor for support

   If found to have an infection
   Once successfully treated you should return to your normal level of functioning & your Parkinson’s medication will not need to be changed during this time

   If you are having difficulties remembering to take your medication on time then speak to your community pharmacist about medication aids to prompt you

   Devised by Kay Bagley, Kathryn Prout, Parkinson’s Nurse Specialists
   Adapted from Barnes, L PDNS, Peninsula Parkinson’s Nurse Specialists
Drugs to Avoid: dopamine antagonists

- Prochlorperazine/Stemetil
- Metoclopramide/Maxolon

Antipsychotics/Neuroleptics e.g. phenothiazines & butyrophenones:
- Chlorpromazine/Largactil, promazine
- Haloperidol/Serenace/Haldol
- Perphenazine/Fentazin
- Fluphenazine/Moditen
- Flupentixol/Fluanxol/Depixol

Antidepressants:
- Perphenazine/Triptafen
- Thioridazine
Adherence / Concordance

• Complex PD med regimens in addition to polypharmacy for concurrent conditions (complexity of drug regimen is inversely related to compliance).

• Under use due to fear of side effects

• Over use to prevent dips in function, dislike ‘wearing off’ so may self escalate meds

• Patient’s can mistake symptoms for side effect so may stop meds [more tremor etc. on initiating therapy]. Patients can mistake side effects for symptoms so take extra meds [dyskinesia]. Hence a need for education.

• Discuss expectations: ensure realistic understanding of benefit of meds i.e. NOT A CURE, e.g. temor v hard to treat.

• Cognitive impairment is common in PD & may influence adherence; compliance aids [dosset box, blister pack] may be difficult to learn

• Some pts stop meds due to poor communication between specialist and GP e.g. GP not informed of change soon enough so no repeat script issued

• Grosset, KA. et al. (2005) found less than half of PwPD take their medication well, only 10% take it as prescribed. 1/5th took less than 80% of their prescribed meds. Even in those that took the correct number of tablets the timings were irregular. 10% took over 110% of the prescribed meds. Poor timing is universal!
Adherence

• Erratic medication taking/variation in number of tablets taken a day will result in fluctuations in plasma levels hence motor fluctuations more likely.

• Other factors for poor compliance include lack of knowledge, younger age, lack of social support & depression. 40% + PwPD experience depression, so may be a significant influencing factor [depressed people are 3 x more likely to be non-compliant]

• Grosset et al. scenarios e.g. a 40yr old who is having problems coming to terms with diagnosis says meds are not working, yet only taking 40% of their prescribed meds.

Management decisions depend on response to therapy therefore assumptions of full compliance may mislead the process.

• Newer therapies e.g. 24 hr delivery (agonists) should help adherence. Salt and base forms of Pramipexole complicate the picture.
Summary

- PD is a progressive neurological condition affecting **numerous systems in the brain** including motor, cognitive and emotional processes and autonomic function so in addition to motor symptoms non-motor symptoms need treating to reduce impact on quality of life.

- **A MDT approach** is key in order to optimally manage patients and provide support to them and their family to help maintain quality of life.
Useful websites

• www.nice.org.uk for NICE PD guidelines
• www.parkinsons.org.uk - the national charity for PD
• www.cureparkinsons.org.uk
• www.cks.library.nhs.uk/parkinsons

References

www.gerimed.co.uk - article in their archive: Aug 2010 “Parkinson’s disease and primary care”
Multiple System Atrophy - MSA

Characterised by: autonomic dysfunction (brain stem), parkinsonism (basal ganglia), ataxia (cerebellum): MSAa, MSAp, MSAc.

- Onset is around 52yrs, slight increase in prevalence in men 1.2:1 Prevalence 6 per 100,000 in UK (1 for every 40 PD patients). Rare over 70yrs and under 30yrs.

- In general more rapid disease progression compared to PD: 5-10 yrs.

- Cause unknown as in PD.

- Key symptoms: Autonomic dysfunction
  - postural hypotension (a drop of 20mmHg on standing) = dizziness & falls, altered consciousness; coat hanger pain
  - male erectile dysfunction
  - bladder dysfunction (urgency, frequency, nocturia, retention)
  - RBD
  - breathing: excessive snoring, inspiratory sighs & stridor, sleep apnoea [may need CPAP, trachy]
  - sweating disturbance
  - constipation
  - cold extremities
Management of MSA

- seek expert advice: Neurologist & Multiple System Atrophy Trust
- treat postural hypotension (mechanical, salt, fluids, drug strategies)
- anti-Parkinson's medication less effective, try higher doses. (Amantadine may help)
- help treat / support antecollis
- manage secretions
- promote independence with physio, OT, (Falls, seating, adaptations at home etc), S&LT (communication and swallowing)
- bladder management (continence nurse specialist may help in assessment and management, anticholinergics, suprapubic catheter)
- treat constipation
- sleep and breathing: sleep studies (polysomnography), CPAP, tracheostomy
- palliative care services, inc respite. Carer support throughout
Progressive Supranuclear Palsy - PSP

- First described 1964 by Steel & Richardson
- Characterised by a progressive slowness/limitation of vertical (upward and downward) gaze, disturbed balance, dysarthria; a % related to mutation on tau gene Ch 17
- Prevalence 5 per 100,000 +
- Incidence 1-2 in 100,000 (5% prev’ PD)
- Onset 50-70 yrs
- Progresses relatively quickly; 5-10 yr illness

Other features:
- early falls, often backward (righting reflex significantly impaired)
- upright posture, unsteady, irregular walk/gait, without insight into severity, spastic speech, swallowing problems, blurred vision resulting in disturbance in vision and in involuntary eye closure, personality change & cognitive impairment
- Tremor is rare
- Poor response to L-dopa (try Amantadine)
Corticobasal degeneration (CBD)

First described by Dr Rebeiz in 1965/68; is a very rare progressive neurodegenerative condition which usually occurs in middle life. Pathology includes neuronal loss in cortex (specifically frontal and parietal lobes) and basal ganglia; cellular accumulation of tau protein as in PSP.

It is characterised by a disturbance in motor function/movement (motor features are similar to those seen in PD) & intellectual impairment. The duration of disease is approximately 7-10 years.

Key features include:
- rigidity/stiffness, poor balance, gait disturbance and clumsiness, often without tremor
- visual perceptual difficulties & limitations in eye movements similar to PSP.
- dysarthria
- tremor and involuntary movements (jerking of the limbs) and abnormal posturing (dystonia)
- personality change: disinhibited and normal personality exaggerated
- memory problems; organizational skills diminish. May progress to dementia
- sensory impairment
- Limb apraxia: ‘alien limb’ - a limb may function in a slow involuntary wandering way (the limb having a mind of its own, won’t do that its owner wants
- Management – supportive, PD drugs rarely help
Lewy Body Dementia (LBD)

- A sporadic condition representing approx 20%+ of dementias. Cause unknown. Degeneration of substantia nigra: dopaminergic cell loss so features of Parkinson’s in addition to dementia.

Features include:
- **Early** visual hallucinations are common.
- Fluctuating episodes of confusion – so behaviour & ability will vary day to day (hour by hour). Confusion is generally worse at night.
- Loss of memory, language, reasoning, and calculation skills.
- Motor signs – as in PD.
- Symptoms of Parkinson’s may develop a couple of years into the illness – hence is often mistaken for Parkinson’s dementia.
- More rapid decline compared with PD.

** Patients with LBD are sensitive to neuroleptics (sedatives such as haloperidol). Severe neuroleptic malignant syndrome (NMS) can develop in patients given these drugs – patients become rigid and akinetic and coma may follow – so AVOID.**