PARKINSON'S DISEASE: DIAGNOSIS AND MANAGMENT

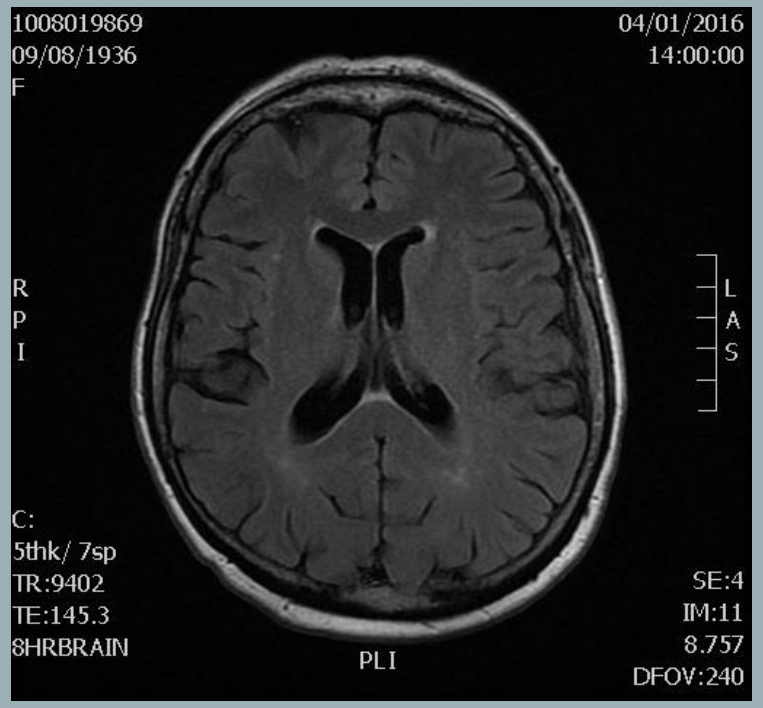
Emke Maréchal

March 2024

CASE, FEMALE 79J

History

- Micrografie
- Right leg drags, balance problems
- Tremor right hand at night
- Reduced smell.
- No memory problems, no autonomic symptoms
- No RBD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PLI
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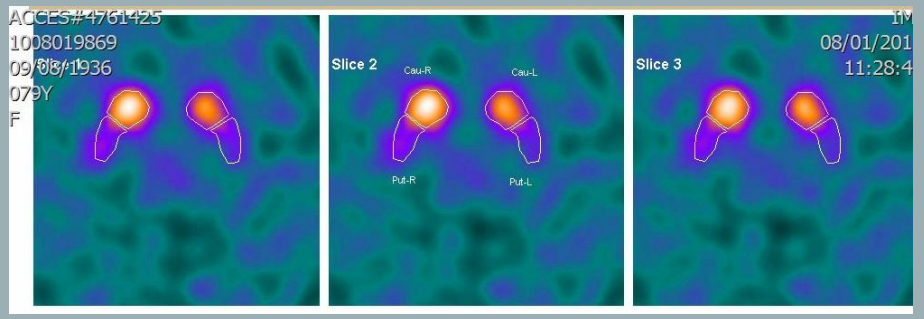
DFOV:240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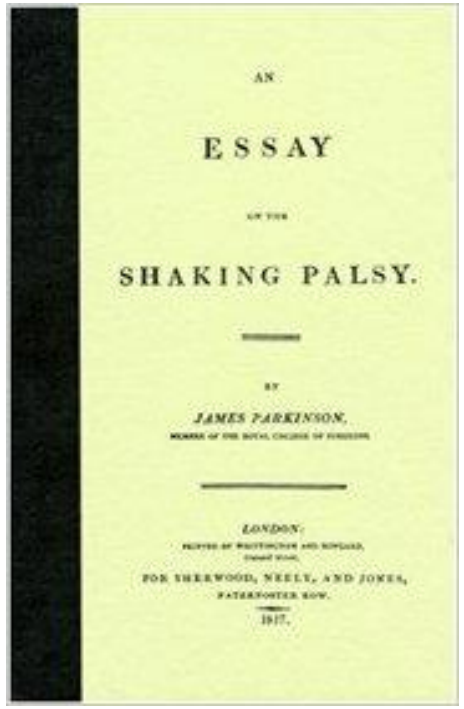
CASE

CONTENT

- Epidemiology
- Pathophysiology
- Diagnosis and disease course
- Parkinsonism – differential diagnosis
- Current treatment options
- Future treatment options


AN

ESSAY

ON THE

SHAKING PALSY.

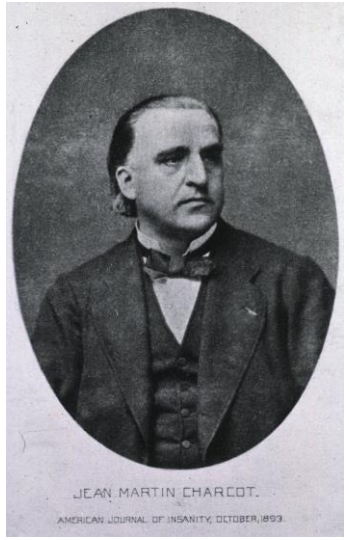
BY

JAMES PARKINSON,

LONDON:

FOR SHERWOOD, NEELY, AND JONES,

1817.



PARKINSON'S DISEASE

- Alfa synucleinopathie, Lewy Bodies
- 2-3% of population >65 j
- James Parkinson (1755-1824)
- Jean Martin Charcot (1825-1893)

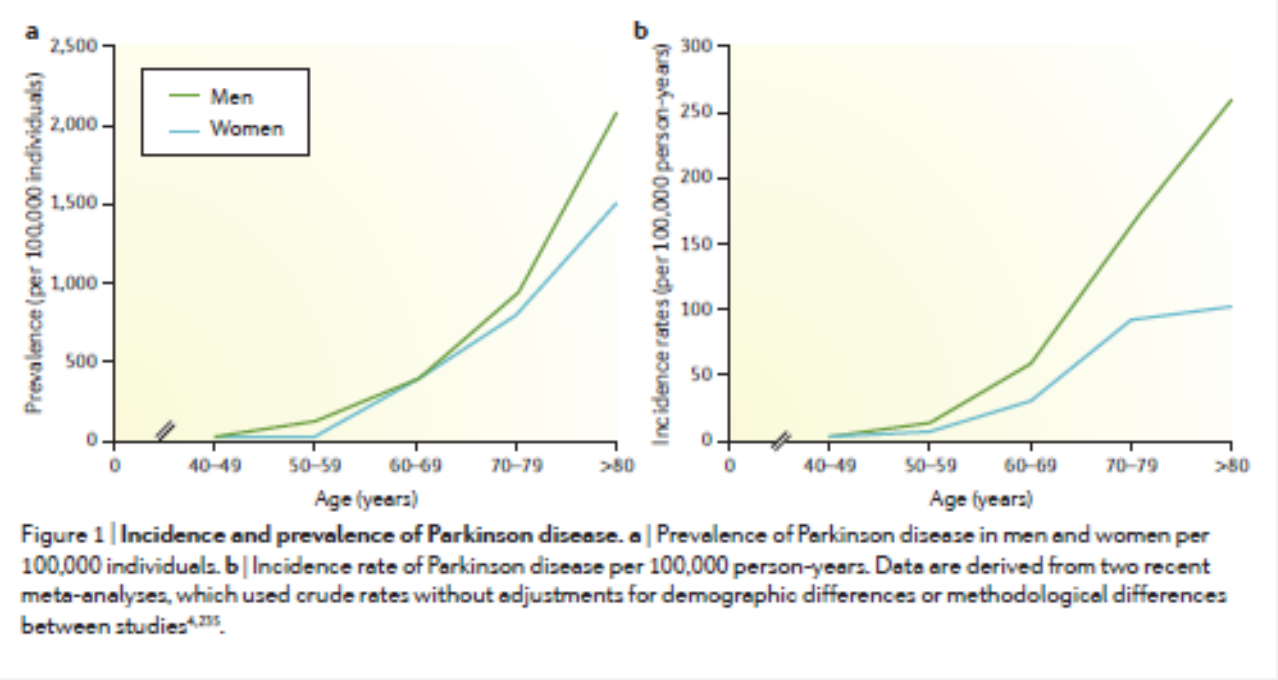


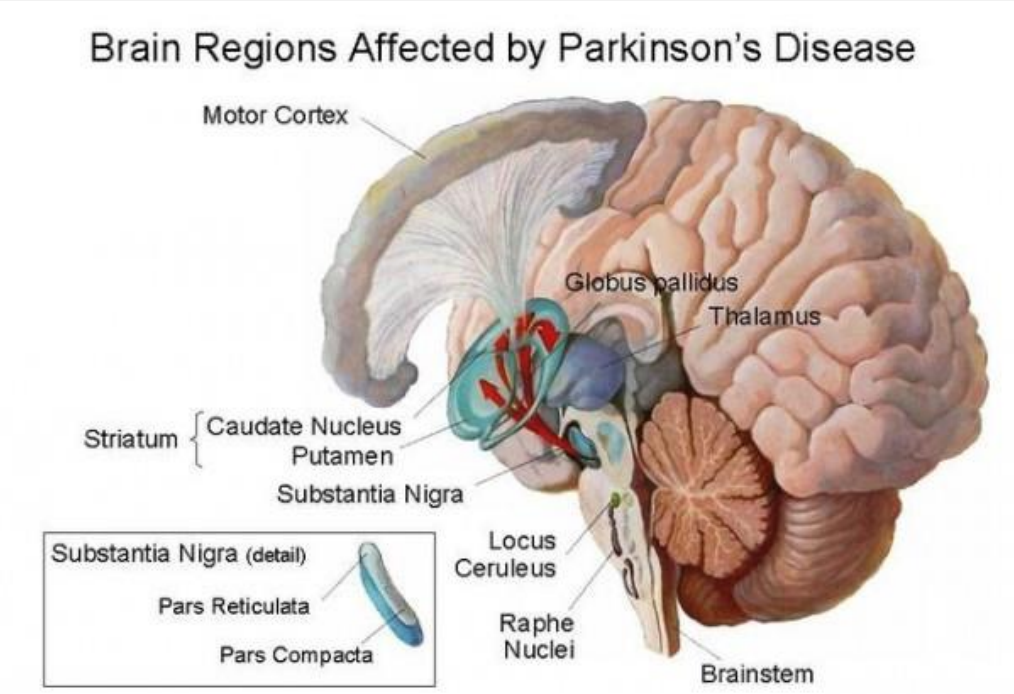
Figure 1 | **Incidence and prevalence of Parkinson disease. a** | Prevalence of Parkinson disease in men and women per 100,000 individuals. **b** | Incidence rate of Parkinson disease per 100,000 person-years. Data are derived from two recent meta-analyses, which used crude rates without adjustments for demographic differences or methodological differences between studies[4,235].

NATURE Reviews 2017; W Poewe et al

- Parkinson disease is rare before 50 years of age, but the incidence increases 5–10-fold from the sixth to the ninth decade of life. The global prevalence, conservatively estimated at 0.3% overall, likewise increases sharply with age to >3% in those >80 years of age
- **Mortality is not increased in the first decade after disease onset**, but increases thereafter, eventually doubling compared with the general population

PATHOPHYSIOLOGY

- Loss of dopaminergic neurons at the substantia nigra
- Ubiquitine misfolded, oxidative stress, mitochondrial dysfunction, inflammation, …

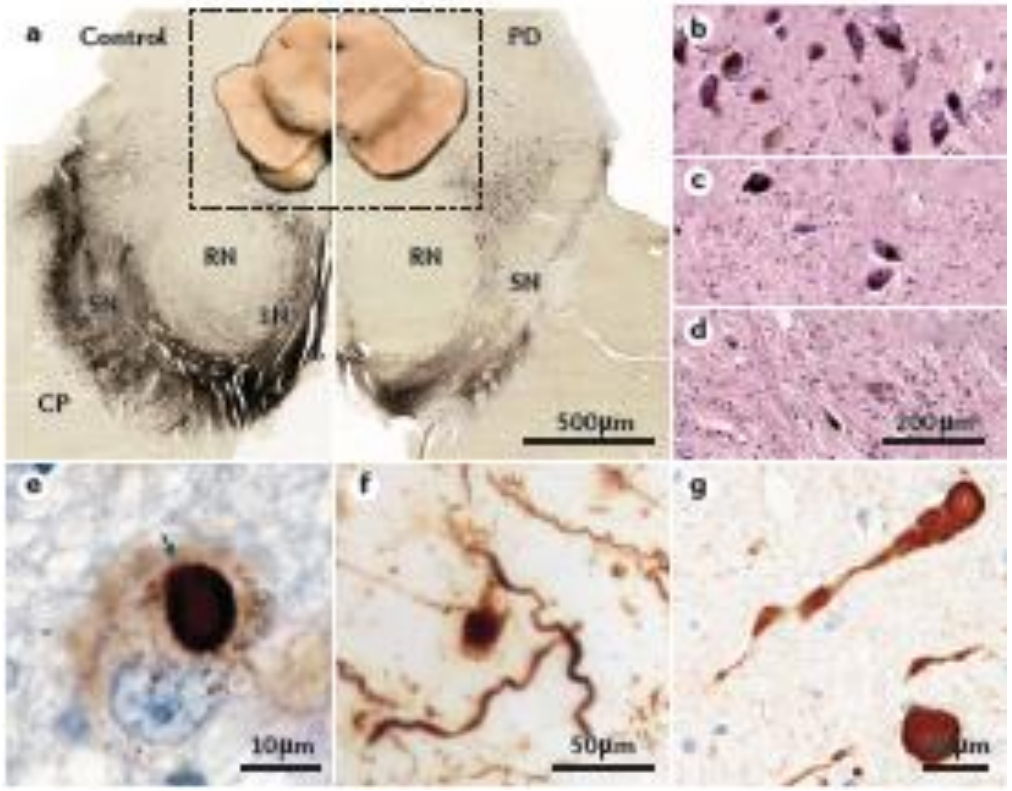


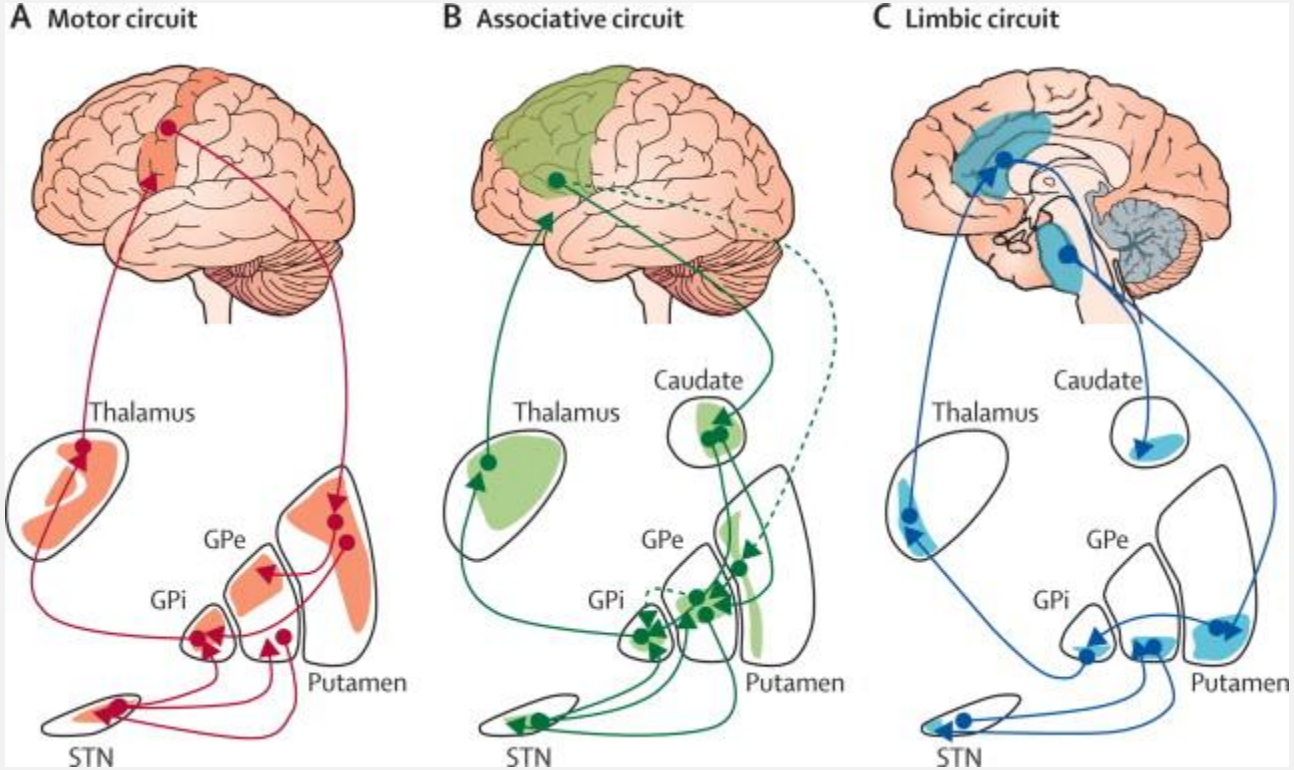
NEUROPATHOLOGY

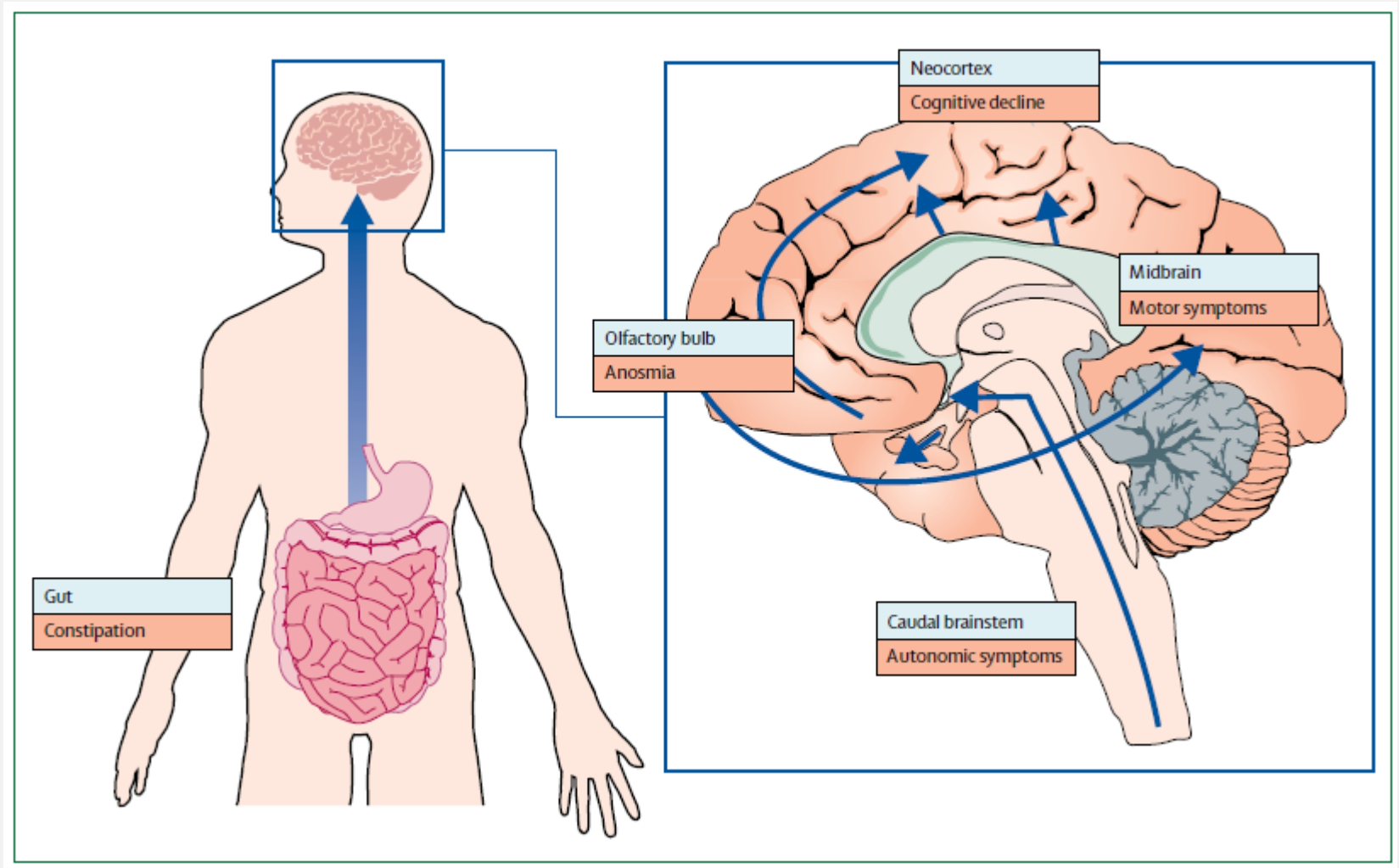
- Depigmentation
  - Substantia nigra
  - Locus coeruleus

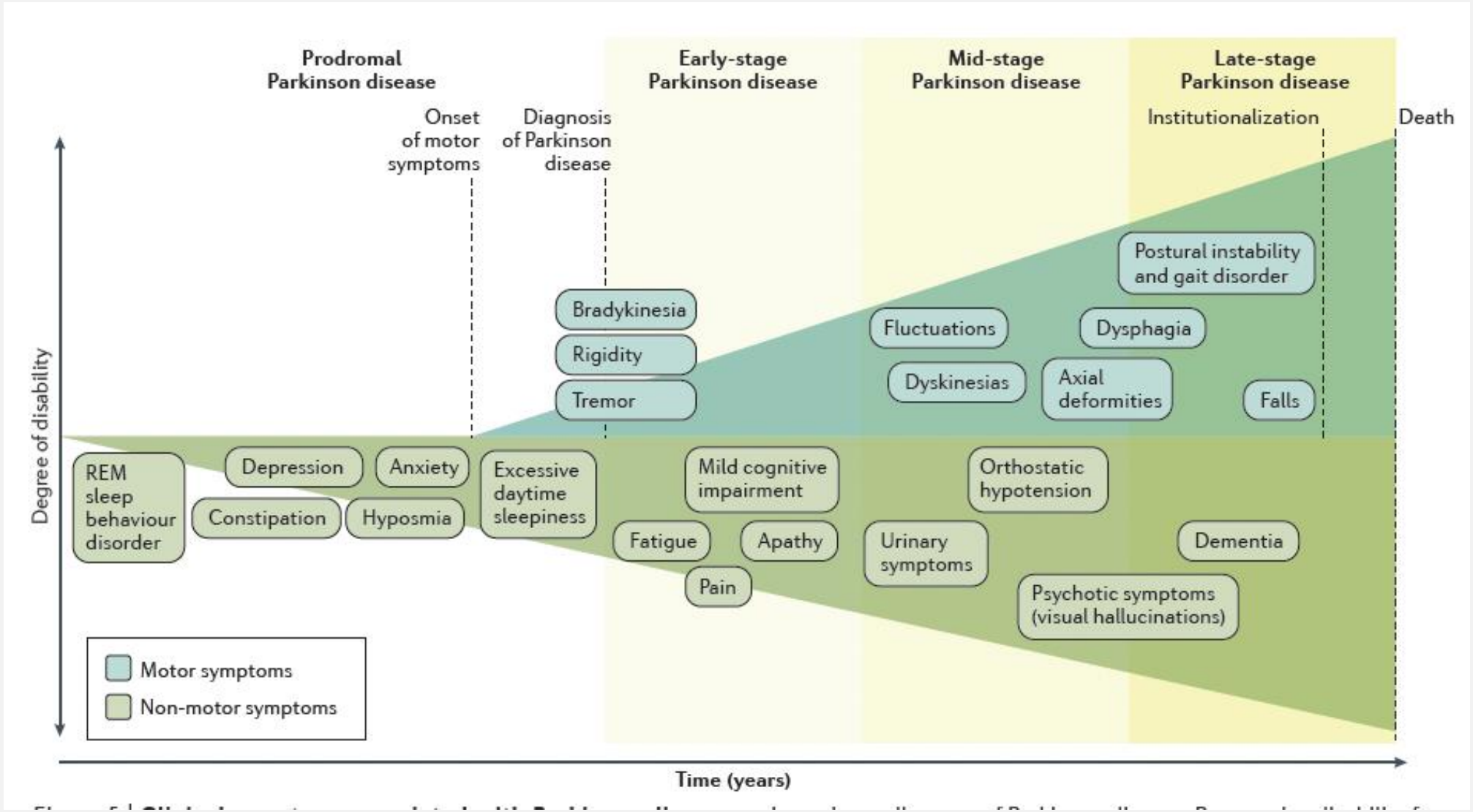
- Neuronal loss
  - Vnl. substantia nigra pars compacta

Lewy bodies in surviving neurons
  - Concentrische hyaliene cytoplasmatische inclusie
  - Immunoreactief voor α-synucleïne




A Motor circuit
Thalamus
GPe
GPi
Putamen
STN
B Associative circuit
Caudate
Thalamus
GPe
GPi
Putamen
STN
C Limbic circuit
Caudate
Thalamus
GPe
GPi
Putamen
STN


Neocortex
Cognitive decline
Midbrain
Motor symptoms
Olfactory bulb
Anosmia
Gut
Constipation
Caudal brainstem
Autonomic symptoms



NATURE Reviews 2017; W Poewe et al

MOTOR SYMPTOMS

- Tremor
- Rigidity
- Bradykinesia
- Postural instability
- Stooped posture, small steps
- Freezing, festinatiion
- Hypofonie, dysarthria
- Hypomimie
- Swallowing problems
- Dystonia
- Eye movements

NON MOTOR SYMPTOMS

- Autonomic dysfunction
  - Obstipation
  - Orthostatism
  - Urgency/frenquency
  - Erectile dysfunction
  - Thermoregulation, sweating
- Mood
- Cognition
- Hyposmie
- Sleep
  - fragmented
  - RBD (REM sleep behaviour disorder)
- Pain

PD TREMOR

- Asymmetrical/unilateral
- Resting tremor 4-6Hz
- Pill rolling
- pro- supination (ET flexie-extensie)
- Leg, lips, chin
- Clinical exam: mental tasks, walking

BRADYKINESIA

- Fingertapping, opening/closing of the hand, foottapping, heeltapping
- Min 10x
- Decrement, hesitations
- DD hypokinesia (arthritis, pareses, dyspraxie, depression, …)

DIAGNOSIS

- Clinical!
- MRI of the brain to exclude other causes
- When in doubt: DAT scan
- In selected cases: MIBG, FDG PET, raclopride

Box 1 | **MDS diagnostic criteria for Parkinson disease**

Step 1: diagnosis of parkinsonism (core feature)

- Presence of bradykinesia as a slowness of movement and a decrement in amplitude or speed (or progressive hesitations or halts) as movements are continued
- In combination with at least one of: rigidity and/or rest tremor

Step 2: determining Parkinson disease as the cause of parkinsonism with two levels of diagnostic certainty

Diagnosis of clinically established Parkinson disease requires all three of the below parameters:

- Absence of absolute exclusion criteria. These criteria include clinical or imaging evidence for alternate diagnoses of parkinsonism, such as atypical parkinsonism, drug-induced parkinsonism or essential tremor.
- Two or more supportive criteria. These include L-DOPA responsiveness, the presence of classic rest tremor, the presence of L-DOPA-induced dyskinesias, the presence of either olfactory loss or cardiac sympathetic denervation on metaiodobenzylguanidine (MIBG) scintigraphy.
- No red flags. This refers to features that are unusual but not absolutely exclusionary for Parkinson disease, for example, the rapid progression of gait impairment that requires wheelchair use or the development of severe autonomic failure within 5 years after onset.

Diagnosis of clinically probable Parkinson disease requires:

- Absence of absolute exclusion criteria (mentioned above)
- Presence of red flags (mentioned above) that are counterbalanced by supportive criteria

For a full listing of absolute exclusion criteria, red flags and supportive criteria see REF. 118.

MDS, International Parkinson and Movement Disorder Society.

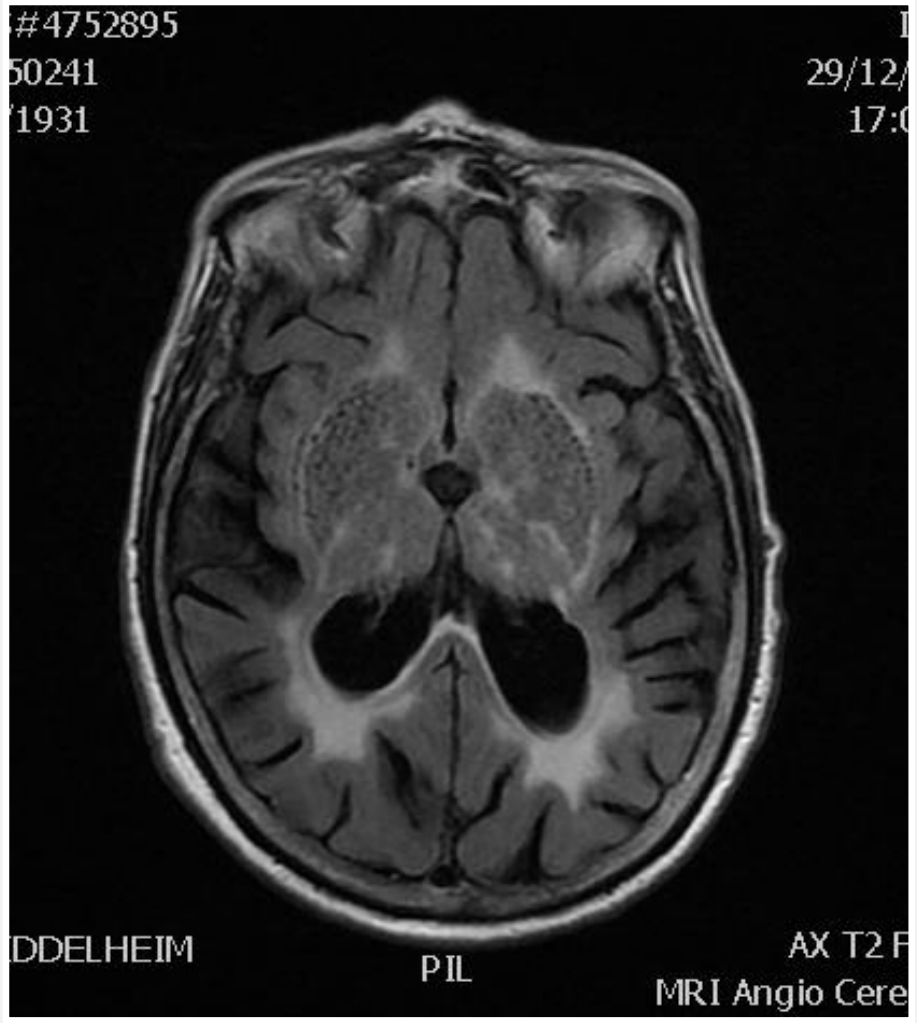
DIAGNOSTIC CRITERIA

RED FLAGS

1. Posture:
   - Pisa syndrome → MSA
   - Antecollis → MSA
   - Retrocollis → PSP
   - Fixed (asymmetrical) dystonia → CBD
2. Cognitive decline
3. Gait and balance
   - Tandem gait
   - bicycle
4. Early and severe autonomic dysfunction → MSA

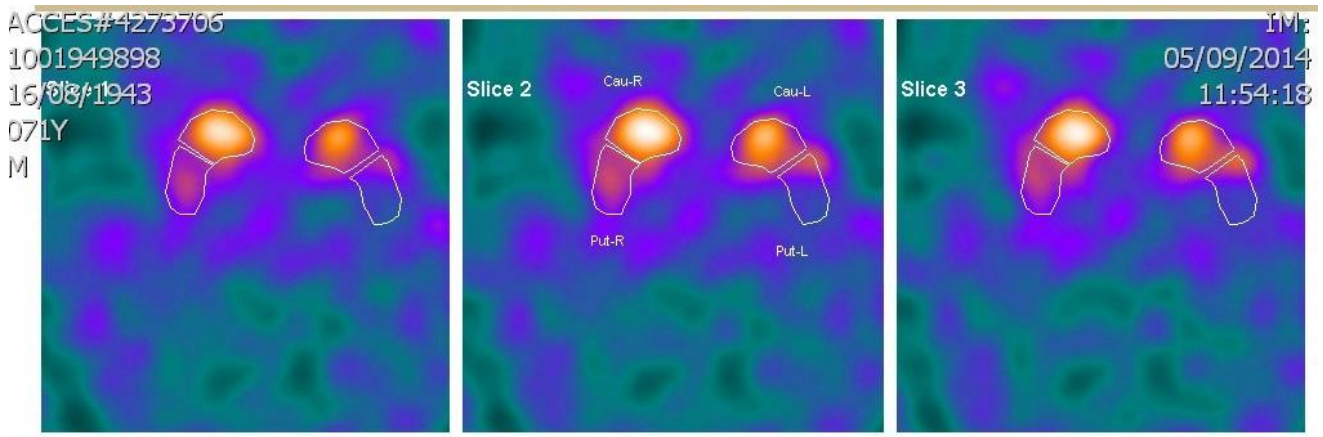
MRI

- Rule out other causes
- vascular, NPH (normal pressure hydrocephalus)
- PSP, MSA (often normal at first)



DAT SCAN

- Presynaptic dopaminergic deficit
- asymmetrical
- False negative with short disease duration
- Putamen < nucleus caudatus



TREATMENT

- symptomatic
- When to start? Depends on burden

TREATMENT OPTIONS

1st line treatment: levodopa, dopamine agonists, (MAO-i)

2nd line: treatment of fluctuations

3d line: Apo Go, pump therapy and DBS

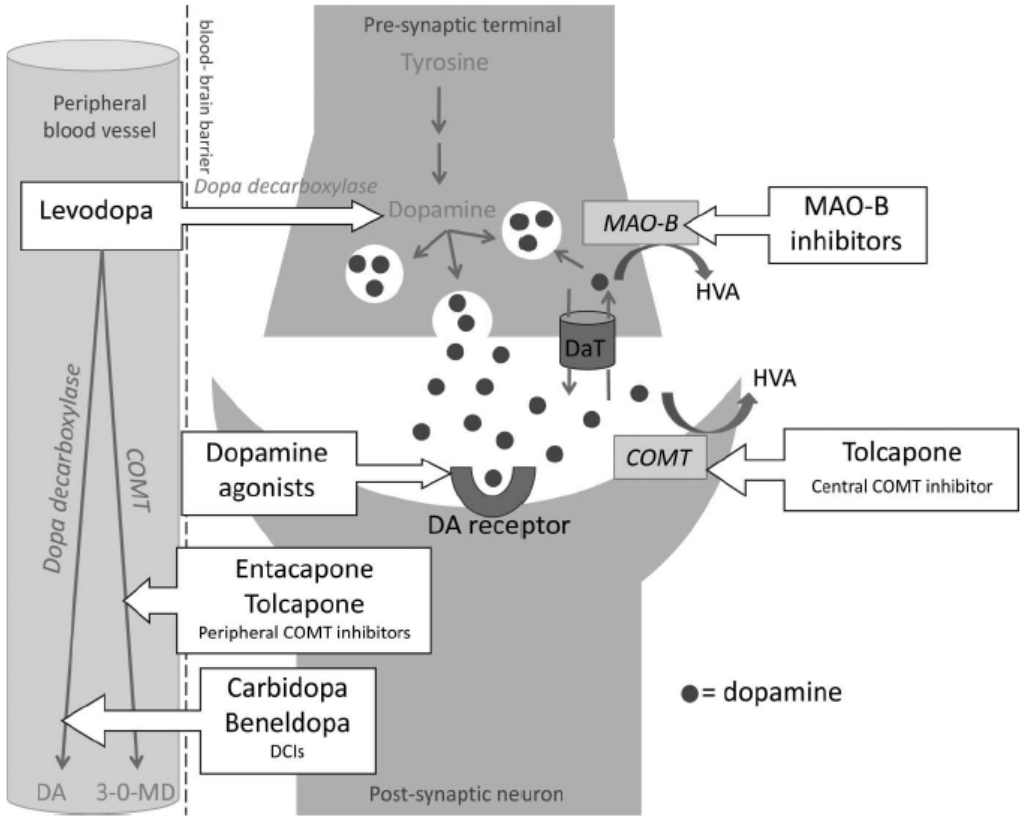
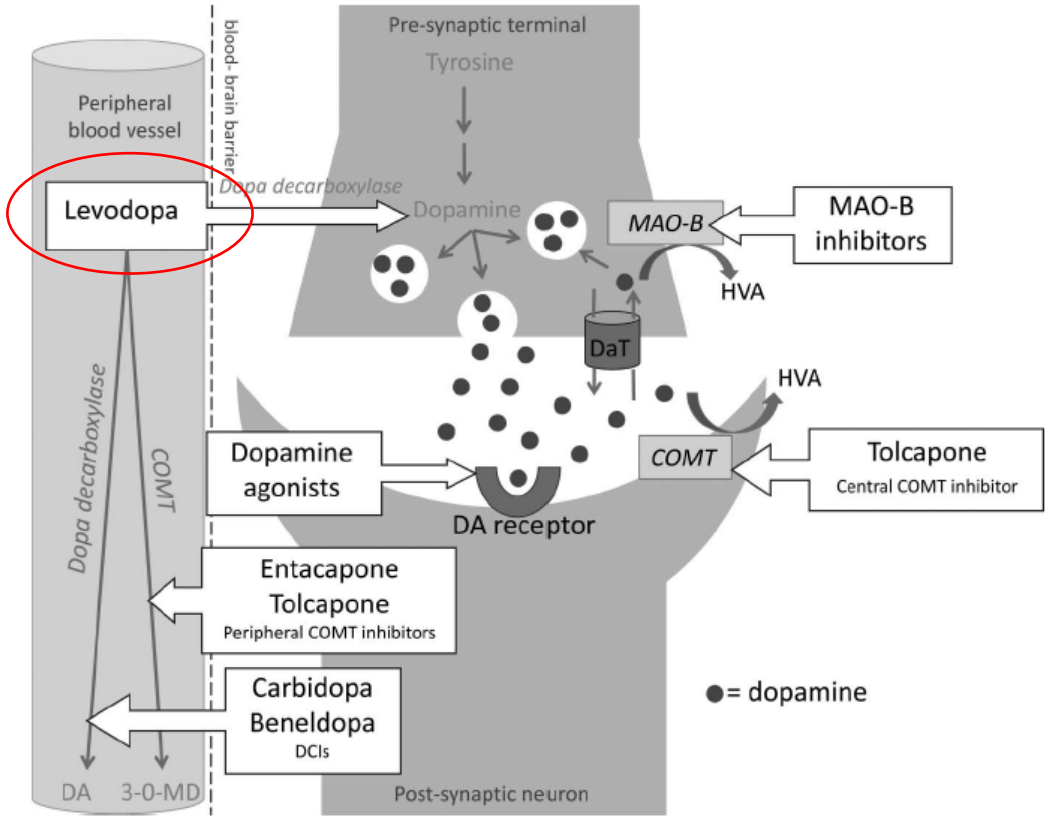


Figure 1 Dopaminergic neurones are activated through a mechanism of dopamine (DA) being released from vesicles at the presynaptic terminal, crossing the synaptic cleft and then binding to postsynaptic dopamine receptors. Excess dopamine is metabolised by monoamine-oxidase-B (MAO-B) at the presynaptic terminal, after first being taken up by the dopamine active transporter (DaT), and also via catechol-O-methyltransferase (COMT) at the postsynaptic terminal. Levodopa, a dopamine precursor that can cross the blood–brain barrier, thus increases the amount of dopamine released at the presynaptic terminal, whereas dopamine agonists directly stimulate the postsynaptic dopamine receptors. MAO-B inhibitors and the COMT inhibitor tolcapone increase dopamine availability in the synaptic cleft by slowing down dopamine metabolism in the central nervous system. Dopa decarboxylase inhibitors (DCIs) and both COMT inhibitors (entacapone and tolcapone) reduce peripheral metabolism of levodopa, thus increasing the amount available to cross the blood–brain barrier, and reducing peripheral side effects. Note: This diagram outlines the *main* actions of Parkinson's disease drugs on dopaminergic neurones but is not exhaustive; most of these drugs have additional roles, especially the enzyme inhibitors on other neurotransmitters' metabolism. HVA, homovanillic acid.

Alty J, *et al. Pract Neurol* 2016;**16**:122–128. doi:10.1136/practneurol-2015-001267



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LEVODOPA: ORAL INTAKE

① **Swallowing oral therapy**
Impaired swallowing (dysphagia) in advanced disease

② **Stomach**
Variable absorption of levodopa due to irregular gastric emptying

③ **Jejunum**
Competition with dietary amino acids for active transport across the intestinal wall

④ **Peripheral tissues**
Reduced levodopa bioavailability due to enzymatic breakdown by AADC and COMT

⑤ **Blood–brain barrier**
Competition for transport across the blood–brain barrier with large neutral amino acids limits the amount of levodopa reaching the striatum

⑥ **Striatum**
Conversion of levodopa to dopamine

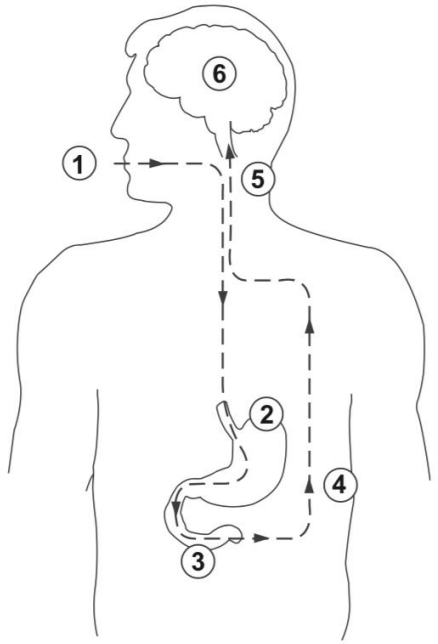
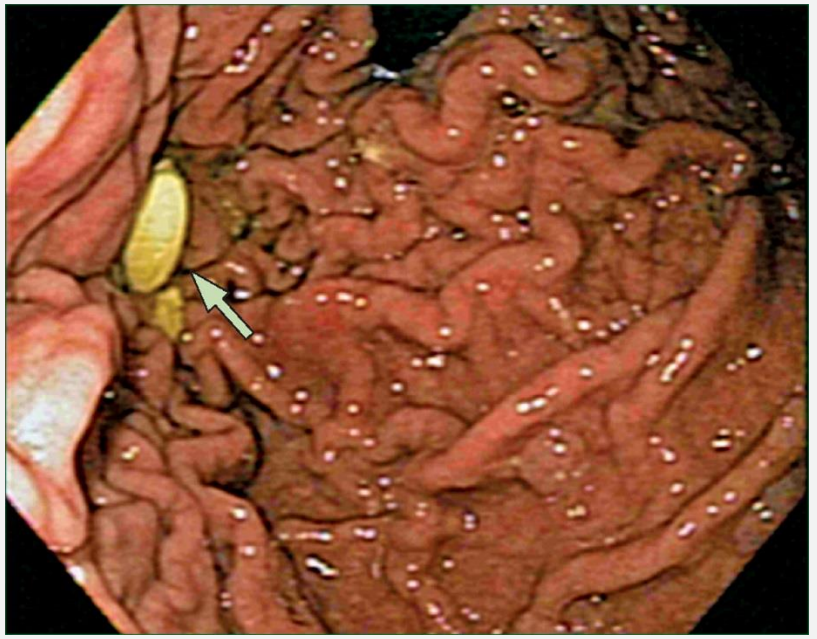


Figure 3 Oral levodopa therapy – hurdles on the route from the mouth to the brain. A number of factors impact on the progress of levodopa from the time of ingestion until it reaches the brain and is converted to dopamine.
Abbreviations: AADC, amino acid decarboxylase; COMT, catechol-*O*-methyl transferase.

Poewe et al. 2010

GASTROPARESIS, OBSTIPATION



LEVODOPA: EMPTY STOMACH!

Protein interaction

Not with dairy, meet, fish, eggs

30 min before or 1-1½ uur hours after the meal

With water, juice (not grapefruit)

LEVODOPA: DIFFERENT FORMULATIONS

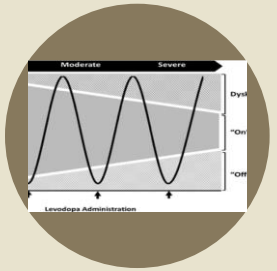
- Prolopa ~~125~~ of 250 (controlled release)
- Prolopa HBS 125
  - Slow release, nighttime
  - Variable absorption
  - Less powerfull
- Prolopa dispersable 125
  - Soluble (stomach tube, swallowing problems)
  - Works faster
  - Works out faster
  - More dyskinesia?

LEVODOPA: SIDE EFFECTS

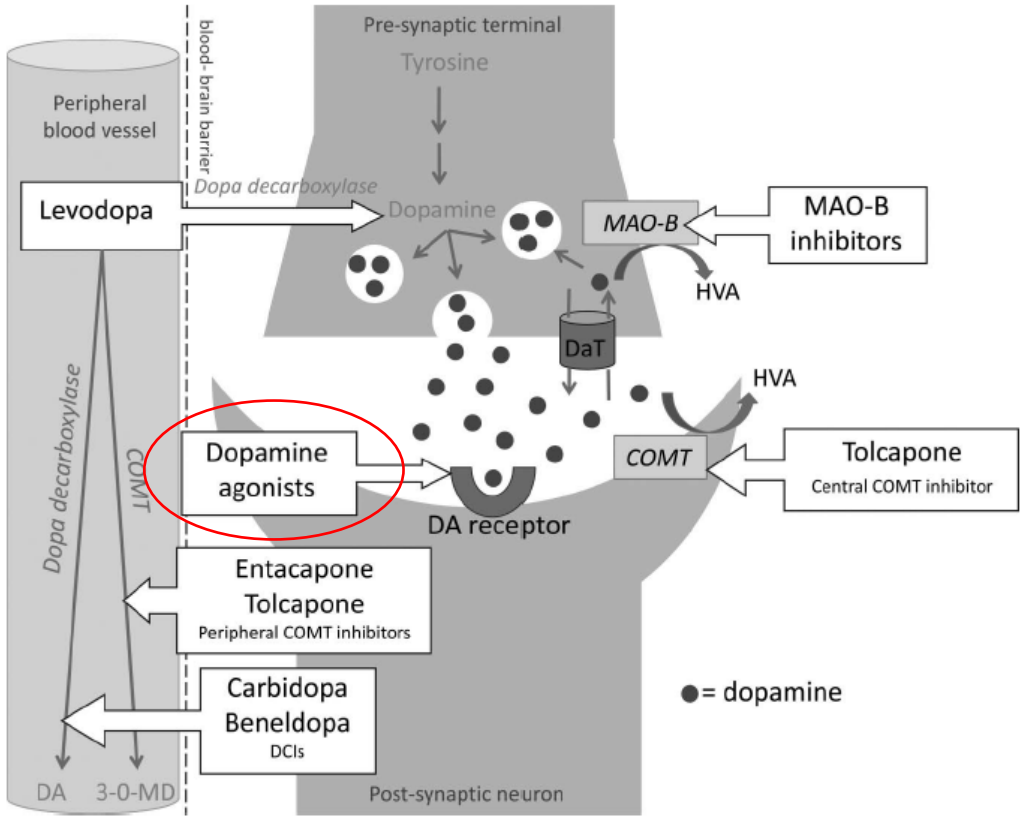
NAUSEA

LOW BP

CONFUSION



LONG TERM: FLUCTUATIONS, DYSKINESIAS



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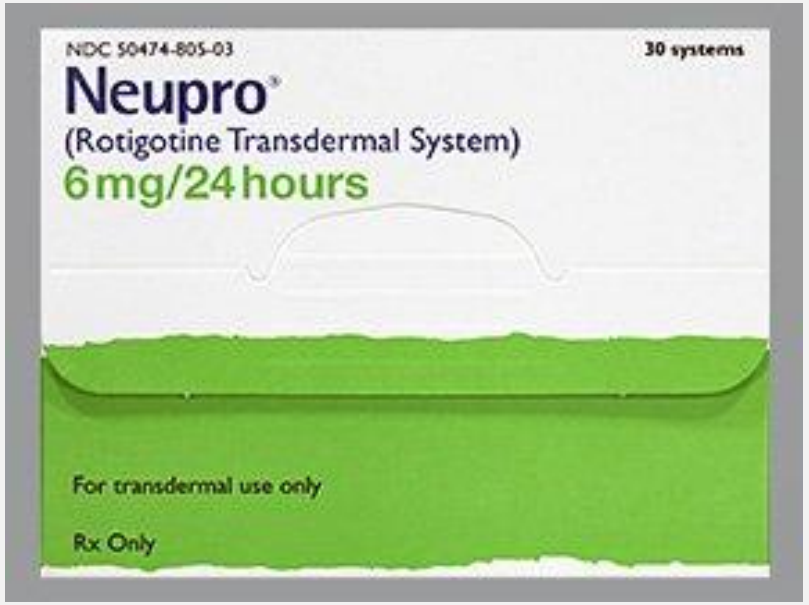
DOPAMINE AGONISTS

- ✓Mirapexin^{R}, Requip^{R}
- ✓less efficient than levodopa
  - add-on
  - Monotherapy in young patients
  - Once daily
- ✓Side effects
  - Same as Prolopa^{R} sometimes more pronounced
  - Daytime sleepiness
  - Malleolar oedema
  - ICD



- Not reimbursed
- Only parental option

Neupro (UCB)

rotigotine

per groepsnaam —
vergelijken

28 x 2 mg / 24 u (4,5 mg / 10 cm²)	Rx	€ 108,54
28 x 4 mg / 24 u (9 mg / 20 cm²)	Rx	€ 130,15
28 x 6 mg / 24 u (13,5 mg / 30 cm²)	Rx	€ 151,76
28 x 8 mg / 24 u (18 mg / 40 cm²)	Rx	€ 173,44

(bevat aluminium)

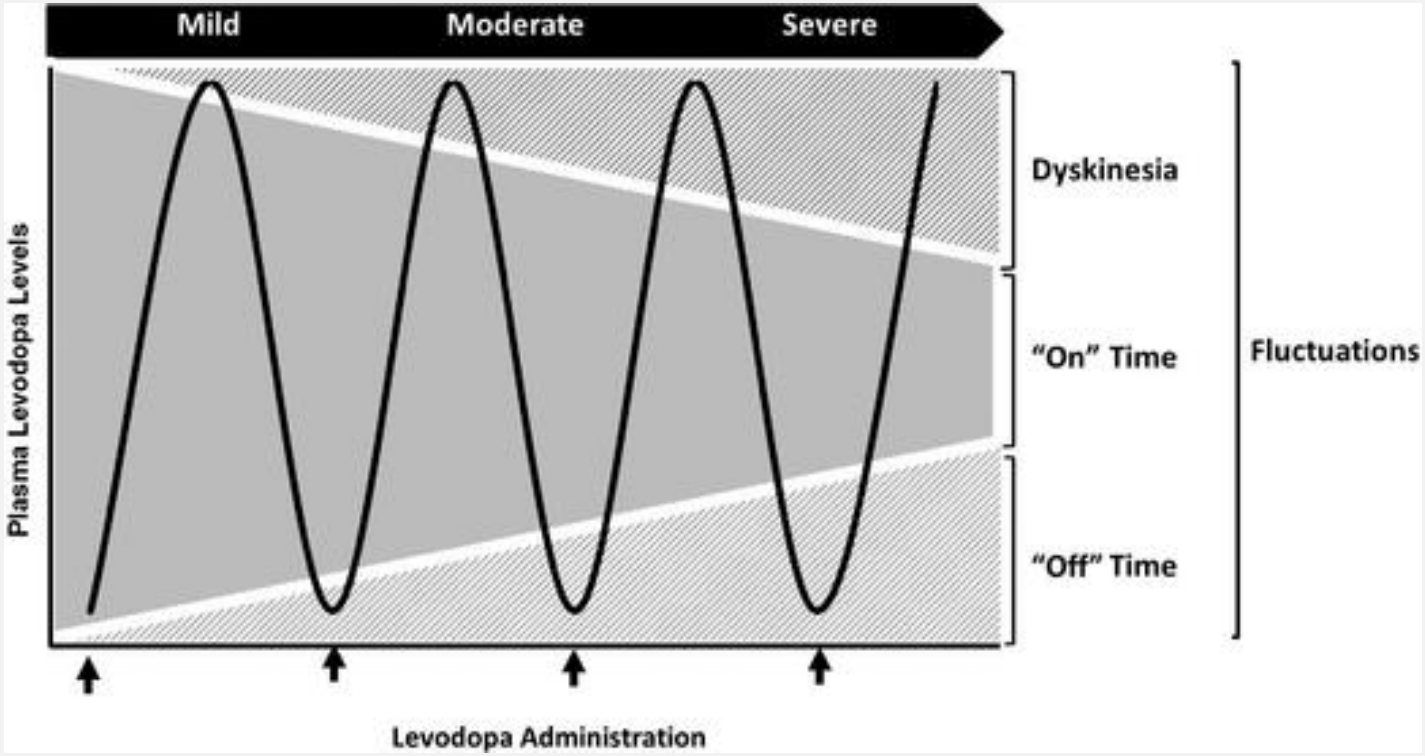
TREATMENT OPTIONS

1st line treatment: levodopa, dopamine agonists, (MAO-i)

2nd line: treatment of fluctuations

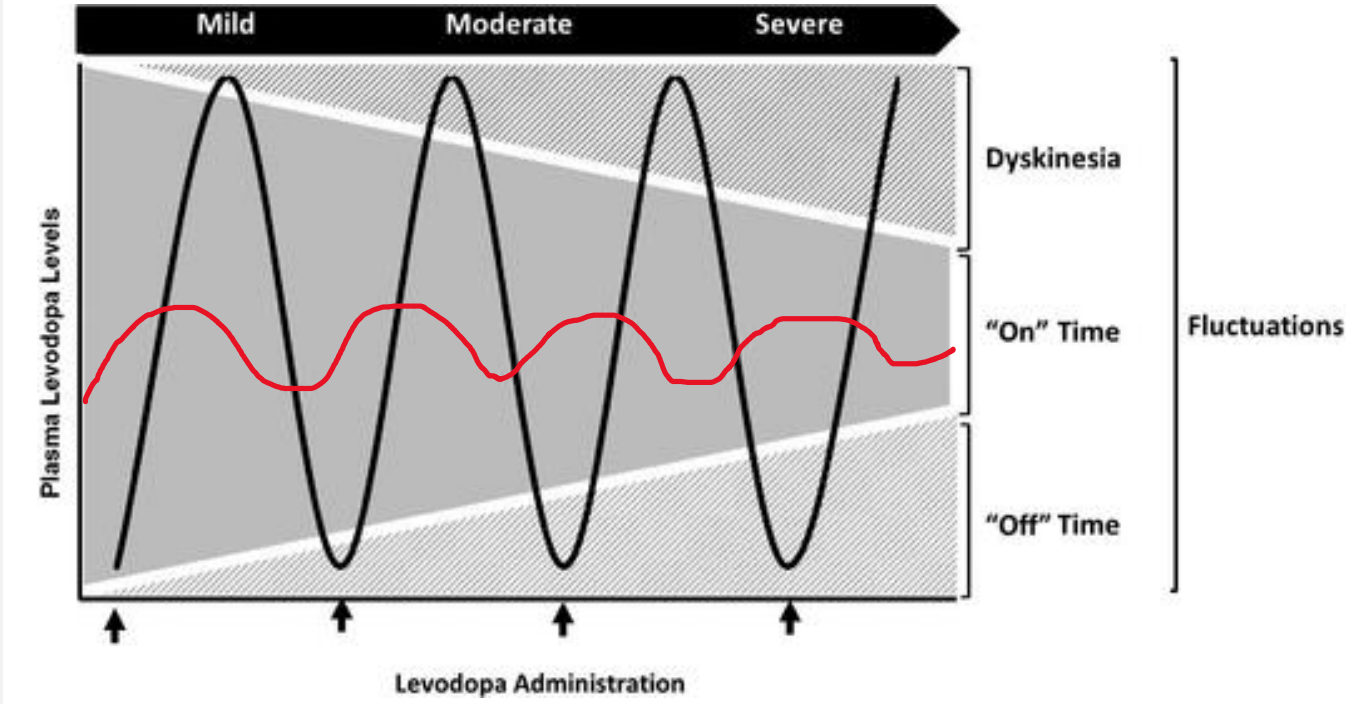
3d line: Apo Go, pump therapy and DBS

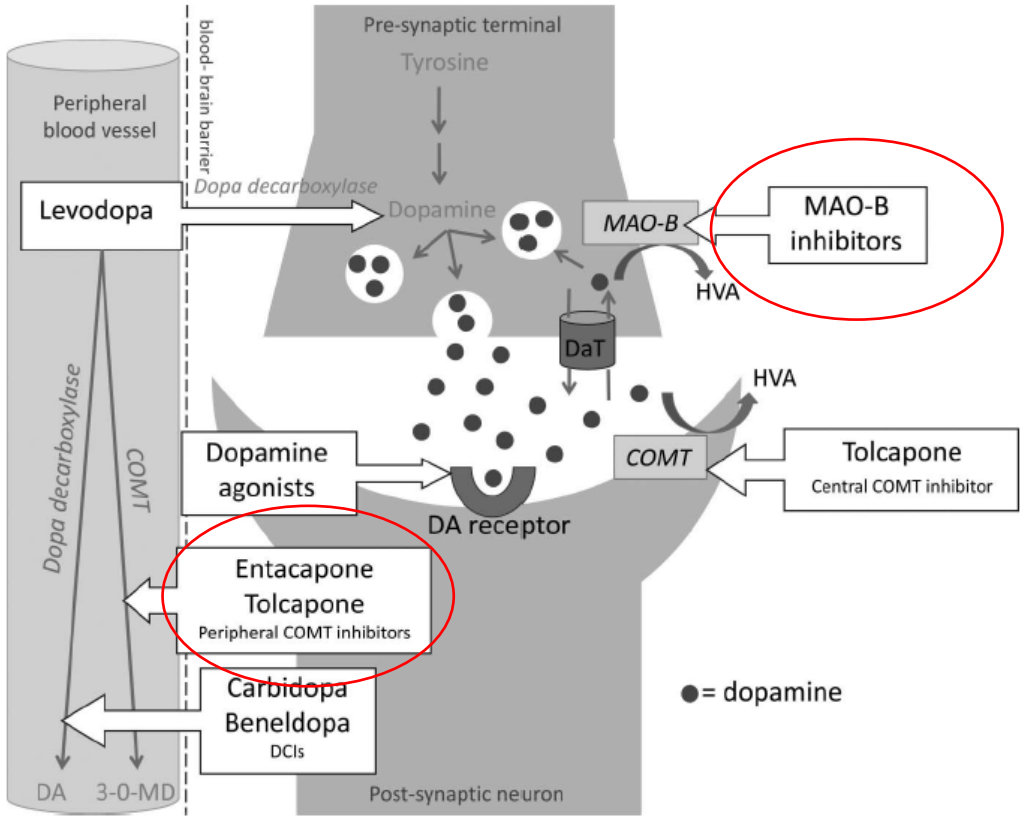
MOTOR FLUCTUATIONS



TREATMENT OF WEARING - OFF

- Increase frequency, decrease dosage
- COMT inhibitor (+2u on per day, more dyskinesias)
- MAO-B inhibitor (“)
- Dopamine agonist





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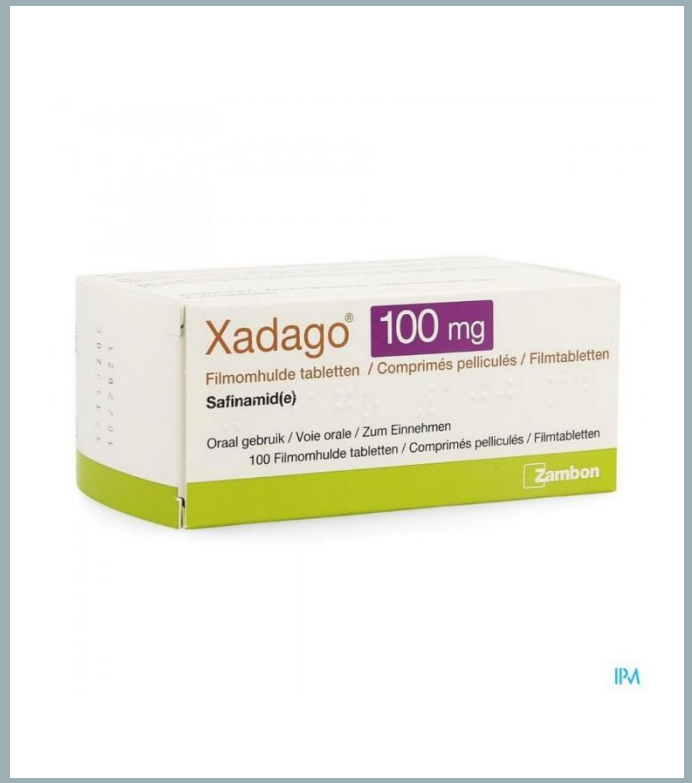
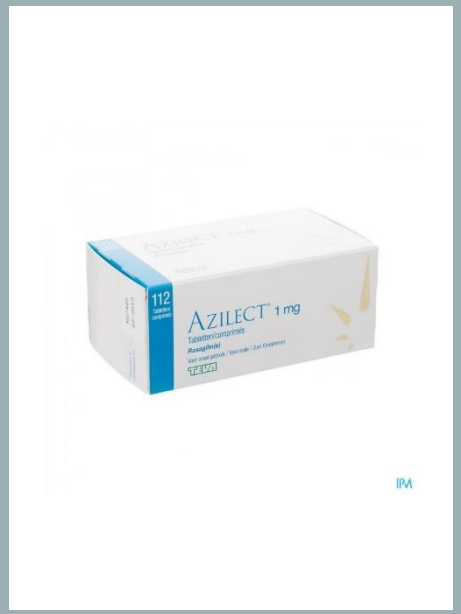
COMT-INHIBITOREN

- ✓Entacapone: Comtan^{R}, (tolcapone/Tasmar$^{R)}$
- ✓Levodopa + entacapone = Stalevo^{R}-Corbilta^{R}
- ✓Side effects
  - Prolopa^{R}
  - Urine discoloration
  - Diarree
  - Tasmar: liver funtion
- ✓With every Prolopa^{R}

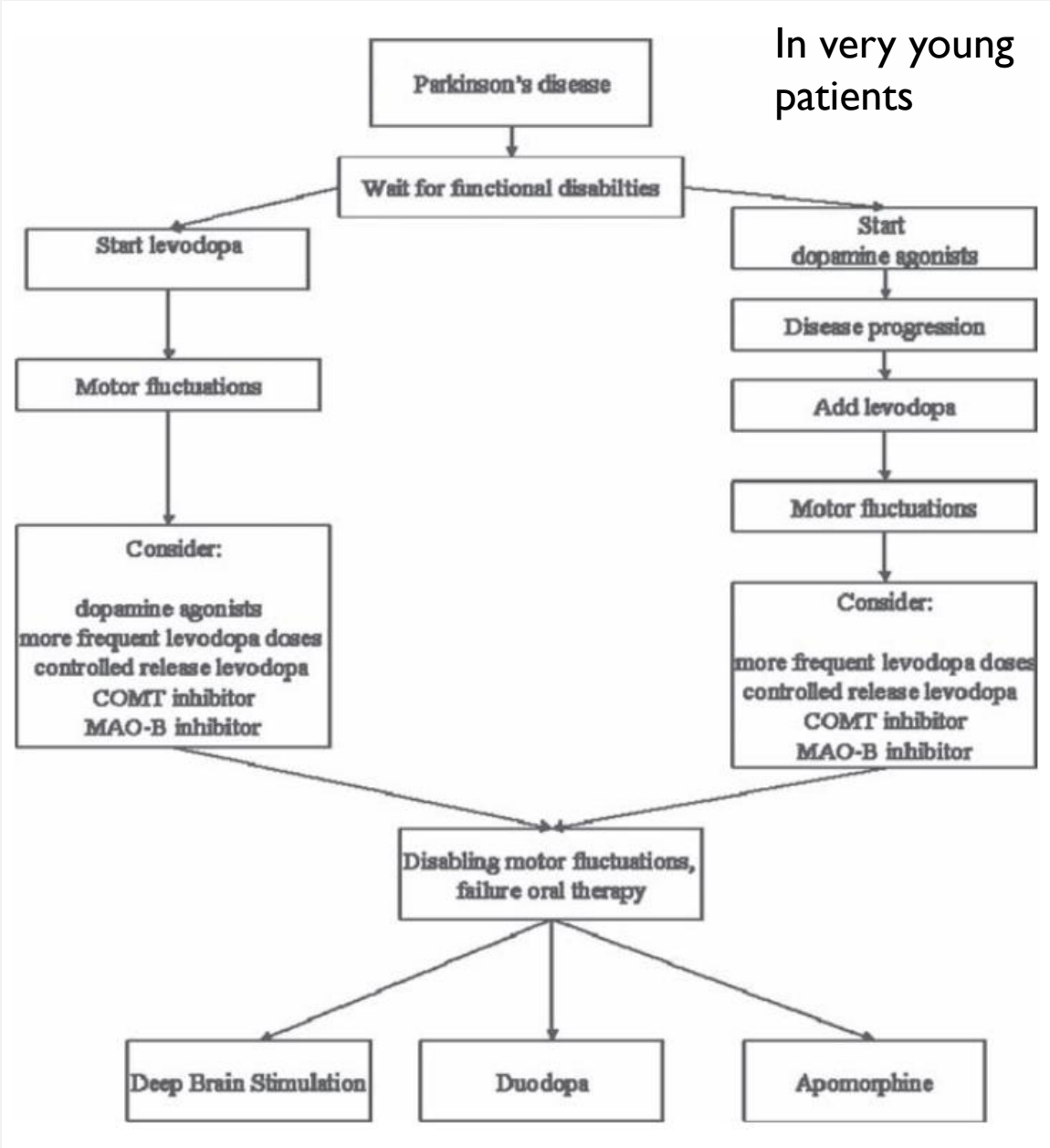




MAO-B INHIBITOREN

- ✓ Eldepryl^{R}, Azilect^{R}, Xadago^{R}
- ✓ Side effects
  - Well tolerated
  - headache, nausea, hallucinatons, dyskinesias

In very young patients



TREATMENT DYSKINESIA

- Lowering dosage (subtherapeutic)
- Amantadine 200-300mg, cave cognition
- Not avalable in Belgium – artsenverklaring

NON-DOPAMINERGIC MEDICATION

- Anticholinergics
  - Artane^{R}, Akineton^{R} ...
  - Tremor reduction
  - dystonia, Salivation, Urgency

  - Side effects:
    - Confusion, dry mouth, urine retention, glaucoma

TREATMENT OPTIONS

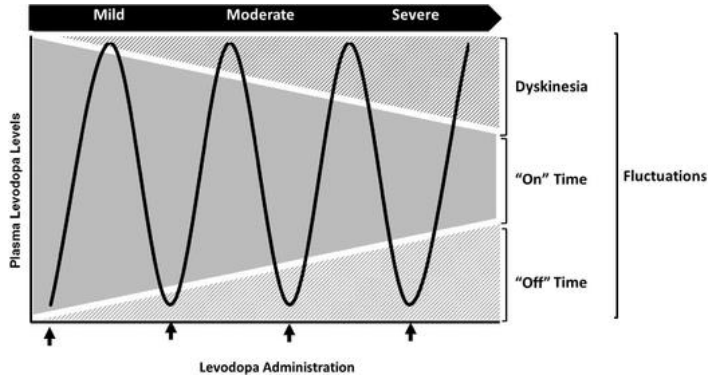
1st line treatment: levodopa, dopamine agonists, (MAO-i)

2nd line: treatment of fluctuations

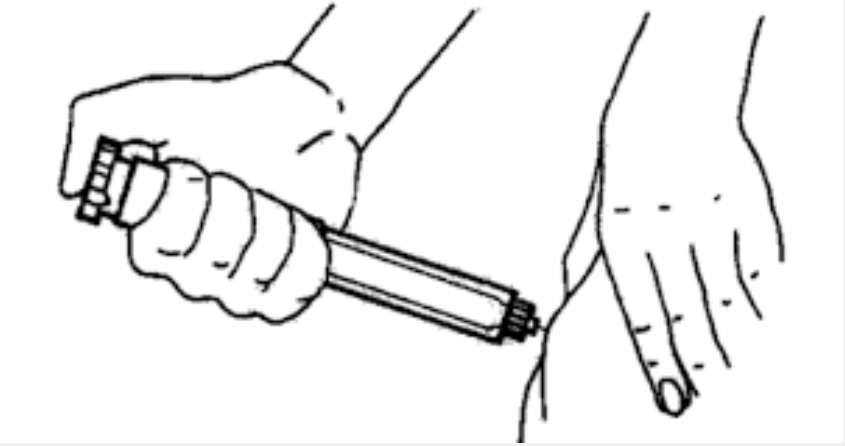
3d line: Apo Go, pump therapy and DBS

UNPREDICTABLE OFF PERIODS - THERAPY RESISTANT FLUCTUATIONS

- Rescue medicatie:
  - Apomorfine SC 1-10mg, effect at 5-10 min
  - Dispersable levodopa: effect at 10-30 min
- ~~Apomorfinepomp~~
- Ctue levodopa intestinal infusion (Duodopa, Lecigimon)
- DBS
- Vanaf 01/09/24: ProDuodopa

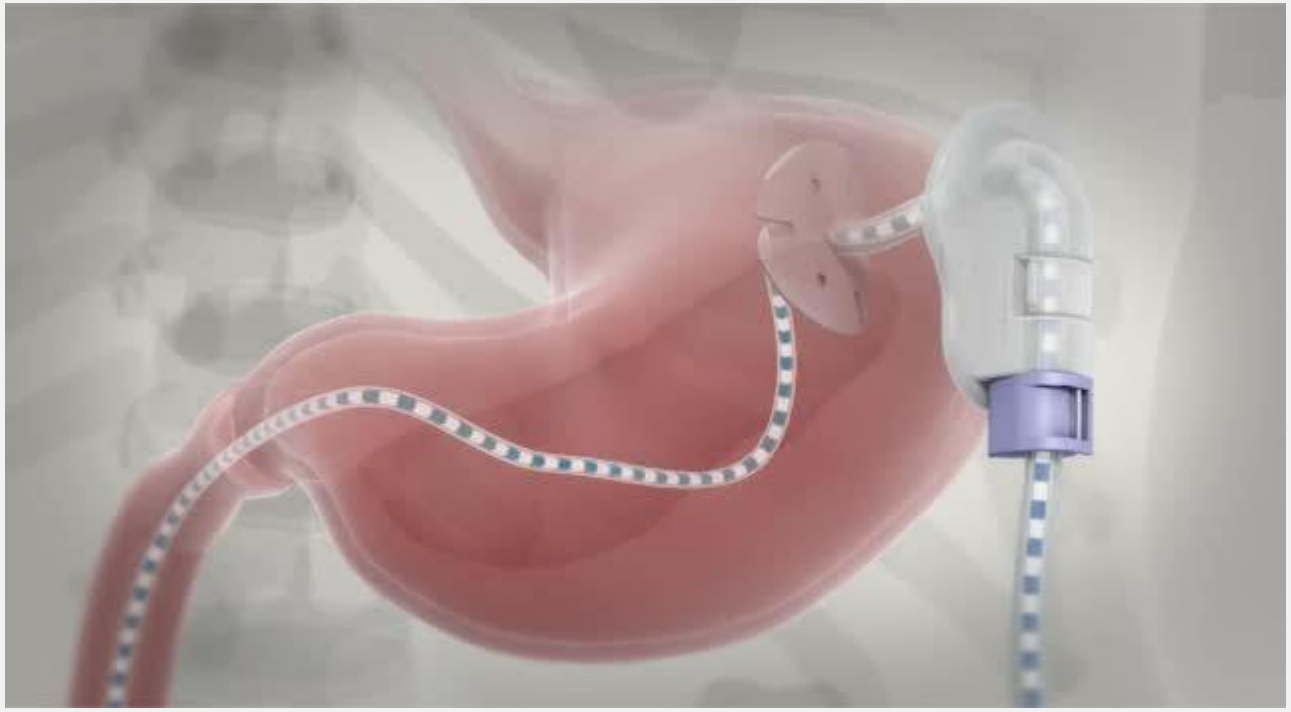


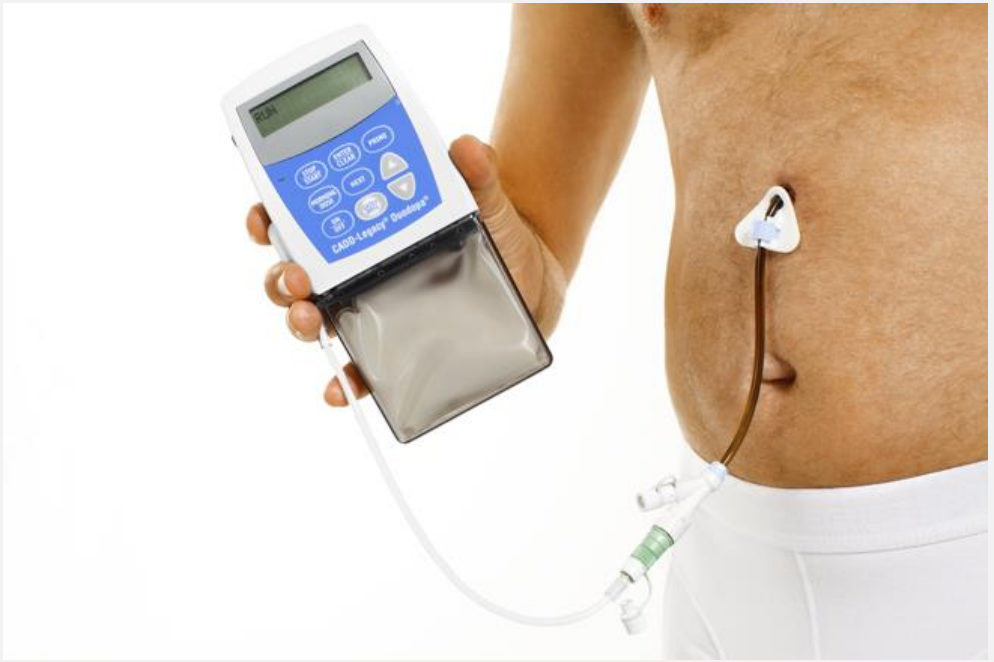
APOMORFINE: APO GO



DUODOPA

- Cassette with 100 ml gel which contains 2000 mg levodopa and 500 mg carbidopa

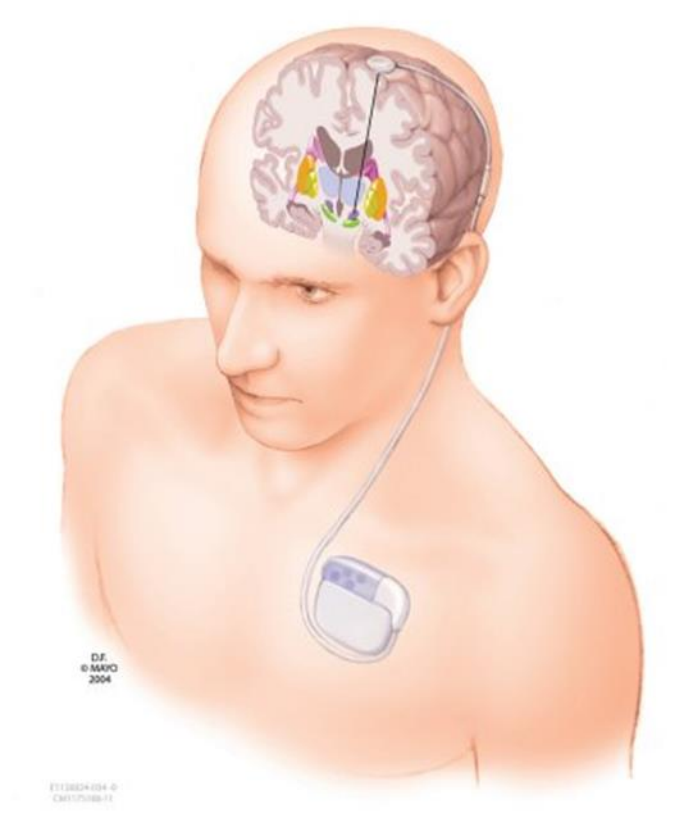




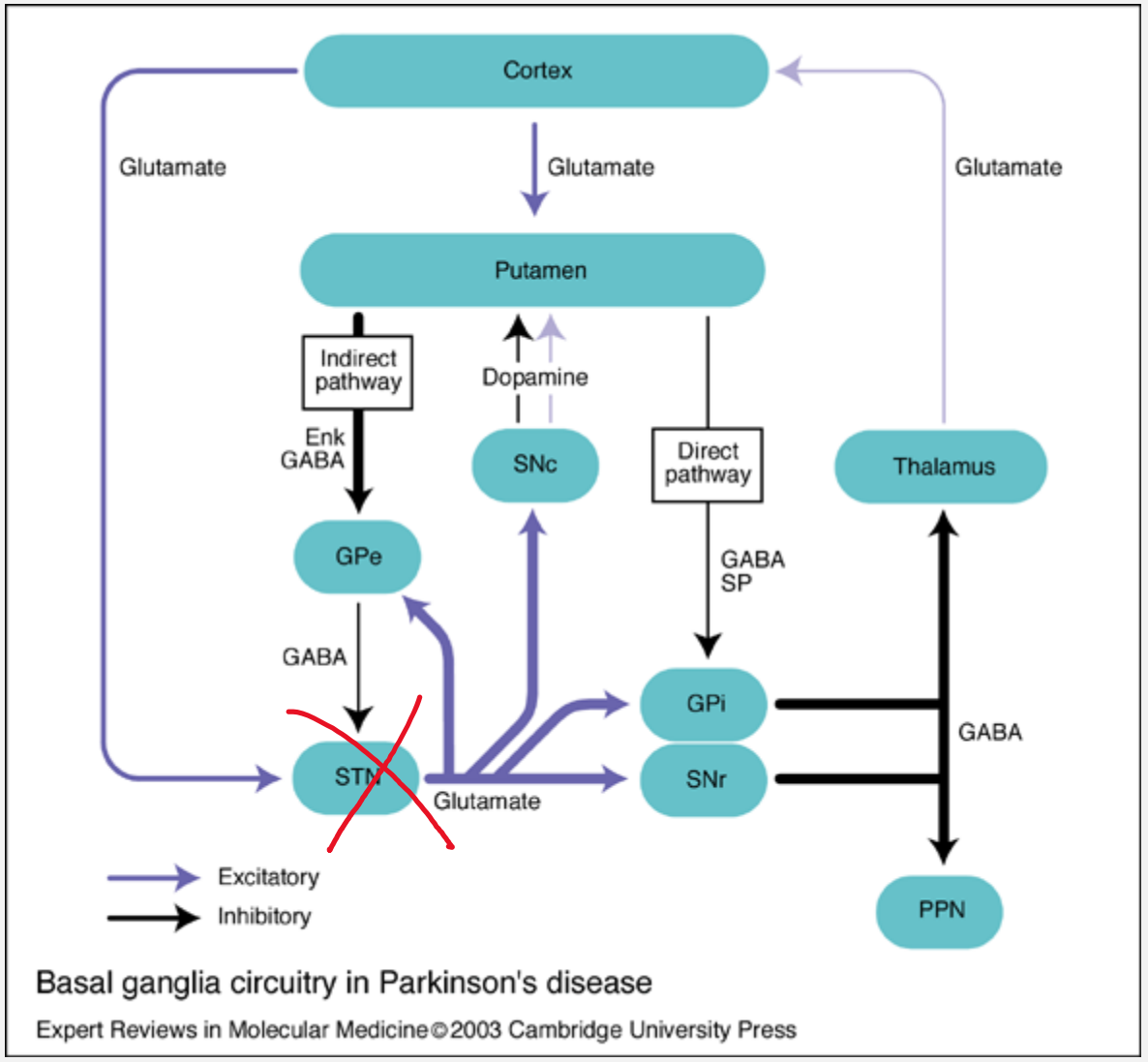
DUODOPA®: BELGIAN REIMBURSEMENT CRITERIA

- Non-eligible patient for DBS
- Parkinson's Disease > 5 years
- Exclusion of structural causes of Parkinsonism by MRI
- Motor fluctuations / dyskinesias despite optimal oral dr
- Absence of dementia: MMSE > 24/30; no dementia drugs; no dementia DSM-IV criteria; neuropsychological tests exclude dementia
- Patient or family can handle the Duodopa® system
- Absence of serious psychiatric disorder
- Levodopa sensitivity: UPDRS III improvement > 50% "on" levodopa vs "off"
- Response to Duodopa® via naso-intestinal tube: UPDRS III improvement > 50% "on" Duodopa® vs "off"

DBS (DEEP BRAIN STIMULATION)



- STN (subthalamic nucleus)
- Gpi (globus pallidus interna)
- Vim (tremor, elderly)



Basal ganglia circuitry in Parkinson's disease

DBS INCLUSION CRITERIA

PD Patient Selection **Inclusion Criteria**

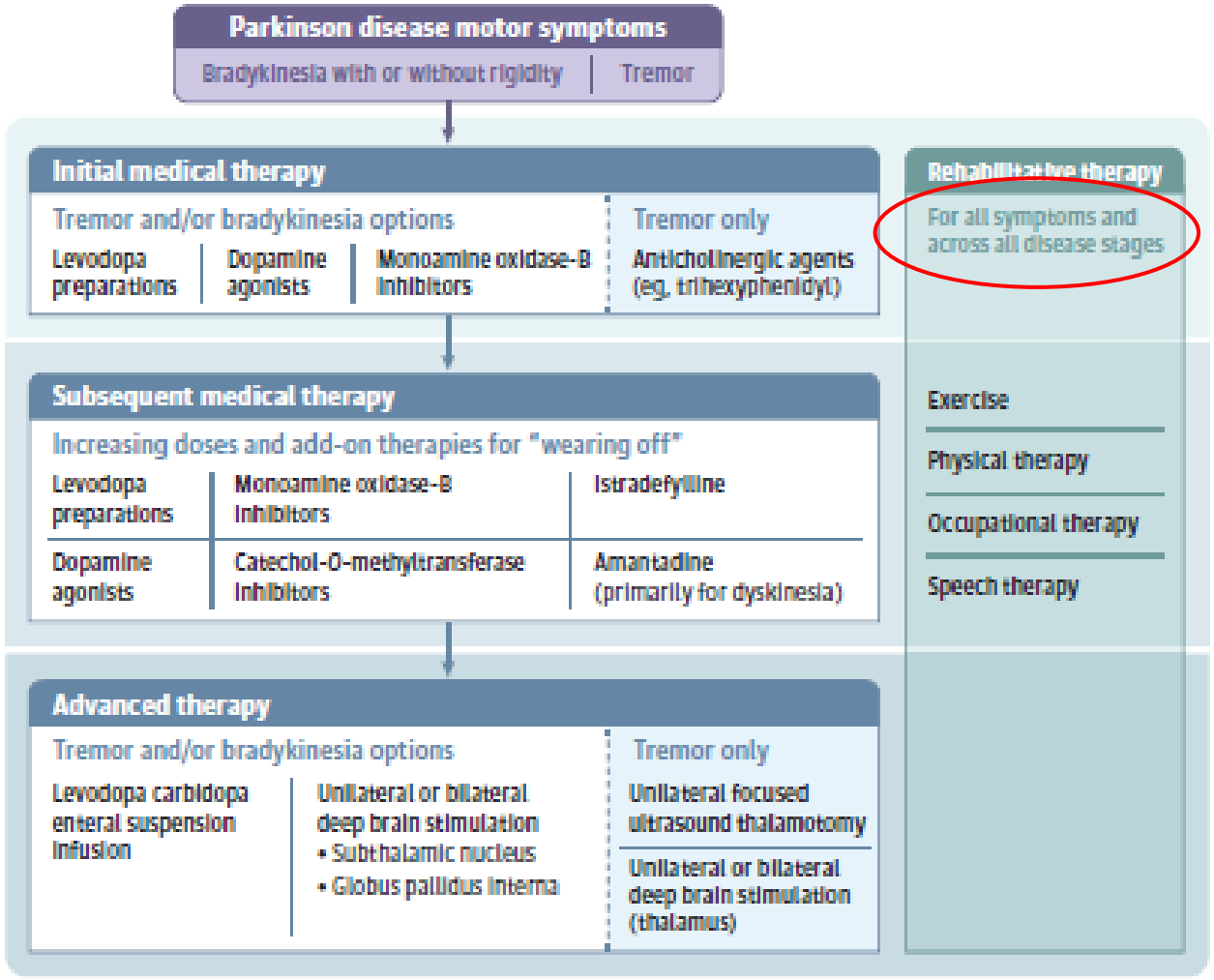
1. **Idiopathic Advanced PD**
2. **Levodopa responsive, with good "on" period function**
3. **Troublesome symptoms, despite optimized pharmacotherapy**
   - Off periods with troublesome bradykinesia, rigidity, tremor, and/or gait difficulty and/or
   - Unpredictable on-off phenomena and/or
   - Motor fluctuations and/or
   - Bothersome dyskinesia and/or
   - Refractory tremor
4. **Response to Dopaminergic Therapy Predicts response to DBS**
5. **No dementia or significant untreated depression**
6. **Realistic expectations**

[1]Lang AE, *et al*. *Mov Disord* 2006
[2]Machado A, *et al. Mov Disord* 2006

WHAT NOT TO DO

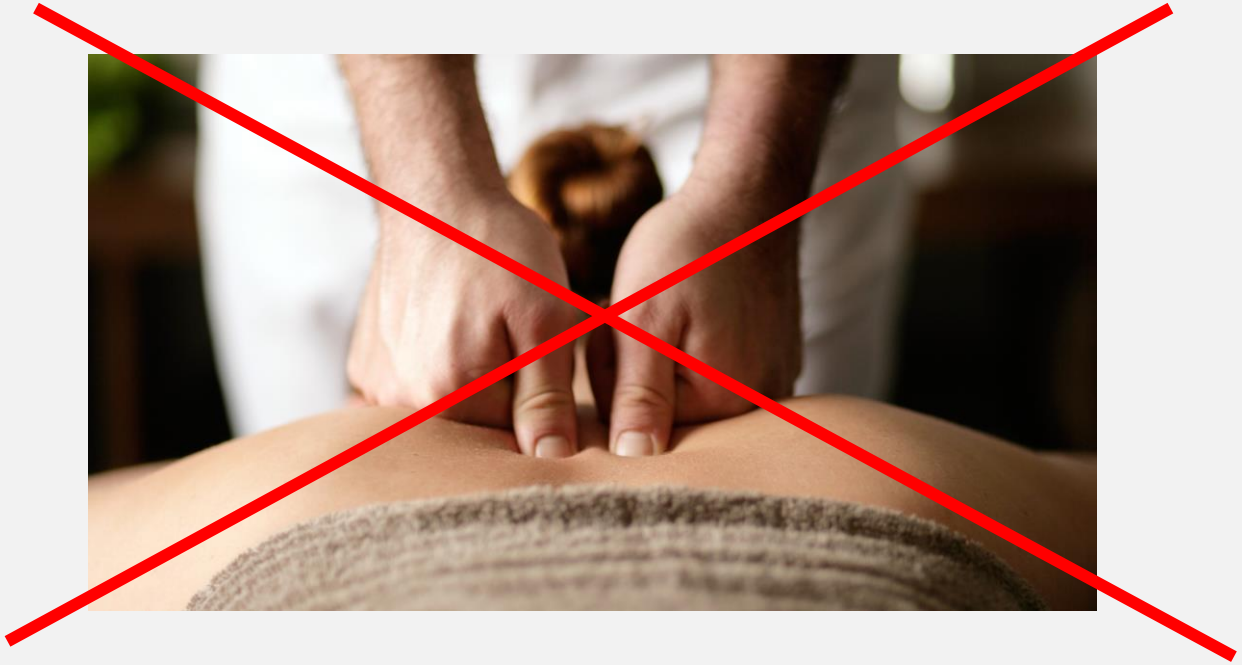
- Typical neuroleptics: Haldol, clopixol, etumine → only safe options: seroquel/quetiapine and leponex/clozapine
- anti-emetics: metoclopramide, alizapride, DHBP → most safe option: domperidone

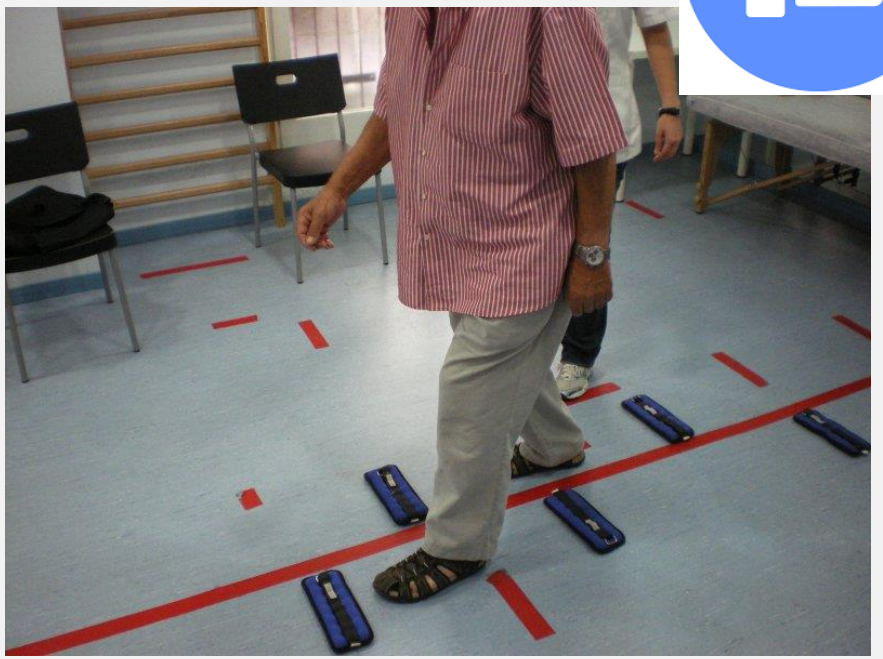
Figure 5. Proposed General Approach to Treating Motor Symptoms in Parkinson Disease



PARAMEDICAL TREATMENT

- physiotherapy
- Occupational therapy
- Speech and swallowing therapy

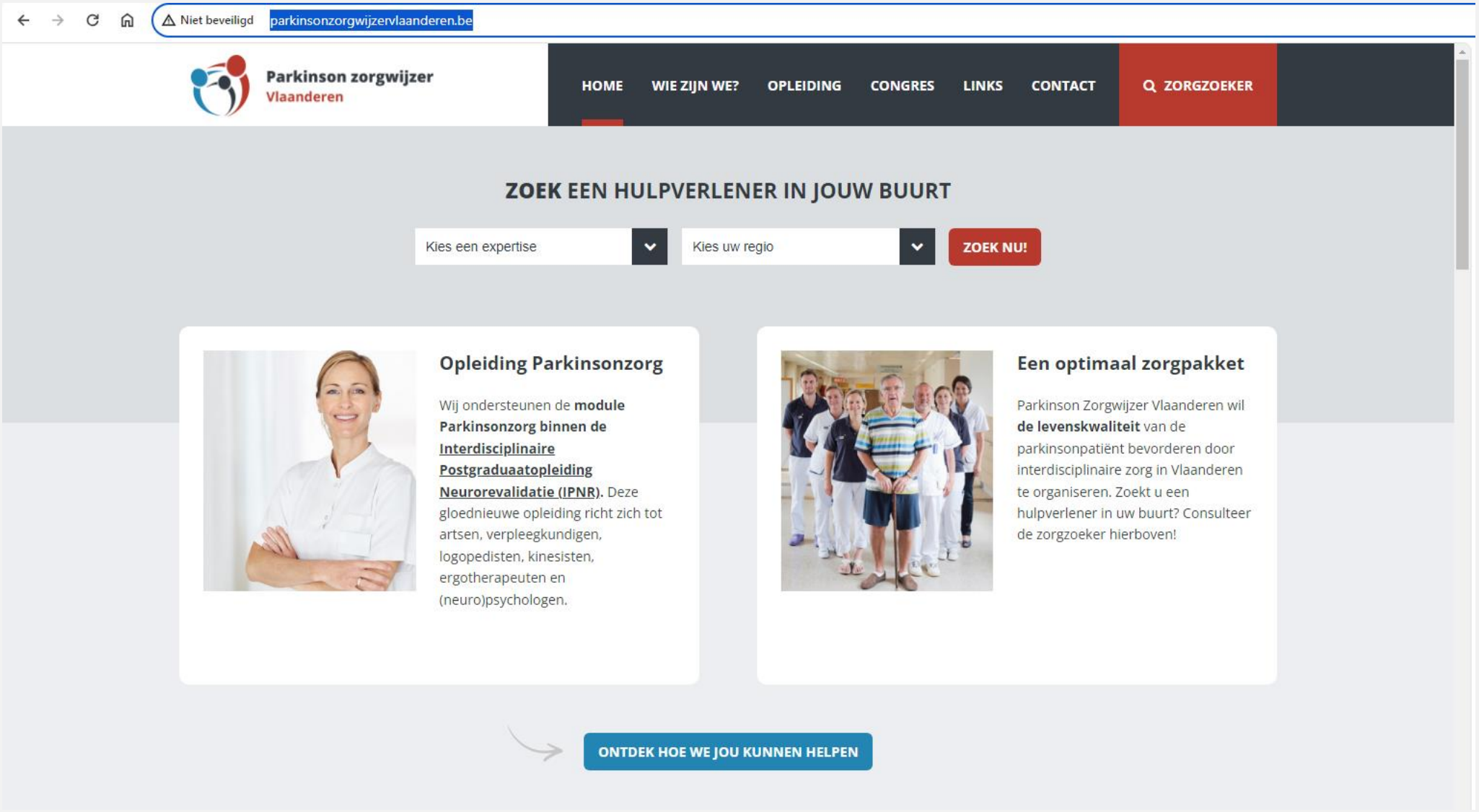




Appendix A. Post-hoc analysis of the separate PD related complications. [a]

Percentage of patients with hospital visit or admission for PD-related complications	**Specialised physiotherapy N = 2,129**	**Usual care physiotherapy N = 2,252**		
	Proportion (n)	Proportion (n)	Difference (95% CI)	p-value
Fractures (e.g. hip or wrist)	6.1 (129)	7.6 (172)	1.6 (0.1;3.3)	0.0390
Other orthopaedic injuries	9.5 (203)	11.5 (259)	1.9 (0.1;3.8)	0.0342
Pneumonia	2.4 (52)	3.5 (79)	1.1 (0.1;2.1)	0.0385
At least one PD-related complication (fractures, other orthopaedic injuries, or pneumonia)	17.3 (368)	21.3 (480)	4.0 (1.7;6.4)	0.001

[a] Our primary endpoint included patients that had at least one of three possible PD-related complications (fractures; other orthopedic injuries; or pneumonia). Some patients had more than one complication (e.g. a hip fracture as well as pneumonia). Therefore, the sum of the number of patients reported for the separate components is higher than the total number of patients with at least one any PD-related complication.



- http://www.parkinsonzorgwijzervlaanderen.be/

BEHANDELING NON MOTORE SYMPTOMEN

- Reageren op levodopa
  - Sialloree
  - Zweten
  - Seksuele dysfunctie
- Reageren op fysieke inspanning:
  - Orthostatisme
  - Slaap
  - Obstipatie
- Reageren op DBS:
  - Urinaire dysfunctie
  - zweten

TREATMENT ORTHOSTATIC HYPOTENSION

TABLE 9-2 **Nonpharmacologic Treatments for Orthostatic Hypotension**

- Liberalization of salt consumption
- Liberalization of water intake (up to 2.5 L/d)
- Acute water bolus (drinking 500 mL water)
- Sleeping with the head of the bed raised 30 to 45 degrees with the help of an electric bed or mattress
- Physical activity with recumbent exercises (eg, stationary bicycle, rowing machine) or in a swimming pool
- Physical countermaneuvers (eg, standing up slowly, leg crossing, buttock clenching)[52]
- Abdominal binder[53]
- Waist-high compression stockings producing at least 15 mm Hg to 20 mm Hg pressure[54] (knee-high or thigh-high stockings are typically not useful)

Treatment	Recommended Dosage	Mechanism of Action	Side Effects
Specifically approved for orthostatic hypotension			
Midodrine	2.5–15 mg 2 or 3 times a day (dosed morning, midday, and 3–4 hours before bedtime) or tailored to the patient's needs	Direct α_1-adrenergic receptor agonist	Supine hypertension, piloerection ("goose bumps"), scalp itching, urinary retention; caution in congestive heart failure and chronic renal failure
Droxidopa	100–600 mg 3 times a day (dosed morning, midday, and 3–4 hours before bedtime) or tailored to the patient's needs	Synthetic norepinephrine precursor	Supine hypertension, headache, nausea, fatigue; caution in congestive heart failure and chronic renal failure
Not specifically approved for orthostatic hypotension			
Atomoxetine	10–18 mg 2 times a day	Norepinephrine reuptake inhibitor	Supine hypertension, insomnia, irritability, decreased appetite
Fludrocortisone	0.05–0.2 mg once a day; no benefit with dosages higher than 0.2 mg/d	Synthetic mineralocorticoid, volume expander that increases sodium and water reabsorption	Supine hypertension, hypokalemia, renal failure, edema, target organ damage; caution in congestive heart failure
Pyridostigmine	30–60 mg 2 or 3 times a day	Acetylcholinesterase inhibitor	Abdominal cramps, diarrhea, sialorrhea, excessive sweating, urinary incontinence

DROOLING

	Dose	Evidence in patients with Parkinson's disease	Outcomes	Side-effects
Systemic anticholinergics				
Glycopyrrolate	1–2 mg twice or three times daily	4-week randomised double-blind, placebo-controlled crossover trial in 23 patients[112]	Significant change in sialorrhoea scoring scale	No difference from placebo (blood–brain barrier not crossed); behavioural changes reported in children and young adults with cerebral palsy;[113] could cause peripheral side-effects (eg, constipation)
Topical anticholinergics				
Sublingual atropine	0·5 mg twice daily (eg, 1 drop of 1% atropine solution)	1-week open-label study in six patients[114]	Improvement in objective and subjective measures	Can cross the blood–brain barrier, producing the same side-effects as systemic administration, including confusion[114]
Sublingual ipratropium bromide	21–42 µg (1–2 sublingual sprays) up to four times daily	5-week randomised double-blind, placebo-controlled, crossover study in 17 patients[115]	No improvement in objective evaluation (primary endpoint) but improvement in subjective ratings	No difference from placebo (blood–brain barrier not crossed)
Tropicamide	Intra-oral films up to 3 mg, one dose	Single-dose pilot study with a randomised double-blind, placebo-controlled, crossover design in 12 patients[116]	A non-significant improvement was seen in the 0·3 mg and 1 mg dosage groups	No difference from placebo
Systemic alpha-2 agonists				
Clonidine	0·15 mg per day	12-week double-blind, placebo-controlled study in 32 patients[117]	Significant improvement of the frequency of clearing saliva	Diurnal somnolence, dizziness, and dry mouth
Systemic alpha-1 agonists				
Modafinil	100 mg per day	A study in patients with Parkinson's disease and Hoehn and Yahr grade 2–3 with moderate to severe drooling;[118] study type not available	Reduction in drooling severity score and patient-reported improvement	Positive effect on drooling might be related to the improvement of dysphagia[22]
Botulinum neurotoxin serotype A and B				
Onabotulinumtoxin A	Parotid gland 5–50 U; submandibular gland 5 U	A case series;[119] three open-label studies;[120–122] an open-label case-control study;[123] a randomised placebo-controlled study;[124] and a randomised double-blind, placebo-controlled study[125]	Similar positive outcomes for botulinum neurotoxin A (abobotulinumtoxin A) and botulinum neurotoxin B, but a faster effect after botulinum neurotoxin B, probably in view of an increased affinity of rimabotulinumtoxin B for autonomic terminals[126]	Safe; only minimum side-effects reported—dryness of mouth and increased saliva viscosity, which might depend on the distribution of botulinum neurotoxin in the major salivary glands
Abobotulinumtoxin A	Parotid gland 75–146·2 U; submandibular gland 78·7 U	A case series;[127] two randomised double-blind, placebo-controlled studies;[128,129] and a randomised double-blind, crossover trial compared with rimabotulinumtoxin B[126]		
Rimabotulinumtoxin B	Parotid gland 500–2000 U; submandibular gland 250 U	Two open-label studies;[130,131] three randomised double-blind, placebo-controlled studies;[132–134] and a randomised double-blind, crossover trial compared with abobotulinumtoxin A[126]		

Table 2: **Pharmacological treatments for drooling in patients with Parkinson's disease**

COGNITION

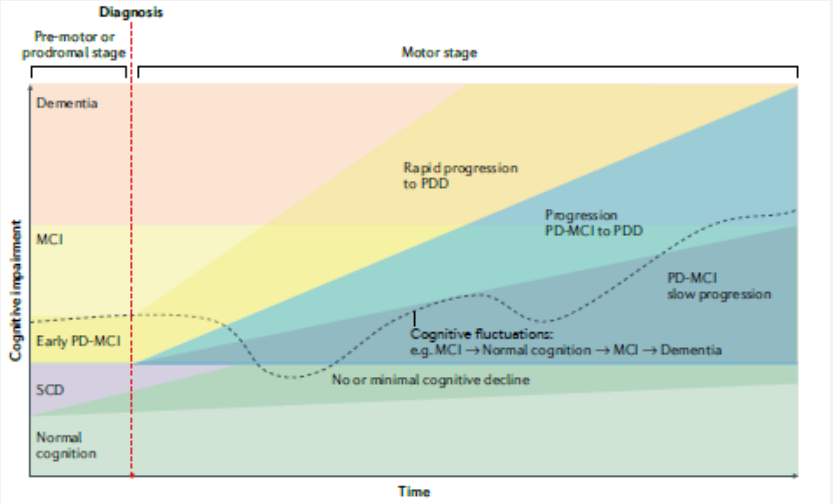


Fig. 1 | **The cognitive spectrum and the heterogeneity of progression of cognitive impairment in Parkinson disease.** Cognitive changes, mostly in the form of subjective cognitive decline (SCD) or mild cognitive impairment (MCI) can occur prior to or at the time of Parkinson disease (PD) diagnosis or even decades later, with high variability in the rate of progression. Cognitive fluctuations may also occur, in which, for example, some patients with PD-associated MCI (PD-MCI) may revert to normal cognition and then develop cognitive impairment later in the disease course, typically accompanied by motor progression and the occurrence of other non-motor symptoms. PDD, Parkinson disease dementia.

NATURE REVIEWS | DISEASE PRIMERS | Article citation ID: (2021) 7:47

- To date, the only unequivocally positive RCT for PDD was for the cholinesterase inhibitor (ChEI) rivastigmine
- Although donepezil and galantamine have insufficient evidence for the treatment of PDD, they have been rated as ‘possibly useful’ by the International Parkinson and Movement Disorder Society Evidence-Based Medicine Committee

BEHAVIOUR

- minor psychosis occurs in 25–40% of patients with Parkinson's disease, visual hallucinations in 15–30%, non-visual hallucinations (eg, auditory, tactile, and olfactory) in up to 35%, and delusions in about 4%.

- **Quetiapine**
- **Clozapine**, cave agranulocytose
- AChR inhibitors
- Stop/reduce dopaminergic medication: anticholinergics, amantadine > MAOi > COMTi > DA > levodopa

- *Quetiapine has little robust evidence to support its use, but is the most commonly prescribed antipsychotic*
- *in patients with Parkinson's disease, whereas clozapine is efficacious but rarely used*

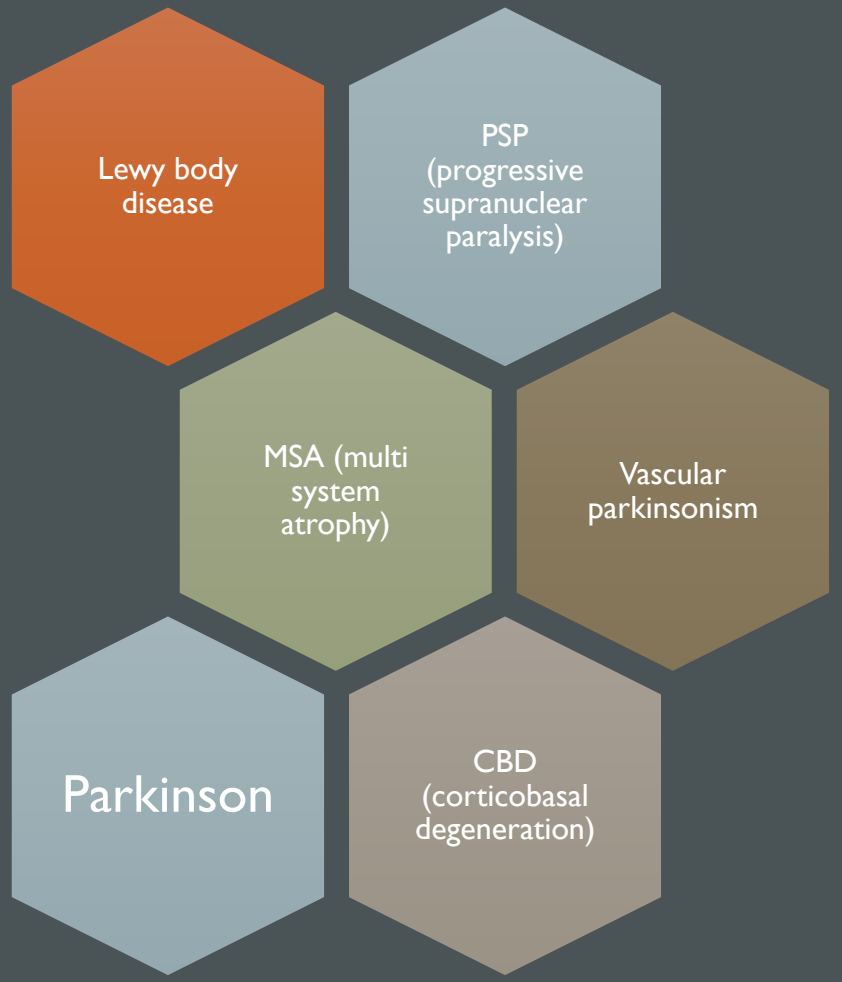
TREATMENT OF DEPRESSION

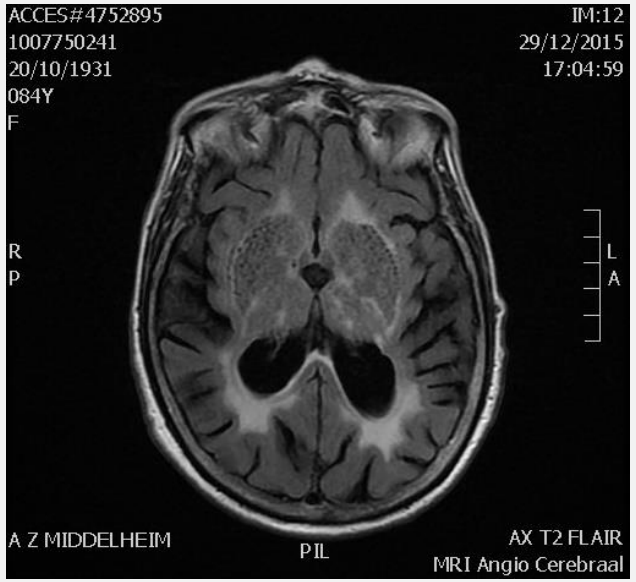
- Findings support the safety and efficacy of several classes of antidepressants in patients with Parkinson's disease, including **selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, and tricyclic antidepressants**, with comparable effect sizes across drug classes.
- Dopamine agonists might confer a mood benefit
- Psychologist/psychiatrist
- Don't forget the caregiver

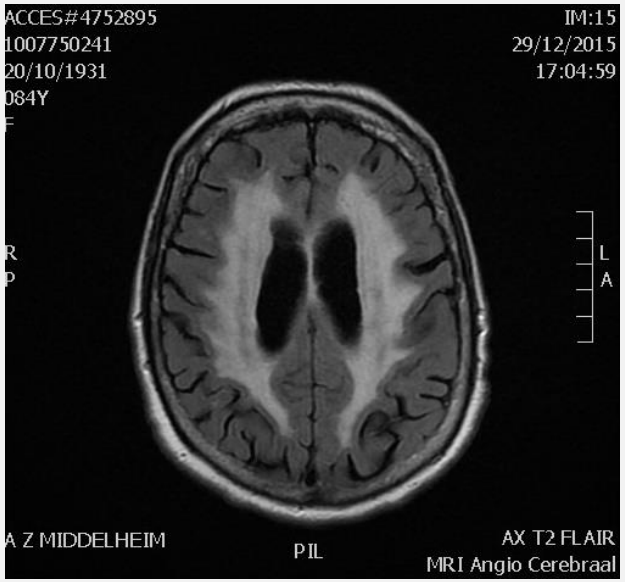
WHAT'S COMING?

- Other administration formulas of levodopa: SC, inhalation
- Biomarkers → start therapy at prodromal stages
- Disease modifying strategies:
  - Gene therapy
  - Stem cells
  - Vaccination – alfa-synuclein as a target

PARKINSON PLUS SYNDROMES







VASCULAR PARKINSONISM

Lower body parkinsonisme

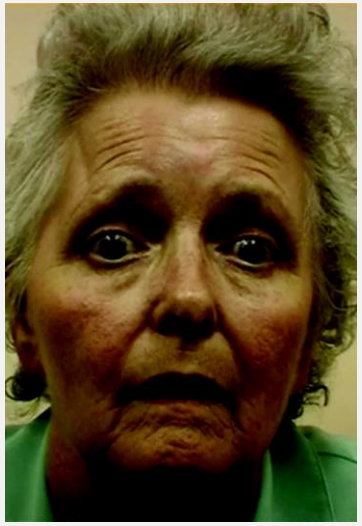
limited levodoparespons

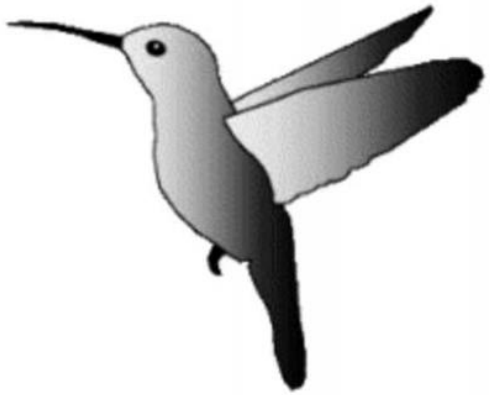
Strategic infarcts nigrostriatal pathway

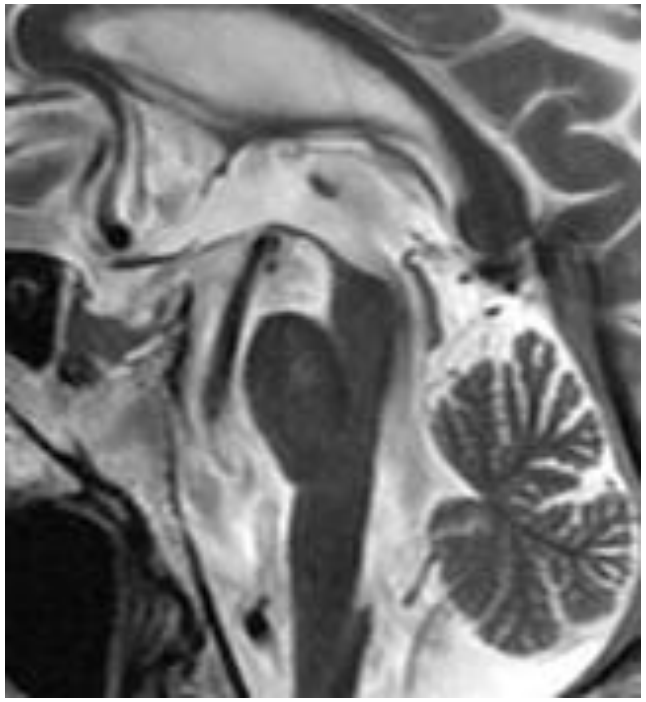
presynaptic dopamine transporter (DAT) deficiëntie

PROGRESSIVE SUPRANUCLEAR PALSY

- Tauopathie? (CBD, TDP43, C9Orf72)
- Age of onset >60j
- Gait: normale stride length, straight posture, hyperextension
- Disturbed balance, retropulsion
- Symmetrical
- Axial rigidity
- Supranuclear gaze palsy
- Overactivity M frontalis (procerus)
- Eye lid retraction





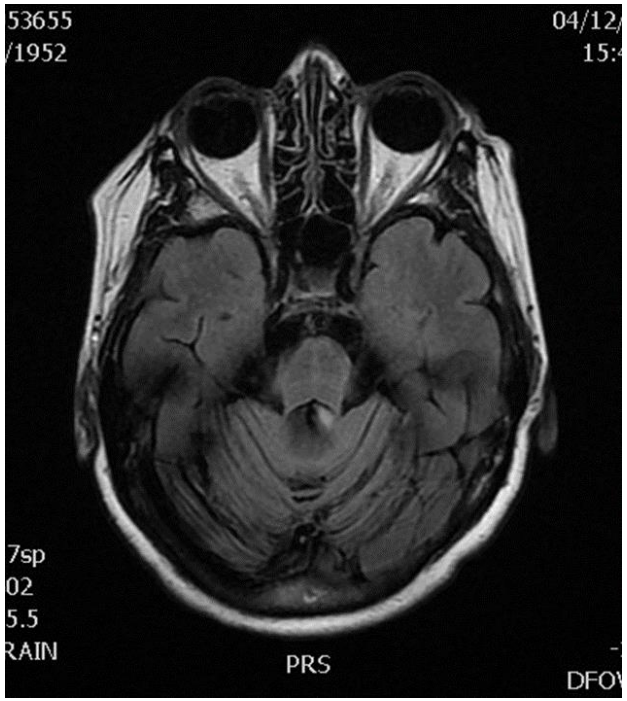


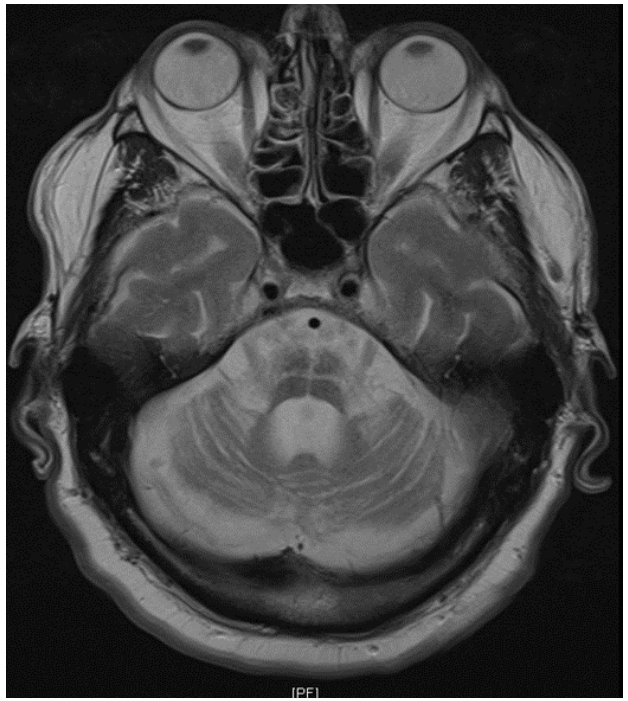
HUMMINGBURD SIGN

MULTISYSTEM ATROPHY

- Alfa synucleinopathy
- Asymmetrical (rigidity, bradykinesia)
- Sometimes good initial levodoparespons
- Gait problems, wide based
- Autonomic dysfunction : severe and early
- Cerebellar signs

- Pyramidal syndrome (Babinski's)
- Antecollis
- dysarthria, swallowing problems
- Stridor, slaapapnoe
- rarely dementia
- mostly 6th decade, late onset not impossible!
- MSA-P
- MSA-C

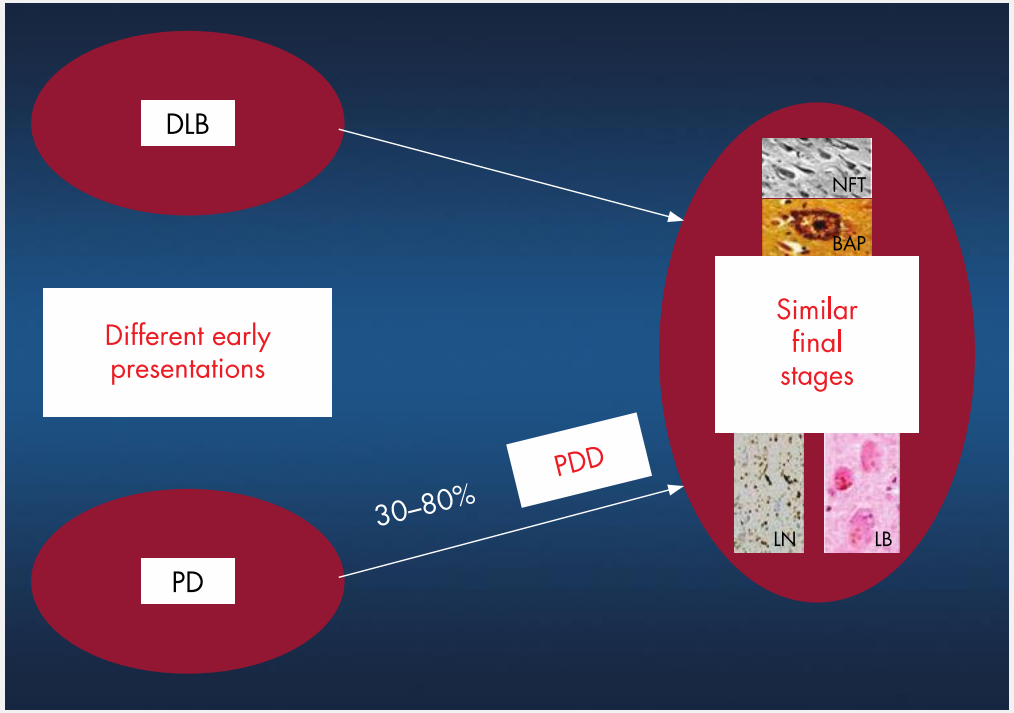




HOT CROSS BUN SIGN

LEWY BODY DISEASE

- primary dementia characterised by visuoperceptual and executive dysfunction accompanied by (1) prominent visual hallucinations, (2) fluctuating attention and (3) parkinsonism.
- Dementia together with or before parkinsonism <-> PD dementia (>1y)



TAKE HOME MESSAGES

- Every patient is different
- Fluctuations, freezing
- Intake Prolopa on an empty stomach, correct timing!
- Prolopa ≠ Prolopa HBS ≠ Prolopa disp
- No typical neuroleptics! Cave anti-emetics!
- Importance of physiotherapy

THANK YOU FOR YOUR ATTENTION

QUESTIONS:

EMKE.MARECHAL@ZNA.BE

SAVE THE DATE

- vrijdagnamiddag 18/10/2024
- ZAS Cadix

MS symposium