
The Many Faces of Parkinson's Disease

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Parkinson's Disease

- Parkinson's disease was first described in 1817 by James Parkinson, a British physician. He called it “The Shaking Palsy”

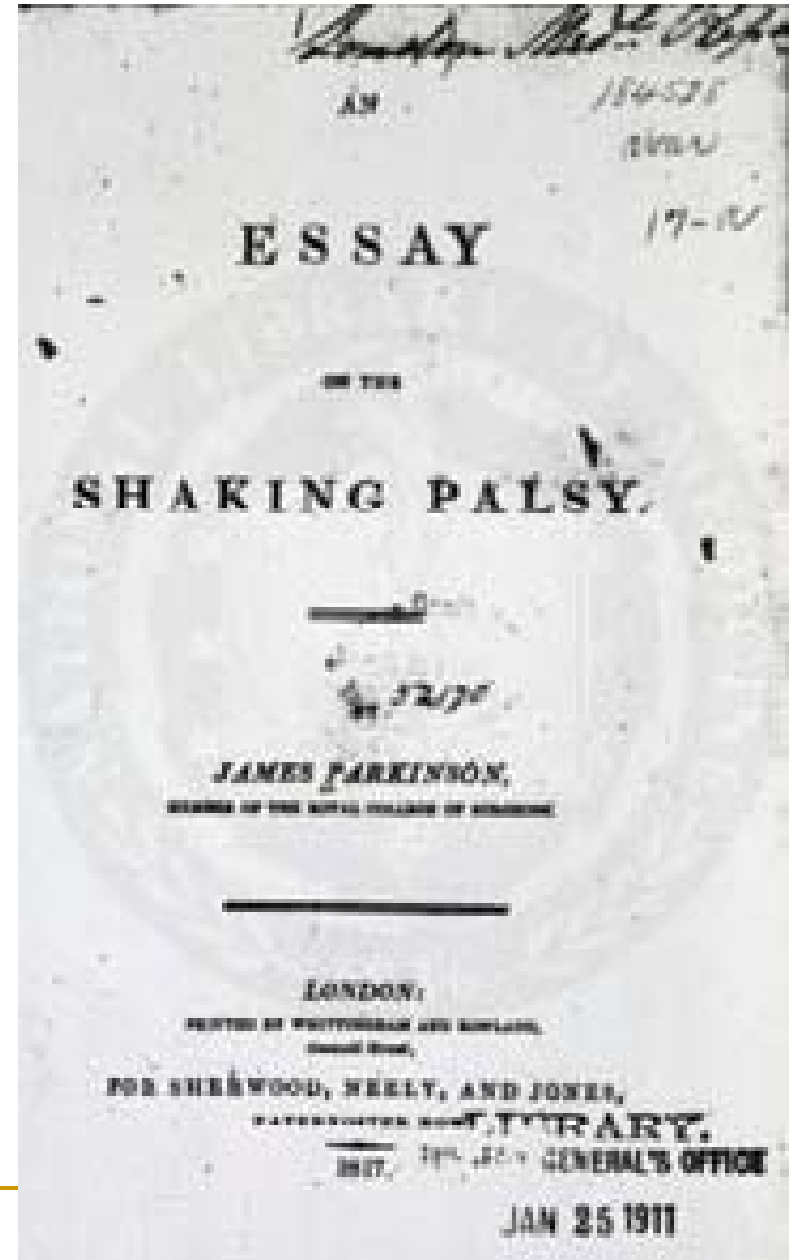
Parkinson's Disease



James C. Parkinson

**DR. JAMES PARKINSON
(1755 - 1828)**

**ESSAY ON THE SHAKING PALSY
(1817)**



Parkinson's Disease

- Parkinson's disease is a chronic, progressive, neurodegenerative disease
 - Associated with significant increase in morbidity and disability
 - Substantial burden on pts, families and caregivers
 - Lower life expectancy than general population
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Parkinsonism

- Parkinsonism is also a major feature of several dementing diseases
 - Many Parkinsonian disorders have “frontal-subcortical” cognitive deficits including mental slowness, inertia, lack of initiative, forgetfulness, visuospatial deficits, decreased executive functioning and mood sx
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Epidemiology of Parkinson's Disease

- The usual age of onset b/w ages of 50-65 years
 - Incidence appears to be lower in African Americans than Caucasians
 - Male : female ratio is 3:2
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Background

- Idiopathic Parkinson Disease (also referred to as primary or classical Parkinson disease), is a progressive neurodegenerative disorder associated with decrease dopamine in parts of the brain (nigrostriatal neurons).
 - Affecting about 0.4% people >40y
 - 1% people >65y
 - 10% people >80y
 - Cardinal features: Resting tremor, Rigidity, Bradykinesia, and postural instability.
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Background (...)

- **Prevalence:** 120 per 100,000
 - **Age:** incidence and prevalence increase with age. average age of onset is approximately 57 years
 - **Etiology of Idiopathic PD:** interplay of
 - **Genetic;** several genetic form of the disease have been identified.
 - **Environmental factors;** toxins (e.g,pesticides), oxidative stress and viral infections.
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Clinical Features of PD

Cardinal features

Resting tremors

Rigidity

Bradykinesia

Postural instability

Symptoms and signs

■ Premotor phase:

- Initial symptoms may be nonspecific; fatigue, depression, Constipation, decreased sense of smell and sleep problem, daytime sleepiness, REM behavior disorder (RBD) , in one study,38% of 50y/o men with RBD and no neurological signs went on to develop parkinsonism.

■ Motor signs:

- A subtle decrease in dexterity, Difficulty with specific tasks; turning in bed, opening jars, rising from a chair, a lack of coordination with activities such as playing golf or dressing, complain of aching or tightness in the calf or shoulder region, the first affected arm may not swing fully when walking, and the foot on the same side may scrape the floor.
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Symptoms and signs

- **A resting tremor** of one hand is often the first symptom. The tremor is characterized as follows:
 - • Slow and coarse
 - • Maximal at rest, lessening during movement, and absent during sleep
 - • Amplitude increased by emotional tension or fatigue
 - • Often involving the wrist and fingers in movements similar to those used to manipulate small objects or pills (**pill-rolling tremor**)
 - Usually, the hands, arms, and legs are most affected, in that order. The jaw, tongue, forehead, and eyelids may also be affected, but not the voice. Tremor may become less prominent as the disease progresses.
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Symptoms and signs

- **Rigidity** develops without tremor in many patients. When a clinician moves a rigid joint, sudden, rhythmic jerks due to variations in the intensity of the rigidity occur, producing a ratchet-like effect (**cogwheel rigidity**).
-

Symptoms and signs

- **Slow movements** (*bradykinesia*) are typical as rigidity progresses. Movement also becomes decreased (*hypokinesia*) and difficult to initiate (*akinesia*).
 - Rigidity and hypokinesia may contribute to; **muscular aches** and sensations of **fatigue**.
 - **masklike face** , with an open mouth, drooling, and reduced blinking.
 - patients may appear depressed due to masklike face and bradykinesia.
 - Speech becomes hypophonic, with characteristic monotonous, **stuttering dysarthria**.
 - **micrographia** (writing in very small letters) due to hypokinesia and impaired control of distal musculature (that make activities of daily living increasingly difficult).
 - Without warning, voluntary movement, including walking, may suddenly halt (called **freezing**).
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Symptoms and signs

- **Postural instability** develops, resulting in gait abnormalities. Patients have difficulty starting to walk, turning, and stopping; the gait becomes **shuffling** with short steps, and the arms are held flexed to the waist and do not swing with the stride. Steps may inadvertently quicken, and patients may break into a run to keep from falling (**festination**). A tendency to fall forward (**propulsion**) or backward (**retropulsion**) when the center of gravity is displaced results from loss of postural reflexes. Posture becomes **stooped**.
-



Symptoms and signs

- **Dementia**; generally occurs late in the disease and affects 15-30% of patients. short term memory and visuospatial function may be impaired, but aphasia is not present.
 - Cognitive dysfunction within a year of onset of motor features suggests a diagnosis of Lewy body disease, a disease closely related to parkinson disease and marked by the presence of cortical Lewy bodies
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Parkinson's disease dementia

- Pts with PD have increased incidence of dementia. Risk of dementia is 6 times higher than seen in general population
 - 30-50% pts with Parkinson's disease develop dementia particularly common in 65 or older
 - Dementia with PD cause rapid functional decline, reduced quality of life, early death
-

Parkinson's disease dementia

- Age of onset of PD after 60
 - Low educational attainment
 - Increased severity of movement disturbances especially Bradykinesia
 - Family history of dementia
 - Presence of depression may contribute in cognitive impairment
 - APOE4 is not a risk factor for PD/PDD
-

Cognitive features

- PDD pts can have a long period of subtle cognitive changes preceding dementia
 - Usually develop 5-6 y after the beginning of Parkinsonian sx
 - Insidious development of bradyphrenia (cognitive slowing), attentional deficits, impairment in executive functions and visuospatial tasks
-

Cognitive features

- Although memory impairment is not as severe as in AD. As dementia progresses, more impairment in short term memory and retrieval of information
 - Visuospatial abilities are particularly impaired, more in PDD than AD
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Cognitive features

- Deficits occur in planning, frontal executive functions, set shifting & response initiation and poor performance on timed tasks
 - Deterioration in abstraction and in problem solving
 - Problems on tasks of delayed recall, semantic memory, speech and language
-

Cognitive features

- Speech changes:

Poor verbal fluency, volume is reduced, speech is monotonous and dysarthric

Impairment of word list generation tasks, reduced sentence length and comprehension, but usually intact naming until late stage

Symptoms and signs

- **Neurologic symptoms unrelated to parkinsonism** commonly develop because synucleinopathy (Lewy bodies) occurs in other areas of the central, peripheral, and autonomic nervous systems. It may have the following effects:
 - • Almost universal sympathetic denervation of the heart, contributing to **orthostatic hypotension**
 - • Esophageal dysmotility, contributing to **dysphagia** and increased risk of **aspiration**
 - • Lower bowel dysmotility, contributing to **constipation**
 - • Commonly, **anosmia** and **urinary hesitancy** and/or **urgency**.
 - **Seborrheic dermatitis** is also common.
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Stages of idiopathic PD

Stage I : One-sided resting tremor, with or without slowed movements (bradykinesia). Mildly affected patients may not need treatment, whereas those with moderate disability will be more comfortable with therapy.

Stage II : Moderate bilateral tremor or rigidity, plus bradykinesia. Symptoms improve with treatment. Median time from onset of symptoms: 25 months.

Stage III : Significant tremor, rigidity and/or bradykinesia, **plus** mobility and balance problems: difficulties in postural control; unsteadiness on turns; hesitations, halts, and freezes when starting to walk. Functional levels fluctuate during the day. Drug-induced dyskinesias may arise. Median time from onset: 42 months.

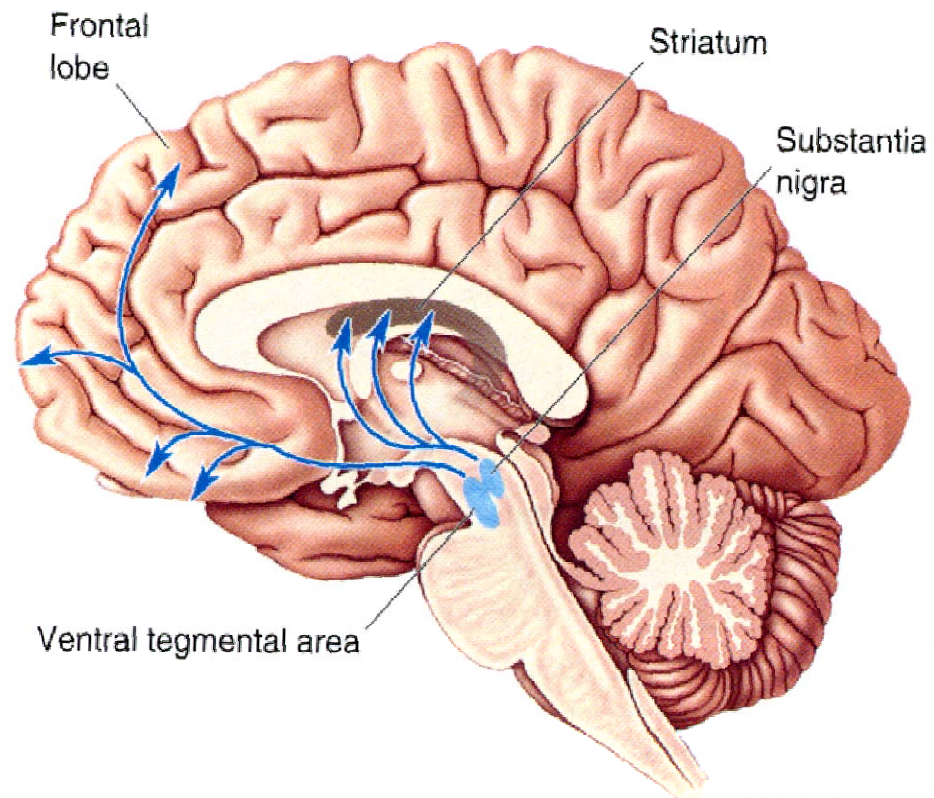
Stage IV : More severe disability, but still able to walk. More severe bradykinesia, often resulting in an inability to dress (e.g., button shirt), to cut food, etc. Assistance with daily activities needed. Fluctuations more severe. Median time from onset: 55 months.

Stage V : Unable to function independently. Severe postural instability. Independent mobility impossible. Median time from onset: 62 months.

Pathophysiology

- The major neuropathologic findings in Parkinson disease are:
 - a **loss of Pigmented dopaminergic neurons** in the substantia nigra (approximately 60-80% are lost before the motor signs of Parkinson disease emerge)
 - the **presence of synuclein-filled Lewy bodies** within the pigmented neurons of the substantia nigra (Lewy bodies also are found in The other parts of the CNS, are not specific to parkinson disease, the prevalence of incidental Lewy bodies increases with age and are hypothesized to represent the presymptomatic phase of parkinson disease.)
 - No standard criteria exist for the neuropathologic diagnosis of parkinson disease so far.
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Dopamine system

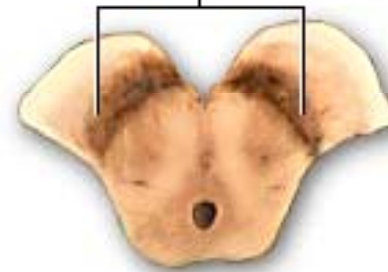




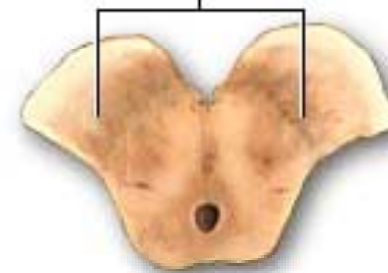
Cut section
of the midbrain
where a portion
of the substantia
nigra is visible



Substantia nigra



Diminished substantia
nigra as seen in
Parkinson's disease

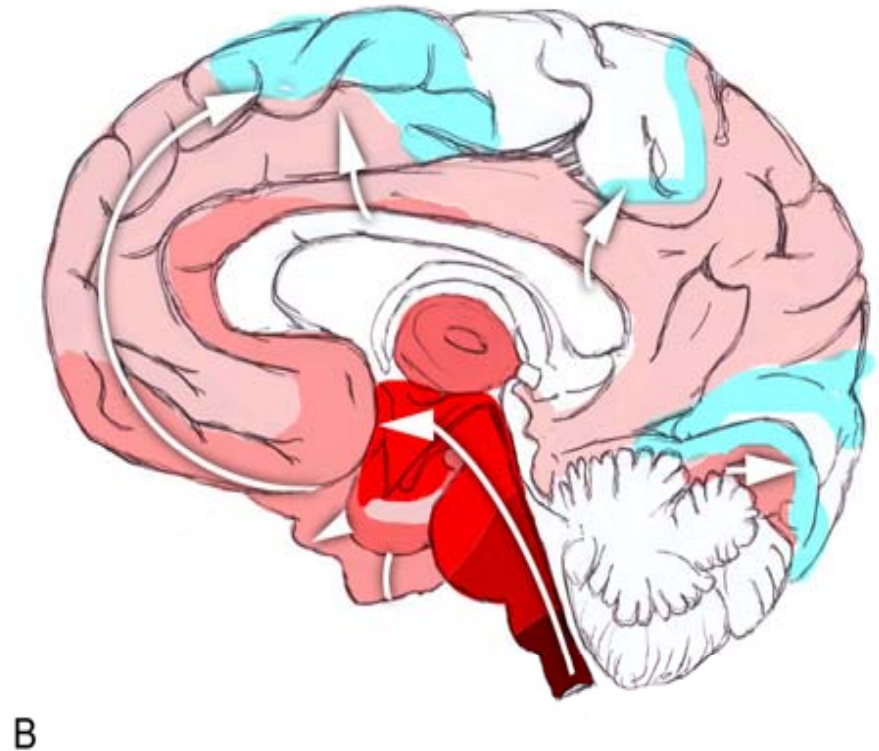
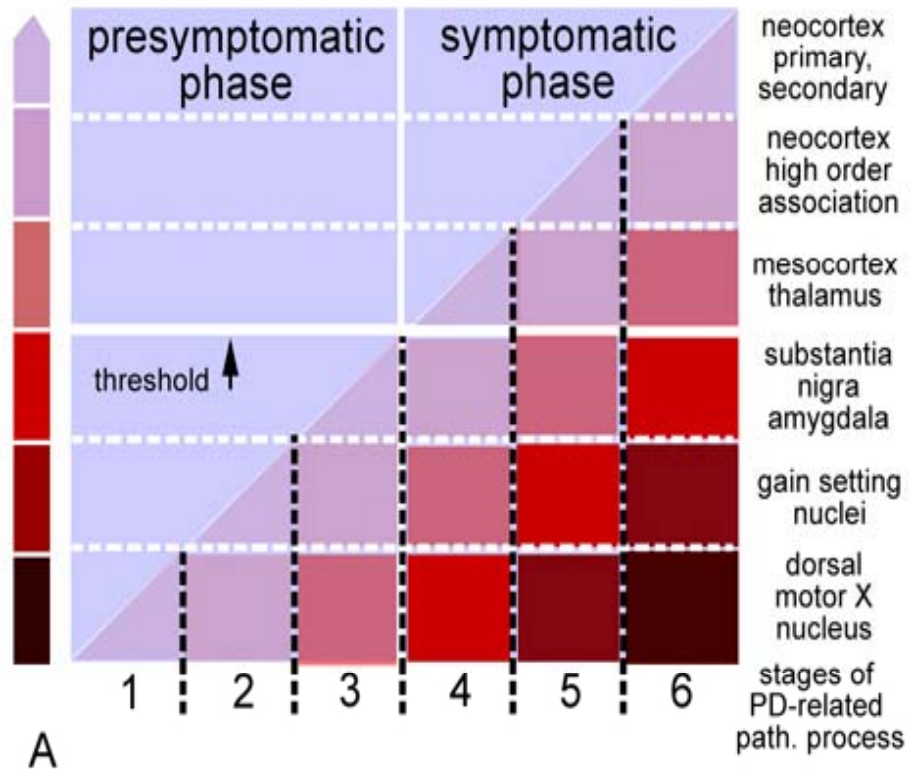


ADAM.

Pathophysiology (...)

- **Lewy body pathology in parkinson disease begins; in the olfactory bulb and lower brainstem** (associated with premotor symptoms such as loss of sense of smell and rapid eye movement (REM) sleep behavior disorder)
 - **Pathology ascends up to the brain stem to involve the midbrain and nigrostriatal dopaminergic neurons** (correlate with onset of motor phase of disease; Bradykinesia, rigidity, and tremor).
 - **Pathology continues to ascend late in the disease to affect the cortex** (patient may exhibit cognitive dysfunction and dementia.)
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Pathophysiology (...)



Pathophysiology (Motor circuit in Parkinson disease)

- The basal ganglia motor circuit modulates cortical output necessary for normal movement.
 - Signals from the cerebral cortex are processed through the basal ganglia-thalamocortical motor circuit and return to the same area via a feedback pathway.
 - In Parkinson disease, decreased striatal dopamine causes increased inhibitory output from basal ganglia which suppresses movement
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FIG. 4. In the laboratory of the neurology clinic, Munich: 1, F. Lotmar; 2, Frau Grombach; 3, St. Rosenthal; 4, Ugo Cerletti; 5, Allers(?); 6, F. Bonfiglio; 7, A. Alzheimer; 8, N. Achucarro; 9, G. Perusini; 10, F. H. Lewy.

Differential diagnosis of Parkinsonism
(General classification)

1-Idiopathic PD

2-Atypical PD

3- Essential Tremor (ET)

DDX

■ 2-Atypical PD

Neurodegenerative disorders other than idiopathic PD, including dementia with Lewy bodies, corticobasal degeneration, multiple system atrophy, progressive supranuclear palsy.

Secondary parkinsonism; a wide variety of conditions can cause secondary parkinsonism, including;

- **Drugs**(classic and atypical antipsychotic agents, haloperidol, pimozide, chlorpromazine, droperidole, fluphenazine, trifluoperazine, Metoclopramide, domperidone, flunarizine, , Reserpine prochlorperazine, illegal or street drugs)
- **Toxins** ; carbon disulfide, carbon monoxide, cyanide, MPTP, manganese, organic solvents.

DDX (detailed classification...)

- **Head trauma**; isolated or repeated (e.g., boxing)
 - **Structural brain lesions that affect striatonigral circuits**, e.g, Hydrocephalus, chronic subdural hematoma, tumors
 - **Metabolic and miscellaneous disorders** (e.g, wilson disease, hypoparathyroidism and pseudohypoparathyroidism, chronic liver failure, extrapontine myelinolysis, neurodegeneration with brain iron accumulation, neuroacanthocytosis.)
 - **Infections**; encephalitis lethargica or economo's encephalitis, HIV/AIDS, neurosyphilis, prion disease, progressive multifocal leukoencephalopathy, toxoplasmosis.
-

DDX (detailed classification...

- **Small vessel disease**; vascular parkinsonism, multiple lacunar infarcts in the basal ganglia and /or Binswanger's disease (this entity is controversial, because most basal ganglia infarcts are not associated with parkinsonian signs.)



Diagnosis ...

- Diagnosis is based on HX and PE
 - Neuroimaging (CT,MRI) may be used to R/O other abnormalities , thus help identify secondary causes of parkinsonism.
 - **Response to levodopa treatment**; Idiopathic PD usually have a good response, comparing to atypical PD who have a poor and transient response. also they show a higher incidence of side effects to anti parkinson medications (particularly confusion, agitation, and hallucinations)
-

Parkinson – Plus Syndromes

Parkinson – Plus Syndromes

- Look like but different from primary Parkinson's disease.
 - A heterogeneous group of diseases presenting as a parkinsonian syndrome associated with other neurological signs, reflecting degeneration in various neuronal systems.
 - Often confused with primary P.D., but prognosis and response to treatment differ greatly.
-

Parkinson – Plus Syndromes

- Progressive supra-nuclear palsy. PSP
 - Multi-system atrophy. MSA :
 - Shy Drager syndrome SDS
 - Olivopontocerebellar atrophy. OPCA
 - Striatonigral degeneration. SND
 - Corticobasal ganglionic degeneration. CBGD
-

Progressive supra-nuclear palsy. PSP

- Steele, Richardson, Olzweski in 1964
 - Most common p-plus syndrome
 - Insidious beginning with :
 - Postural instability
 - Bradykinesia
 - Axial rigidity
 - Ophthalmoplegia – vertical gaze palsy
 - Dementia with fronto-temporal features
-

Progressive supra-nuclear palsy. PSP

- ? Not primary P.D.
 - Sx usually symmetrical
 - Rigidity : axial > limb
 - Tremor rare
 - Cervical/facial dystonia
 - Corticospinal signs
 - Corticobulbar signs
-

Progressive supra-nuclear palsy. PSP

- Diagnosis made clinically and confirmed at autopsy.
 - Imaging shows midbrain atrophy, and cortical and frontal hypometabolism.
 - Tx – trial of dopaminergic agent, botox for dystonia, AchE inhibitors ineffective.
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Parkinson – Plus Syndromes

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Multi-system atrophy. MSA

- Three subtypes which commonly overlap:
 - ❑ Shy Drager syndrome
 - ❑ Autonomic signs
 - ❑ Olivopontocerebellar atrophy
 - ❑ Cerebellar signs
 - ❑ Striatonigral degeneration
 - ❑ Pyramidal signs
-

MSA : Shy Drager syndrome

- Similar to P.D. with autonomic symptoms, but ANS impairment is much more severe.
 - Orthostatic hypotension > 30 mm drop syst.
 - GU symptoms often early
 - Autonomic signs usually in the 50's
 - Neurologic signs usually in the 60's
-

MSA : Shy Drager syndrome

- Cognitive and pyramidal degeneration
 - Rigidity – bilateral and symmetrical
 - Respiratory obstruction and stridor
 - CT shows atrophy of the brainstem and cerebellum, MRI often shows hyperintensity of the putamen
 - Non-pharmacological measures first
-

Parkinson – Plus Syndromes

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-

MSA

Olivopontocerebellar atrophy. OPCA

- Characterized by neuronal loss in the cerebellum and brainstem.
 - Ataxia (lower limb involvement) usually seen first, followed by the upper limbs, and then dysarthria
 - Kinetic tremor
 - Imaging shows diffuse cerebellar and brainstem atrophy
-

Striatonigral degeneration. SND

- The most difficult to differentiate from primary P.D.
 - Predominant symptoms involve extrapyramidal movements.
 - Tremor rare, no response to levo-dopa
 - Pseudo-bulbar degeneration
 - Sleep apnea
-

Parkinson – Plus Syndromes

- Progressive supra-nuclear palsy. PSP
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 - Corticobasal ganglionic degeneration. CBGD
-

Corticobasal ganglionic degeneration.

CBGD

- Rare form of p-plus
 - Presentation is asymmetric and most commonly begins in an upper limb with slowing of movement and clumsiness.
 - Limb apraxia, movements become jerky and uncontrolled, ultimately leading to a dystonic/rigid “alien” limb.
 - Usually stays limited for 2 – 5 years before generalizing.
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Corticobasal ganglionic degeneration.

CBGD

- Gait impairment
 - Ocular movement changes
 - Cognition often intact
 - Imaging may show asymmetrical atrophy corresponding to the symptomatic side
-

FLAGS for P-PLUS

- Severe degeneration and quick progression
 - Symmetry of symptoms
 - Irregular, sudden, or absent tremors
 - Early autonomic dysfunction
 - Early falls
 - Severe dysarthria/dysphonia
 - Non-response to levo-dopa
 - Oculomotor problems
-

Diagnosis ...

- The major differences between atypical and idiopathic PD
 - Absence of resting tremor
 - Earlier onset and more rapid progression to gait disorder and postural instability
 - Rigidity that is greater in the trunk than the limbs (axial rigidity) or is very severe
 - Early onset of falls, dementia, dysphagia, or autonomic instability (e.g., dizziness associated with postural hypotension; urinary retention and incontinence; constipation; impotence; impaired thermoregulation; sweating)
 - Poor, transient, or absent motor symptom response to levodopa
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DDX ...

- **Essential tremor (ET)**; most common neurologic cause of **action tremor** (frequency 8-12/sec), estimated worldwide prevalence 5%, most often **symmetrical**, usually affects the hands and arms, but can also affect the head, voice, chin, trunk, and legs. Immediately apparent in the arms when they are held outstretched. Head tremor is more likely to be a manifestation of ET, whereas tremor of the jaw or lips is more typically parkinsonian. Other PD symptoms (rigidity, bradykinesia) are absent, Family Hx is common, benefit from treatment with beta-blocker.
-

Management

- Parkinson disease (PD) is a chronic disorder that requires broad-based management including patient and family education, support group services, general wellness maintenance, exercise, and nutrition. Treatment of PD can be divided into :
 - pharmacologic
 - Non-pharmacologic
 - surgical therapy.
-

Management (cont..)

Non-Pharmacologic treatment of Parkinson disease

Education : essential in order to provide the patient and family with some understanding and control over the disorder , available through books written for the lay audience; national and regional Parkinson disease organizations, which publish educational pamphlets and organize symposia for patients and families; and the Internet. A useful central information resource is the "We Move" Foundation at www.wemove.org.

Support : The emotional and psychological needs of the patient and family should be addressed. Normal reactions of anger, depression, anxiety, and social and economic concerns often begin with the onset of the disease and evolve as it progresses. Support for the caregiver is particularly important. Referral of the patient and/or family to a psychologist or psychiatric social worker experienced in dealing with chronic illness may be appropriate in some cases. In other instances, referral for legal, financial, or occupational counseling is indicated.

Management (cont..)

Non-Pharmacologic treatment of Parkinson disease

- **EXERCISE AND PHYSICAL THERAPY** : Exercise will not slow the progression of akinesia, rigidity, or gait disturbance, but it can prevent or alleviate some secondary orthopedic effects of rigidity and flexed posture such as shoulder, hip, and back pain, and it may also improve function in some motor tasks. Brisk walks, swimming, and water aerobic exercises are particularly useful. Referral to a physical therapist or exercise group may be a good way to get patients started in such activities.
- **Speech therapy** : Dysarthria and hypophonia are common manifestations of Parkinson disease (PD).
- **Nutrition** : Elderly patients with chronic illness are at risk for poor nutrition and weight loss. Prompt recognition and management of this problem is important to avoid loss of bone and muscle mass. No specific diet influences the course of Parkinson disease (PD), A high fiber diet and adequate hydration help manage the constipation of PD. Large, high-fat meals that slow gastric emptying and interfere with medication absorption should be avoided. Dietary protein restriction is not necessary except in some patients with advanced disease and motor fluctuations in whom competition with other amino acids interferes with L-dopa absorption.

Management (cont..)

Pharmacologic treatment of Parkinson disease

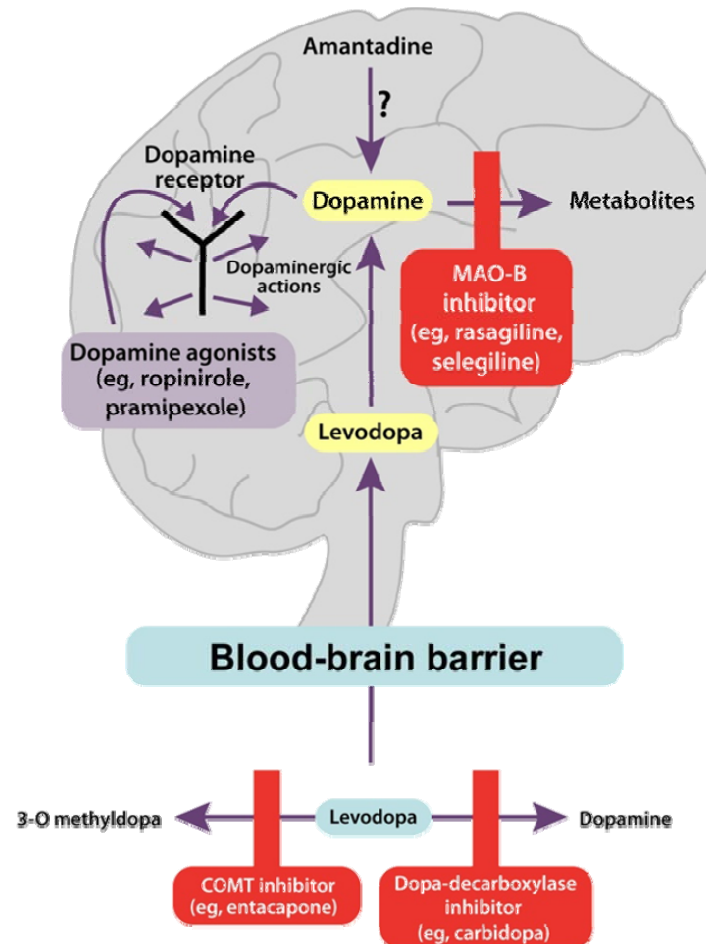
- The major drugs available for symptomatic therapy include:
 - Levodopa
 - MAO B inhibitors
 - Dopamine agonists
 - COMT inhibitors
 - Anticholinergic agents
 - Amantadine
-

Management (cont..)

Pharmacologic treatment of Parkinson disease

Generic name	Trade name	Usual starting dose	Usual maintenance dose	Mechanism
Trihexyphenidyl	Artane	1 mg BID	2 mg BID-TID	Anticholinergic
Benzotropine	Cogentin	0.5 mg BID	1 to 2 mg BID-TID	Anticholinergic
Amantadine	Symmetrel	100 mg BID	100 mg BID-TID	?
Selegiline	Eldepryl	5 mg	5 mg q am	MAO B inhibitor
Carbidopa/levodopa	Sinemet	25/100 mg TID	25/250 mg TID-QID	Dopamine precursor
Carbidopa/levodopa	Sinemet CR	25/100 mg TID	50/200 mg TID	Dopamine precursor
Apomorphine	Apokyn	2 mg SC test dose	2 to 10 mg SC TID	Dopamine agonist
Bromocriptine	Parlodel	2.5 mg daily	5 to 10 mg QID	Dopamine agonist
Pergolide	Permax	0.05 mg daily	0.5 to 1.0 mg TID	Dopamine agonist
Pramipexole	Mirapex	0.125 mg TID	1.5 mg TID	Dopamine agonist
Ropinirole	Requip	0.25 mg TID	1.0 mg TID	Dopamine agonist
Entacapone	Comtan	200 mg with L-dopa	600 to 800 mg a day	COMT inhibitor
Tolcapone	Tasmar	100 mg TID	100 to 200 mg TID	COMT inhibitor

Current Therapies for Parkinson's Disease



COMT = catechol-O-methyltransferase; MAO-B = monoamine oxidase B

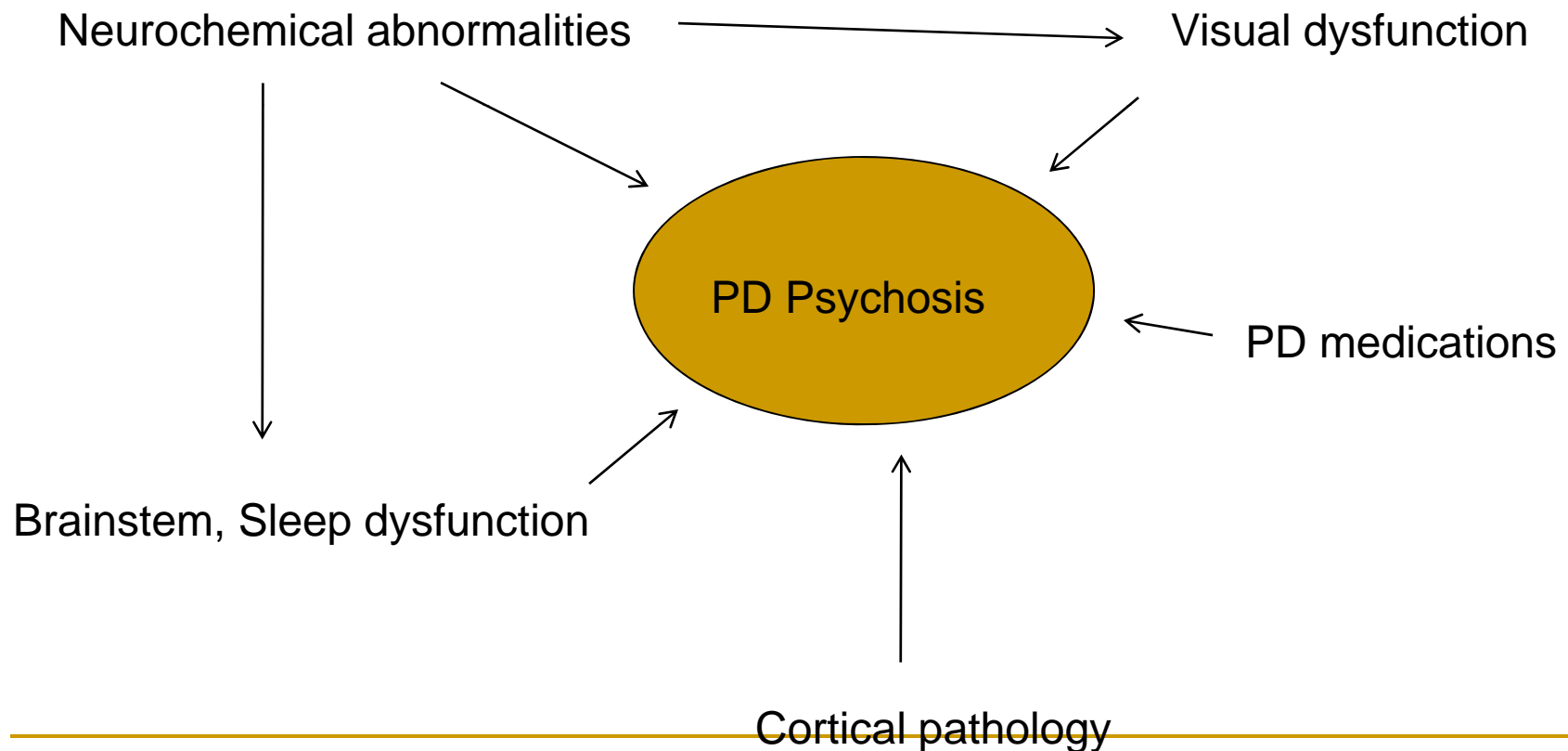
Management (cont..)

Management of comorbid problems associated with Parkinson dz

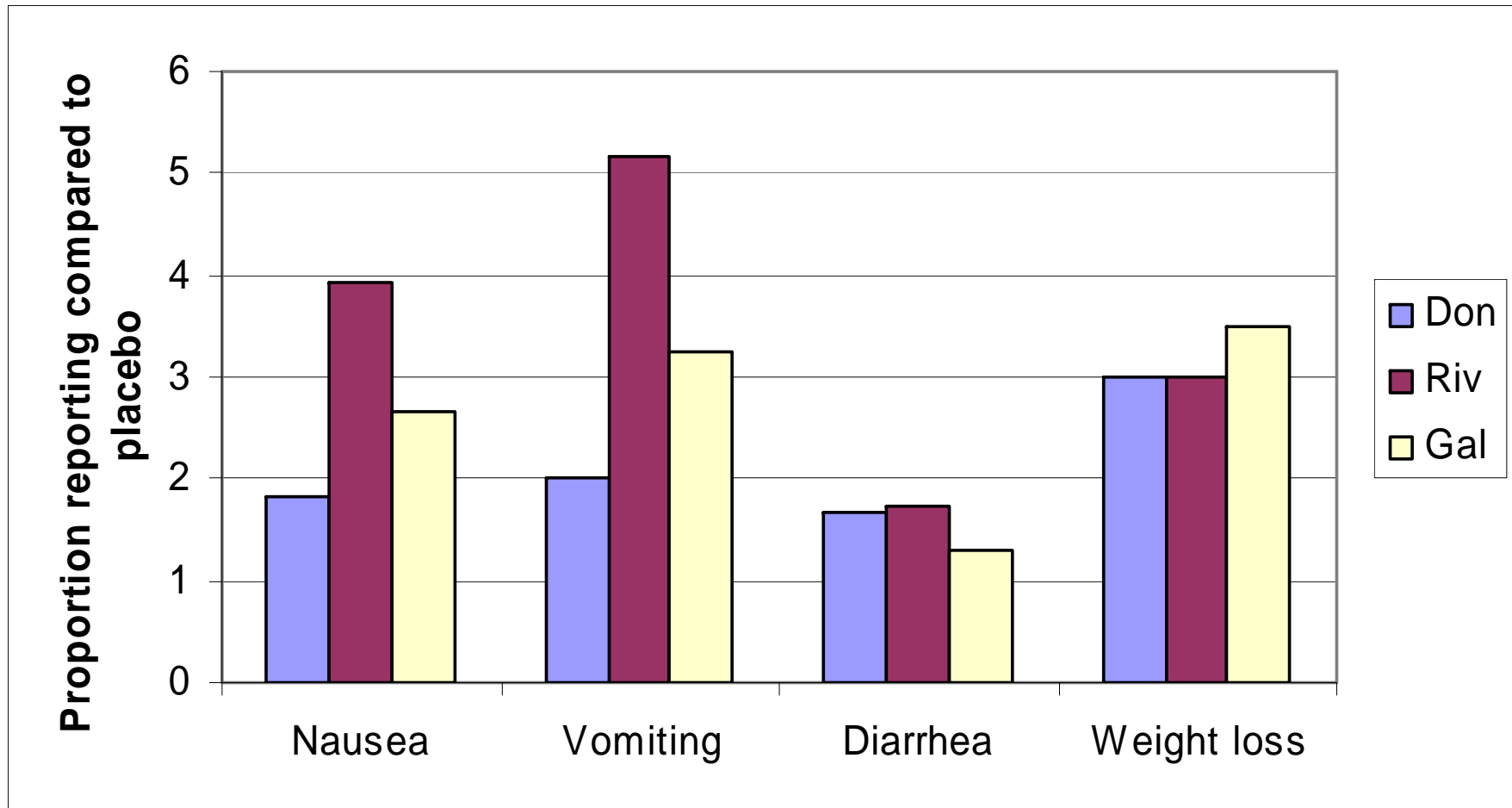
- patients with PD may experience problems related to the disease itself or to the medications used to treat it. These comorbid problems include psychosis, hallucinations, daytime sleepiness, depression, fatigue and dementia.
 - **PSYCHOSIS AND HALLUCINATIONS** : visual hallucinations and delusions, and paranoia. Antiparkinsonian drugs can be reduced or stopped in reverse order of their potency and effectiveness if hallucinations are causing disability; the suggested sequence begins with anticholinergic drugs, followed by amantadine, COMT inhibitors, and, lastly, dopamine agonists. Levodopa is essential for almost all patients with PD.
 - The atypical neuroleptics clozapine and quetiapine may be helpful in low doses . They do not appear to worsen parkinsonism as do other atypical neuroleptics, such as risperidone and olanzapine, and the typical neuroleptics.
 - **DEMENTIA** :
 - **DAYTIME SLEEPINESS** : involves efforts to improve nocturnal sleep hygiene and to treat causes of poor nocturnal sleep.
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Pathophysiology of Psychosis in PD

(from Zahodne & Fernandez, Drugs Aging 2008)

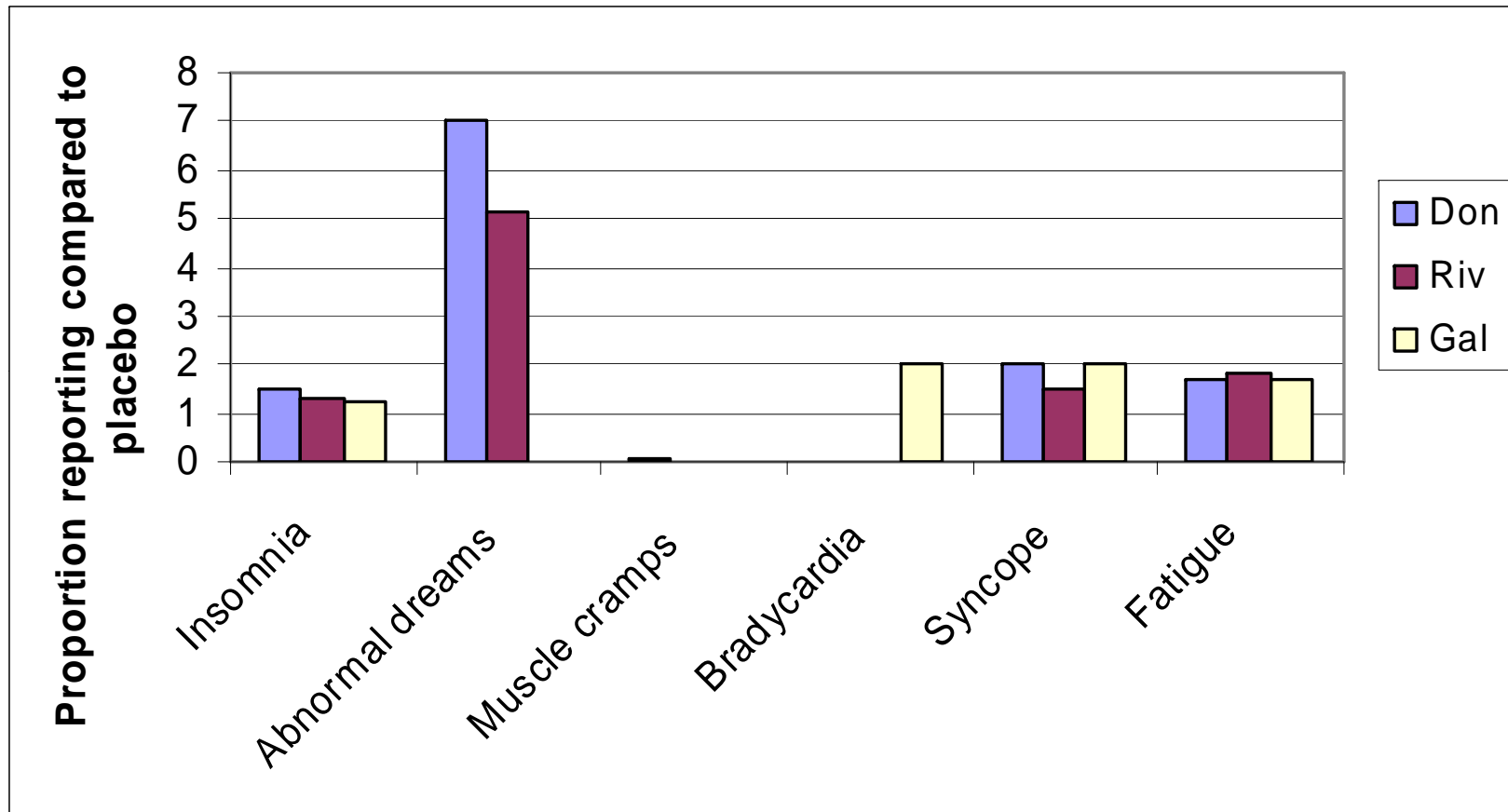


Adverse Effects Of Cholinesterase Inhibitors



Cummings JL. Use of cholinesterase inhibitors in clinical practice: evidence based recommendations. *Am J Geriatr Psychiatry* 2003;11:131-145.

Adverse Effects Of ChE-Is



Abnormal dreams reported in 3 compared to 0 in donepezil group, but arbitrarily assigned a score of 7 for this graph.

Cummings JL. Use of cholinesterase inhibitors in clinical practice: evidence based recommendations. Am J Geriatr Psychiatry 2003;11:131-145.

Dose and EPS Related to the Treatment of Psychosis In PD

Antipsychotic	Dose	Efficacy	EPS Potential
Aripiprazole	1-10	Modest	Medium to high
Clozapine	6.25-50	Good	Low
Olanzapine	2.5-10	Low	High
Quetiapine	25-150	Modest to low	Low
Risperidone	0.5-1.5	Modest	Medium
Ziprasidone	Limited info.	Limited info.	Limited info.

Hasnain et al. Pharmacological management of psychosis in elderly patients with parkinsonism. Am J Medicine 2009;122:614-622

Management (cont..)

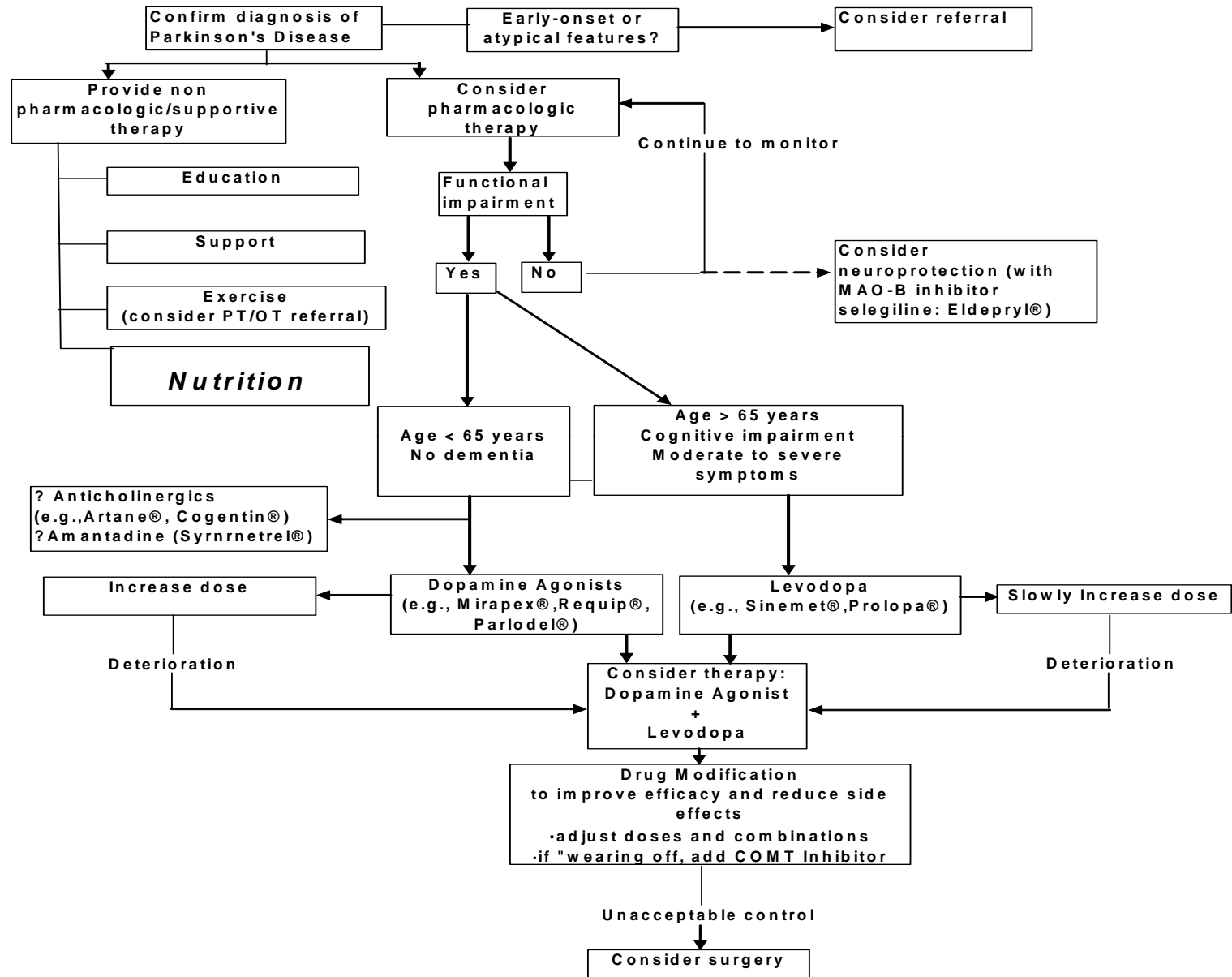
Management of comorbid problems associated with Parkinson disease

- **FATIGUE** : ? modafinil
 - **DEPRESSION** : No clear first choice for treating depression associated with PD, drug selection should be based on potential advantages versus potential side effects .
 - **Serotonin syndrome** ; avoid co administration of selegiline (MAO B inhibitor) and SSRI's
 - **Aggravating motor symptoms** : SSRI's may exacerbate the motor symptoms of parkinson's disease, mostly associated with Fluoxetine and Paroxetine, sertraline has been associated with relatively few cases .
-

Depression: Treatment-ECT

- ECT benefits both depression and motor symptoms of PD.
 - Post ECT delirium is common, particularly in those with pre-existing cognitive impairment.
 - Maintenance ECT is an option.
-

Management



Adopted from: Olanow CW, Koller WC. An algorithm (decision tree) for the management of Parkinson's disease