Idiopathic Parkinson's Disease (IPD)

Objectives

- Definition
- Aetiology
- Pathology
- Clinical features
- Investigations
- Differential diagnoses
- Management

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Defining IPD

- Named after James Parkinson who published 'An Essay on the Shaking Palsy' in 1817, which established Parkinson’s as a recognised medical condition.

- He studied at the London Hospital Medical College, qualifying as a surgeon in 1784 when he was 29.

Defining IPD

-Degenerative, progressive disease affecting the basal ganglia.

Movement disorders:

1) Akinetic-rigid syndromes

- Slowed movement.
- Increased tone.

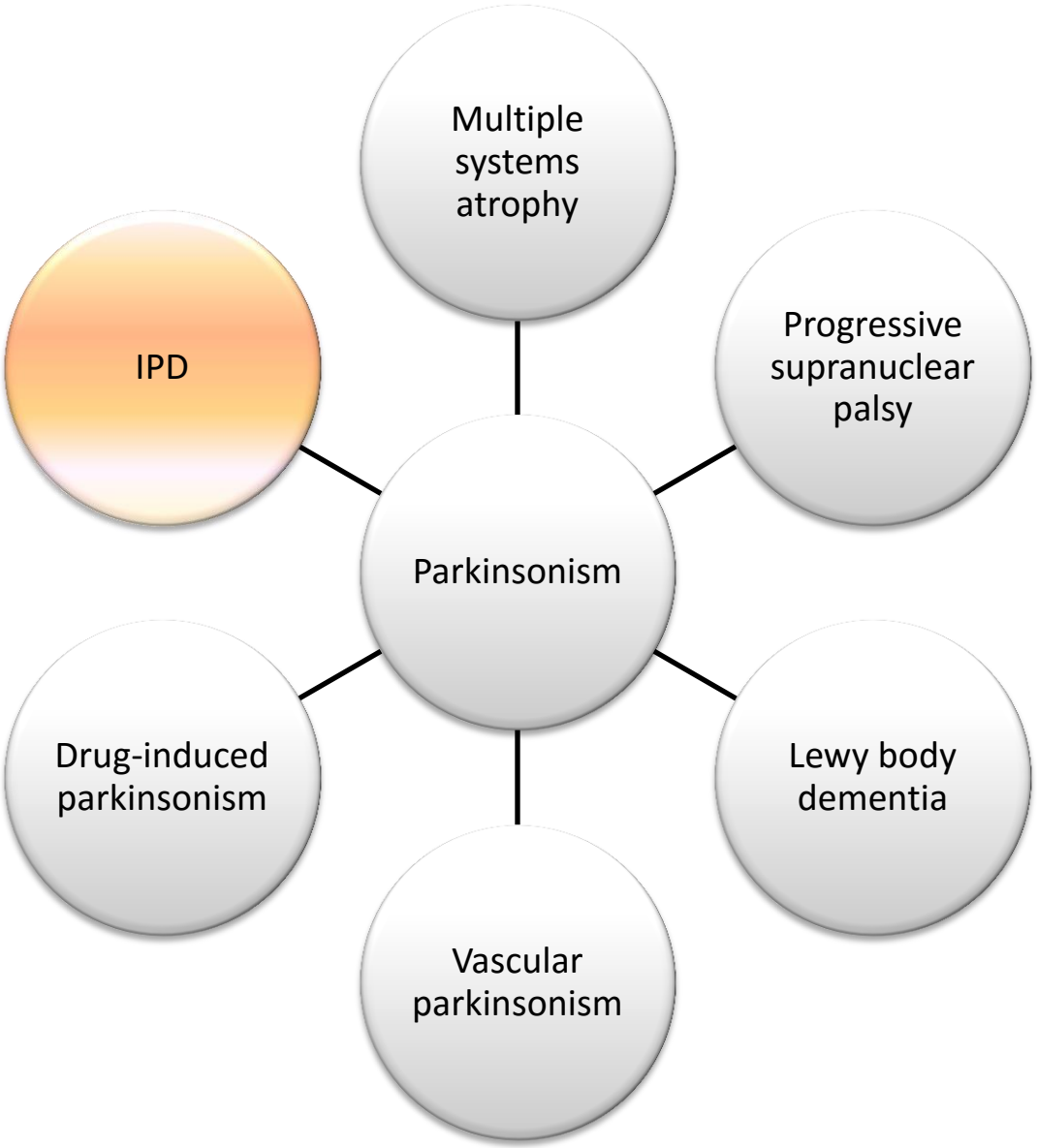
-> IPD, drug-induced parkinsonism, multiple systems atrophy, progressive supranuclear palsy.

2) Dyskinesias

-Added, uncontrollable movements.

-> Essential tremor, chorea, myoclonus, tics.

Defining IPD



Defining IPD

- Annual incidence- 0.2/1000.
- Prevalence- 1/500 (127 000 people in the UK).
- Tends to affect ≥50 years.
- 1/20 is under the age of 20 years.
- Incidence and prevalence increase with age.
- Equal sex incidence.

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Aetiology

- Unknown aetiology.

Several theories:

- Nicotine- IPD is less prevalent in smokers than lifelong abstainers.
- MPTP- caused severe parkinsonism in young drug abusers.
- Genetic factors- clustering of early-onset PD in some families.

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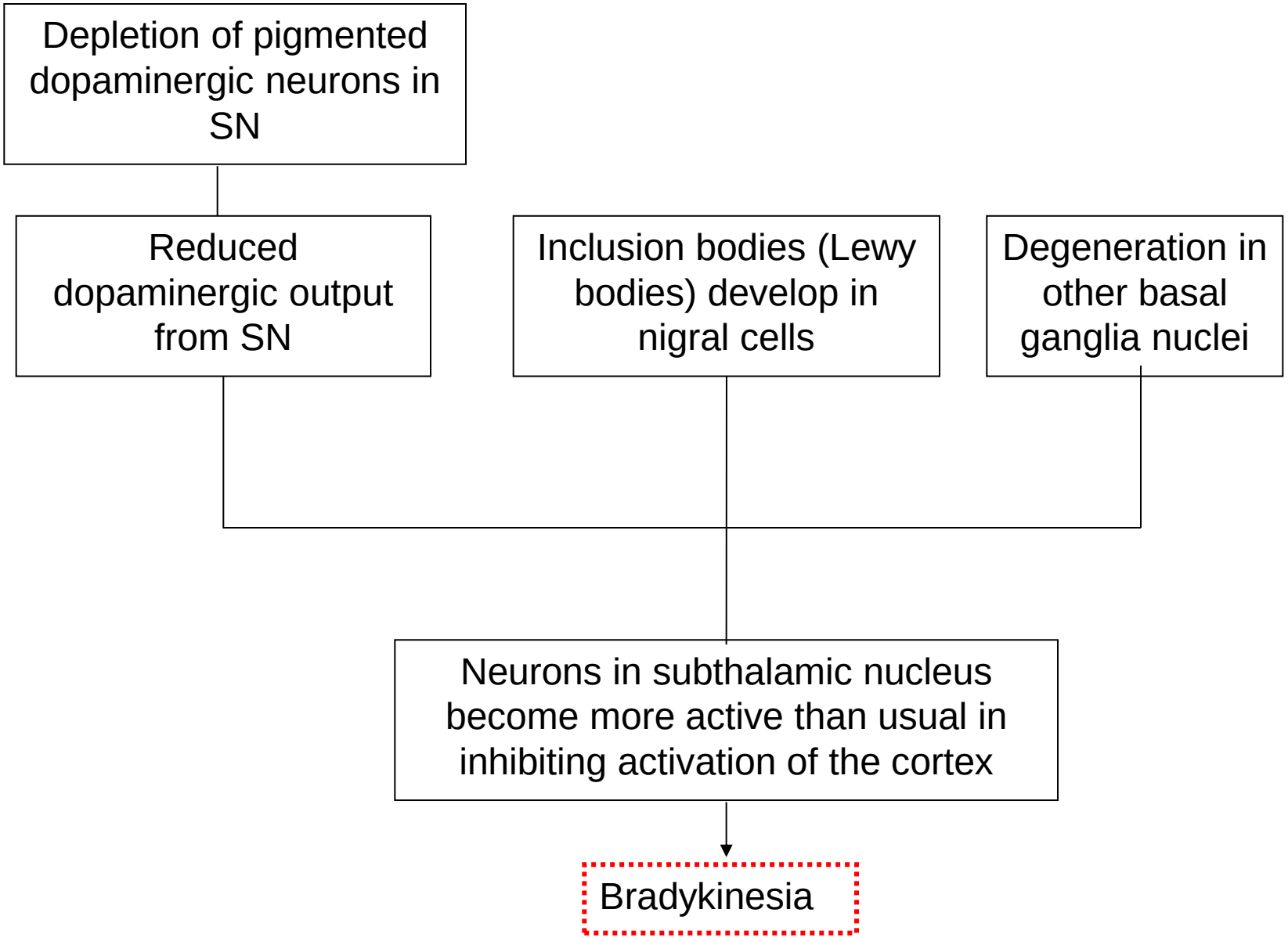
Pathology

Basal ganglia:

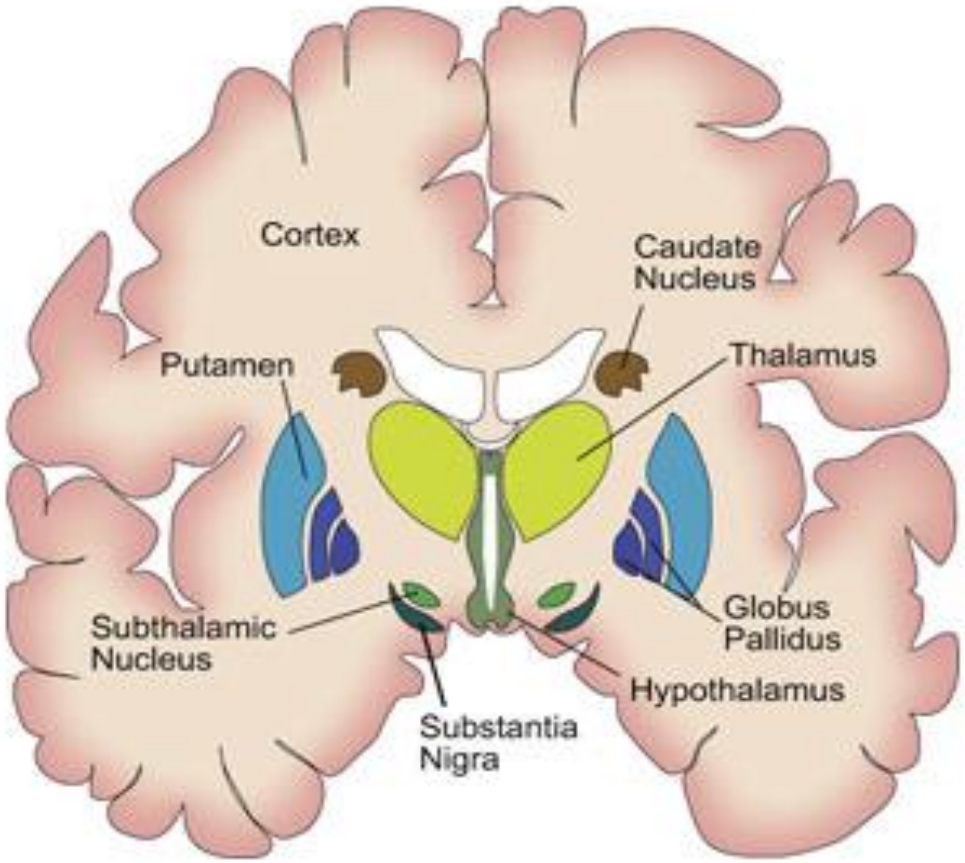
- Group of nuclei in the brain situated at the base of the forebrain (striatum, globus pallidus, substantia nigra [SN], nucleus accumbens, subthalamic nucleus).

- Associated with voluntary motor control, procedural learning, eye movements, cognitive and emotional functions.

Pathology


Depletion of pigmented dopaminergic neurons in SN
Reduced dopaminergic output from SN
Inclusion bodies (Lewy bodies) develop in nigral cells
Degeneration in other basal ganglia nuclei
Neurons in subthalamic nucleus become more active than usual in inhibiting activation of the cortex
Bradykinesia

Pathology


Cortex
Caudate Nucleus
Putamen
Thalamus
Subthalamic Nucleus
Globus Pallidus
Hypothalamus
Substantia Nigra

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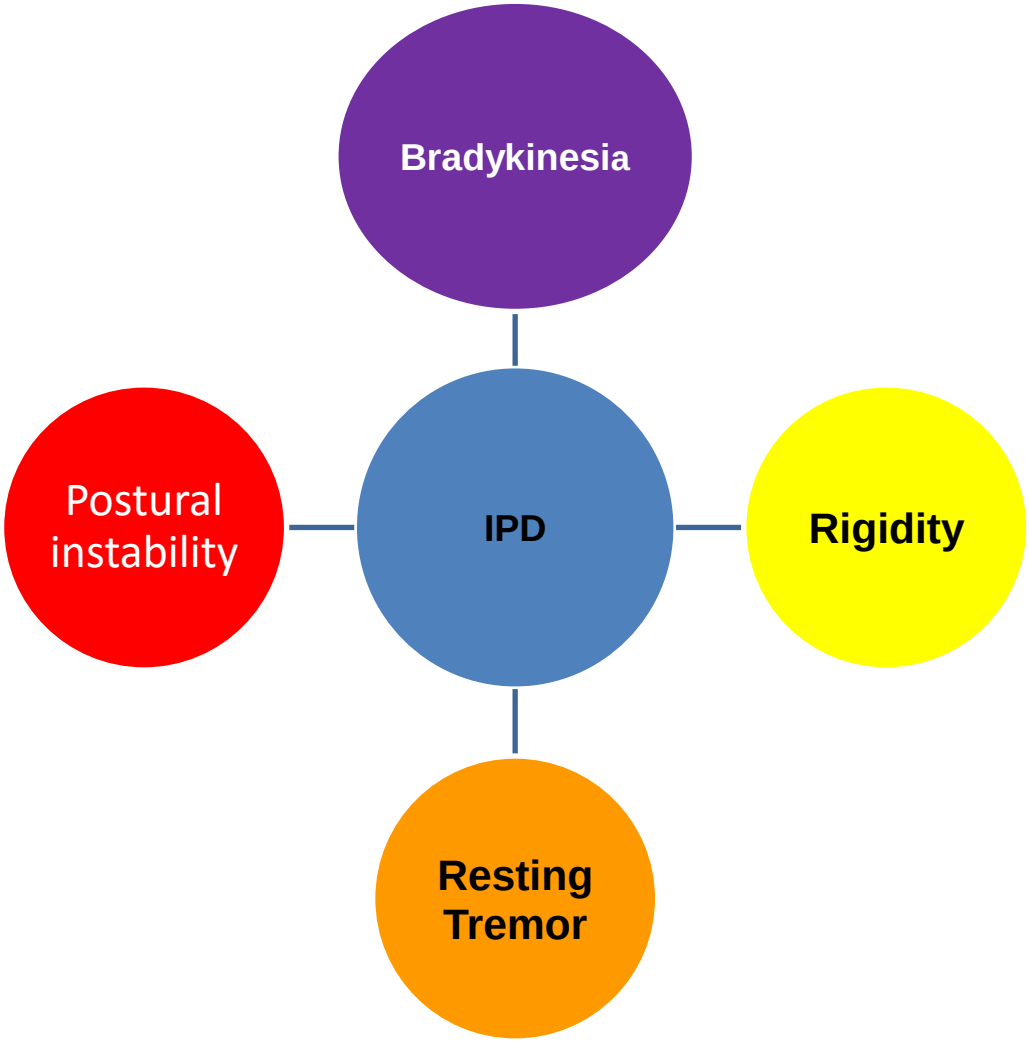
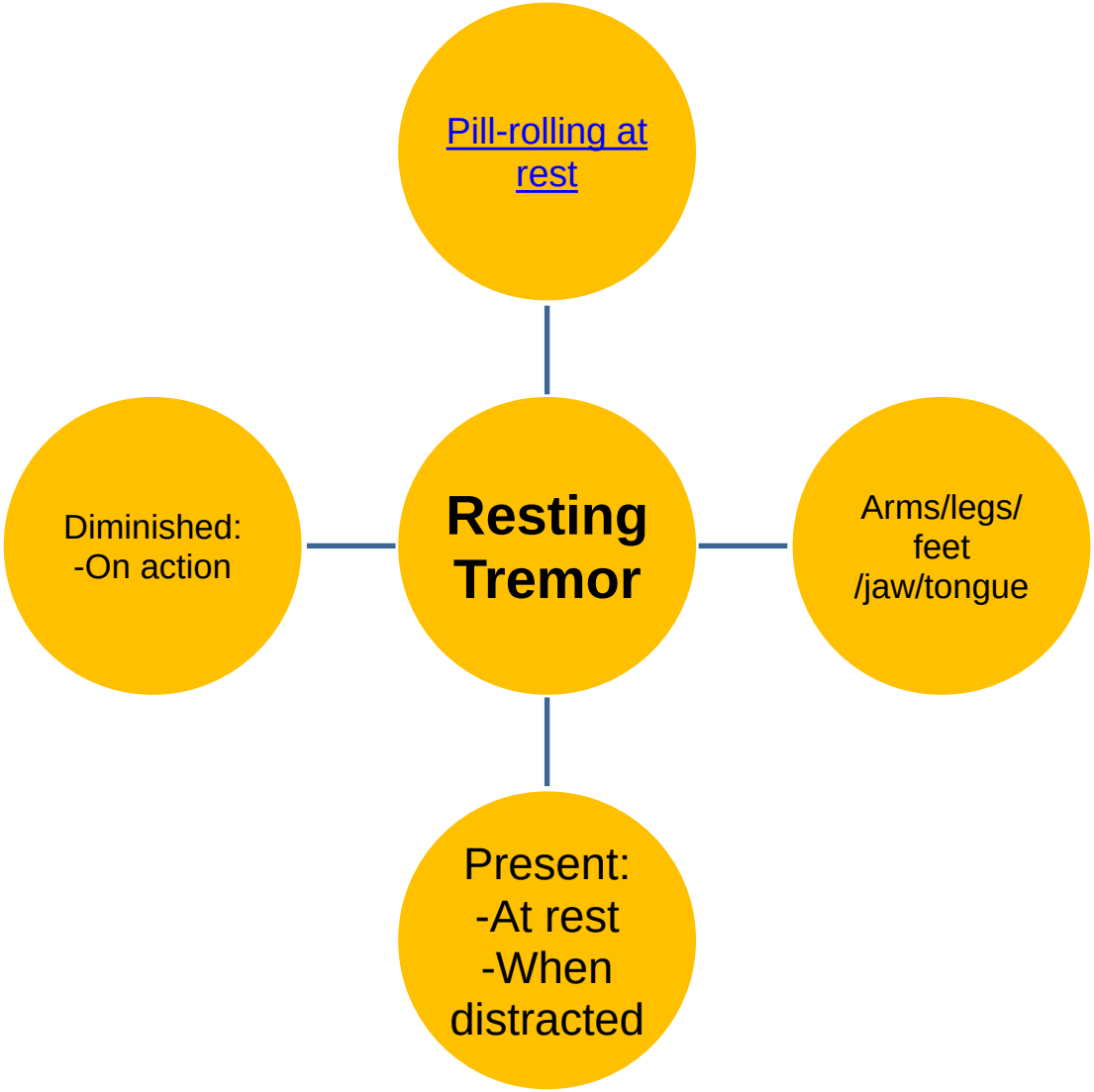
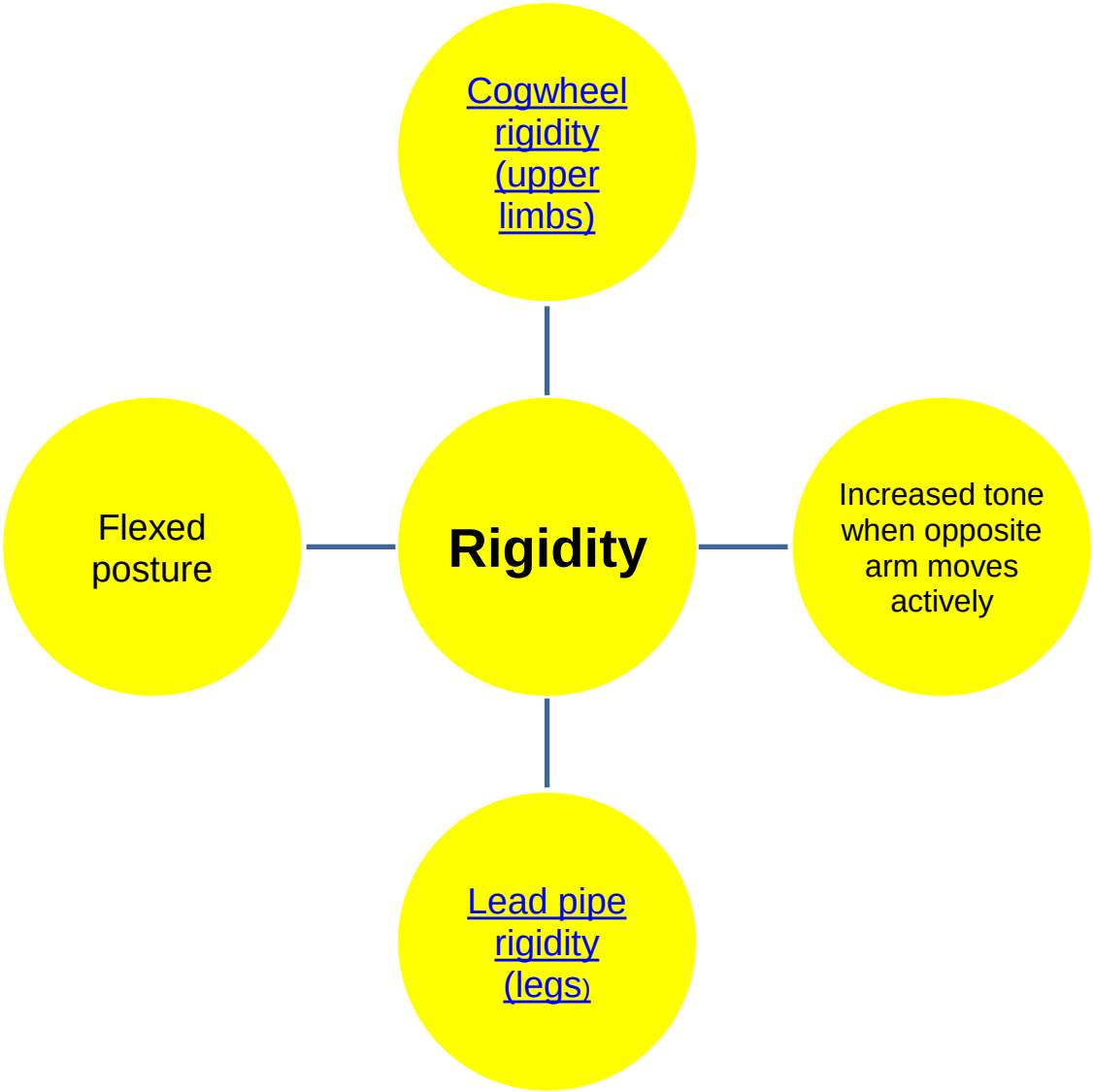
Clinical features



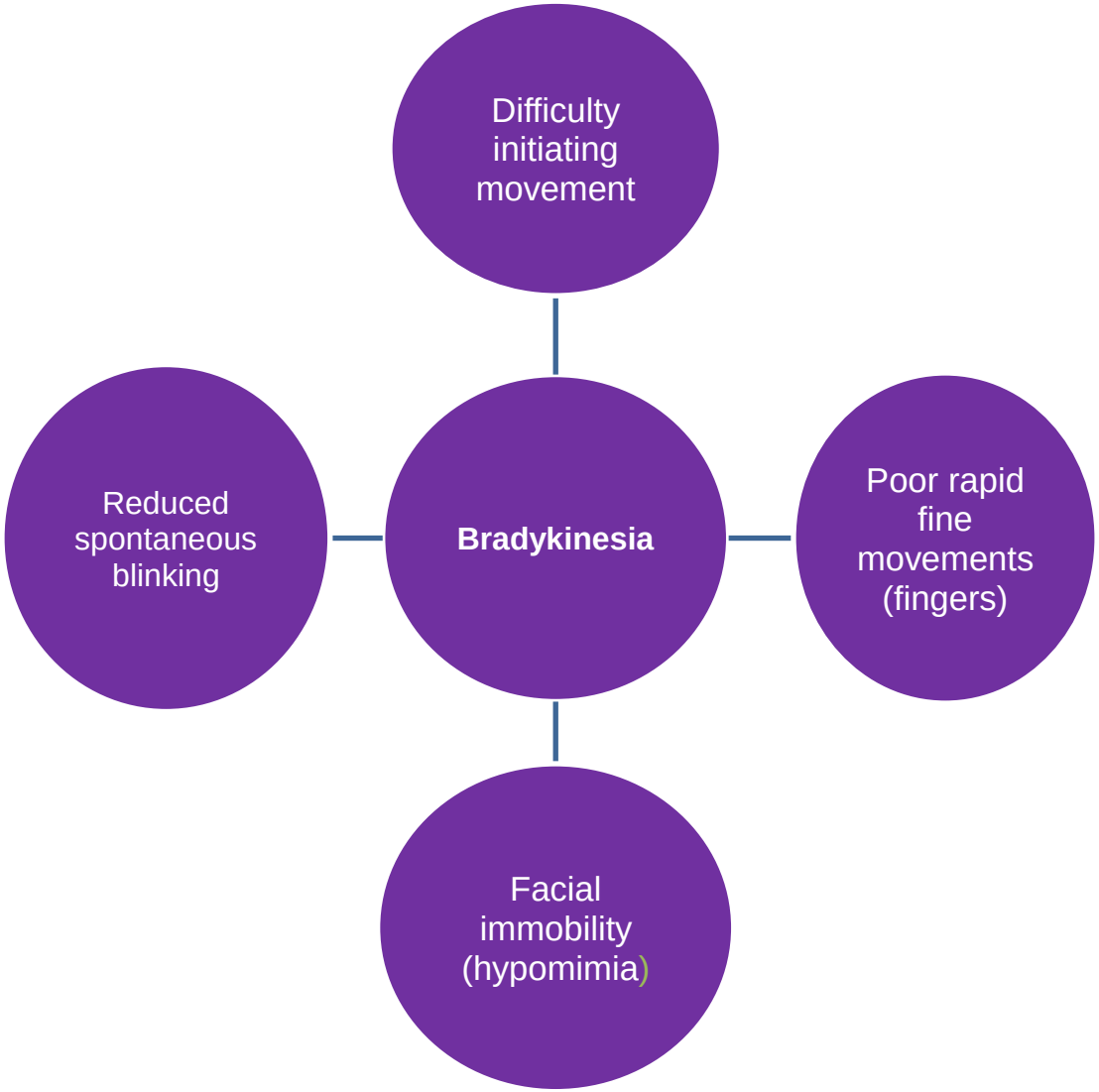
Clinical features



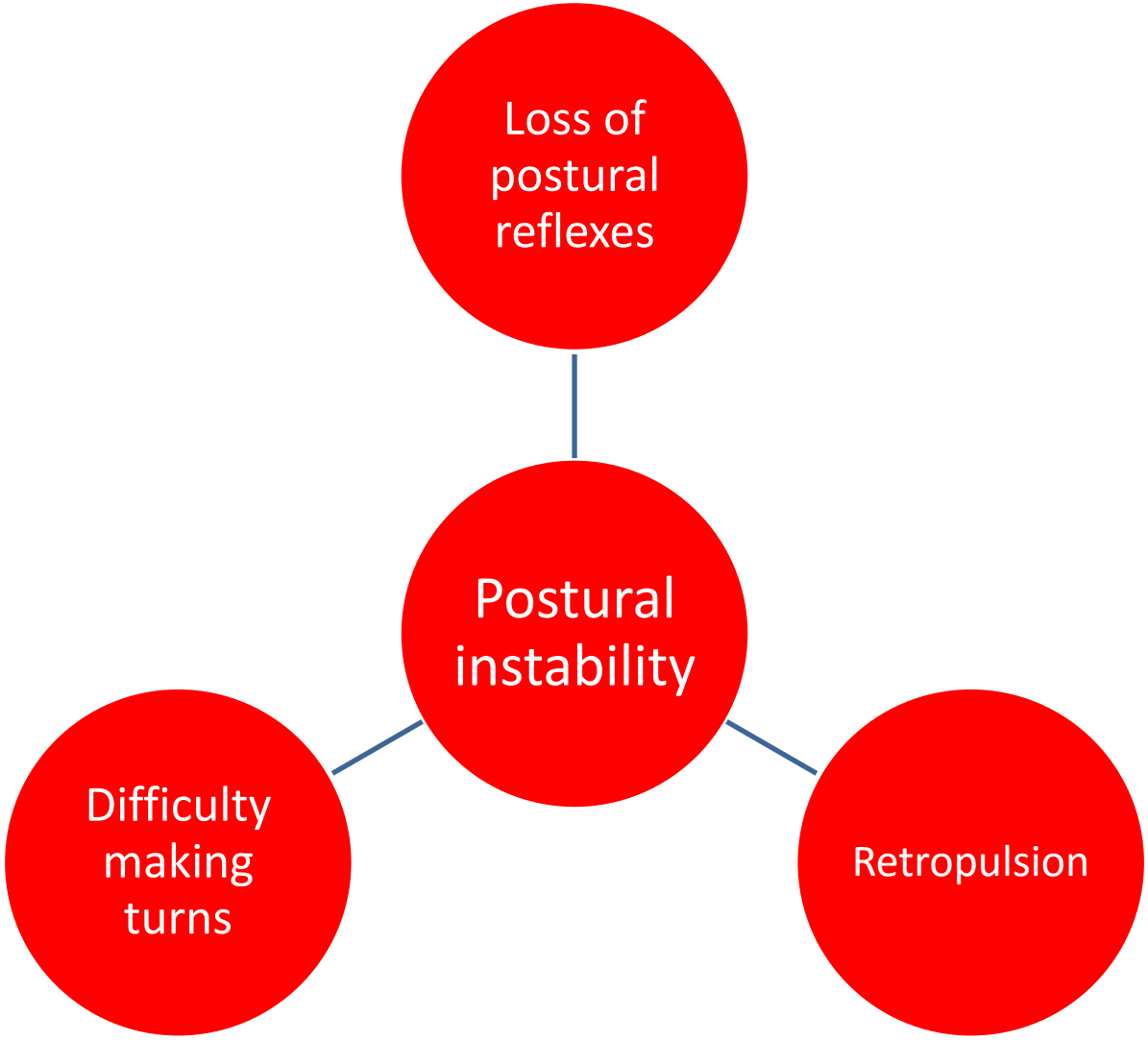
Clinical features



Clinical features



Clinical features



Clinical features

Gait:

i) Stooping
ii) Slow to initiate walking
iii) Shortened stride
iv) Rapid small steps (shuffling)
v) Tendency to run (festinating)
vi) Reduced arm swing
vii) Impaired balance on turning

- Falls common in later stages.
- Parkinson's gait

Clinical features

- Speech

-Monotone →tremulous, slurring dysarthria.

-Soft, rapid, indistinct.

- Cognitive

-Cognitive impairment in 1/3 of patients (loss of executive functions including planning/decision-making/controlling emotions).

-Depression.

Clinical features

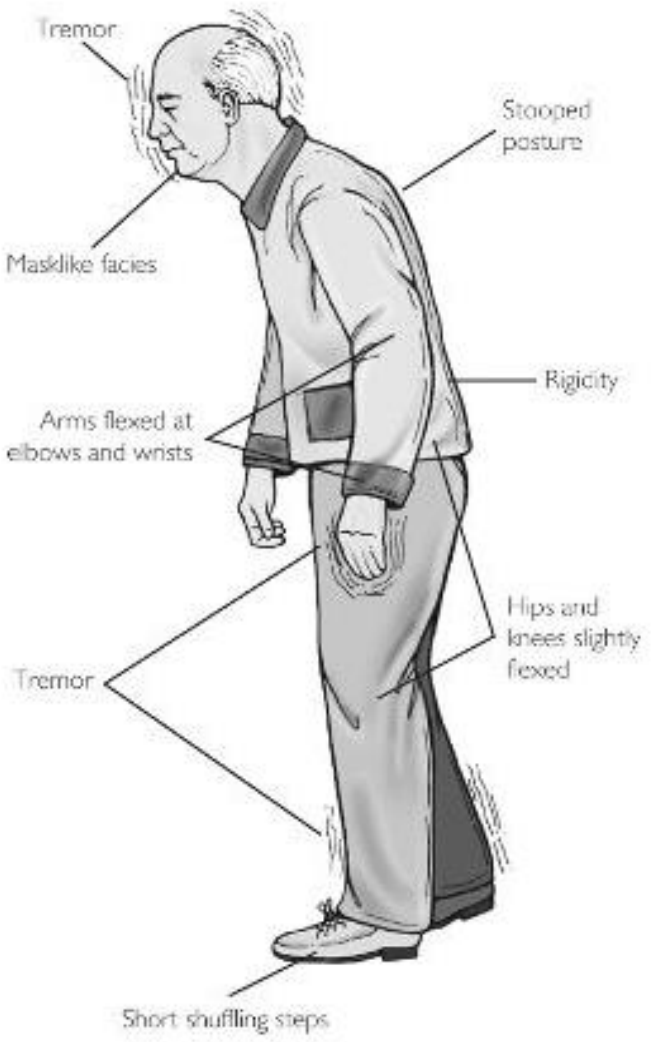
- GI/others

-Constipation/heartburn/dribbling/ dysphagia/weight loss.

-Greasy skin.

-Micrographia.

Clinical features



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Investigations

- Clinical diagnosis.

- <50 years: Test for Wilson's disease.

- CT head scan if:

- Pyramidal/cerebellar/autonomic involvement.
- Diagnosis is in doubt.

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Differential diagnoses

- Multiple systems atrophy
- Progressive supranuclear palsy
- Lewy body dementia
- Drug-induced parkinsonism
- Vascular parkinsonism

Differential diagnosis	Features		Others
Multiple systems atrophy	Parkinsonism	-Autonomic failure -Cerebellar involvement -Pyramidal tract degeneration	-Postural hypotension -Sphincter disturbance (impotence/urinary sxx) -Cerebellar signs
Progressive supranuclear palsy		-Supranuclear paralysis of eye movements -pyramidal signs -cognitive impairment	-Axial rigidity -Failure of vertical gaze
Lewy body dementia		-Early progressive dementia -Nocturnal wanderings +/- confusion	
Drug-induced parkinsonism		-Symmetrical disease -Younger patient -Taking dopamine antagonists/lithium	
Vascular parkinsonism		Sudden onset -Stuttering progression -Minimal tremor -Lower limbs affected >upper limbs	-MRI diagnosis

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Management

Medical:

- Levodopa + peripheral decarboxylase inhibitor (E.g. Carbidopa, Benserazide).

-Levodopa: Precursor of dopamine stimulates remaining neurons to produce more dopamine.

-Decarboxylase inhibitor: Prevents peripheral decarboxylation to dopamine and .:. peripheral SE's.

Management

- Side effects of levodopa:

-N&V
-Confusion
-Visual hallucinations
-Delusions
-Chorea

LT effects:
-Levodopa-induced involuntary movements.
-Gradually ineffective after several years.
-Episodes of immobility (freezing).

THEREFORE→ drugs are avoided until clinically necessary (significant disability) because of delayed unwanted effects.

Management

- Other medical treatment options:

-Dopamine receptor agonists (Bromocriptine/Cabergoline).

-Amantadine.

-Rivastigmine (cognitive changes).

-Antioxidant compounds (Vitamins C & E- possible neuroprotective agents).

Management

- Surgical

-Stereotactic thalamotomy- temporary improvement of symptoms.

- Physiotherapy

- Reduces rigidity & corrects abnormal posture.

- Speech therapy

-Dysarthria/dysphonia.

- Neuropsychiatric

- SSRI’s for depression.

Management

Natural history:

- Slowly progressive (10-15 years).
- Bradykinesia & tremor worsen.
- Late deterioration despite Levodopa Rx occurs in 1/3-1/2 of patients after 3-5 years. This includes the ‘on-off’ phenomenon.
- Patient’s c/o limb & joint discomfort.

Management

- Prognosis

-Partly related to age of onset e.g. if symptoms start in middle life → disease likely to shorten lifespan (complications of immobility & tendency to fall).

Onset >70 years of age unlikely to shorten life/become severe.

Summary

- IPD is the most common cause of parkinsonism.
- Degenerative, progressive disease affecting the basal ganglia.
- Classical features include tremor, rigidity and bradykinesia.
- Mainstay of treatment is with levodopa & a PDI. Treatment is delayed until clinically necessary because of unwanted delayed effects of levodopa.
- Multi-disciplinary approach to management.

Thank you! ☺

Questions?