Parkinson's Disease

NORMAL PHYSIOLOGY

Dopaminergic neurons in the pars compacta of the substantia nigra modulate the activity of the corpus striatum (caudate nucleus, putamen and globus pallidus) in the direct and indirect pathway. Overall effect is to faciliate selection, preparation and execution of voluntary movements.

CAUSES OF PARKINSONISM

- Idiopathic Parkinson's disease (this summary focuses on)
- Drug-induced
- Multiple systems atrophy
- Progressive supranuclear palsy
- Cerebrovascular disease
- Wilson's disease
- Carbon monoxide poisoning
- Hydrocephalus

AETIOLOGY AND PATHOLOGY

Genetic mutations:

- Autosomal dominant alpha-synuclein gene
- Autosomal recessive Parkin gene

Environmental factors

Increasing age

Degeneration of the dopaminergic neurons in the pars compacta of the substantia nigra, with Lewy inclusion bodies

FEATURES

- Tremor- at rest, upper limb predominant, asymmetric, pill-rolling
  - NEVER have head tremor
- Bradykinesia
- Rigidity- lead pipe
- Cogwheeling- combination of tremor and rigidity
- Micrographia
- Hypophonia
- Mask face- lack of expression
- Dementia
- REM sleep behaviour disorder- vocal and thrash out in sleep
- Autonomic sx- postural hypotension etc

INVESTIGATION AND MANAGEMENT

A clinical diagnosis- response to levodopa contributes. SPECT scans not routinely used (image pre-synaptic dopamine transporter)

- Levodopa
- Dopamine agonists (ropinirole)- may be used alone in young-onset
- COMT inhibitors (entacapone)- prolongs levodopa for wearing-off
- MAOI-B (selegiline)
- Apomorphine- DA agonist
- Amantidine- used in mild disease
- Anti-muscarinics (procyclidine)
- Deep brain stimulation

Soton Brain Hub