

MemoCharts Pharmacology: Drug Therapy for Parkinsons Disease (Review chart)

DRUG THERAPY FOR PARKINSON'S DISEASE
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Neuronal circuitry of the basal ganglia that facilitate cortical locomotor initiation via thalamic stimulation

Characteristics and Treatment of Parkinson's Disease (PD)

Clinical Features	Pathophysiology	Treatment
Onset onset, usually after age 40 yrs, progressive	A. Loss of striatal dopamine neurons in the nigrostriatal pathway (substantia nigra (SNpc)) in the midbrain. It is associated with characteristic neurodegenerative inclusions called Lewy bodies.	1. Levodopa (L-Dopa) is the gold standard. It is converted to dopamine in the striatum.
Classic cardinal signs are: rest, rigidity, bradykinesia and postural instability.	B. Dopaminergic depletion in the nigrostriatal pathway and pretectal nucleus of the basal ganglia due to A.	2. Dopamine agonists (DA agonists) such as pramipexole and ropinirole.
Motoric signs: shuffling gait (bradykinesia), masked face, stooped posture and festinating gait.	C. In the neostriatum, striatal cholinergic interneurons (acetylcholine (ACh) interneurons) control (inhibit) dopamine neurons.	3. Anticholinergics (ACh antagonists) such as benztropine and trihexyphenidyl.
Depressive symptoms.	D. Reduced dopamine reuptake of striatal neurons, caused by C. results in a cortical locomotor initiation and the symptoms and signs of the disease.	4. MAO-B inhibitors (MAO-Bi) such as rasagiline and selegiline.
Responsive to L-Dopa therapy.		5. COMT inhibitors (COMTi) such as entacapone and tolcapone.

Drugs Used in PD

Classification (e.g.)	Mechanisms of Action	Clinical Applications	Major Side Effects
1. Dopamine Precursors (Levodopa)	Converted to dopamine (DA) in the striatum after being converted to dopamine in the striatum by AADC.	The therapy for PD. Used for reducing bradykinesia and rigidity.	Motor fluctuations, dyskinesia (involuntary movements), hallucinations (if combined with a D2 antagonist), orthostatic hypotension, urinary incontinence, weight gain, constipation, and peripheral edema.
2. AAD Inhibitors (Selegiline)	1. MAO-Bi that is a selective MAO-B inhibitor. It is converted to selegiline in the striatum. 2. MAO-Bi that is a selective MAO-B inhibitor. It is converted to selegiline in the striatum.	Used with levodopa to prevent levodopa's degradation and peripheral side effects by a monoamine oxidase (MAO) inhibitor.	Headache, dizziness, insomnia, and constipation.
3. COMT Inhibitors (Tolcapone)	1. Inhibits COMT, the enzyme that metabolizes levodopa to 3-O-methyldopa in the striatum. 2. Inhibits COMT, the enzyme that metabolizes levodopa to 3-O-methyldopa in the striatum.	Used with levodopa to prevent levodopa's degradation and peripheral side effects by a COMT inhibitor.	Headache, dizziness, insomnia, and constipation.
4. MAO-B Inhibitors (Rasagiline)	1. Selective MAO-B inhibitor that blocks the conversion of levodopa to dopamine in the striatum. 2. Selective MAO-B inhibitor that blocks the conversion of levodopa to dopamine in the striatum.	Used as an adjunct to levodopa therapy. Adjusted to levodopa therapy.	Headache, dizziness, insomnia, and constipation.
5. Dopamine Agonists (Ropinirole)	1. Selective D2 receptor agonist that mimics the action of dopamine in the striatum. 2. Selective D2 receptor agonist that mimics the action of dopamine in the striatum.	Used as an adjunct to levodopa therapy. Also good for a bradykinesia.	Headache, dizziness, insomnia, and constipation.
6. Anticholinergics (Benztropine)	1. Inhibits ACh receptors in the striatum. 2. Inhibits ACh receptors in the striatum.	Used for reducing rigidity and tremor.	Headache, dizziness, insomnia, and constipation.

An integrated mini review of the drug therapy for Parkinsons disease, illustrated with visually appealing tables and diagrams. A quick visual aid for the course study and board review

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