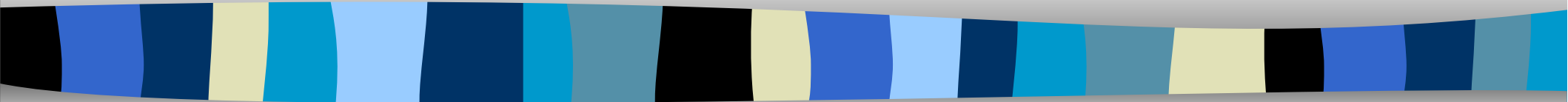


Basal Ganglia





Basal ganglia

■ Consists of

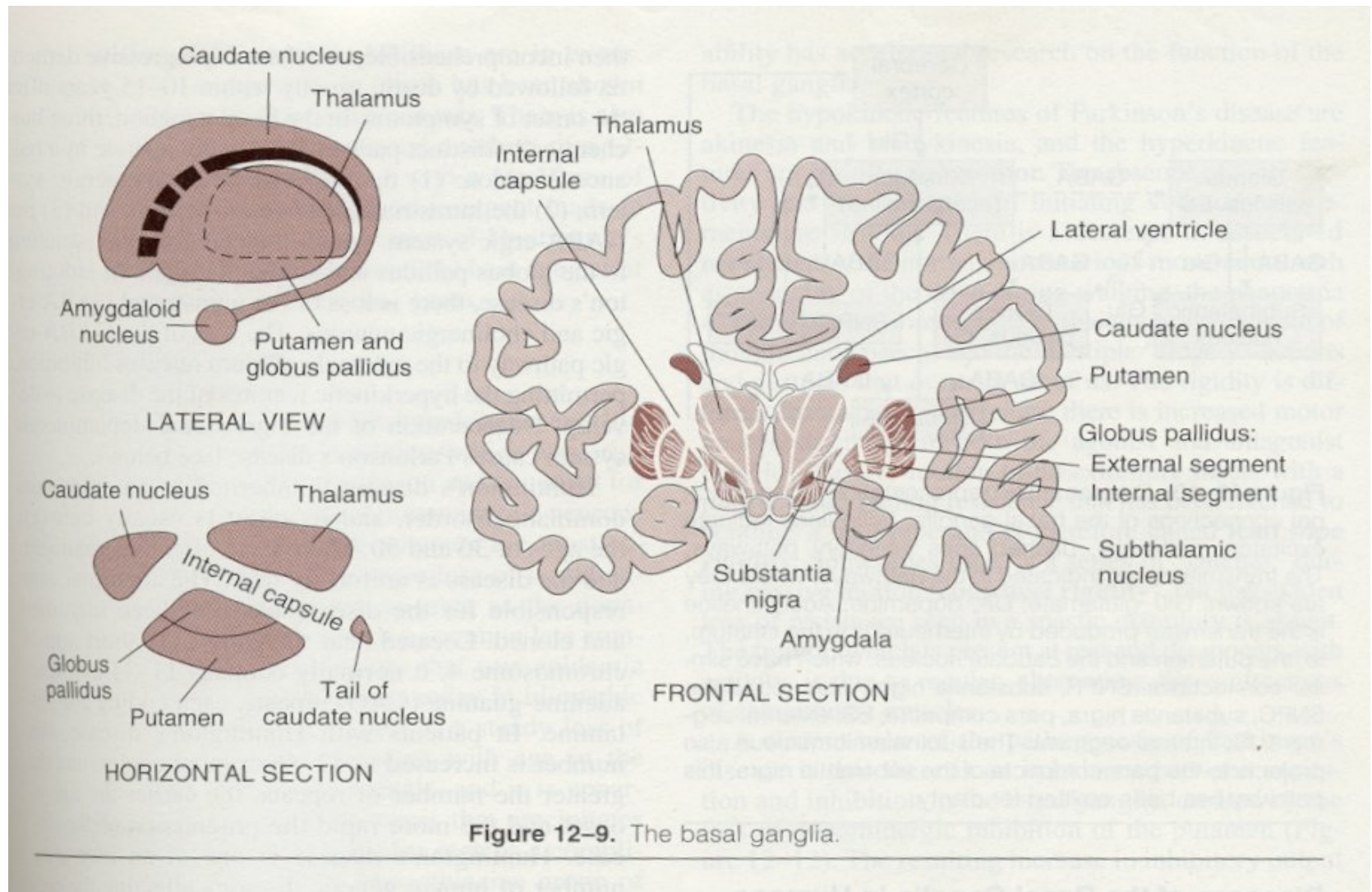
1. Caudate nucleus
2. Putamen
3. Globus pallidus - Internal and external segments
4. Subthalamic nucleus
5. Substantia nigra - pars compacta
pars reticulata



Basal ganglia

- Lenticular nucleus
 - Globus palidus and putamen
- Striatum
 - Caudate nucleus and putamen
- Parts of the thalamus are intimately related to the basal ganglia

Basal ganglia





Connections

- These nuclei have complex interconnections
- No direct connection with the spinal cord
- Afferents
 1. Corticostriate projections
 - are the main afferent fibers
 - terminate in the striatum
 - have afferents from all parts of the cerebral cortex



Connections

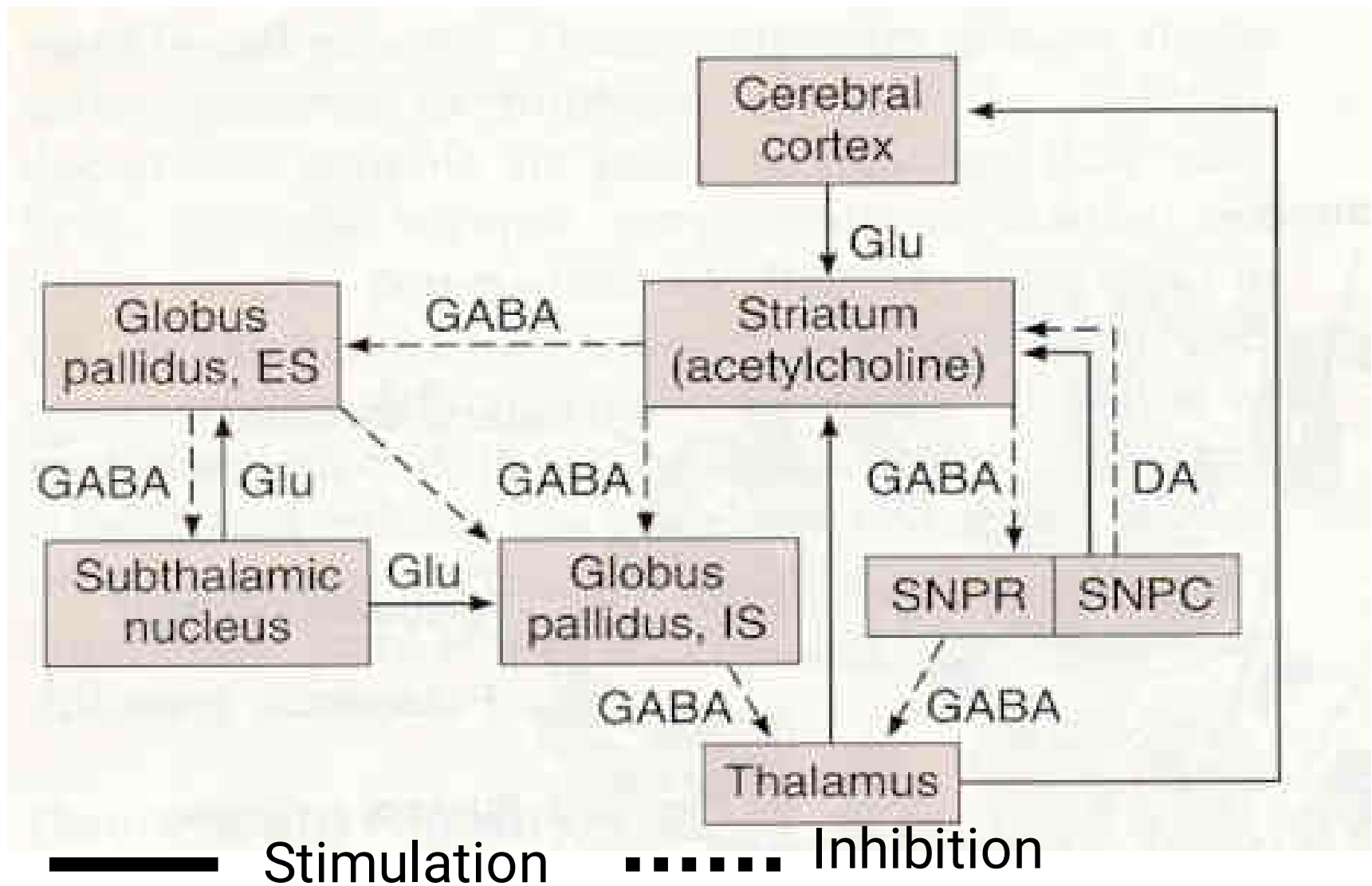
2. Dopaminergic nigrostriatal projections
 - from pars compacta of substantia nigra
3. GABA-ergic projections
 - from striatum to the pars reticulata of substantia nigra
4. Complex interconnections between each others



Connections

- Efferents
- from internal segment of globus palidus via thalamic fasciculus to
 - ventral lateral
 - ventral anterior
 - centromediannuclei of thalamus

Connections





Functions of basal ganglia

- Planning and programming of the movements
 - abstract thought is converted to voluntary action
 - sends information to motor neurons via
 - thalamus
 - motor cortex
 - corticospinal tract



Functions of basal ganglia

- Cognitive control of motor activity
 - particularly by caudate nucleus
 - determine the pattern and sequences of movements
- Determination of the timing and scaling the intensity of a movement



Diseases of the basal ganglia

Produce marked and characteristic abnormalities of the motor functions

Generally two types of motor disorders

Hyperkinetic

Hypokinetic

■ Hyperkinetic disorders

there is excessive abnormal movements

include

chorea

athetosis

ballism



Diseases of basal ganglia

■ Hypokinetic disorders

– Include

- akinesia
- bradykinesia



Features of basal ganglia disease

Hyperkinetic disorders

1. Chorea

- rapid involuntary dancing movements

2. Athetosis

- continuous, slow writhing movements

3. Balismus

- sudden, intense, violent jerky movements of the body
- due to damage to subthalamic nuclei



Features of basal ganglia disease

■ Hypokinetic disorders

1. Akinesia

- difficulty of initiating movements

2. Bradykinesia

- slowness of movements

3. Rigidity- lead pipe rigidity

- Due to increased motor discharge to both agonist and antagonist muscles

4. Cogwheel rigidity

- Rigidity interrupted by tremors



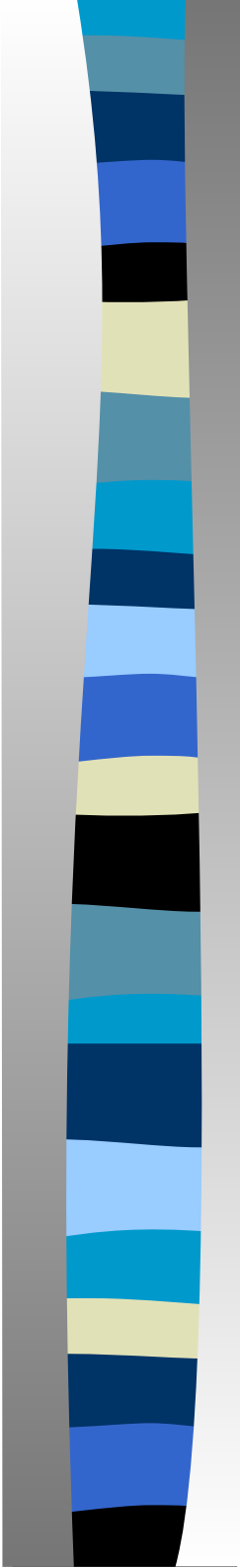
Parkinson's disease

- Has both hypo and hyperkinetic features
 - Hyperkinetic features are
 - Resting Tremor
 - Muscle Rigidity
 - Hypokinetic features are
 - Akinesia
 - bradykinesia
 - With Postural instability
 - Decreased associated movements
 - lack of swinging movements with walk
 - Lack of facial expressions and gestures
- Remember TRAP

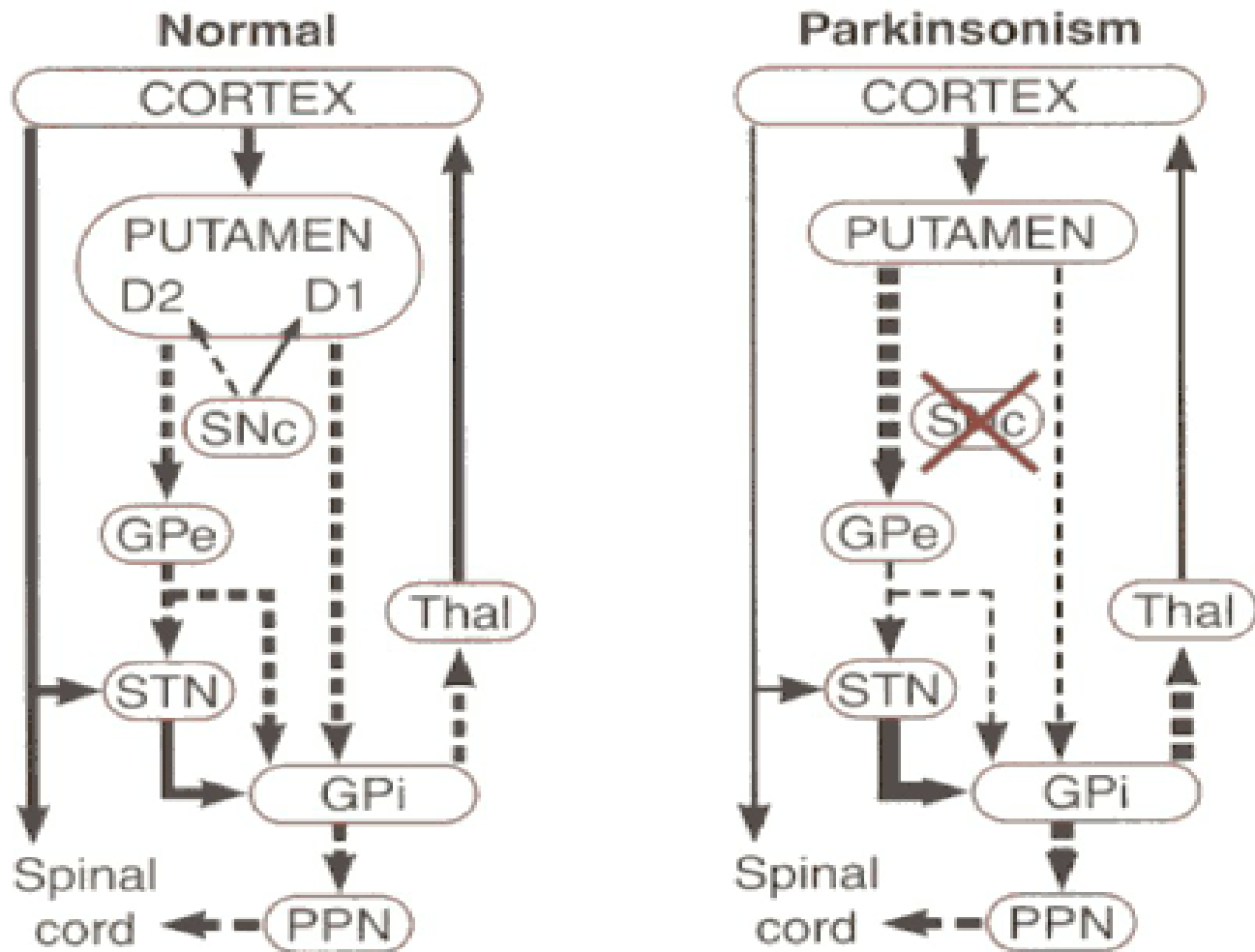


Causes(Aetiology)

- Idiopathic
- Old age
- Viral infections –influenza
- Drugs causes features of Parkinson's disease
 - D₂ receptor blockers

- 
- Usually there is a balance between
 - Dopaminergic inhibition
 - Cholinergic excitation in the basal ganglia
 - Effects are due to
 - Loss of dopaminergic neurons in nigrostriatal system
 - Creating loss of dopaminergic inhibition of putamen
 - Resulting imbalance between excitation and inhibition
 - towards increase in inhibitory output to cerebral excitation and cortical drive
 - Resulting akinesia and bradykinesia

Parkinson disease





Parkinson's disease

Treatment

- By increasing dopaminergic stimulation
- Dopamine does not cross blood brain barrier
- Therefore L-dopa is used
 - Cross the blood brain barrier
 - Converted to dopamine by dopa decarboxylase
- To minimize the side effects of dopamine at peripheral tissues a dopa decarboxylase inhibitor is also given
- This inhibitor does not cross the blood brain barrier



Parkinson's disease

Treatment

- Other dopamine agonists
 - Bromocriptine
- Anticholinergic drugs
- Mono amine oxidase-B inhibitors
 - Eg. Selagiline