Basal ganglia

Bustami

24 A

Caudate | Putamen | globus pallidus | amygdaloid nucleus | Claustrum

Caudate + Putamen + globus pallidus ⇩

corpus Striatum

(appear striated in myelin preparations because of the myelinated fibres traversing these gray masses)

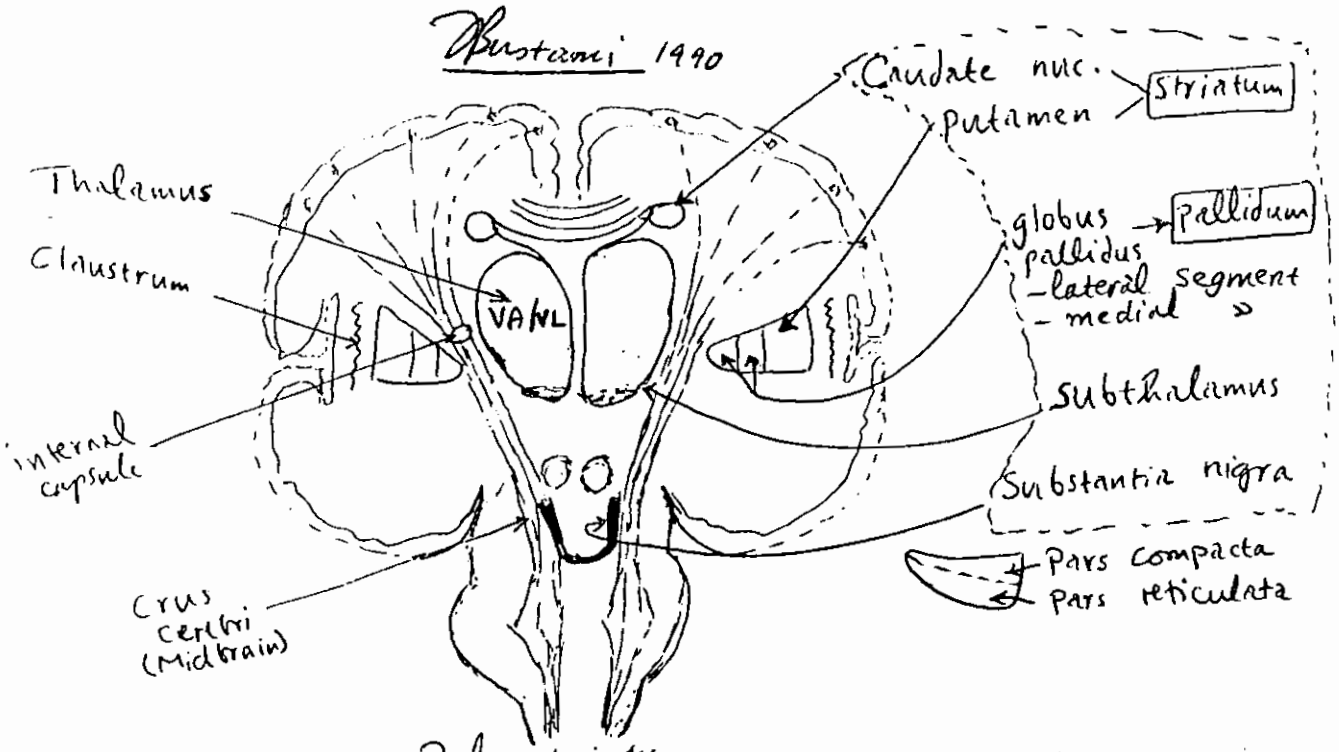
amygdaloid nucleus ⇩

functionally a part of limbic system

Emotional behaviour | memory

Claustrum ⇩

functions and connections are not well understood



Corpus striatum
- Paleostriatum (Pallidum) = globus pallidus
- Neostriatum (Striatum) = Putamen + caudate nucleus

} Lentiform nucleus

The nuclei that are functionally related to the basal ganglia

(a) Subthalamic nucleus (Part of diencephalon)

(b) Substantia nigra (Part of mesencephalon)

The Basal Ganglia

24
B

Movement is controlled by the upper motor neuron (UMN) system in the cerebral cortex

Control of UMN motor commands comes from two distinct systems < Basal ganglia, cerebellum

These structures ... influences UMNs so that a precisely planned & executed motor commands can be conveyed to the LMNs and the muscle

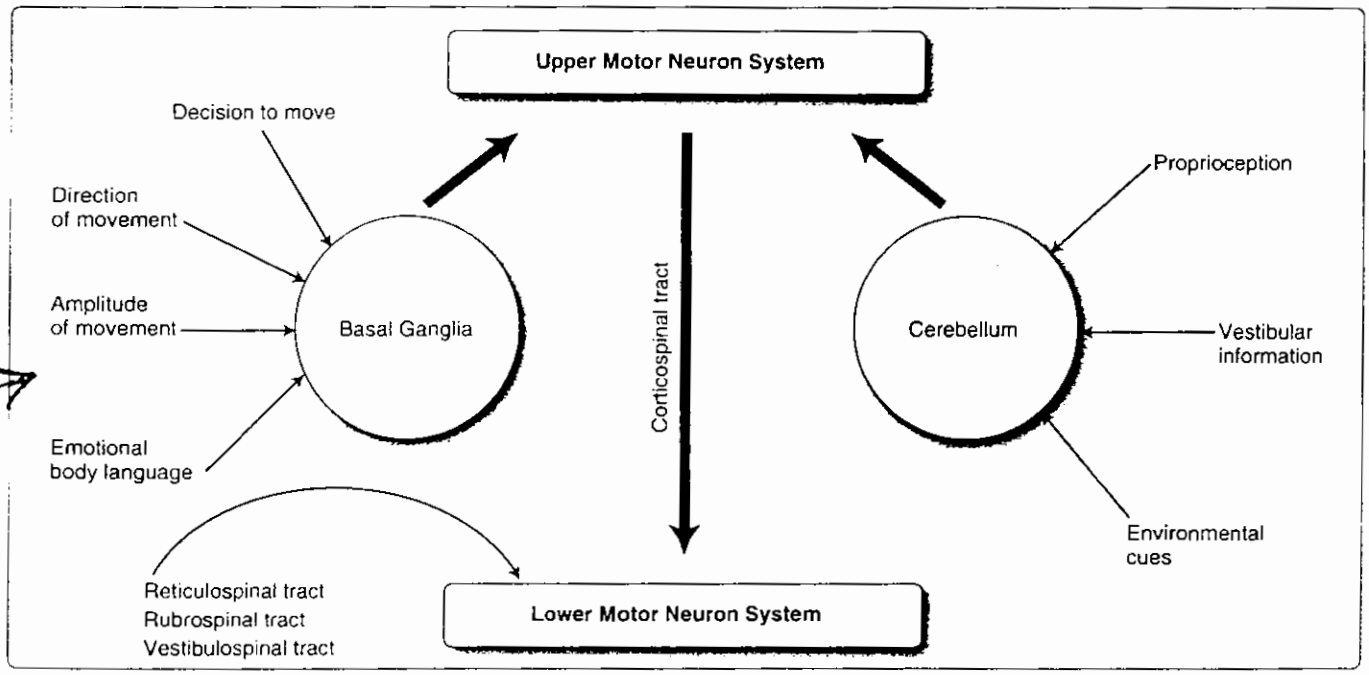


Figure 16.1
Conceptual overview of motor control.

- The decision to move
- The direction of movement
- The amplitude of movement
- The motor expression of emotions (Figure 16.1)

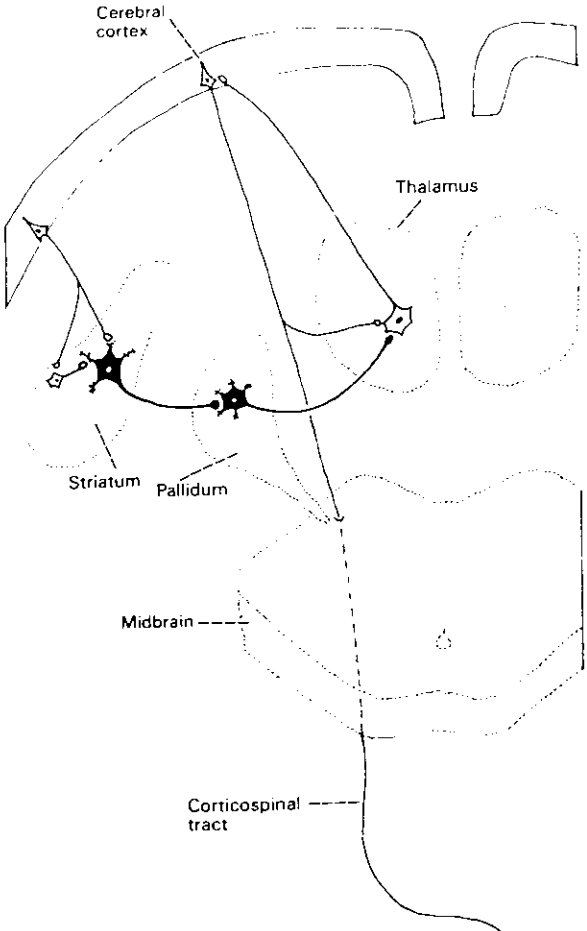


Fig. 18-4 The cortico-strio-pallido-thalamo-cortical loop.

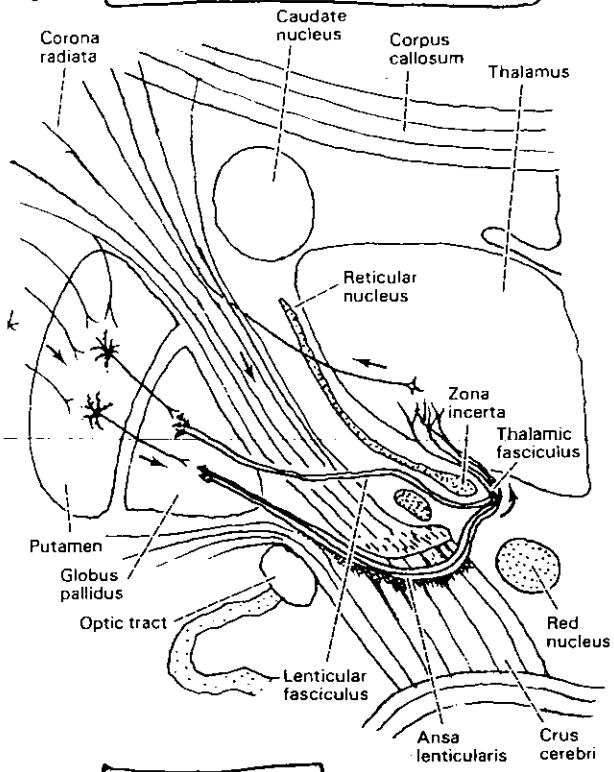


Fig. 18-5 Pallidothalamic fibers.

Basic circuits

1) From all parts of the cerebral cortex axons run into the Striatum (caudate putamen)

Striatum contains: excitatory cholinergic neurons; inhibitory GABAergic neurons

2) The largest projection from the striatum is from inhibitory GABAergic to all parts of the pallidum (and to substantia nigra)

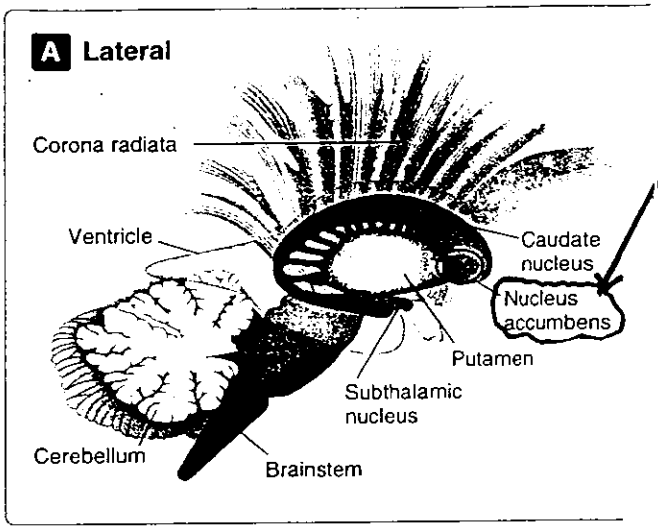
Axons of pallidum run to the thalamus in the pallidothalamic tract

formed of 2 parts:
- Ansa lenticularis
- lenticular fasciculus

3) Synapse on ventral anterior (VA) and ventral lateral (VL) nuclei of the thalamus (like the striopallidal fibres, the pallidothalamic fibres are inhibitory and GABAergic !!! ??

From VA & VL EXCITATORY fibres run to premotor, supplementary motor & primary motor areas of the cerebral cortex

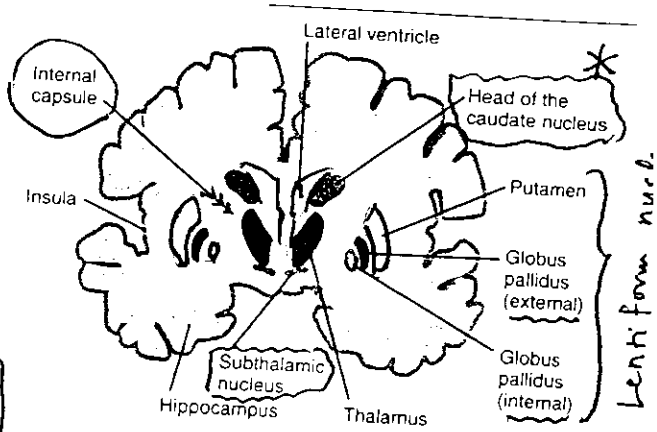
The motor cortex gives rise to the bulk of the pyramidal tract which generates contralateral movement in response to thalamocortical stimulation



The **nucleus accumbens** is the anterior & ventral of the Striatum where the head of the caudate & the Putamen are continuous with each other. It receives extensive dopaminergic input and is an integral part of the limbic system.

* **Putamen and caudate** → together form the **Striatum**, separated by the anterior limb of the internal capsule
* **INPUT NUCLEI** form to the basal ganglia and receive mainly EXCITATORY input from wide areas of the cerebral cortex

* **globus pallidus** → medial to the Putamen and lateral to the thalamus
  - subdivided into an external part (GPe) and an internal part (GPi) → The two parts are **functionally different** & have different connections within the basal ganglia
  - is the **OUTPUT NUCLEUS** of the basal ganglia SENDING INHIBITORY PROJECTIONS TO THE THALAMUS



Subthalamic nucleus → inferior to the thalamus
receive input from basal ganglia ??
- its output is **Excitatory** through glutamatergic fibres to the globus pallidus as well as substantia nigra

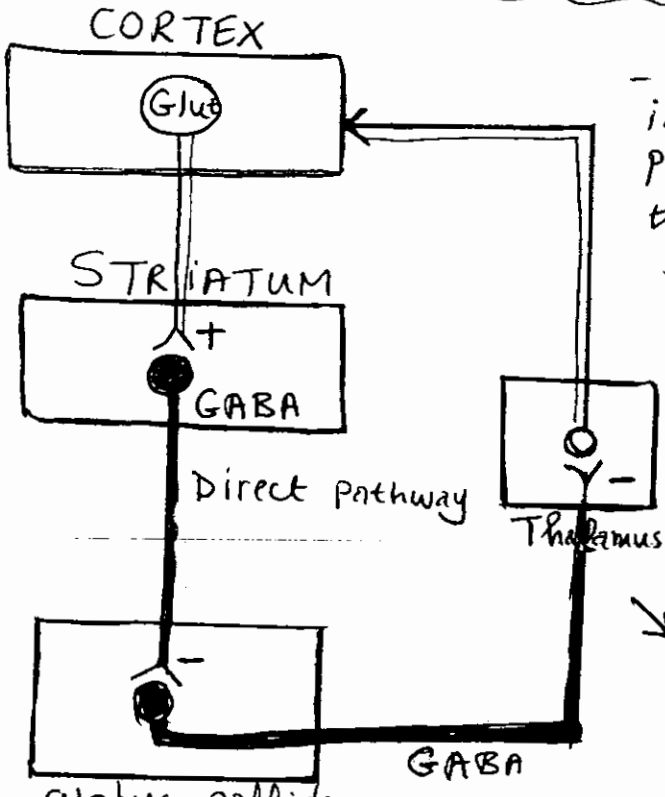
Functional organization of the Basal ganglia

- The basal ganglia exert their motor actions largely via RECIPROCAL connections with the cerebral cortex.

→ Nearly all areas of the cerebral cortex PROJECT to the STRIATUM (caudate & putamen). The cortical inputs to the striatum are EXCITATORY and mediated by GLUTAMATE

→ The output from the basal ganglia is via INHIBITORY (gamma-aminobutyric acid, GABA) neurons from the INTERNAL SEGMENT OF the GLOBUS PALLIDUS to the THALAMUS AND THEN → via Excitatory pathways to the motor & premotor cortices

The flow and Processing of cortical signals within the basal ganglia involve 2 major pathways ⟨ direct / indirect

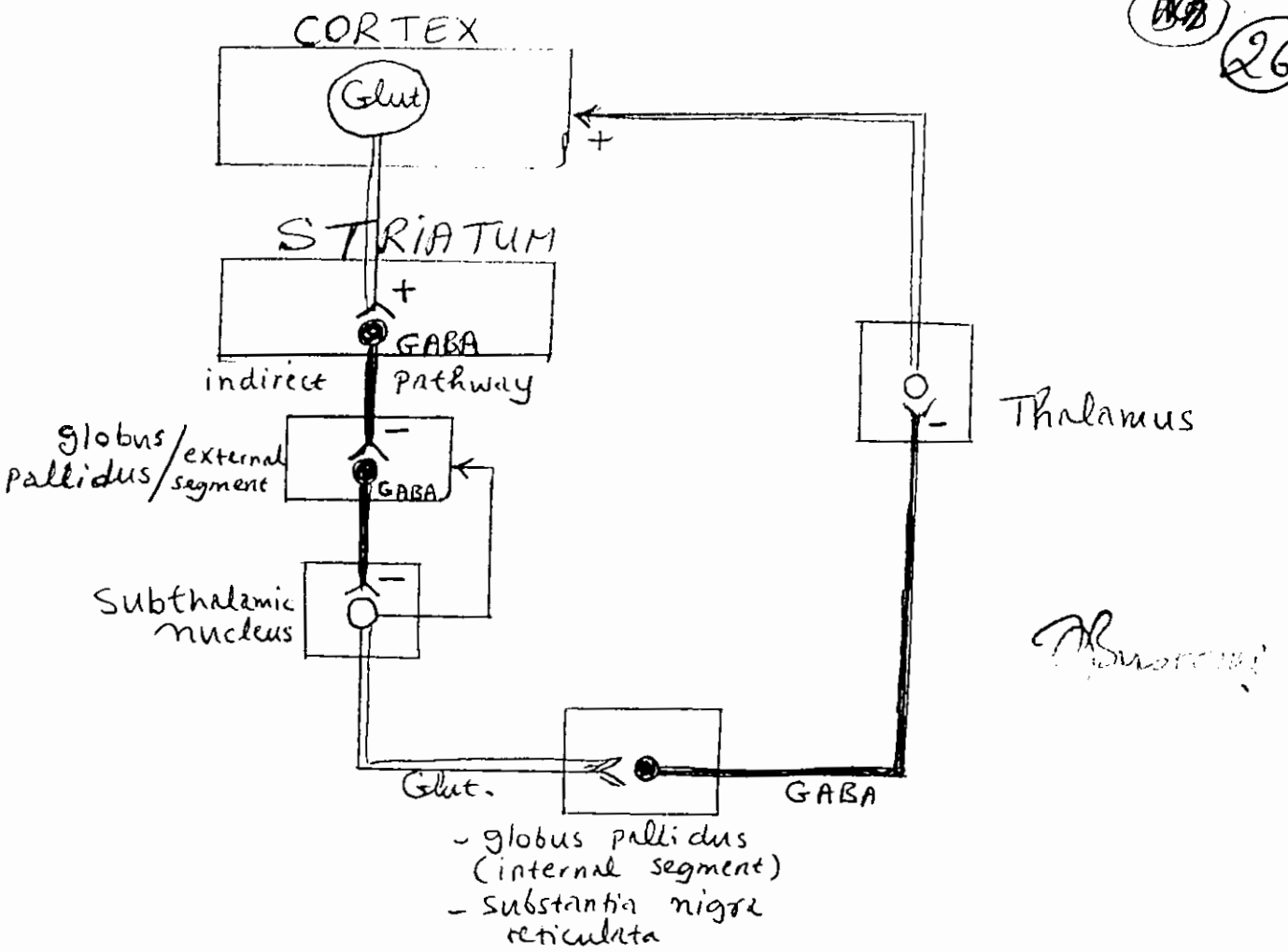


- globus pallidus internal segment (GPi)
- Substantia nigra reticulata (SNR)

- The DIRECT pathway involves inhibitory GABAergic projection from the Striatum to the internal segment of globus pallidus → Activation of this pathway results in inhibition of inhibitory pallidal output neurons & hence DISINHIBITION of the thalamic neurons

↓

This is thought to FACILITATE movement by exciting premotor & supplementary motor cortical areas



The INDIRECT pathway involves a distinct group of Striatal GABAergic neurons that project to the EXTERNAL SEGMENT of the globus pallidus and inhibit an inhibitory GABAergic Projection to the Subthalamic nucleus, From which excitatory (glutamatergic) neurons Project to the internal segment of globus pallidus PROVIDING EXCITATORY effect to the inhibitory GABAergic pallidothalamic output neurons

↓

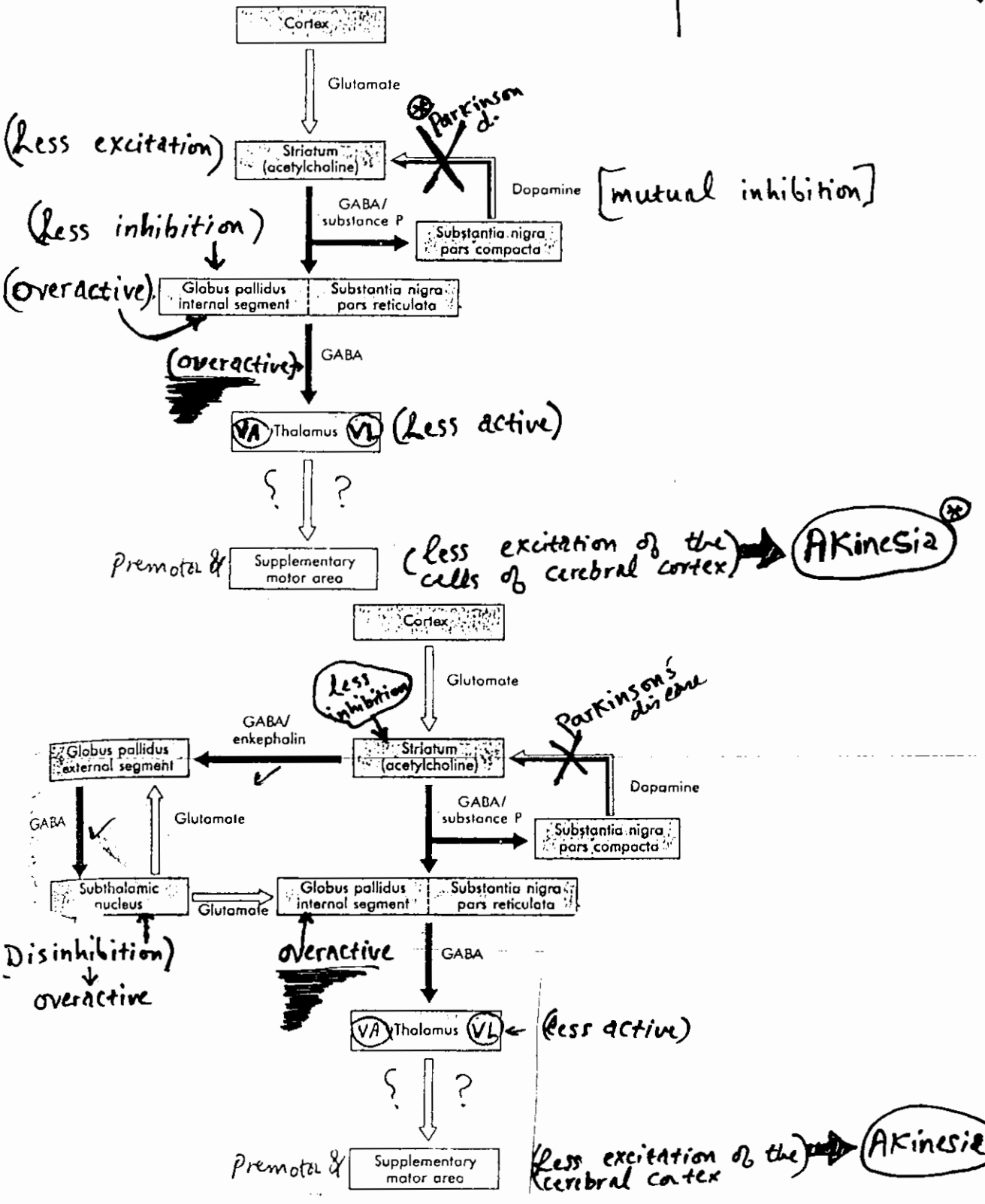
The neteffect of activation of this Pathway is the SUPPRESSION of thalamic neurons activity → DISFACILITATION of the motor cortical neurons →

INHIBITION OF MOVEMENT

Dopamine has excitatory action on the striatal neurons that control the DIRECT Pathway

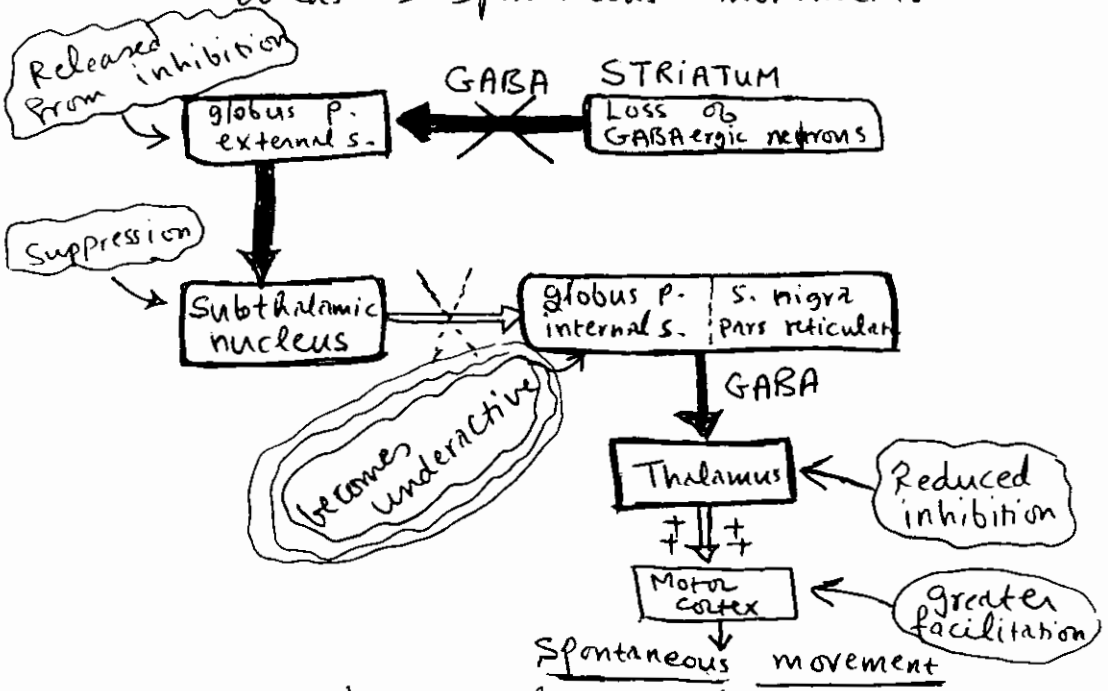
inhibitory action on the striatal neurons that control the INDIRECT Pathway

In Parkinsonism → Loss of Striatal dopamine



Choreas: a group of disorders characterized by rapid (dancelike) involuntary movements (diskinesia) largely restricted to muscles of distal extremities

Lesion → loss of Striatal GABAergic neurons that project to the external segment of the globus pallidus (indirect pathway) → This releases the inhibition of the external pallidal segment → suppression of the subthalamic activity → Reduced inhibition of thalamic neurons → greater facilitation of cortical areas → spontaneous movements



Functions of the basal ganglia

1. The Corpus striatum (caudate, putamen, globus pallidus) ⊕ Substantia nigra ⊕ Subthalamic nucleus
   ⇒ are FUNCTIONALLY INTERDEPENDANT
   * Disease in any part of this complex of extrapyramidal nuclei ⇒ UPSETS TOTAL FUNCTION and the symptoms reflect general derangement
   * Dysfunction of one component may result in over-activity in another part of the complex ⇒ RELEASE PHENOMENON.
2. The exact role of the basal ganglia in movement is far from clear. One speculation is that the Striatum contains LEARNED MOTOR PROGRAMS imprinted there by the multiplicity of cortical inputs. In Parkinson's disease patients find it hard to initiate movements learned in early life (such as standing and turning around) and in carrying them through.
3. Inhibit muscle tone throughout the body ???

In Parkinson disease

Overactive GPi (internal segment of globus pallidus which is INHIBITORY)

- Inhibition of Midbrain extrapyramidal area
  - Inhibition of Pontine Reticulospinal tract
    - Disinhibition of α and γ motoneurons
      - Hyperactive
        - Hyperactive Stretch reflex
          - Hypertonia in Axial & Proximal muscles (Extensors, flexors)
  - Inhibition of rubrospinal tract
    - Disinhibition of α and γ motoneurons
      - To flexor muscles Proximal & distal
        - ↑ tone in flexor muscles of limbs
- Suppression of thalamus
  - Disfacilitation of cortex
    - ⊗ HYPOKINESIA

Tremor → overactive globus pallidus ??

Programming of eye movements appears to occur Not only in the frontal eye feild but also in basal ganglia

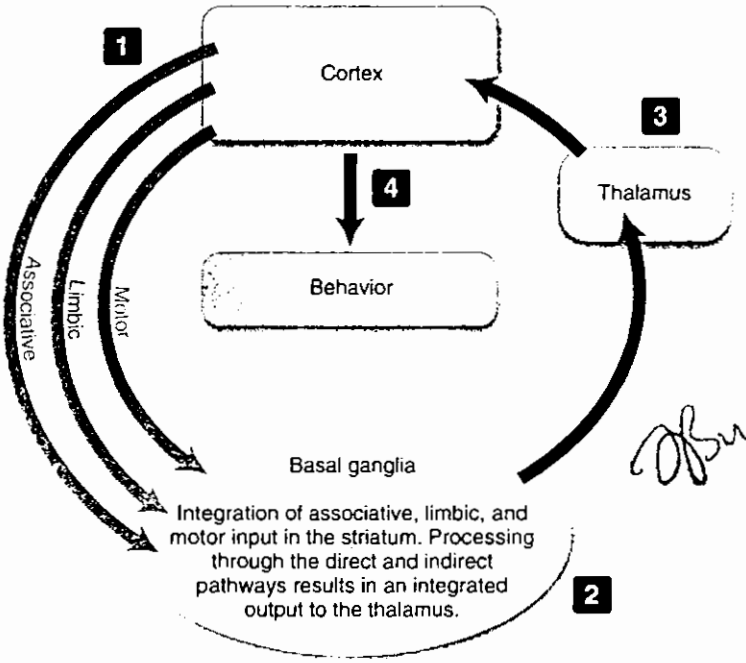
↓

INPUT: reaches the basal ganglia via corticostriate fibres from the frontal eye feild and Posterior Parietal cortex

Output: from the globus pallidus(i) and substantia nigra (Pars reticulata) to VA thalamic nucleus → directly influence the frontal eye field

Bustami.

In Parkinson disease: Normal spontaneous eye movements are lacking or seldom occur ⊕ infrequent blinking → Staring appearance



(1) The input to the basal ganglia can be described as three parallel streams of information from the cortex { Motor, associative, limbic }

(2) The Striatum integrates these inputs

(3) from the striatum the activity of the thalamus is determined via the direct & indirect pathways

(4) The thalamus then sends Projections back to the cortex

The basal ganglia therefore INTEGRATE these { Sensory, motor, emotional, motivational } inputs that result in a final common pathway which determines the complex pattern of behaviour we display.

Motor circuit

Inputs related to motor Performance come from widespread areas of the cortex including: Primary motor area, Premotor & supplementary motor areas as well as primary somatosensory & sensory association areas ⇒ ALL ARE INTEGRATED IN THE PUTAMEN → The Motor circuit is mediated through both the direct and indirect Pathways within the basal ganglia ⇒ the balance of these two Pathways results in coordinated motor Performance

An imbalance in these Pathways causes movement disorders characterized by TOO LITTLE MOVEMENT (Without Paralysis) or uncoordinated EXCESSIVE MOVEMENTS

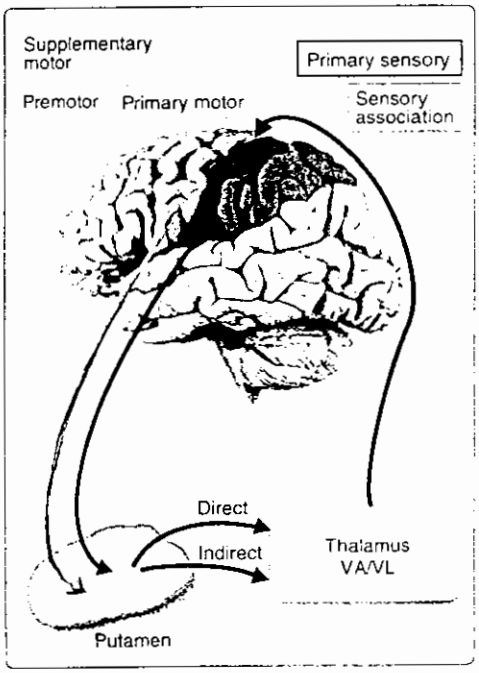
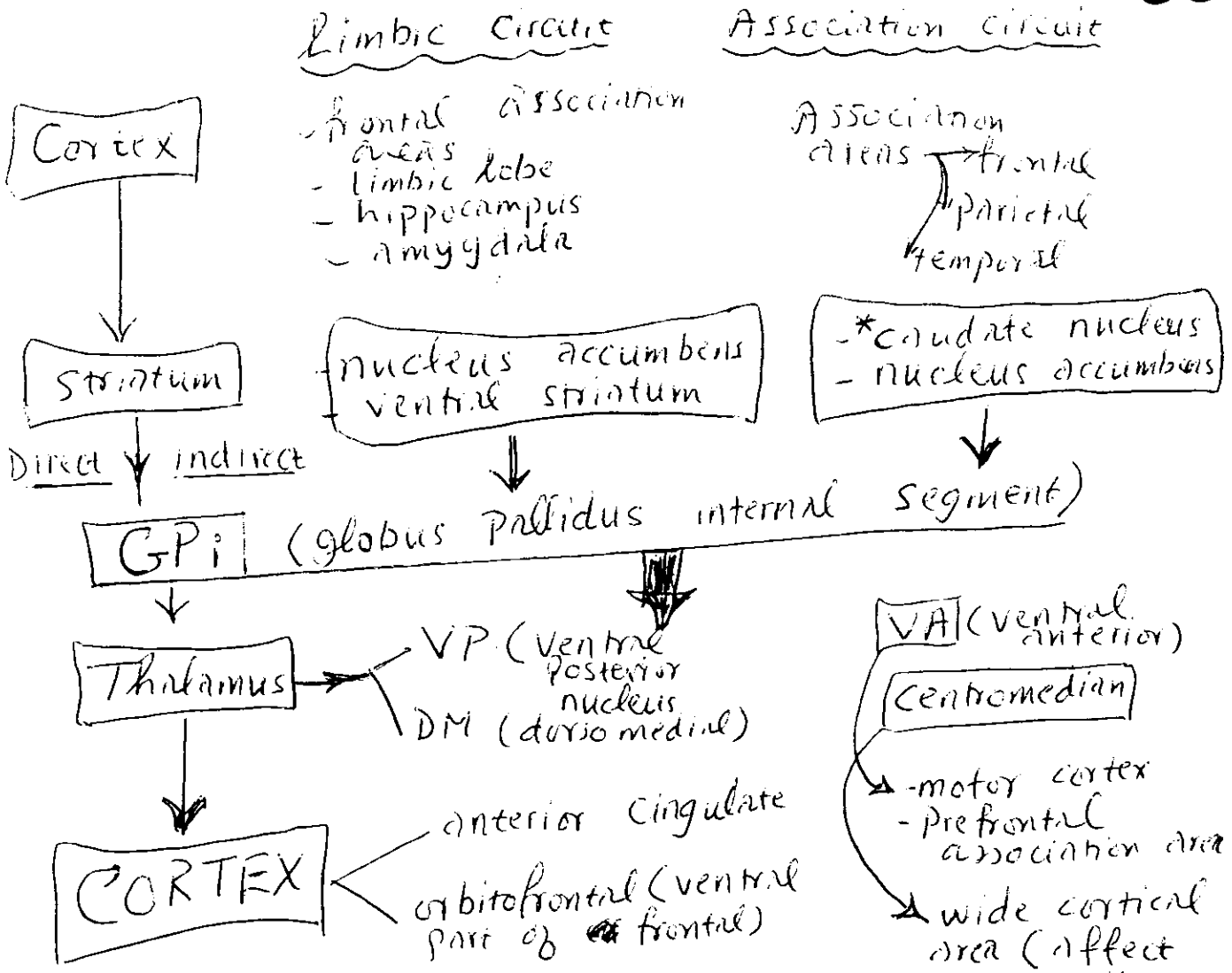


Figure 16.13
Schematic representation of the motor circuit. VA = ventral anterior nucleus; VL = ventral lateral nucleus.



* when a new task has been practiced and well learned → activity in the associative circuit decreases & the motor circuit becomes active instead

THE NEUROLOGY OF THE BASAL GANGLIA

The basal ganglia have great neurological importance because several common diseases have been correlated with specific lesions to this area. Damage to the basal ganglia produces movement disorders, or dyskinesia [G. *dys,* bad, and *kinesis,* movement]. Dyskinesia, a motor disorder that entails some loss of voluntary control and regulation, falls into two classes: those that result in spontaneous movements, or hyperkinesia, and those that result in poverty of movement, or hypokinesia. Hyperkinesia is expressed as *involuntary spontaneous movements.* Hypokinesia causes the opposite effect, *the lack of spontaneous movements and a slowing of voluntary movement.* It is important to note that the motor system is otherwise intact, as are the knowledge and will to initiate and perform the motor act.

Dyskinesia differs from paralysis and paresis in two major respects. First, unlike paralysis or paresis, dyskinesia involves no dysfunction of the upper or lower motor neuron systems. Consequently, there is no weakness. Second, dyskinesia is not apraxia, the inability to plan or execute a complex motor act. Apraxia follows a lesion to the cerebral cortex and affects one's ability to conceptualize the task.

Parkinson's disease (paralysis agitans)

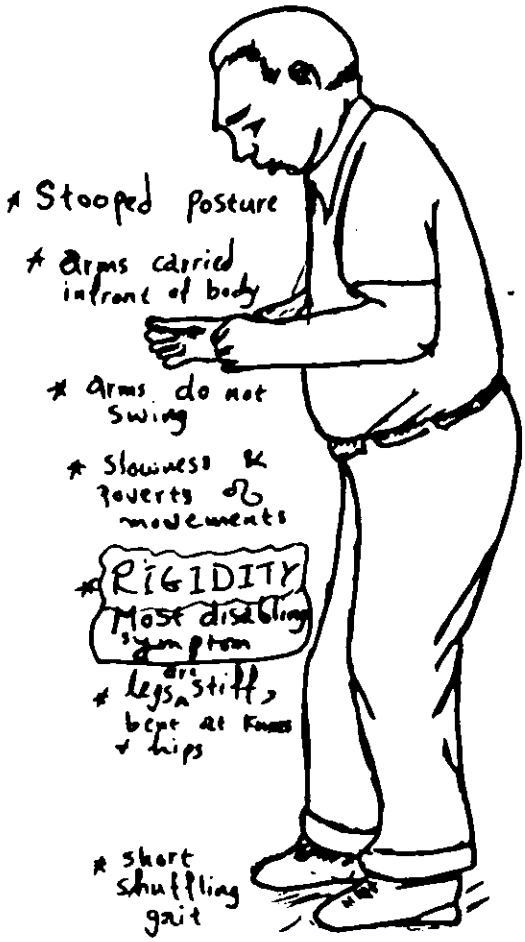
- widespread destruction of substantia nigra → (loss of dopamine at Striatum)*
- Clinical signs → hypokinesia (or akinesia) ① / Rest tremor ③ / rigidity ②
- Hypokinesia : difficulty in initiating movements, in carrying them through (freezing) or in terminating them
- Rigidity : increase muscle tone affecting both flexors & extensors (i.e bidirectional)
- Rest tremors → not always present
  - affect muscles of fingers (pill-rolling)
  - disappear during movement (unlike the intention tremors of cerebellar disease)

- Chemical changes in Parkinson's disease
  - ↓ DA/Ach ratio (decrease dopamine/Acetyl choline ratio)
- Treatment : - L-dopa → crosses the blood brain barrier and changes into dopamine within the living dopaminergic neurons
  - Anticholinergic drugs
  - Surgical destruction of the overactive pallidum

34

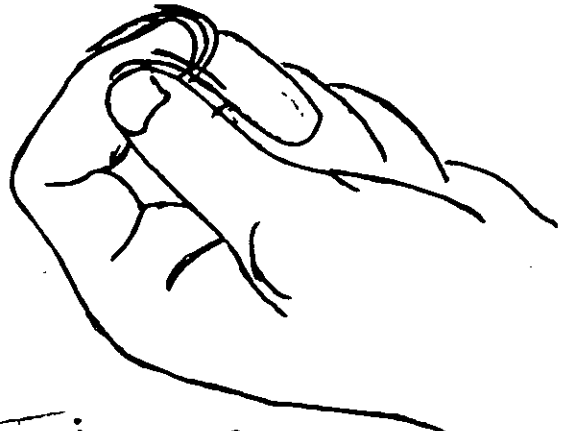
PARKINSON'S DISEASE

(Degenerative changes are present in substantia nigra & Globus Pallidus)



* Stooped posture
* Arms carried infront of body
* Arms do not swing
* Slowness & poverty of movements
* RIGIDITY Most disabling symptom
* Legs are stiff, bent at knees & hips
* short shuffling gait

Unblinking mask-like face

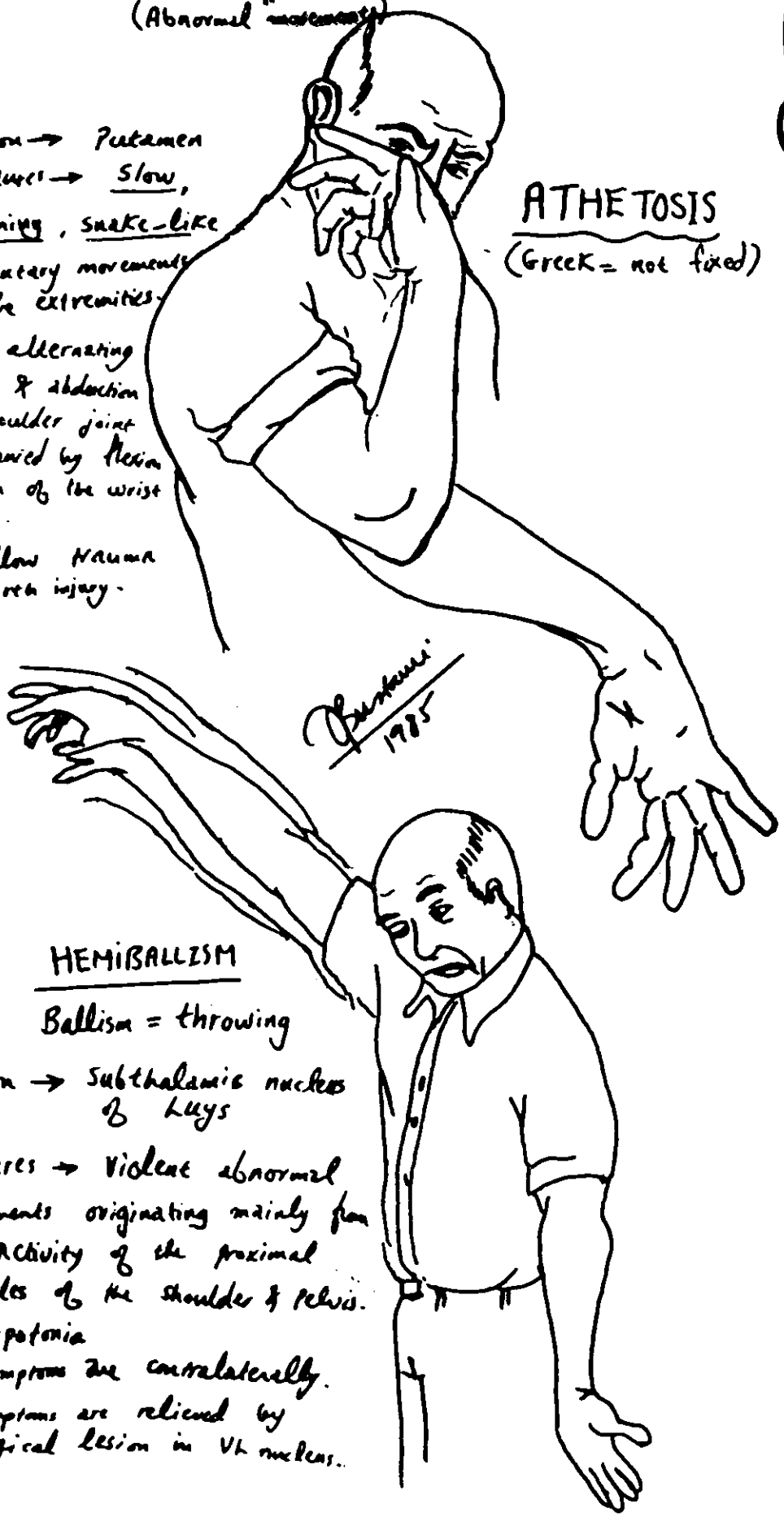


"rest-tremor" often "pill-rolling tremor in thumb & fingers

Bastani 1985

- Dyskinesias -

(Abnormal "movements")



ATHETOSIS

(Greek = not fixed)

* lesion → Putamen
* features → Slow, Writhing, snake-like involuntary movements of the extremities.
* The alternating adduction & abduction of the shoulder joint is accompanied by flexion & extension of the wrist & fingers.
* may follow trauma & a birth injury.

HEMIBALLISM

Ballism = throwing

* Lesion → Subthalamic nucleus of Luys
* features → Violent abnormal movements originating mainly from the activity of the proximal muscles of the shoulder & pelvis.
* Hypotonia
* Symptoms are contralaterally.
* Symptoms are relieved by surgical lesion in Vl nucleus.