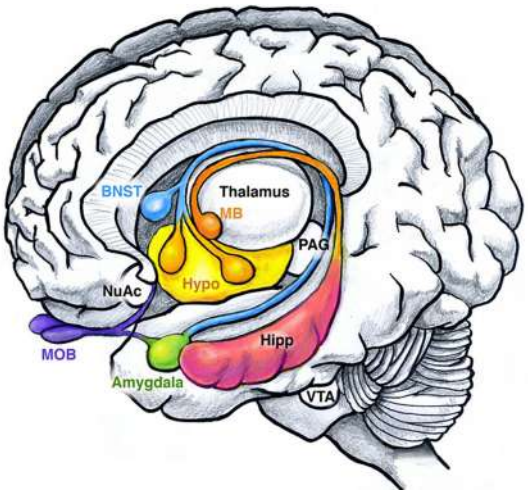
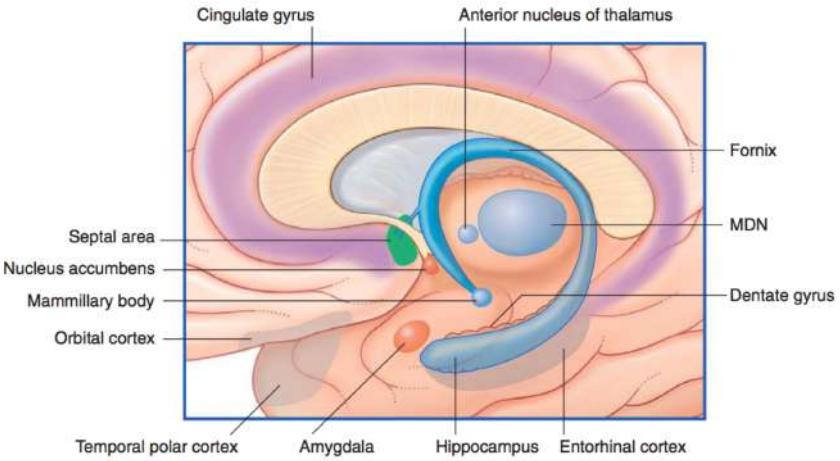
The Limbic System

1 Anatomy of the Limbic System

A



Saggital section of the brain



Cingulate gyrus –

Septal nuclei –

Nucleus accumbens –

Amygdala –

Thalamus -

Hippocampus –

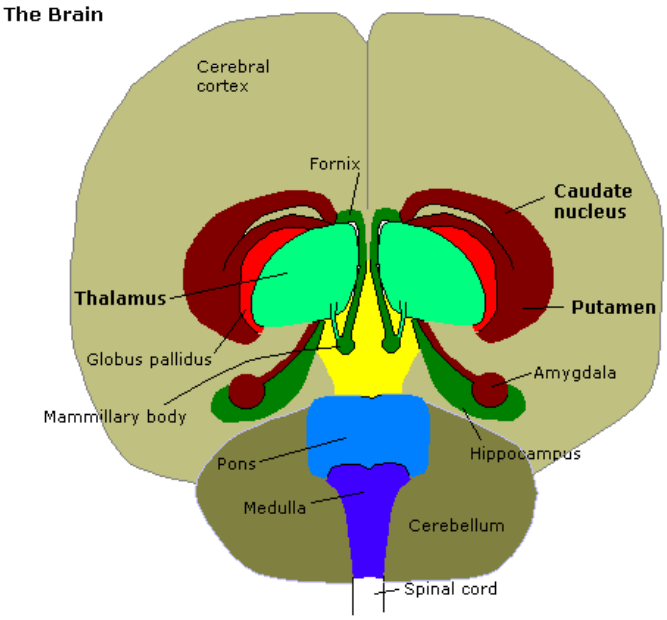
Fornix –

Mammilary bodies –

Parahippocampal gyrus -

Nuclei –

Coronal View of Limbic System



2 Function of the Limbic System

Main function = **memory** and **emotion**

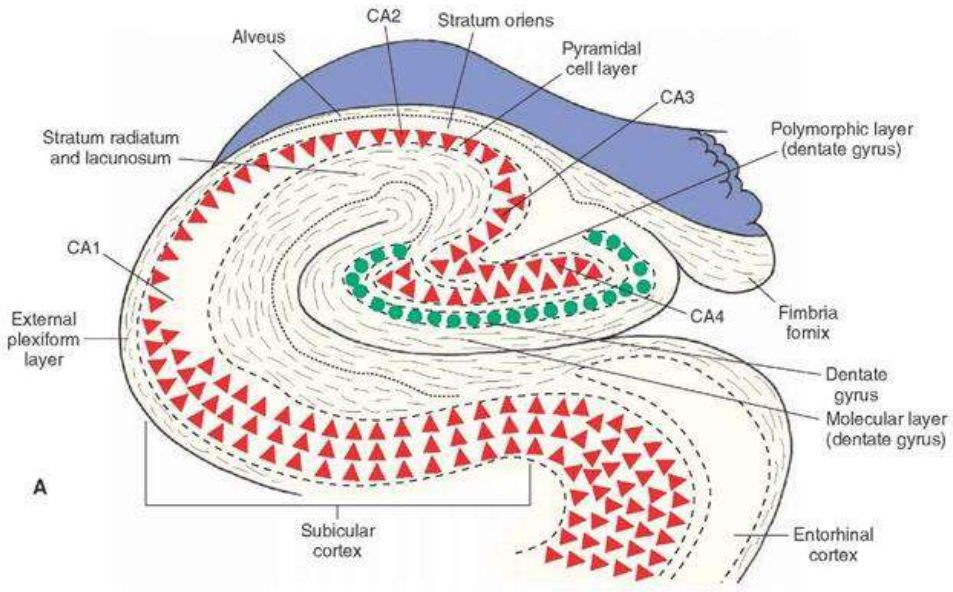
- F
- F
- F
- F
- F

3 Hippocampal Formation

The hippocampal formation consists of three parts:

- _____
- _____
- _____

Cornu Ammonis



Types of cell:

- Dentate gyrus = _____
- Rest of the Hippocampal formation = _____

4 Pathways of the Limbic System

Direct Pathway

Input: sensory association cortex

Output: Fornix and Sensory cortex

Entorhinal Cortex

Subiculum

CA1

CA2

CA3

CA4

Dentate Gyrus

Perforant Pathway

Input: sensory association cortex

Output: Fornix and Sensory cortex

Entorhinal Cortex

Subiculum

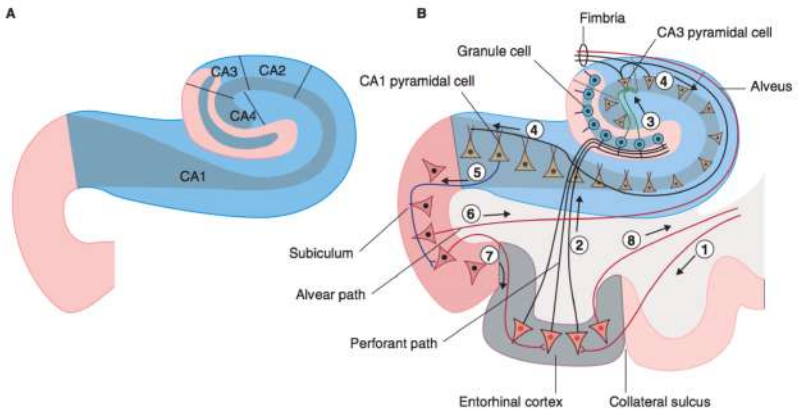
CA1

CA2

CA3

CA4

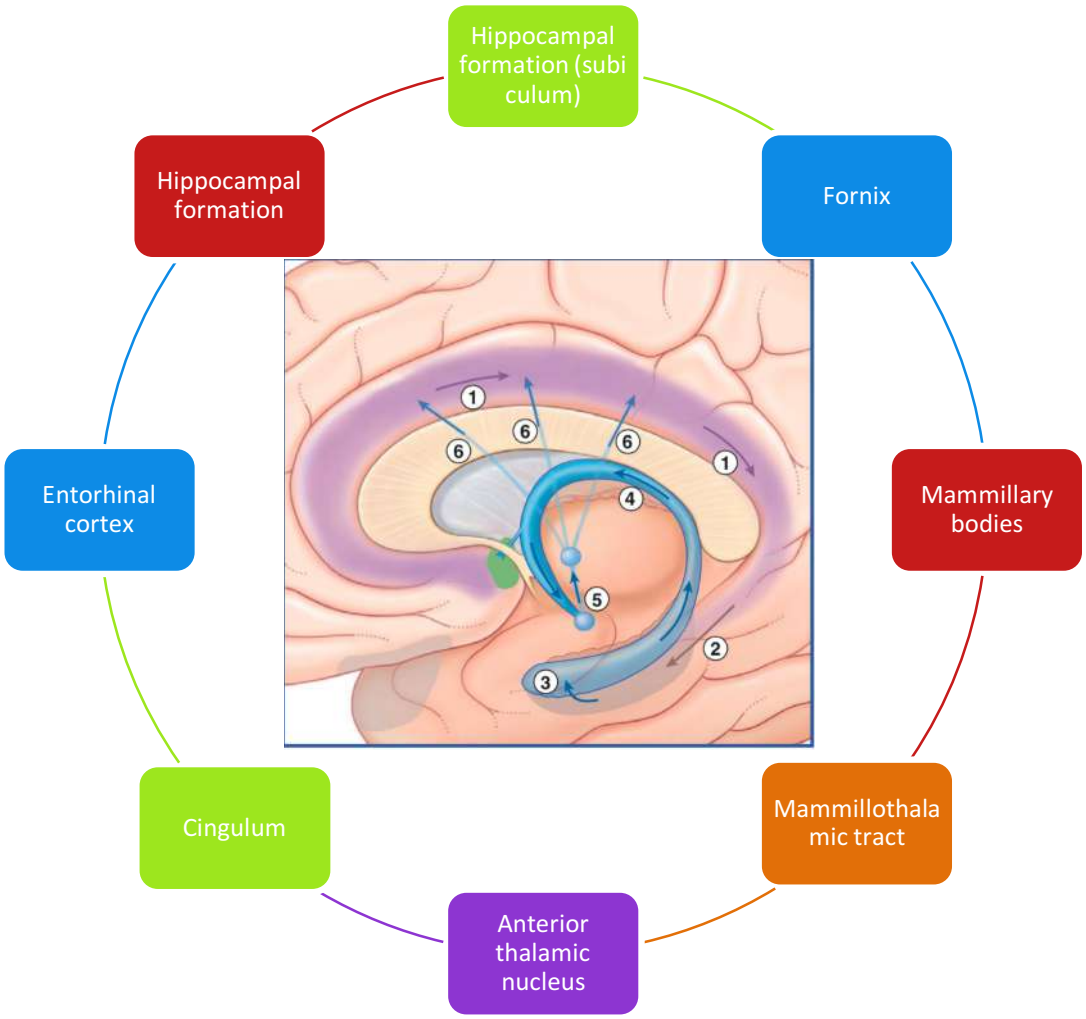
Dentate Gyrus



Perforant Pathway

Rhino Dentists are a Proper Odd Sub-speciality!

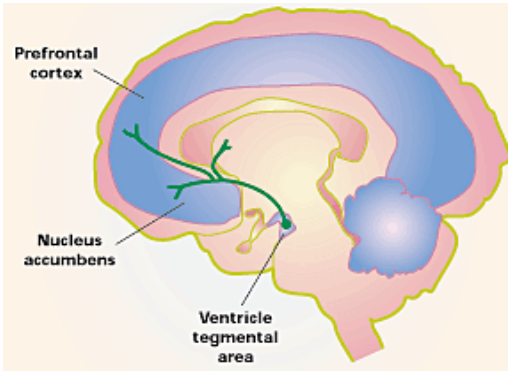
Papez Circuit



Function of the papez circuit = involved in memory and integration of the limbic system

Reward/ Mesolimbic Pathway

Function – this pathway has a role in reward + addiction



Ventral Tegmental Area

↓

Nucleus Accumbens

↓

Orbitofrontal Cortex (part of the prefrontal cortex)

Orbitofrontal cortex – brake/accelerator.

Anatomy of the Basal Ganglia

What is the **BASAL GANGLIA**?

Function of the Basal Ganglia

M

O

L

C

Structures of the Basal Ganglia

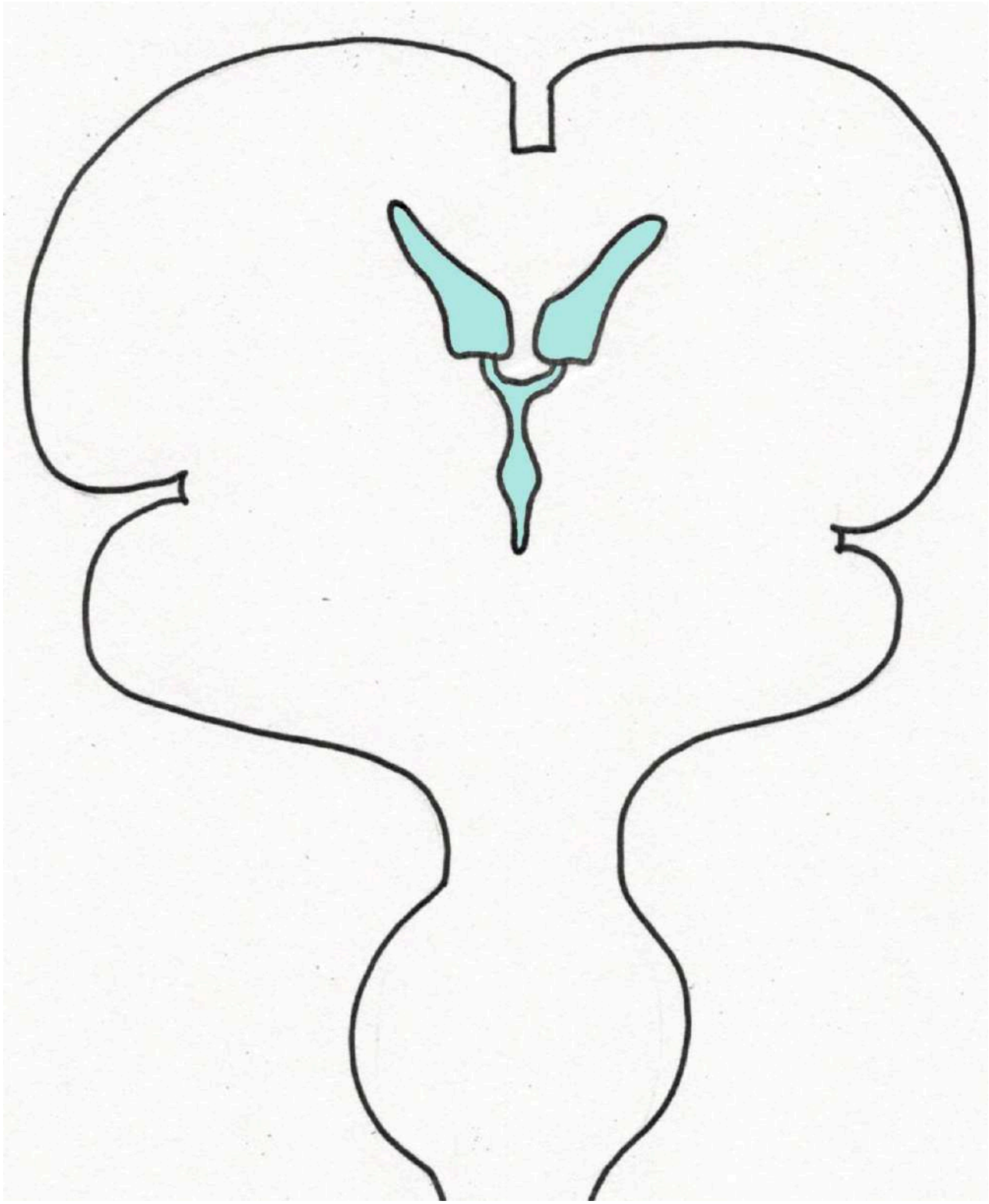
1)

2)

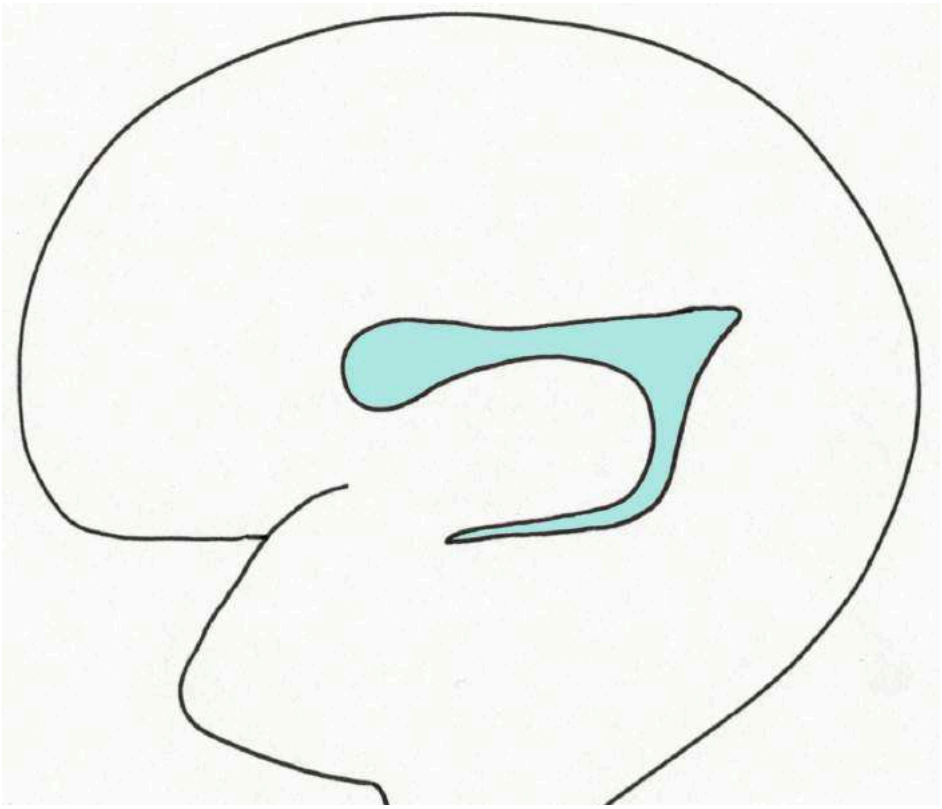
3)

4)

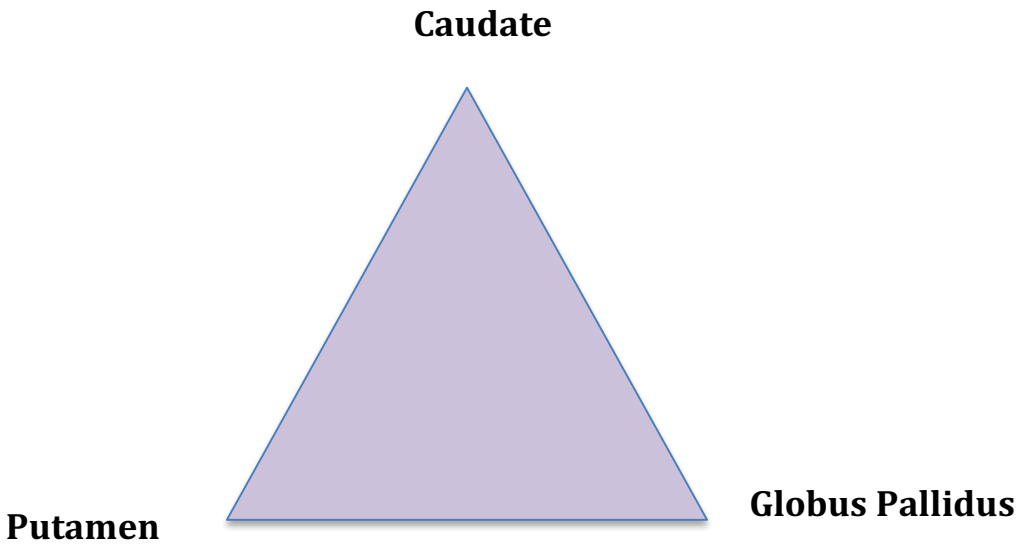
Location of key structures



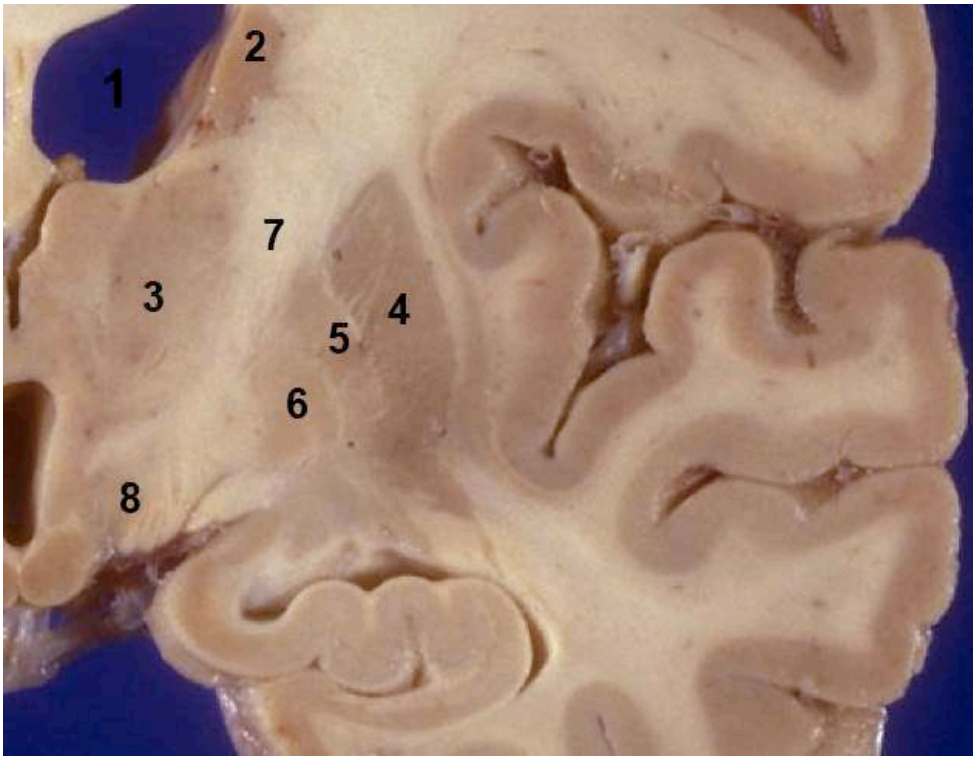
Caudate nucleus



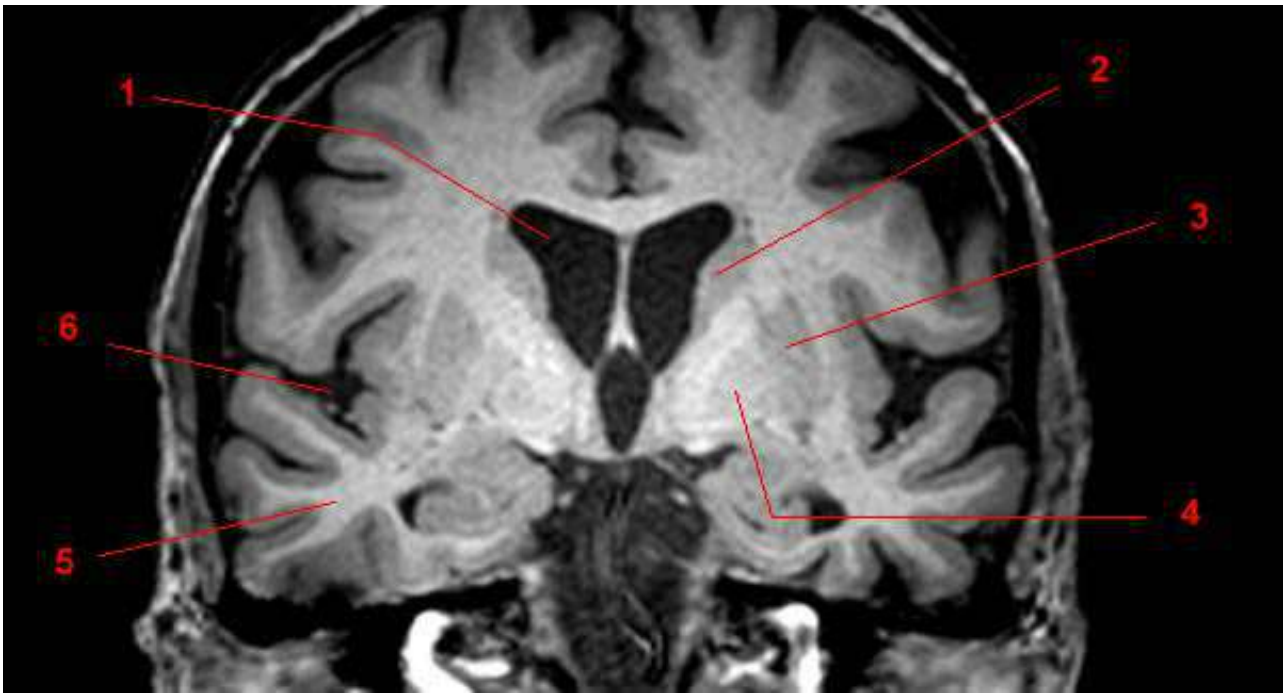
Nomenclature and classification



Coronal section of brain



Coronal MRI of the brain



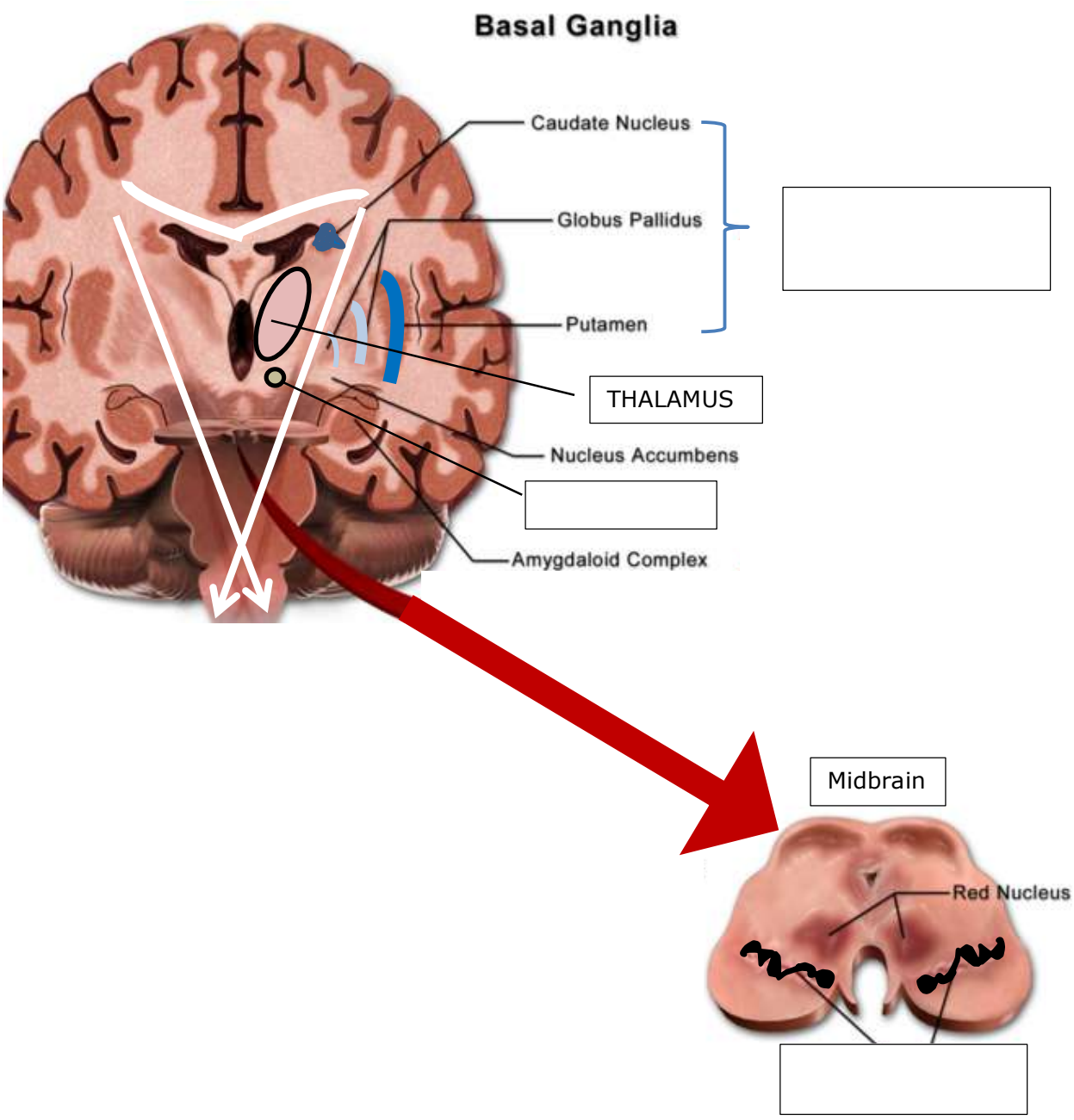
Basal ganglia (function and circuitry and integrated disease)

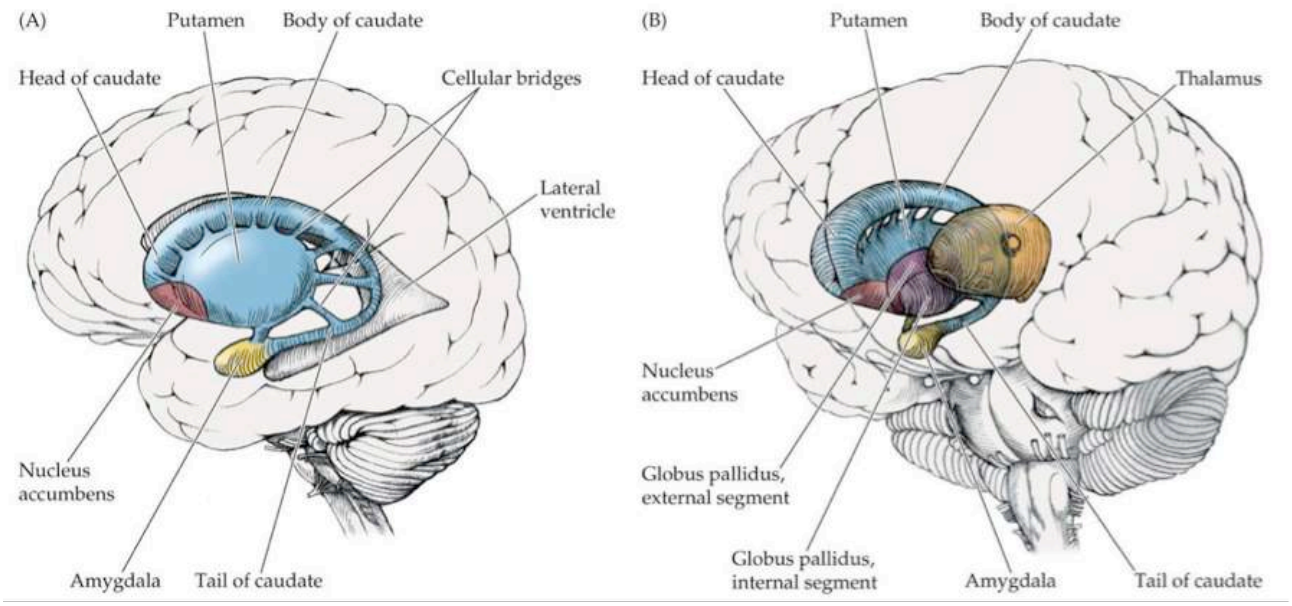
A quick recap:

ANATOMY:

BASAL GANGLIA SURROUND THE THALAMUS IN BOTH CORONAL AND TRANSVERSE SECTIONS

USE THE VENTRICLES AND THALAMUS AS YOUR GUIDES





CAUDATE NUCLEUS and PUTAMEN are ANTOMICALLY AND FUNCTIONALLY linked. Collectively called STRIATUM (because interrupted by INTERNAL CAPSULE WHITE FIBERS)

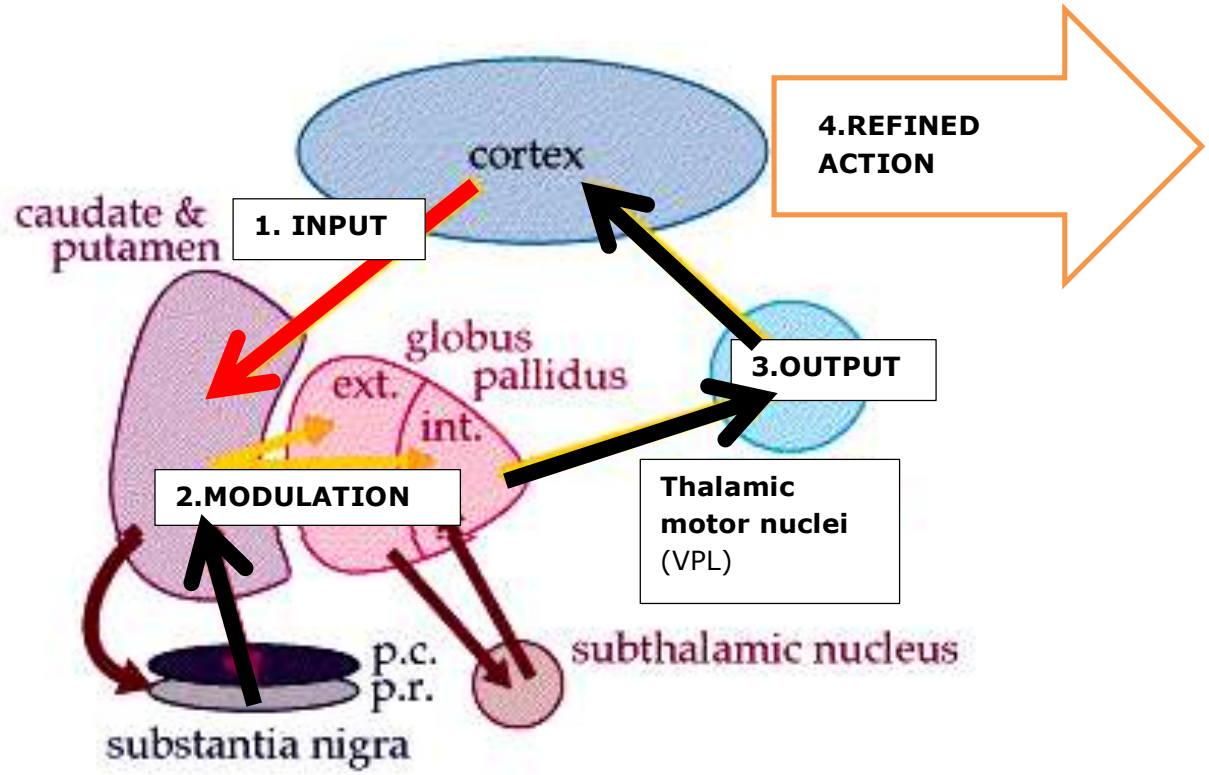
MAIN FUNCTION OF BASAL GANGLIA: REFINE MOVEMENTS ie SMOOTHER, MORE PRECISE

1.INPUT: from MOTOR CORTEX **to the STRIATUM (ie Sensory component of BASAL GANGLIA)**

OUTPUT: to the THALAMUS via:

1. GLOBUS PALLIDUS Internus
2. Substantia Nigra Pars reticulata

NB: this is a closed LOOP as thalamocortical projections will relay information to the cortex.



MODULATION:

2 opposing pathways:

DIRECT ---Drives MOVEMENT (D1-R on STRIATUM)

NB: directly connected to GLOBUS PALLIDUS internus (1 motor output)

Entirely GABAnergic

IN-DIRECT: INHIBITORY of MOVEMENT (D2 R on striatum)

NB: indirectly connected to Globus pallidus internus and Substantia nigra pars reticulata **(via SUBTHALAMIC NUCLEUS)**

DISINHIBITION: INHIBITION OF AN INHIBITORY NEURON leads to EXCITATION drives the circuits in the basal ganglia

(DOUBLE NEGATIVE is +)

DIRECT PATHWAY:

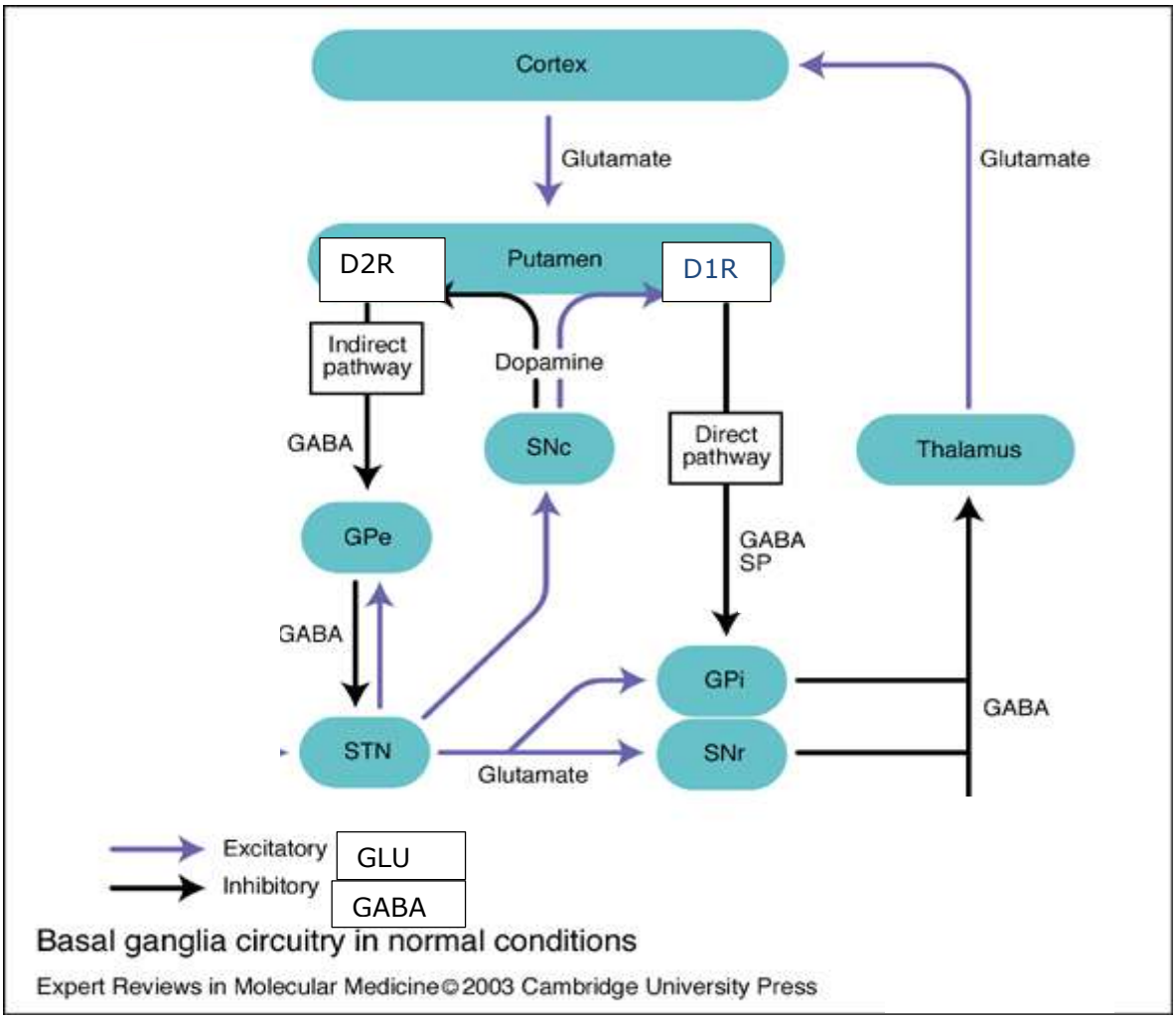
OVERALL EFFECT: DISINHIBITION of the Thalamic Neurons- INCREASE EXCITATORY OUTPUT TO THE CORTEX-DRIVES MOVEMENT

NB: Globus Pallidus INternus has a INHIBITORY ACTION ON MOTOR THALAMIC NUCLEI. GPi inhibition, consequently lead to THALAMIC EXCITATION.

DOPAMINE PRODUCED in the SUBSTANTIA NIGRA Pars Compacta stimulates the activation of the D1R ECT pathway in the STRIATUM and INHIBITS the INDIRECT pahtway. **DOPAMINE PROMOTES MOVEMENT.**

INDIRECT PATHWAY:

DISINHIBITION of SUBTHALAMIC NUCLEUS activates GPi and Substantia Nigra pars reticulata and lead to THALAMIC SUPPRESSION.



Basal ganglia circuitry in normal conditions

CLINICAL PERSPECTIVE-Semplified

Any imbalance in BASAL GANGLIA CIRCUITS will lead to non-smooth movements and involuntary movements (DYSKINESIAs)

Too little stimulation-DIFFICULT INITIATING MOVEMENTS, TREMOR AND BRADYKINESIA (Parkinson s disease)

Too much stimualation- ATHETHOSIS AND CHOREA (Hungtington s disease)

AT REST, which pathway predominates?

ANSWER: NO MOVEMENT, must be INDIRECT, hence to initiate movement we need to stimulate the DIRECT PATHWAY.

PARKINSON's DISEASE is characterised by DECREASED DOPAMINE PRODUCTION in the Substantia Nigra Pars Compacta:

- DIFFICULT IN INITIATING MOVEMENENT
- TREMOR
- BRADIKINESIA

Treatment is with **L-DOPA which is converted to DOPAMINE and is therefore used to reduce tremor and promote movement.**

Treatment:

D1R stimulation (little receptors)

D2R activation- INHIBITION OF INDIRECT---form of DISINHIBITION---MORE MOVEMENT

SEs: Too much stimulation (CHOREA)

HUNGTINTON's DISEASE (previously known as CHOREA)

Genetic disease: AUTOSOMAL DOMINANT (CAG expansion)

Leads to death of GABAnergic neurons in striatum (MOVEMENT DISORDERS) and Ach in the brain (DEMENTIA)

CAG= Caudate (striatum) loses Ach and GABA

- **CHOREIC MOVEMENTS are characteristic sudden jerky, dance-like movements**
- **ATHETOSIS: slow writhing movements.**

Treatment:

None at present

SUMMARY

D1RECT PATHWAY:

Drives movement via THALAMIC DISINHIBITION (GPi suppressed)

Dopamine stimulates it (D1R)- DOPA deficiency- PARKINSON's DISEASE

IN-DIRECT

Inhibits movement via SUBTHALAMIC DISHINHIBITION and activation og GPi and SNr

Inhibited by Dopamine (D2R)